CLINICO-BIOCHEMICAL AND ULTRASONOGRAPHIC EVALUATION OF DISEASES OF PROSTATE IN DOGS

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DECLARATION

I hereby declare that this thesis, entitled **"CLINICO – BIOCHEMICAL AND ULTRASONOGRAPHIC EVALUATION OF DISEASES OF PROSTATE IN DOGS"** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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Certified that this thesis, entitled **"CLINICO – BIOCHEMICAL AND ULTRASONOGRAPHIC EVALUATION OF DISEASES OF PROSTATE IN DOGS"** is a record of research work done independently by **Sindhu. K. Rajan.,** under my guidance and supervision and it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to her.

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1. INTRODUCTION

Prostate gland is an androgen dependent accessory sex gland in dogs. It is an ovoid, bilobed gland composed of glandular and stromal elements and encircles the urethra of the male dogs caudal to the neck of urinary bladder. Prostate gland contributes fluids to the first and third fractions of ejaculate, which aids to both support and transport sperm during ejaculation.

The prostatic growth as well as its secretion are under the influence of hormone testosterone throughout its life and tend to increase in size with progressive advancement of age. Hence, the increase in size of the prostate with age predisposes the gland to many disease processes such as benign prostatic hyperplasia, prostatitis, prostatic abscess, prostatic cyst, squamous metaplasia and prostatic neoplasia. Majority of these diseases manifest only when it exert effects on the nearby structures which result into the classic signs of prostatic diseases such as tenesmus, ribbon like feces, dysuria, haematuria, preputial discharges, and abnormal gait. However the case may present with clinical signs referable to gastrointestinal or urinary tract disorders due to the overlapping of clinical signs. Perineal and inguinal hernia could also occur due to prostatomegaly. Hence a thorough evaluation is required to localize the problem to the prostate and to identify the specific disease.

Therapeutic approaches vary according to the condition. Therefore, a specific diagnosis is essential for proper therapy and prognosis. Routine history and physical examination may alert the veterinarian to early diagnosis of prostatic disease. However, accurate diagnosis of prostatic diseases requires a variety of diagnostic techniques such as rectal palpation, ultrasound imaging of the prostate, prostatic fluid cytology, hematology and serum biochemistry.

Studies regarding the canine prostatic disorders are very scanty in India when compared to other countries. This might be due to the misinterpretation of diseases due to the overlapping nature of clinical signs of this disease as well as inappropriate diagnostic tools and methods. In most of the cases, prostatic diseases are misinterpreted as gastrointestinal diseases or urinary disorders or toxemia. Prostatic diseases are one of the major etiologies for renal failure in old male dogs, as this disease can further lead to renal failure, if untreated. Hence, the present study was undertaken with the following objectives.

1. To study the clinico – pathological findings associated with diseases of prostate

2. To evaluate the response to the treatment adopted.

Review of Literature

2. REVIEW OF LITERATURE

2.1. ANATOMY OF PROSTATE GLAND

2.1.1. Development of the Canine Prostate

O'Shea (1962) divided the development of the canine prostate into three phases, as a phase of normality (1 to 5 years), hyperplasia growth (6 to 10 years) and senile involution (11 years and over).

Evans and Christensen (1993) described that testosterone produced by the testis during foetal development gets converted into Dihydrotestosterone (DHT), which stimulate the development of the prostate gland.

2.1.2. Location and Structure

Gordon (1961) suggested that the age, androgenic stimulus, diseases, and the degree of urinary bladder distension affect the position of the prostate gland. The author also described that at about 8 months of age when sexual maturity is achieved, due to the androgenic stimuli, prostate gets repositioned cranially over the pelvic brim and gradually increases in size to a total / partial intrabdominal position.

Ellenport (1975) also stated that the degree of distension of the bladder could affect the position of the prostate gland, like; a full bladder displaces the gland intrabdominally where as, an empty bladder facilitates an intrapelvic position.

Dyce *et al.* (1996) stated that a dorsal groove and internal septum divided the prostate gland into right and left lobes, which were subdivided into lobules by finer septa that radiate outwards to the capsule. Atalan *et al.* (1999b) observed a round shape for normal and hyperplastic canine prostate, while, Johnston *et al.*

(2000) stated that canine prostate is an androgen-dependent, ovoid, bilobed gland composed of glandular and stromal elements, which encircles the urethra of the male dog caudal to the neck of the urinary bladder.

Kustritz and Klausner (2000) stated that the prostate is the only accessory sex gland of the male dog. It is surrounded by a fibromuscular capsule and divided into two lobes by the median raphe. Histologically the gland is divided into different lobules by the smooth muscle and the glandular lining cells differed from low cuboidal to high columnar type.

Smith (2008) described that the prostate gland lies in close apposition to the bladder cranially, rectum dorsally, pubic symphysis ventrally and abdominal wall laterally.

2.1.3. Prostatic Vascularization

Gordon (1960) described artery umbilicalis and artery urogenitalis as the main arteries in the pelvic cavity. Later Hodson (1968) identified two ramifications of artery urogenitalis, supplying the prostate and adjacent organs like, artery prostatica- vesicalis and artery prostatica- urethralis. Stefanov *et al.* (1996, 1999) stated that the artery prostatica gives of three main branches viz. cranial, middle and caudal to each of the prostate lobes as independent vascularization and the middle, ventro-cranial and ventrocaudal veins draining the prostate.

Stefanov (2004) opined that the prostate cancer most frequently damaged one of the prostate lobes only and author also stated that the independent vascularization of the prostate lobes permitted partial prostatectomy of the diseased lobes.

2.2. PHYSIOLOGY OF PROSTATE

Prostatic fluid makes up the first and third fraction of the canine ejaculate and its function is to increase the volume of the semen and aid in sperm transport (Barsanti and Finco, 1979).

Prostatic fluid is secreted continuously in intact male dogs, and flows retrograde into the urinary bladder or antegrade out the external urethral orifice in volumes ranging from a few drops to several milliliters, depending on the size of prostate (Johnston *et al.*, 2000).

Romagnoli (2007) opined that as a dog ages, the prostate gland undergoes increase, in both number (hyperplasia) and size (hypertrophy) of its epithelial cells wherein, the increase in number is more marked. This process could be visualized in dogs as early as 2.5 years of age. The basal prostatic secretion constantly enters the prostatic excretory duct and prostatic urethra. In the absence of micturition or ejaculation, urethral pressure moved prostatic fluid cranially into the bladder by a mechanism called prostatic fluid reflux.

The third fraction of semen was solely prostatic fluid that should be clear in appearance and the volume of this fraction was the most variable and could exceed 15 ml in normal dogs (Smith, 2008).

2.3. PROSTATIC WEIGHT AND SPECIFIC GRAVITY

The results of the study conducted by Lowseth *et al.* (1990) suggested that the increases in weight and size of the prostate were primarily attributable to increases in the inflammatory and interstitial tissues with the inflammation being located primarily within the interstitial tissues.

Atalan *et al.* (1999b) calculated prostatic density in canine cadavers, and it varied from 0.86 to 1.15 g cm^{3-1} with a median of 1.00 g cm^{3-1}.

Kamolpatana *et al.* (2000) determined prostate specific gravity in dogs by dividing prostate weight in grams by prostatic volume in cm^3 and found that the mean specific gravity of the prostate gland in dogs as $1 \pm 0.05 g/cm^3$ with range 0.9 -1.09 g/cm^3. The author also observed that the specific gravity was less than one in two cases.

2.4. EFFECT OF DIETARY FAT INTAKE ON PROSTATE

The study conducted by Attar-Bashi *et al.* (2003) showed that the canine prostate had a low level of n-3 fatty acids as well as n-3/n-6 ratio and authors also observed that diet of the dogs also contained low n-3 content and n-3/n-6 ratio.

2.5. INCIDENCE

Barsanti and Finco (1986), Black *et al.* (1998) and Kustritz and Klausner (2000) stated that prostatic disease affects approximately 80 % of male dogs of more than 10 years of age. Krawiec and Heflin (1992) reported that prostatic diseases in male dogs represented 2.5 per cent of the total number of males seen during their five-year study. Histopathological studies conducted by Amorim *et al.* (2004a) revealed that 81.3% of the dogs had some kind of prostatic hyperplasia upon screening between five and fifteen years of age.

Prostatic disorders occurred more frequently in dogs than in cats or any other domestic species. This could be due to the continued expansion of the gland throughout life in the dog that resulted in the development of prostatic hyperplasia in this species (Parry, 2007).

2.6. PREDISPOSING FACTORS

2.6.1. Size of Breed

O'Shea (1962) reported that Scottish Terriers had prostate size four times than that of dogs of other breeds. Branam *et al.* (1984) observed that bacterial prostatitis was absent in small breeds. According to Krawiec and Heflin (1992),

middle aged and large breeds were more prone to the development of prostatic diseases, with Doberman pinscher and German shepherd appeared to be affected more frequently than other breeds. According to Johnston *et al.* (2001) there was no breed predilection for prostate disease, but large-breed dogs such as German shepherds and Dobermans seemed to have an increased prevalence although a causal relationship had not been established.

2.6.2. Effects of Age and Body Weight on Prostate

Bloom (1954) opined that weight and size of the prostate gland was variable depending on the age and breed. The normal ratio recorded varied from 0.1 to 0.7 g with an average of 0.4 g of prostate to 1 kg of body weight.

O'Shea (1962) found out a positive correlation between prostatic weight and body weight in dogs. The prostatic size correlated with the breed as cited by O'Shea (1962) and the body weight and age of the animal as reported by Berry *et al.* (1986).

O'Shea (1962) and Berry *et al.* (1986) reported an association between benign prostatic hyperplasia (BPH) and increase in size of the prostate gland with age. Lowseth *et al.* (1990) reported that prostatic diseases were more common in dogs over six years and found that the weight and volume of the prostate increased with age as 1.08 ± 0.22g/kg body weight at 3 years of age which showed an increase to 2.64 ± 0.37g/kg at 14 years of age. The mean age of onset of prostatic disease was 8.9 years, as reported by Krawiec and Heflin (1992).

Waters *et al.* (1996) compared the age at detection of prostate cancer in humans and dogs and concluded that the development of prostate cancer in both the species was influenced by age. Ruel *et al.* (1998) reported that the prostate gland in healthy intact adult dogs increased with the size and age of the dog. Atalan *et al.* (1999b) demonstrated that prostatic weight and volume were related to body weight as well as age in 60 male entire dogs and not in neutered dogs and

the authors also indicated that prostatic width and length were the best predictors of prostatic volume and weight.

Dorso *et al.* (2008) found a positive correlation between absolute prostate weight and age in dogs and stated that development of prostate advanced with the age of the animal.

2.7. DIAGNOSTIC TECHNIQUES FOR PROSTATIC DISORDER

2.7.1. Digital Rectal Palpation

Hastak *et al.* (1982) opined that rectal palpation was a simple method for evaluating prostate size, but permits examination only of the dorsal or dorsocaudal aspects of the prostate.

Dorfman and Barsanti (1995) reported that in the medium sized dogs, the prostate was physiologically the size of a walnut, with a smooth surface, solid consistency, free, isothermic and did not cause pain to the animal during examination.

The prostate gland should be evaluated for size, shape, symmetry and evidence of pain (Johnston *et al.*, 2001; Gobello and Corrada 2002 and Kutzler and Yeager, 2005).

Smith (2008) reviewed the anatomy, pathology, diagnosis and treatment of prostatic disease and opined that transrectal digital palpation was the best method for physical examination of the prostate and the prostate was most easily reached when assisted with caudal abdominal pressure.

2.7.2. Prostatic Ultrasonography

Ultrasound visualization of the prostate allowed more accurate identification and sampling of pathologic area as opined by Ling (1995).

Ruel *et al.* (1998) opined that prostatic measurements from the ultrasonographic images were more accurate and reliable as the contours of the prostate were better outlined, the measurements were performed using integrated electronic calipers and there was no magnification effect as opposed to radiology. The author used 7.5 MHz sectorial transducer for the transabdominal ultrasonography of the prostate in their study and the true sagittal section of the prostate was confirmed by observing the hypoechoic urethral tract.

In the study conducted by Atalan *et al.* (1999b), the bladder was first identified and the transducer was then moved caudally to the neck of the bladder and thence to the prostate. The author also opined that transabdominal, suprapubic ultrasonography was a non-invasive, quick and simple method for estimating prostate size in the dog, unlike transrectal and transurethral ultrasonography.

Kamolpatana *et al.* (2000) measured mean prostatic volume by water displacement method (14.35 ± 8.6 cm^3) in cadavers of 12 intact male dogs. The authors also determined the mean prostate length (L), width (W) and depth (D) sonographically which were comparable to the similar data collected in the study conducted by Ruel *et al.* (1998). The prostatic volume was calculated using the formula of V_E (volume of an ellipsoid) = ($0.524 \times L \times W \times D$) and V_B (volume for a box) = ($L \times W \times D$). The authors derived a formula for calculating prostatic weight from measured prostatic volume (Vm). (Prostate weight = ($0.96 \times Vm$) + 0.43) and the measured prostatic volume could be predicted using the formula, $Vm + [½_{.6} (L \times W \times D)] + 1.8$ cm^3. An excellent correlation was observed between prostatic volume calculated from ultrasonographic measurements and water displacement method.

Normal prostatic ultrasonography was of homogenous echodense pattern (Smith, 2008).

2.7.3. Sampling Technique

Barsanti and Finco (1995) opined that the third fraction of the ejaculate was the clear prostatic fluid and should be collected separately from the rest of the fluid.

Collection of ejaculate fluid, fine needle aspiration and urethral catheterization with prostatic massage were the three most common and effective sampling techniques. Prostatic massage combined with urethral catheterization could be performed if the dog was unable to ejaculate Kraft *et al.* (2008).

2.7.4. Prostatic Biopsy

Chang *et al.* (1996) opined that ultrasound-guided biopsy was a convenient and reliable method for taking tissue from the prostate and the precise monitoring of the needle tract on the ultrasound screen, reduced the chance of inadvertent access to other organ.

Zinkl (1999) opined that fine needle aspiration of the prostate gland under ultrasound guidance was an effective means of localizing and diagnosing prostatic lesions.

Kustritz (2006) stated that a biopsy was warranted in cases in which less invasive diagnostic tests did not render a diagnosis, or case had been unresponsive to initial therapy, or in cases in which an immediate diagnosis was warranted to ensure prompt treatment. Prostatic biopsy yielded a diagnosis in approximately 66% of cases, in comparison to 50% in fine needle aspirates of the prostate.

Smith (2008) stated that the gold standard test for determining disease processes involving the prostate was histological examination of a prostatic biopsy. Peritonitis secondary to seeding of bacteria from an abscess, or dissemination of neoplastic cells along the instrument tract were the

complications. The primary contraindications for performing biopsy of the prostate included potential prostatic abscess and acute prostatitis.

2.7.5. Prostatic Fluid Cytology

Prostatic fluid cytology was only supportive for the final diagnosis and there was substantial variability in shape and structure of epithelial cells as opined by Thrall *et al.* (1985).

Powe *et al.* (2004) and Smith (2008) stated that normal prostatic ejaculate was clear and may contain a few erythrocytes, leucocytes and squamous epithelial cells.

Kraft *et al.* (2008) stated that fine needle aspirate of a normal prostate gland commonly contained clusters of well differentiated cuboidal to low columnar prostatic epithelial cells and the epithelial cytoplasm was finely granular, basophilic, vacuolated with round to oval nuclei. The cells exfoliated in sheets and had honey comb appearance.

Smith (2008) reported that aspiration of any quantity of prostatic fluid from the prostate should be considered abnormal. Normal prostatic fluid was very minimal in amount, light yellow and translucent, and resembled urine. If unsure about the exact location of the needle at the time of aspiration, it is prudent to compare the sample to a urine sample to rule out inadvertent puncture of the urinary bladder or prostatic urethra.

2.7.6. Radiography

Lattimer (1986) stated that the size of the normal prostate should not exceed 50 % of the width of the pelvic inlet on a ventrodorsal radiograph. Later, Lattimer (1994) opined that the exact dimension of the prostate could not always be accurately determined on abdominal radiographs due to superposition of osseous structures as well as lack of abdominal serosal detail.

The study conducted by Atalan *et al.* (1999a) revealed a good correlation between radiographic and ultrasonographic prostate length measurement, provided the radiographic magnification error was eliminated. The authors suggested that radiographic prostatic depth was an unreliable estimate of prostate size and agreed with the findings of O'Shea (1962). The reasons for that might be due to imprecise organ boundaries, particularly on radiographs, or the presence of adjacent viscera, especially the colon, which might obscure the dorsal radiographic border of the prostate.

Smith (2008) observed that survey radiography was of limited value for diagnosis of specific prostatic diseases; however, it could be used to determine the size, shape, contour and location of the prostate and recommended both lateral and ventrodorasl views of the caudal abdomen for the radiography of the prostate.

2.8. CLASSIFICATION

Paclikova *et al.* (2006) reported that prostatic disorder included benign prostatic hyperplasia, squamous metaplasia, prostatic inflammation, prostatic abscess and cysts, as well as neoplasia. Smith (2008) opined that benign prostatic hyperplasia and chronic prostatitis were the most common conditions affecting the canine prostate gland.

2.8.1. Benign Prostatic Hyperplasia (BPH)

Benign prostatic hyperplasia was a spontaneous, age-dependent change in the structure of the gland of humans, dogs and chimpanzees (Berry *et al.*, 1986 and Johnston *et al.*, 2000).

Atalan *et al.* (1999b) reported that the prostate weights in 36 dogs with BPH ranged from 10 to 100 g (median 35 g), with mean prostatic dimensions of 4.5 × 3.2 × 4.5 cm. The author also reported that the size of the prostate gland increased up to 4 times its normal size in a hyperplastic prostate.

Lowseth *et al.* (1990) stated that the prostate in human beings is anatomically fixed between the symphysis pubis and the rectum; and the hyperplastic growth compresses inwardly on the urethra, producing urinary obstruction. Where as in dogs, the prostate is not anatomically fixed and hyperplastic growth expands outwardly in all directions, producing rectal obstruction and constipation. The author recorded more stromal growth than glandular involvement in dogs which supported the use of the dogs as a model for humans.

Kutzler and Yeager (2005) described that BPH becomes a clinical problem once it pressurizes the colon or if it encroaches into the muscles and nerves in the pelvic canal.

2.8.1.1. *Glandular Hyperplasia and Cystic Hyperplasia*

Glandular hyperplasia was characterized by elaborate projections of a hypertrophic and hyperplastic epithelium into the lumina of enlarged alveoli while cystic hyperplasia was characterized by a cuboidal hyperplastic epithelium, with the formation of large cysts and an increase in the ratio of stromal to epithelial cells (Deklerk *et al.,* 1979). The author also suggested that glandular hyperplasia might represent an earlier stage of development of the disease preceding the appearance of cystic hyperplasia.

In dogs, hyperplasia affects mostly epithelial cells, while in humans the process takes place more frequently within the stroma of the gland (Isaacs, 1984).

Lowseth *et al.* (1990) found that the relative lumen of the glandular acini of the prostate was increased with age, which reflected the cystic nature of complex BPH.

Paclikova *et al.* (2006) reported that although the term hyperplasia was widely used, changes in both the number (hyperplasia) and the size (hypertrophy) of cells always happened.

Smith (2008) stated that, initially hyperplasia begins as glandular hyperplasia and subsequently get transformed into cystic hyperplasia, which often lead to formation of cystic structures within the parenchyma of the prostate, giving it a honeycomb appearance.

2.8.1.2. Etiological Factors

Deklerk *et al.* (1979) demonstrated that dihydrotestosterone alone or in combination with 17β-estradiol could stimulate the prostatic growth. It was also observed that administration of 17 β- estradiol maintained the size of the prostate of the intact dog and stimulated stromal growth of the prostate of the castrated dog.

Cochran *et al.* (1981) confirmed that the level of estrogen in both serum and prostatic fluid of dogs suffering from BPH was significantly elevated compared to young animal, while, Barsanti and Finco (1986) opined that an overproduction of dihydrotestosterone within the prostate was the primary cause for BPH.

Berry *et al.* (1986) reported that prostatic growth as well as its secretion was controlled by 5-α dihydrotestosterone, derived from testosterone after cleavage by 5-α reductase.

Bamberg-Thalen and Linde-Forsberg (1993) opined that although the pathogenesis of BPH had not been elucidated, it was clear that BPH begins with an alteration of the androgen: estrogen ratio secreted by the testes. Benign prostatic hyperplasia was androgen-dependent and therefore did not occur in castrated dogs as stated by Dorfman and Barsanti (1995).

Prostatic growth (both stromal and glandular components) and secretion were mediated by dihydrotestosterone (DHT), a metabolite of testosterone formed in the presence of the enzyme, 5α-reductase (Johnston *et al.*, 2000).

Johnston *et al.* (2000), Davidson (2003) and Parry (2007) reported that more than 80 percent of the male dogs over five years of age exhibited BPH and prostatic volume was 2 to 6.55 times greater than that of normal dogs of similar weight.

2.8.1.3. Epidemiology

Deklerk *et al.* (1979) confirmed that the spontaneous prostatic hyperplasia was a disease that increased in frequency with age. The benign prostatic hyperplasia had been reported in dogs as early as 2 years of age and resulted in a homogenous increase in the size of the gland (Berry *et al.*, 1986).

Gobello and Corrada (2002) stated that 16 per cent of male dogs had an evidence of BPH at two years of age, 50 per cent had evidence of BPH at 4.1 to 5 years of age and more than 95 per cent of the animals suffered from the disease by 9 years of age.

Benign prostatic hyperplasia was a spontaneous disease of intact male dogs that begins as glandular hyperplasia as early as 3 years of age (Smith, 2008).

2.8.1.4. Diagnosis

2.8.1.4.1. Clinical Signs

Krawiec and Heflin (1992), Baker and Lumsden, (2000), Parry (2007) and Smith (2008) stated that the clinical signs in affected dogs include sanguineous prostatic fluid dripping from the tip of penis, blood in the urine or semen, constipation, difficult urination and sometimes asymptomatic.

Smith (2008) opined that ribbon or tapered stools were common for dogs with enlarged prostate due to compression of the rectum by enlarged prostate.

2.8.1.4.2. Digital rectal Palpation

Barsanti and Finco (1989), Johnston *et al.* (2000) and Davidson (2003) described that on rectal palpation of a dog with BPH, the prostate was uniform symmetrical enlargement without evincing pain on palpation. Benign prostatic hyperplasia produces a symmetrical prostate with normal contour, although intraparenchymal cyst formation might result in some degree of asymmetry and was smooth, movable and painless.

Johnston *et al.* (2001) described that the prostate gland was palpable per rectum unless prostatomegaly was severe enough to cause cranial movement of the prostate into the abdomen, in which case, simultaneous palpation of the rectum and application of upward pressure on the caudal abdomen or "wheelbarrow" the animal up to stand on its hind legs so as to push the prostate back within the reach of the gloved finger within the rectum. The authors reported that the animal did not evince pain when pressure was applied on the normal or hypertrophied prostate.

2.8.1.4.3. Ultrasonography

Peeling and Griffith (1984) opined that the transrectal ultrasonography caused discomfort when the probe was introduced to the rectum, especially in dogs with prostate enlargement.

Lamb (1990) in a review of abdominal ultrasonography in small animals described the indications, examination techniques and interpretation of abnormal findings of prostatic ultrasonography. Prostatomegaly, signs of urinary tract infection, haematuria, hemospermia and caudal abdominal pain were the indications.

Atalan *et al.* (1999b) derived a formula to estimate prostate weight and volume from physical and ultrasonographic measurements and found that width

and length of the prostate (measured physically or ultrasonographically) were the best predictors of prostatic volume and weight.

In cases of BPH, ultrasound revealed prostatomegaly, with a homogenous parenchyma with or without cavitating cystic lesions (Johnston *et al.,* 2000), where as Smith (2008) stated that in cases of inflammation, hyperplasia or neoplasia the homogenous nature of the prostate was lost, and focal to multifocal areas of hyperechoic and or hypoechoic tissue became apparent.

On sagittal images of the prostate, the mean length and height of prostate were 3.4 ± 1.1cm and 2.8 ± 0.8 cm respectively and on transverse images, the mean width, the mean height and the mean calculated volume were 3.3 ± 0.9, 2.6 ± 0.7 cm and 18.9 ±15.5 cm^3 respectively as reported by Ruel *et al.* (1998). The authors defined the length of the prostate on sagittal images as the maximum diameter of the gland along the urethral axis and height as the maximum diameter perpendicular to the axis of length. On transverse images, the height was defined as the diameter of the prostate on a line separating the two lobes of the gland and the width as the maximum diameter perpendicular to the axis of the height and used the formula length× width×height×0.523 for estimating prostatic ellipsoid volume in their study.

2.8.1.4.4. Radiography

Radiographically, the prostate gland was considered by Feeney *et al.* (1987) to be large if the prostatic dimensions exceed 70 per cent of the distance between the sacral promontory and the pecten of the pubis.

The results of the study conducted by Atalan *et al.* (1999a) suggested that the prostatic enlargement did not occur uniformly, but there was greater increase in length than depth and the authors recommended that the prostatic length rather than depth should be used when evaluating prostatic size from lateral abdominal radiograph.

2.8.1.4.5. Prostatic fluid evaluation

In BPH, the nuclei of prostatic epithelial cells are round to oval with finely reticulated or stippled chromatin patterns (Zinkl, 1999).

Baker and Lumsden (2000) reported an increase in cell size and mild anisokaryosis, with preserved nucleus : cytoplasm ratio in BPH.

The cytology of the prostatic fluid in BPH was normal, but presence of blood in prostatic fluid of the ejaculate or in prostatic fluid emitted from the end of the penis was seen in BPH (Johnston *et al.*, 2000, Davidson, 2003 and Parry, 2007).

Poor cellularity associated with aspiration of fibrotic tissue, masking of mild BPH with inflammation and misinterpreting of dysplastic epithelium as neoplastic epithelium were the limitations of cytologic diagnosis for canine prostatic disorders (Powe *et al.*, 2004).

Smith (2008) reviewed that diagnosis of BPH could be made after biopsy; however, a presumptive diagnosis was usually made based on a thorough history, physical examination findings, and prostatic fluid evaluation via prostatic massage.

2.8.1.4.6. Hemogram

Hematologic and serum biochemical findings were unaffected by hyperplasia of the prostate (Gobello and Corrada, 2002 and Parry, 2007).

2.8.1.4.7. Serum biochemistry

2.8.1.4.7.1. Acid phosphatase

Total acid phosphatase (TAP), prostatic acid phosphatase (PAP) and non prostatic acid phosphatase (NPAP) serum concentrations in dogs suffering from non- prostatic diseases were 3.3 ± 2.0; 1.6 ± 0.9 and 1.7 ± 1.5 U/ litre and TAP,

PAP and NPAP serum concentrations in dogs suffering from benign prostate hypertrophy were 3.8 ± 1.5, 2.9 ± 1.6 and 0.9 ± 0.6 U/ litre respectively. The TAP, PAP and NPAP serum concentration in 19 months- five year old and more than five year old male dogs were 2.8 ± 1.6, 1.4 ± 1.7 and 1.4 ± 0.6 U/ litre and 5.0 ± 2.6, 3.4 ± 2.4 and 1.6 ± 1.2 U/litre respectively. (Corazza *et al.*, 1994).

Amorim *et al.* (2004b) determined mean PAP serum value in healthy dog as 0.7 U/ litre and urinary PAP value as 0.1 U/ litre.

2.8.1.4.7.2. Serum testosterone

Lowseth *et al.* (1990) estimated the serum testosterone levels in healthy beagle dogs aged 3 to 14 years using RIA and found no statistical difference with age, instead a wide range of value (92-2,550 pg/ml) was observed within age groups and also between age groups. The authors also found that the measured levels of testosterone could not be used to predict the degree of BPH present.

2.8.1.4.8. Urinalysis

Urinalysis of a dog with BPH might be normal or contain blood without pyuria or bacteriuria (Gobello and Corrada, 2002).

Parry (2007) stated that urinalysis might be normal in BPH, but some erythrocytes and slightly increased numbers of squamous epithelial cells might be present. The urine culture should be negative and concurrent prostatitis should be considered if positive.

2.8.1.4.9. Pathology

2.8.1.4.9.1. Gross

Bloom (1954) noticed that in canine prostate hyperplasia, the gland was severely enlarged with the outer surface smooth or irregularly nodular. The length of the gland showed a greater increase in dimension than width. On sectioning the

lobules varied in size and were outlined by wide irregular whitish grey bands of stromal tissue, along with cysts containing clear or cloudy fluid, irregularly distributed through out the parenchyma.

O'Shea (1963) stated that diffuse glandular hyperplasia of the prostate might be accompanied by cyst formation, and stromal hyperplasia, was very common in older dogs, but had not been shown to be a preneoplastic change.

Pearson and Gibbs (1971) stated that hyperplasia, the most common pathological change of the canine prostate, was associated with marked enlargement of the gland.

Klausner *et al.* (1995) suggested that canine prostatic hyperplasia occurred in two phases, glandular and complex. Glandular hyperplasia was common in dogs less than five years and was characterized by symmetric enlargement of the prostate. Complex hyperplasia common in dogs over five years and was characterized by asymmetric enlargement of the prostate.

Davidson (2003) opined that hyperplastic prostate was symmetrically, smooth and contained small intra parenchymal cysts with bloody fluid.

2.8.1.4.9.2. Histopathology

Bloom (1954) observed that glandular hyperplasia was characterized by increase in the glandular tissue and irregularity in the size and shape of the acini. The lining epithelial cells were tall columnar and formed villous and papillary intra luminal projections. Cysts lined by flattened cuboidal epithelium characterized cystic hyperplasia. The cystic lumen contained desquamated epithelium and accumulated secretion. The inter lobar stroma frequently increased in connective tissue with accumulation of inflammatory cells.

Leeds and Leav (1969) reported case of hyperplasia with and without concurrent inflammation. Hyperplastic prostate without inflammatory changes contained dilated acini, lined by hyperplastic tall columnar cells that had

eosinophilic granular cytoplasm and ovoid basal nuclei. Dilated acini were surrounded by smooth muscle and fibrous connective tissue. Hyperplasia with concurrent inflammation was characterized by the presence of inflammatory cells.

2.8.1.5. Treatment of Benign Prostatic Hyperplasia

Johnston *et al.* (2000) reported that Diethystilbestrol at the dose of 0.2–1.0 mg, PO, every 2–3 days, for 3–4 weeks was historically, as an effective medical treatment of BPH in dogs, but potential adverse side effects of diethystilbestrol in dogs included bone marrow suppression and pancytopenia, and squamous metaplasia of the prostate, with ductal obstruction and cyst formation. The author opined that only 53% of BPH dogs treated with medroxyprogesterone acetate showed decreased prostatic size by 6 weeks of treatment. The potential adverse sequelae of progesterone administration in dogs were development of diabetes mellitus and mammary nodules. The finasteride treatment in dogs resulted in decreased semen volume, but had no adverse effect on semen quality and serum concentration of testosterone. The author also opined that finasteride-induced prostatic involution, appeared to occur via apoptosis, not necrosis, in BPH dogs.

White (2000) suggested that castration was the most effective treatment for removing the hormonal influence in dogs with BPH.

Surgical castration caused 70 % reduction in size of prostate after surgery. Although the prostate began to shrink within 7-14 days after castration, complete involution might require 4 months (Johnston *et al.*, 2001 and Kutzler and Yeager, 2005).

Sirinarumitr *et al.* (2001) found that the most common medical treatment for BPH was finasteride, a synthetic steroid type-Π 5∞-reductase inhibitor that has been used in human urology for over a decade in the management of BPH.

Zhang *et al.* (2004) reported that hot agarose solution and enzyme injection could significantly ablate prostate tissue without identifiable complications.

2.8.2. Prostatitis

Inflammation of the prostate gland was the second most common prostatic disorder as reported by Barsanti and Finco (1979).

2.8.2.1. Etiology

Escherichia coli was the most common bacterial organism identified in dogs with bacterial prostatitis, followed by *Staphylococcus aureus*, *Klebsiella* spp., *Proteus mirabilis*, *Mycoplasma canis*, *Pseudomonas aeruginosa*, *Enterobacter* spp., *Streptococcus* spp., *Pasteurella* spp., and *Haemophilus* spp. (Ling *et al.,* 1983; Barsanti and Finco 1986 and Johnston *et al.*, 2000).

Brucella canis could spread into prostate gland, but the main target remained testicles and epididymis (Ling *et al.*, 1983; Krawiec and Heflin, 1992 and Johnston *et al.,* 2000).

Brucella canis, Brucella suis and *Brucella abortus* were all capable of causing prostatitis in dogs (Barr *et al.,* 1986).

Barsanti and Finco (1986) Krawiec and Heflin (1992) stated that in most cases, bacteria that ascend up the urethra colonized in the prostate, however there was also hematogenus spread.

In healthy dogs, prostatic tissue discharges contained prostatic antibacterial factor (PAF) (Barsanti and Finco, 1989). Prostatic antibacterial factor is a low molecular peptide, containing zinc, thermo-stable water-soluble substance responding to bacterial infestation and the most frequent etiological factor of prostatitis was ascending infection of an aerobic microflora from the urethra (Paclikova *et. al.,* 2006). Cowan *et al.* (1991) and Duque *et al.* (2009)

reported prostatitis in a castrated dog and might be due to the deficiency of zinc-associated protein (an antibacterial factor) after neutering.

Krawiec and Heflin (1992) and Dorfman and Barsanti (1995) opined that dogs become predisposed to prostatitis by increased number of bacterial organisms in the periprostatic urethra, compromise of local immunity, disease of urinary tract, altered prostatic tissue and fluid flow as in the cases of BPH and cysts. Infection was mostly of ascending nature from the urethral flora, along with hematogenous spread or extension from testes, epididymis or peritoneal cavity.

Jayathangaraj *et al.* (1993) reported a case of prostatitis and secondary acute renal failure in a Pomeranian dog.

Dorfman and Barsanti (1995) reported that the most contaminant bacteriae were gram positive and large numbers of gram-negative bacteriae and degenerated neutrophils with intracytoplasmic bacteriae were indicative of inflammation.

Klausner *et al.* (1995) observed infections with anaerobic bacteria and fungal agents like *Blastomyces dermatitidis*, *Cryptococcus neoformans*, or *Coccidioides immitis* via hematogenous spread, urethral ascent, or penetration through the scrotum with descending prostate infection from a testicular source

Both acute and chronic infections occurred in the canine prostate gland, usually as a result of ascend of normal aerobic urethral bacteria including mycoplasma into a gland with benign prostatic hypertrophy (Johnston *et al.*, 2000).

Paclikova *et al.* (2006) reviewed that *Escherichia coli* had been the most frequently found pathogen so far, but *Staphylococcus aureus, Klebsiella* spp., *Proteus mirabilis, Mycoplasma canis, Pseudomonas aeruginosa, Enterobacter* spp., *Streptococcus* spp., *Pasteurella* spp., and/or *Haemophillus* spp. could also

be found. The major mycoses that affected prostate gland were *Blastomyces dermatitidis, Cryptococcus neoformans* and *Coccidoides immitis.*

Mycoplasma canis and *Ureaplasma* spp. were other possible opportunistic pathogens, and fungal prostatitis occurs rarely (Parry, 2006).

Brennan *et al.* (2008) reported prostatitis in a dog with brucellosis and the lesions were of interstitial type.

2.8.2.2. Epidemiology

Ling *et al.* (1983) Krawiec and Heflin (1992) reported that bacterial prostatitis accounted for more than 38 per cent of the dogs identified with prostatic disease and the mean age of affected dog was 8.2 years.

Cowan and Barsanti (1991) reported the prevalence rate for prostatitis as 20-70%.

2.8.2.3. Diagnosis

The values of prostatic fluid pH, specific gravity, cholesterol and zinc were not reliable aids in the diagnosis of bacterial prostatitis in dogs where as in prostatic fluid iron, showed a significant difference between healthy dogs and dogs with prostatitis (Branam *et al.,* 1984). Johnston *et al.* (2000) stated that *B. canis* serology indicated in male dogs with prostatitis in order to rule out canine brucellosis. The author opined that presumptive diagnosis of acute prostatitis is based on presence of clinical signs in an intact or recently castrated male dog. The presumptive diagnosis of chronic prostatitis was based on signs of infertility or decreased libido.

2.8.2.3.1. Clinical signs

Clinical signs in dogs with acute prostatitis included depression, pain on rectal palpation of the prostate, fever, straining to urinate or defecate, a ''stiff-legged'' gait, haematuria, edema of the scrotum, prepuce or hindlimb, and

pollakiuria (Krawiec and Heflin, 1992; Dorfman and Barsanti, 1995 and Johnston *et al.*, 2000) while, vomiting, caudal abdominal pain, preputial discharge and possibly unwillingness to breed were noticed by Smith (2008).

The clinical signs were absent in dogs with chronic prostatitis, or consist of poor semen quality with infertility or sometimes decreased libido if prostatic contraction was painful (Johnston *et al.*, 2000).

2.8.2.3.2. Digital rectal palpation

Davidson (2003) and Smith (2008) stated that in acute prostatitis, the prostate was normal to slightly enlarged, asymmetrical, and painful on palpation, while in chronic prostatitis, the prostate was symmetrical, nonpainful and firm.

2.8.2.3.3. Ultrasonography

Duque *et al.* (2009) found diffused increase of prostatic echodensity, enlarged prostate (5.28 cm length × 4.9 cm width) and multiple intraprostatic cysts (2.8 cm diameter) on abdominal ultrasonography of dogs with prostatitis.

Diffused increase in prostatic echodensity, which became more pronounced over time, was reported in prostatitis (Smith, 2008).

2.8.2.3.4. Radiography

Atalan *et al.* (1999a) opined that disease processes like prostatic inflammation or neoplasia might affect prostatic outline, but extension into surrounding tissue lead to indistinct prostatic margin on abdominal radiographs.

2.8.2.3.5. Prostatic fluid evaluation

Ling *et al.* (1983) opined that bacteria-laden white blood cells in canine prostatic fluid were indicative of active infection.

Prostatic massage, immediately followed by a prostatic wash was a very good technique to obtain samples for cytology and bacteriology, especially in the dog in which an ejaculate could not be easily obtained, due to lack of interest, fear or pain (Barsanti and Finco, 1986, Johnston *et al.*, 2001. and Kustriz 2006).

Detecting inflammatory exudates in prostatic fluid collected by ejaculation or prostatic massage was the definitive diagnosis of chronic prostatitis (Johnston *et al.*, 2000). The authors also opined that needle aspiration of an infected prostate or prostatic abscess should be avoided to prevent seeding of the needle track with bacteria and the exudates should be examined cytologically and microbiologically for the diagnosis of bacterial and fungal disease as well as determination of bacterial sensitivity to antibiotics.

Powe *et al.* (2004) stated that there was a high (80 per cent) concordance between cytologic and histologic diagnoses of prostatic disease and canine prostatic cytology was a more sensitive method than histology for the detection of sepsis, as the thin monolayer obtained with cytologic smears often allowed improved detection of etiologic agents as well as better assessment of individual cytomorphology.

Smith (2008) stated that fine needle aspiration could be used to collect both fluid and tissue for cytological evaluation, microbial culture, and to drain fluid from cystic lesions within or exterior to the prostate. The coffee-ground appearance to the prostatic fluid indicated chronic heamorrhage.

2.8.2.3.5.1. Culture and sensitivity

Barsanti and Finco (1995) reported that the healthy dogs had less than 100 bacteria / ml of prostatic fluid and the undetected urethral contamination accounted for up to 10^5 bacteria / ml without evidence of inflammation.

Johnston *et al.* (2000) stated that normal canine semen and or prostatic fluid should contain less than 10,000 bacteria per milliliter, and sediment should not contain significant numbers of inflammatory cells.

Rohleder and Jones (2002) reported a case of emphysematous prostatitis from which *Escherichia coli* was isolated.

Culture of gram positive bacteria of several species at concentrations < 100,000/ml should be viewed with caution, as should growth and conversely, heavy growth of a pure culture of gram-negative bacteria often indicated infection (Smith, 2008).

2.8.2.3.6. Hemogram

Johnston *et al.* (2000) reported that laboratory finding in most of the dogs with acute prostatitis included regenerative leucocytosis, although occasionally animals were leukopenic.

Davidson (2003) and Smith (2008) reported that neutrophilic leucocytosis with left shift in acute prostatitis or prostatic abscess with low-grade sepsis while, the hemogram of dogs with chronic prostatitis was usually normal. The former also stated that, leucopenia or degenerative shift might be present with septic shock.

Duque *et al.* (2009) reported a mild nonregenerative anemia (5.1×10^6 cells/µl) in a castrated dog with acute prostatitis while elevated leucocytosis (19.6 × 103 cells/µl) was noticed in an intact male dog with prostatitis and preputial oedema.

2.8.2.3.7. Serum biochemistry

Jayathangaraj *et al.* (1993) reported elevated serum creatinine (8 mg/dl) in a Pomeranian dog with prostatitis.

Duque *et al.* (2009) found elevated BUN (229 mg/dl), creatinine (2.6 mg/dl) and alkaline phosphatase (416 UI/L) in a dog with prostatitis and preputial oedema. There is paucity of literature regarding the serum testosterone and acid phosphatase.

2.8.2.3.8. Urinalysis

In acute prostatitis, urinalysis typically revealed pyuria, haematuria and bacteriuria and these abnormalities were absent in dog with chronic prostatitis (Davidson, 2003 and Smith, 2008).

In acute prostatitis, urine collected by cystocentesis might contain blood, bacteria, and leukocytes, because prostatic fluid constantly drips retrograde from the prostatic urethra into the urinary bladder in the intact male dog. In dogs with chronic prostatitis, urine might contain blood, bacteria and leukocytes as with acute prostatitis, and the case usually presented to a veterinarian for suspected lower urinary tract disease (Johnston *et al.*, 2000). Duque *et al.* (2009) reported proteinuria, pyuria and haematuria on urinalysis of a dog with prostatitis.

2.8.2.3.9. Pathology

2.8.2.3.9.1. Gross

Mapes (1987) reported a case of prostatic abscess that caudally displaced the urinary bladder and resulted in perineal herniation.

Davidson (2003) noticed that in acute prostatitis the prostate was enlarged and asymmetrical with fluctuant areas of abscesses, along with firm areas and adhesions.

2.8.2.3.9.2. Histopathology

Bloom (1954) observed that in suppurative prostatitis the lumen of the ducts and acini, as well as the stroma contained infiltrations of polymorphonuclear leukocytes, occasional lymphocytes and histiocytes. In

chronc prostatitis there was focal or diffused proliferation of fibrous tissue, which contained varying numbers of lymphocytes and plasma cells.

Ladds (1993) observed that in acute prostatitis, there was focal or diffuse suppurative inflammation with accumulation of neutrophils in the acini and stroma. In chronic prostatitis, lymphocytes were the predominating type of inflammatory cell that invaded the stroma.

Klausner *et al.* (1995) suggested that suppurative and chronic prostatitis were the most common types. They revealed the presence of epithelial cells, degenerated neutrophils, lymphocytes, plasma cells and bacteria in exfoliative cytology in cases of canine prostatitis

2.8.2.4. Treatment

Castration resulted in prostatic atrophy, and had been shown to reduce duration of chronic bacterial prostatitis and number of bacterial colony-forming units per milliliter of urine in experimentally infected dogs (Cowan *et al.*, 1991).

Dorfman *et al.* (1995) reported that antibiotics known to diffuse into the prostatic fluid of the normal dog in therapeutic concentrations included trimethoprim-sulfa, chloramphenicol, and enrofloxacin, all of which were effective in treating most aerobic bacterial infections of the canine prostate.

Treatment strategies for dogs with prostatitis included specific antimicrobial therapy, and consideration of castration or antiandrogen therapy to decrease prostatic size. Antibiotic therapy should be selected based on sensitivity of bacteria cultured .The blood–prostate barrier in the normal dog prevents diffusion of drugs with low lipid solubility or those that were highly protein bound in plasma from entering the prostatic fluid in therapeutic concentrations. In addition, pH gradients between blood and prostatic fluid may influence ionized drug trapping in prostatic fluid. Enrofloxacin was effective against mycoplasma infections also. Trimethoprim-sulfa and enrofloxacin were not effective against

anaerobic infections, while chloramphenicol was indicated in these cases. Inflammatory compromise of the blood–prostate barrier in acute and chronic prostatitis, which might permit therapeutic concentrations of other antibiotics to reach the site of infection, had not been well studied. Fungal prostatitis usually occurred as a part of systemic fungal infection in the dog, which should be treated with systemic antifungal regimens. Castration should be considered in dogs with prostatitis if the disorder was a recurrent one, or infection was associated with a hyperplastic or abscessed gland, or reproductive potential was unimportant to the client. Castration was not recommended in the presence of acute infection; as such surgery might result in scirrhous spermatic cords. Therapy with megestrol acetate 0.11 mg / kg per orally once daily or finasteride 5 mg orally once daily might be used to accomplish decrease in prostate size temporarily, until the infection is controlled with antibiotics and castration, or until breeding, if desired (Johnston *et al.*, 2000).

Duque *et al.* (2009) observed a complete recovery from a dog with acute prostatitis when enrofloxacin was given @ 10 mg/kg/ day per orally.

2.8.3. Prostatic abscess

2.8.3.1. Etiology

Infection of prostatic cysts or early stages of neoplasia were the factors, associated with formation of prostatic abscess (Christie, 1983 and Barsanti and Finco, 1989). Prostatic abscesses developed as a result of bacterial contamination spreading from another part of the urinary tract, by hematogenous route, or from cysts that become secondarily infected as opined by Barsanti and Finco (1986) and Basinger *et al.* (1993).

Krawiec and Heflin (1992) and Johnston *et al.* (2001) stated that prostatic abcessation was a sequelae to chronic prostatitis where cavities of purulent fluid were found within the parenchyma of the prostate. According to Baker and

Lumsden, (1999), prostatic abscesses developed either after fusion of small areas of infection within the gland, or after infection of prostatic cysts.

2.8.3.2. Epidemiology

Krawiec and Heflin (1992) reported that out of 33 dogs diagnosed with prostatitis, four (12.12 per cent) had abscesses.

2.8.3.3. Diagnosis

2.8.3.3.1. Clinical signs

Baker and Lumsden (2000), Johnston *et al.* (2000) and Davidson (2003) stated that a fulminating infection or peritonitis secondary to rupture of the abscess could end up in septic shock and cardiovascular shock. Boland *et al.* (2003) reported clinical signs like mild to moderate illness, inappetance, abdominal pain, pyrexia, dysuria, stranguria, penile discharge, tenesmus and ribbon like faeces in dogs with prostatic abscess. Kutzler and Yeager (2005) and Smith (2008) reported the clinical signs of prostatic abscess as anorexia, fever, depression, gait abnormalities, tenesmus, dysuria and caudal abdominal pain.

2.8.3.3.2. Digital rectal palpation

Boland *et al.* (2003) reported that the rectal examination of dog with prostatic abscess revealed asymmetrical enlargement of prostate and pain on palpation while, palpable fluctuant areas were present in prostatic abscess as reported by Davidson (2003).

2.8.3.3.3. Ultrasonography

Boland *et al.* (2003) stated that typical prostatic abscess appeared as hypoehoic cavitary lesions with irregularly defined margins. The fluid within the lesion might have mixed echogenisity or a flocculent appearance. In contrast, typical prostatic cysts appeared as anechoic cavitary lesions with more regularly defined margins.

Ultrasonography of abscess revealed focal hypoechoic to anechoic areas in prostatic parenchyma and prostatic abscess often had irregular borders and the surrounding parenchyma had decreased echogenicity (Davidson, 2003).

2.8.3.3.4. Prostatic fluid evaluation

Baker and Lumsden (2000) opined that, if sampling had already begun and purulent fluid was noted, aspiration should continue until all pressure reduced to prevent leakage of material.

Boland *et al.* (2003) found that the fluid aspirated from abscess was either purulent or serosanguineous and cytopathological examination revealed degenerate neutrophil and bacteriae.

2.8.3.3.5. Hematobiochemistry

Boland *et al.* (2003) found that hematology and serum biochemical results of dog with prostatic abscess were variable and the most common abnormalities included mild to moderate leucocytosis, lymphopenia, or both. Alkaline phosphatase was elevated in three dogs with prostatic abscess.

Davidson (2003) recorded that dogs with prostaic abscess had elevated serum alkaline phosphatase, hypoglycemia, elevated serum alanine amino transaminase, hyperglobulinemia and azotemia. There was paucity of literature regarding the serum testosterone and acid phosphatase.

2.8.3.3.6. Urinalysis

In the study conducted by Boland *et al.* (2003), urinalysis was found to be normal and cultures were negative in three dogs with prostatic abscess, whereas urine culture of four dogs with prostatic abscess yielded pure growth of *E. coli* and *Staphylococcus intermedius*. The authors also observed blood pigment and protein in urine sample of a dog with prostatic abscess.

2.8.3.4. Treatment

In the study conducted by Mullen *et al.* (1990) 92 dogs treated with multiple penrose drain applications, three dogs died during surgery, and 19 died or euthanized in the immediate postoperative period because of sepsis, shock and peritonitis.

Surgical drainage had been recommended for prostatic abscessation, as antibiotic therapy alone would not result in cure (Mullen *et al.,* 1990 and Glenon and Flanders, 1993).

Postoperative complications included painful abdomen, scrotal / preputial / hindlimb edema, hypoproteinemia, hypoglycemia, anemia, sepsis / shock, hypokalemia and urine leakage from penrose drains. Poor results in surgical drainage of abscessed prostates in the dog prompted some clinicians to substitute aggressive antibiotic and antiandrogen finasteride therapy for this procedure (Johnston *et al.,* 2000).

In another study conducted by Boland *et al.* (2003) in 13 dogs with either prostatic abscess or cysts in which the primary treatment was percutaneous ultrasound-guided drainage, no complications was observed after drainage.

2.8.4. Prostatic Cysts

Hoffer *et al.* (1977) classified prostatic cysts into multiple cysts associated with prostatic hyperplasia (small cysts within the prostate), prostatic retention cysts (cysts developed within the prostate as a result of obstruction of prostatic ducts), paraprostatic cysts (no contact with the prostate and arise from a uterus masculinus, hematoma, or a serosal cyst.), cystic prostate associated with squamous metaplasia (arise secondary to squamous metaplasia) and paraprostatic lymphatic cysts.

2.8.4.1. Retention Cysts

Retention cysts were present within the glandular tissue and were encapsulated, containing either clear or cloudy fluid (White *et al.,* 1987).

Pathogenesis was unknown, but the observation of retention cysts occurring concurrently with estrogen-secreting Sertoli cell tumors had prompted speculation that they might occur as dilation of prostatic acini secondary to estrogen-induced squamous metaplasia (Johnston *et al.*, 2000).

Retention cysts were formed when cavitating lesions, filled with fluid, or created within the parenchyma of the prostate and were typically communicated with the urethra (Smith, 2008).

2.8.4.2. Paraprostatic Cysts

Paraprostatic cysts were single or multiple structures often invading the space in between prostate gland and urinary bladder. It might sometimes-obstructing pelvic inlet (Olson *et al.*, 1987).

Evans and Christensen (1993) stated that paraprostatic cysts were found outside the prostate and had been associated with remanants of the uterus masculinus. Barsanti (1995) stated that paraprostatic cysts probably originated from persistent uterus masculinus.

Paraprostatic cysts usually arise craniolateral to the prostate, displacing the bladder cranially and ventrally, or caudal to the gland within the pelvis, as possible dilated embryonal remnants of Wolffian duct (Johnston *et al.*, 2000).

Head and Francis (2002) reported that mineralized paraprostatic cyst was a potential contributing factor in developing perineal herniation. Bakalov *et al.* (2004) recorded a case of paraprostatic cysts in 6 years old male boxer dog.

Paraprostatic cysts were often very large and could be palpated transabdominally, generally attached to the prostate via a stalk of tissue or adhesions (Smith, 2008).

2.8.4.3. Etiology

Barsanti and Finco (1986) stated that prostatic cysts might be associated with BPH and fluid retention caused by obstruction of canaliculi, leading to accumulation of prostatic fluid.

Prostatic cysts in the dog included diffuse cystic change associated with androgen-dependent BPH, as well as retention or paraprostatic cysts, which were cavitating lesions with a distinct wall, containing clear to turbid fluid, either within (retention) or outside (paraprostatic) the prostatic parenchyma (White *et al.*, 1987).

Hyperestrogenisation caused stasis of prostatic fluid within excretory ducts and acted as a triggering factor for squamous metaplasia which further increased the risk of developing prostatic retention cysts (Barsanti and Finco, 1989).

Ruel *et al.* (1998) opined that after 4 years of age, intraparenchymal cysts formation might be accompanied by BPH due to obstruction of the parenchymal ducts and subsequent accumulation of prostatic secretions, which then predisposed to infection and abscess formation. The author also stated that the prevalence of intraparenchymal cyst formation was 14 per cent.

Prostatic cysts were cavitary lesions filled with secretions from the glandular epithelium of the prostate (Krawiec and Heflin, 1992). According to Basinger *et al.* (1993) and Purswell *et al.* (2000) the prostatic cysts originated from an embryonic structure known as *uterus masculinus*.

Black *et al.* (1998) isolated *Mycoplasma* spp from the prostatic cystic fluid and reported that small cysts found in the parenchyma of a hyperplastic

prostate communicated with each other and get manifested macroscopically as prostatic cysts.

The remnant of the uterus masculinus occasionally resulted in enlarged cysts that were attached with the prostate by a stalk or adhesions (Johnston *et al.*, 2000).

Kustritz and Klausner (2000) opined that cysts might develop as a result of obstruction of ducts leading to accumulation of prostatic secretion within the parenchyma of the gland.

Paclikova *et al.* (2006) stated that prostatic cysts were most often observed as a result of benign prostatic hyperplasia.

2.8.4.4. Epidemiology

Black *et al.* (1998) and Ruel *et al.* (1998) observed that the prevalence of prostatic cysts in adult, intact male dogs was 14%. The former also reported that the infection rate was 42% and all the dogs were nonclinical.

2.8.4.5. Diagnosis

2.8.4.5.1. Clinical signs

Spackman (1988), Krawiec and Heflin (1992), Closa *et al.* (1995) and Johnston *et al.* (2000), described clinical signs as lethargy, straining to defecate, anorexia, intermittent urethral discharge and signs of estrogen toxicity like feminization and anemia in those associated with a sertoli cell tumor. However, Henson (2001) reported that clinical signs were uncommon unless the cyst or cysts become secondarily infected.

Affected dogs might be asymptomatic, or presented with signs referable to concurrent BPH or to physical displacement of abdominal viscera (Johnston *et al.*, 2000).

Gobello and Corrada (2002) stated that the affected dogs might be asymptomatic or might develop signs referable to concurrent BPH or to physical displacement of abdominal viscera.

Dog with prostatic cysts, most commonly was an incidental finding and exhibited no clinical signs (Boland *et al.*, 2003).

2.8.4.5.2. Digital rectal palpation

Boland *et al.* (2003) reported that prostatic cysts were typically enlarged, asymmetrical and non painful on rectal examination.

Small cysts could be palpated rectally as asymmetric prostatomegaly with soft, fluctuant areas where as large, discrete cysts might be palpable in the caudal abdomen or perineal area and very large cysts might cause abdominal distension (Davidson, 2003).

2.8.4.5.3. Ultrasonography

Closa *et al.* (1995) reported the importance of distension of the urinary bladder with saline to differentiate it with a large cyst. Ultrasound might identify mildly echogenic fluid (Davidson, 2003).

Small cystic structures did not alter the contour of the prostate, making them difficult to detect with transrectal palpation or radiology (Smith, 2008).

2.8.4.5.4. Radiography

Feeny *et al.* (1987) suggested that prostatic enlargement on lateral radiographs of greater than 90% of the pubic brim-sacral promontory distance was suggestive of neoplasia, abscessation or paraprostatic cysts.

Survey radiographs revealed a discrete fluid density in the caudal abdomen and there might be mineralized areas in the cyst wall. Excretory

urography revealed displacement of the ureters and help to identify the urinary bladder (Davidson, 2003).

Smith (2008) stated that contrast radiography could be used to determine whether a cystic structure was located within the prostate gland or paraprostatic.

2.8.4.5.5. Prostatic fluid analysis

In prostatic cysts, cytology revealed a modified transudate with erythrocytes and occasional epithelial cell and inflammatory cells (Baker and Lumsden, 2000 and Davidson, 2003).

Boland *et al.* (2003) found that the fluid aspirated from prostatic cysts was clear or straw coloured and cytopathlogical examination revealed predominately a modified transudate of low or moderate cellularity, containing a mixed population of macrophages, degenerated neutrophils, lymphocytes and without bacteriae.

2.8.4.5.5.1. Culture and sensitivity

Black *et al.* (1998) observed a correlation between the urine culture and prostatic fluid culture and stated that urine culture was helpful to predict the infection present in prostatic cysts.

2.8.4.5.6. Hematobiochemistry

Boland *et al.* (2003) also observed no abnormalities in hematological and serum biochemical values in dogs with prostatic cysts.

Hemogram and serum biochemistry were usually normal, although a neutrophilic leucocytosis with or without a left shift, and toxic neutrophilic changes might be present (Parry, 2007). There is paucity of literature regarding the serum testosterone and acid phosphatase.

2.8.4.5.7. Urinalysis

Boland *et al.* (2003) observed normal urinalysis and negative cultures in dogs with prostatic cysts.

Urinalysis was usually normal in prostatic cysts, but increased red blood cells or inflammatory cells might be seen if the cyst had communication with the urethra, hemorrhagic or associated with inflammation (Davidson, 2003 and Parry, 2007).

2.8.4.5.8. Pathology

2.8.4.5.8.1. Gross pathology

Weaver (1978) described prostatic cysts in 12 dogs. The content of the cysts varied from colorless pink or red serous material through a grey or cloudy appearance to a dark brown viscid material with fibro necrotic debris. The smallest cyst had a volume of 90 ml and the largest approximately a volume of 800 ml. The degree of attachment to the prostate varied from relatively limited pedicle to extensive adhesions.

Krawiec and Heflin (1992) stated that paraprostatic cysts were most commonly seen in the craniolateral or caudal aspect of the bladder and prostate in older large breed dogs.

2.8.4.5.8.2. Histopathology

Histologically, normal glandular cells lined these cysts. Rapid enlargement of cysts resulted in attenuation of epithelium (Black *et al.*, 1998).

2.8.4.6. Treatment

White and Williams (1991) and Barsanti and Finco (1995) recommended surgical excision or omentectomy as the treatment of choice for prostatic cysts.

Surgical resection, with or without concurrent castration, was the recommended treatment (Johnston *et al.*, 2000).

White (2000) observed that traditional therapy for prostatic cysts included surgical debridement, omentalization, marsupialization and placement of surgical drains.

2.8.5. Squamous Metaplasia of the Prostate

2.8.5.1. Etiology

According to the literature by Leeds and Leav (1969), squamous metaplasia had been diagnosed in about 67% of patients after treatment by estradiol-cyclopentylpropionate and also observed squamous metaplasia in patients suffering from estrogen producing sertoli cell tumor of testicles.

Brendler *et al.* (1983) concluded that squamous metaplasia was induced by increased estrogen concentration either by exogenous administration or from relative increase in serum estrogen concentration that occurred in normal aged dogs as androgen secretion declined.

Metaplastic cells were subsequently becoming inactive and might lead to stasis of prostatic fluid within the gland, the situation likely responsible for creating cysts and or abscess (Johnston, 1985 and Barsanti and Finco, 1989).

Retention cysts developed as a consequence of the dilation of prostatic acini secondary to estrogen-induced squamous metaplasia. The oestrogen secreting tumor could cause squamous metaplasia of prostate and thus predisposing to infection (Johnston *et al.*, 2000).

Gobello and Corrada (2002) stated the prostatic squamous metaplasia occured as a result of exogenous estrogen administration or estrogen – secreting sertoli cell tumors and these excessive serum estrogen concentrations caused the

epithelial cells of the prostate to undergo squamous metaplasia and decreased secretion of prostatic fluids.

Paclikova *et al.* (2006) stated that short term administration of estrogen results in metaplasia of region around the prostatic urethra and periurethral ductal tissue, while long term exposure made whole gland metaplastic. The term squamous metaplasia was described as pathophysiological as well as pathomorphological entity and might be a part of a more complex prostatic disease, with some other disorder dominating. Squamous metaplasia developed after exposure of the estrogen receptor of prostatic urethra and stroma of the gland to estrogen stimulation.

2.8.5.2. Diagnosis

2.8.5.2.1. Clinical signs

Clinical signs minimal except for potential hemorrhagic urethral discharge and a hyperestrogenic skin pattern and the rectal palpation revealed an enlarged gland without concomitant signs unless cysts present (Gobello and Corrada, 2002).

2.8.5.2.2. Digital rectal palpation

Gobello and Corrada (2002) stated that rectal palpation of dog with squamous metaplasia revealed an enlarged gland without concomitant signs unless cysts were present.

2.8.5.2.3. Ultrasonography

In squamous metaplasia, ultrasonography revealed prostatomegaly. Hypoechoic fluid filled cavities might also be seen, if cyst or abscess formation had resulted (Parry, 2007).

2.8.5.2.4. Prostatic fluid evaluation

Cytology of prostaic fluid from a dog with squamous metaplasia revealed squamous epithelial cells and possible hemorrhage (Gobello and Corrada, 2002).

Kraft *et al.* (2008) opined that the identification of squamous epithelial cells from fine needle aspiration of the prostate supported the diagnosis of squamous metaplasia or squamous cell carcinoma; whereas, same cells in fluid samples obtained from prostatic massage represent normal lower urinary tract squamous epithelial cells.

2.8.5.2.5. Hemogram

Parry (2007) stated that the hemogram findings of squamous metaplasia was consistent with oestrogen toxicity and included non-regenerative anemia, thrombocytopenia, granulocytosis or granulocytopenia.

2.8.5.2.6. Serum biochemistry

Isaacs (1984) observed no significant serum biochemical alteration in squamous metaplasia.

2.8.5.2.7. Histopathology

Leeds and Leav (1969) stated that the identifying characteristics of squamous metaplasia of prostate was the presence of concentrically arranged squamous cells that were flattened towards the acini, which contained eosinophilic material with pyknotic nuclei

2.8.5.3. Treatment

Treatment is focused on the elimination of estrogenic source, castration of neoplastic testis or interruption of exogenous estrogen administration (Gobello and Corrada, 2002).

2.8.6. Prostatic Neoplasia

The reported prostatic neoplasia included squamous cell carcinoma (Leib, 1986), transitional cell carcinoma of ductal epithelium, prostatic adenocarcinoma (Barsanti and Finco, 1989), prostatic adenomas, leiomyomas, fibromas and sarcomas (Turrel, 1989) and leiomyosarcoma and lymphoma (Mainwaring, 1990).

Cornell *et al.* (2000) examined 76 cases and classified 27 as being adenocarcinoma and 40 as mixed adeno- and transitional cell carcinoma. It was found that carcinomas with transitional cell differentiation tend to occur more commonly in castrated dogs whereas neutered dogs were evenly split between those with and without transitional differentiation.

Gobello and Corrada (2002) stated that prostatic malignancy was uncommon, adenocarcinoma was the most common prostatic tumor followed by locally invasive transitional cell carcinomas.

Bryan *et al.* (2007) found that castration had no protective effect on canine prostate with regards to neoplasia, while castration had the same or greater prevalence of prostatic neoplasia when compared to intact dogs.

Smith (2008) stated that adenocarcinoma of the prostate and transitional cell carcinomas of the prostatic urethra were the most frequently diagnosed prostatic tumors in dogs.

2.8.6.1. Prostatic Intraepithelial Neoplasia (PIN)

Waters *et al.* (1997a) found that a high grade of prostate intraepithelial neoplasia (PIN) a known precursor for human prostate cancer was frequently present in the prostates of elderly intact dogs and its appearance was influenced by testicular androgens as it did not appeared in castrated animals. Waters *et al.* (1997b) opined that canine PIN was similar in its morphology and immunophenotype to its human counterpart, which, appeared to be a precursor to

adenocarcinoma in dogs as well. Canine and human PIN were similar in basal cell disruption, proliferative index, and microvessel density, suggesting that the canine prostate might be a useful model for studying carcinogenesis and prostate cancer progression in humans.

Cytologic features of Prostatic intraepithelial neoplasia were cell crowding, loss of polarity, and nuclear and nucleolar enlargement (Johnston *et al.*, 2000).

Parry (2007) stated that high-grade prostatic intraepithelial neoplasia also occurred in many older dogs with or without prostatic adenocarcinoma, suggested to be a precursor to adenocarcinoma in the dog, as in man.

2.8.6.2. Etiopathogenesis

Unlike in humans, malignant prostatic tumour growth in dogs was not affected by decrease of androgen level in serum (anti-androgen therapy, castration). As a result, low level of androgen receptors expression in canine prostatic tumors had been suggested (Bell *et al.*, 1991 and Johnston *et al.*, 2000). The latter also reported that glandular basal cells played a role in BPH, accelerating their growth with age, while from ductal basal cells prostatic cancer could develop.

Significance of environmental chemicals that have hormonal activity (environmental estrogens) in the pathogenesis of spontaneous prostatic adenocarcinoma in the dog was unknown (Johnston *et al.*, 2000). Teske *et al.* (2002) reported that there were no known causes, although there was a slightly increased risk in castrated dogs.

2.8.6.3. Epidemiology

Barsanti and Finco, (1989) reported that about 5% of patients with prostatic disorder had malignant tumors and the most frequent type of neoplasia observed in dogs as well as humans was malignant prostatic adenocarcinoma.

Bell *et al.* (1991) reported a prevalence of 0.2 - 0.6% prostatic neoplasia from necropsy studies and found that the risk of a castrated dog for developing prostatic neoplasia was 2.38 times greater than that of an intact dog.

The average age of dogs with prostatic neoplasia was about 9 years (Bell *et al.*, 1991 and Krawiec and Heflin, 1992).

2.8.6.4. Diagnosis

Prostatic tumors are often not diagnosed until clinical signs were observed, and by this time, local or regional metastasis has already occurred (Barsanti and Finco, 1986; Johnston *et al.*, 2001 and Gobello and Corrada, 2002).

Gobello and Corrada (2002) opined that, in prostatic neoplasia, the prostate was so enlarged to drop over the pelvic brim and create a palpable mass in the caudal abdomen and the prostate gland should not be detectable in castrated dogs, and the finding of a normal prostate was suggestive of malignancy.

2.8.6.4.1. Clinical signs

Metastasis of prostatic neoplasia to regional lymph nodes and pelvic bones were commonly associated with pain and neurological deficits in the pelvic limbs (Barasanti and Finco, 1986).

Gobello and Corrada (2002) stated that the prostatic neoplasia had a great potential for secondary spread to pelvic lymph nodes, lumbar vertebrae, pelvic bones and more distant sites. The initial clinical manifestations of malignancy included bone metastasis leading to myelopathy, pain, neurologic deficits of the hind limbs and lameness.

Clinical signs of prostatic adenocarcinoma included fever, lethargy, anorexia, weight loss, tenesmus, dyschezia, hemorrhagic urethral discharge, hematuria, stranguria, incontinence and rare limb weakness. Gait abnormalities due to bone pain, were more likely to occur with prostatic neoplasia than with

other types of prostatic disease. Impaired venous and lymphatic flow could also cause hind limb oedema (Davidson, 2003).

Clinical signs exhibited by dog with prostatic neoplasia included fecal tenesmus, dysuria, haematuria, anorexia weight loss and signs of myelopathy or lameness as the manifestations of skeletal metastases (Parry, 2007).

Smith (2008) stated that clinical signs associated with prostatic neoplasia could vary depending on the time of diagnosis, degree of invasiveness, and potential metastasis.

2.8.6.4.2. Digital rectal palpation

The prostate was firm, irregular, immobile, asymmetrical, and painful on rectal examination in dogs with prostatic neoplasia (Johnston *et al.* 2000). Very large prostate might be palpable in the caudal abdomen. (Davidson, 2003)

2.8.6.4.3. Ultrasonography

Feeney *et al.* (1987) reported that changes in canine prostatic ultrasonography were common to variety of prostatic diseases as prostatic adenocarcinoma produced either a focal or diffuse hyperechoic pattern similar to that observed with bacterial prostatitis or benign hypertrophy.

Ultrasonographic appearance of the neoplastic canine prostate included prostatomegaly, mineralization of the parenchyma, presence of focal to diffuse hyperechoic areas, and irregular/ discontinuous prostatic contour (Finn and Wrigley, 1989; Bell *et al.*, 1991 and Klaunsner *et al.*, 1995).

Bell *et al.* (1991) reported that 58 percent of the dogs with prostatic carcinoma had an irregular/ discontinuous prostatic contour.

Smith (2008) stated that prostatic neoplasia gave the picture of focal to diffuse hyperechoic areas, suggestive of mineralization.

2.8.6.4.4. Radiographic findings

Feeney *et al.* (1987) observed that retrograde urethrocystography revealed periurethral asymmetry as well as narrowing, distortion, or destruction of the prostatic urethra.

According to Johnston *et al.* (2000), radiographic findings from 185 dogs with prostatic neoplasia included: prostatic enlargement (82%), prostatic mineralization (32%), sublumbar lymphadenopathy (24%), axial skeletal metastasis (16%), lung metastasis (15%) and appendicular skeletal metastasis (8%).

Gobello and Corrada (2002) stated that the radiographic findings in dogs with prostatic adenocarcinoma included prostatic enlargement and mineralization, sublumbar lymphadenopathy, and lung and appendicular skeletal metastasis.

Smith (2008) reviewed that thoracic and abdominal survey films should always be performed in any dog in which there was evidence of prostatic neoplasia, due to the common metastasis to pelvic lymph nodes, vertebral bodies and lungs.

2.8.6.4.5. Prostatic fluid evaluation

Cowan and Barsanti (1991) stated that prostatic massage combined with urethral catheterization was useful especially in identifying neoplastic cells.

Klaunsner *et al.* (1995) stated that exfoliative cytology by transrectal or transabdominal aspiration or needle core biopsy was a successful method for diagnosing prostatic adenocarcinoma.

Collection of prostatic cells by transrectal or transabdominal fine needle aspiration, with or without ultrasound guided placement, was reported to diagnose prostatic carcinoma correctly in about 80 % of affected dogs (Johnston *et al.*, 2000).

Smith (2008) stated that prostatic massage and wash were more likely to obtain neoplastic cells than in an ejaculated sample.

2.8.6.4.6. Hemogram

Leucocytosis with neutrophilia might be present, also regenerative or nonregenerative anemia reported in prostatic neoplasia (Davidson, 2003).

2.8.6.4.7. Serum biochemistry

Corazza *et al.* (1994) opined that low serum concentrations of total acid phosphatase (TAP) and prostatic acid phosphatase (PAP) did not rule out prostatic adenocarcinoma in the dog, but elevated concentrations in prostatic adenocarcinoma compared to BPH could be a useful criteria for differentiating the tumor from BPH. The serum concentrations of TAP, PAP and NPAP in normal male dogs and that of dogs suffering from prostatic adenocarcinoma were 3.2 ± 2, 1.8 ± 2, 1.4 ± 0.8, 21.4 ± 18.9, 11.9 ± 12.8 and 9.5 ± 9.2 U/ liter respectively. The authors observed no difference among TAP, PAP and NPAP serum concentrations of normal male dogs and dogs with non- prostatic diseases. Also the dogs with the tumor had higher TAP, PAP and NPAP than dogs with benign prostate hypertrophy, normal dogs and dogs with non-prostatic disease. The authors also opined that PAP should not be considered as a specific marker for prostatic disease, since its levels were higher in gastrointestinal tract cancer, BPH, inflammation of the prostate and after prostatic massage.

Abnormalities in serum biochemistry of dog with prostatic neoplasia included increased alkaline phosphatase, varying levels of serum calcium, hypoalbuminemia and azotemia (Davidson, 2003).

There was paucity of literature regarding the serum testosterone and acid phosphatase.

2.8.6.4.8. Pathology

2.8.6.4.8.1. Gross

Bloom (1954) noticed that the neoplastic prostate was small, normal, or of larger size, hard and nodular with dense capsule and adherence to adjacent structures. The cut surface was grayish or yellowish, granular, dry, opaque, stony, dense and relatively homogenous.

O'Shea (1963) observed that the neoplastic prostate was enlarged and irregular, or small and firm which contained fluid filled cysts, abscess and areas of hemorrhage.

Kraweic and Heflin (1992) stated that prostatic cancer was not always associated with prostatomegaly. The carcinomatous gland was of normal size, firm, irregular and adhered to the pelvic canal.

2.8.6.4.8.2. Histopathology

Bloom (1954) observed that prostatic adenocarcinoma was the commonest among the prostatic neoplasms. The acini were large or small, lined with cuboidal or columnar cells. Multiacinar arrangement that consisted of atypical cells forming secondary acini, tubular and papillary type was also present. The stroma was scanty or abundant and contained irregular cell nests and sparsely scattered small acini. In some lesions, the cells were anaplastic and contained irregular giant sized, hyperchromatic, bizarre nuclei with many mitosis.

O'Shea (1963) classified prostatic adenocarcinoma in to anaplastic, small acinar and large acinar types. Anaplastic regions were composed of solid sheets of loosely or densely packed cells, small acinar type showed the presence of small rounded acini, that lacked papillary infolding whereas, the large acinar type revealed large acini with epithelial infoldings that subdivided the lumen. Simple, stratified, columnar, or cuboidal epithelium with eosinophilic vacuolated cytoplasm that contained large nuclei with one or more nucleoli, lined the acini.

2.8.6.5. Metastasis

Bell *et al.* (1991) reported a prevalence of metastasis as high as 80 % for canine prostatic adenocarcinoma in neutered affected dogs and the pulmonary metastases were significantly more common in neutered than intact dogs with prostatic adenocarcinoma.

Johnston *et al.* (2000) reported sites of metastasis of prostatic carcinoma, from most to least common were lungs, regional lymph nodes, liver, urethra, spleen, colon and rectum, urinary bladder, bone, heart, kidney, distant lymph nodes, and adrenal glands. Skeletal metastasis most frequently affected lumbar vertebrae and pelvis and only 7% of the lesions occurred distal to the elbow or stifle. Bone metastasis leading to myelopathy or lameness was the initial clinical manifestation of malignancy.

2.8.6.6. Treatment

Chemotherapy should be used only as a last resort for diffuse metastatic disease of prostate (Turrel, 1989). In contrast to prostatic adenocarcinoma in men, canine prostatic adenocarcinoma did not appear to be androgen responsive as androgen deprivation had not been beneficial in its management and external beam radiation therapy was reported to cause shrinkage of some canine prostatic tumors with relief of urinary outflow obstruction and obstipation (Klaunsner *et al.*, 1995)

Treatment of canine prostate cancer generally was unrewarding. Castration resulted in involution of the non-neoplastic portion of the prostate, but did not affect progression of the neoplastic disease. Surgical resection usually was not recommended, because the disease was not usually diagnosed at an early stage, and prostatic surgery often resulted in urinary incontinence (Johnston *et al.*, 2000).

2.8.6.7. Prostatic Adenocarcinoma

Obradovich *et al.* (1987) observed that the prevalence of adenocarcinoma in dogs castrated at a young age was equal to that of intact dogs. Adenocarcinoma was usually found in 8-10 year old dogs and only sporadically in very young dogs. The risk factor of developing prostatic adenocarcinoma was 2.38 fold higher in castrated dogs than in intact ones (Bell *et al.*, 1991).

Krawiec and Heflin (1992), Johnston *et al.* (2001) and Smith (2008) opined that the most common conditions found in the prostate of neutered dogs were adenocarcinoma.

Johnston *et al.* (2000) stated that unlike in human, prostatic adenocarcinomas in dogs were not responding to androgen deprivation, antiandrogens or castration. Prostatic adenocarcinoma was an uncommon, highly invasive malignant tumor of intact and castrated male dogs.

Gobello and Corrada (2002) stated that concomitant prostatic hyperplasia usually occurred with adenocarcinoma in intact dogs.

Prostatic adenocarcinoma tends to metastasize to the iliac lymph nodes, urinary bladder, rectum, lung, pelvic musculature and bone and the most frequent sites of bone metastasis were the pelvis and lumbar vertebrae (Davidson, 2003).

Powe *et al.* (2004) suggested that canine prostatic cytologic evaluation could be used to accurately differentiate transitional cell carcinoma from adenocarcinoma based on features of urothelial cells.

Borbil (2006) reported a paraneoplastic thrombocytopenia a male dog with prostatic adenocarcinoma.

2.8.6.8. Prostatic Hemangiosarcoma

Hayden *et al.* (1992) reported a case of prostatic hemangiosarcoma which metastasized to other organ in an 11- year old male miniature poodle and described the clinical and pathological findings.

Hematology revealed a normochromic normocytic anemia (PCV 23%) without significant regenerative response. Urinalysis was consistent with urinary tract infection and urine culture revealed *klebsiella pneumoniae* and *Proteus mirabilis* (> 105 colony forming units/ml).

Prostatomegaly was identified on abdominal radiographs. Ultrasonography revealed diffuse prostatic hyperechogenicity with irregular multifocal hypoechoic masses.

Microscopic examination of prostatic biopsy sample revealed neoplastic polyhedral and spindle cells of uncertain tissue type. The right lobe of the prostate was found to be enlarged on necropsy. The prostate was 10.5 × 6.0 × 4.0 cm and weighed 125 g. Transverse sections of the prostate revealed a large yellow core encircled by a rim of red tissue. Histopathologic examination of prostate gland revealed necrohemorrhagic tissue and viable tissue consisted of neoplastic cells which formed septa that delineated a series of plexiform blood- filled channels and the tumor cells arranged in solid sheets with a pavemented epithelium like appearance.

Metastases with similar variation in morphologic pattern were found in the kidneys, small and large intestine, pancreas, skeletal muscle, tongue, diaphragm, heart and lungs. Diagnosis of hemangiosarcoma of the prostate was made from areas of tumor with a typical vascular growth pattern supported by cells with positive immunoreactivity for vimentin and F VIII R Ag.

2.8.6.9. Prostatic Leiomyosarcoma

Hayden *et al.* (1999) reported prostatic leiomyosarcoma with metastasis in a 10-year-old intact male Boxer, which had a history of stranguria and urinary incontinence. Clinicopathological studies revealed mature neutrophilic leukocytosis (25.7× 10^3 neutrophils/μl), increased serum creatinine (3 mg/dl), increased serum urea nitrogen (49 mg/dl) and hyperglycemia (213 mg/dl).

The prostate was asymmetrical, firm and enlarged. The cut surface revealed a pale capsular region and mottled beige to tan parenchyma with multiple cysts. A large periprostatic cyst was situated at the base of the prostate dorsocaudal to the urinary bladder. The cyst wall was mineralized, and the lumen contained many hard spike-like concretions and thick brown fluid. The urinary bladder was dilated and had mucosal hyperemia and focal hemorrhages.

The capsular surfaces of the kidneys were irregular. Pronounced hydronephrosis revealed on sectioning the left kidney.

Microscopically, the prostate gland was largely effaced by interlacing fascicles of neoplastic spindle cells interspersed with sheets of haphazardly arrayed tumor cells. Tumor cells were hyperchromatic with oval or elongated nuclei, clumped chromatin, one or more nucleoli, and abundant cytoplasm. Multinucleated tumor giant cells and pleomorphic tumor cells often commingled with smaller spindle cells Trichrome stain confirmed the distribution of collagen and revealed scant fuchsinophilic myofilaments in the cytoplasm of some tumor cells. Residual prostatic tissue consisted of atrophic lobules with variably dilated secretory ducts and bundles of smooth muscle. Intense interstitial infiltrates of lymphocytes and plasma cells inter- faced focally with the advancing tumor cells.

Metastases were confirmed histopathologically in the regional lymphnodes, mesentery, kidneys, and lungs. Typically, metastatic sites contained spindle and polyhedral tumor cells, abundant collagen deposits, and neoplastic thromboemboli.

Microscopic lesions in the urinary tract included, severe acute cystitis and for the left kidney, segmental cortical tubular atrophy and interstial fibrosis secondary to hydronephrosis. Pale foci in the renal cortices consisted of an amalgamation of tumor cells and fibrous connective tissue or fibrosis alone.

Immunohistochemically, prostate tumor cells stained intensely with vimentin and were negative for cytokeratin. Lymph node metastasis showed strong positive staining for both vimentin and actin and no labeling for cytokeratin the diagnosis of prostatic leiomyosarcoma was established with a panel of antibodies that revealed positive staining for vimentin, alpha smooth muscle actin, and desmin while eliciting no immunoreactivity for cytokeratin or myoglobin.

2.8.6.10. Lymphoma of Prostate

Mainwaring (1990) reported a case of lymphoma that only was found in the prostate and adrenal.

2.9. PERINEAL HERNIA AND POSTATIC DISEASE

Sereda *et al.* (2002) reported a case of iatrogenic proximal urethral obstruction after inadvertent prostatectomy during bilateral perineal herniorrhaphy.

Kumar *et al.* (2008) advocated surgical removal of hyperplastic prostate gland in a Pomeranian dog with perineal hernia and observed complete relief.

2.10. PROSTATIC MARKERS

Bell *et al.* (1981) stated that, although serum Canine prostate specific antigen activities were significantly higher in dog's with BPH than in normal dogs, mean serum activitiy in dogs with BPH, bacterial prostatitis and prostatic carcinoma were not significantly different from each other.

Frenette *et al.* (1983) and Juniewitz *et al.* (1990) stated that Canine prostate specific arginine esterase (CPSE) was produced by prostatic epithelial cells under androgenic control and could be inhibited by antiandrogen treatment or surgical castration. Additionally, Frenette *et al.* (1983) observed that, the effects of either surgical or chemical castration on this enzyme were reversible following exogenous androgen administration.

Klaunsner *et al.* (1995) found that CPSE was significantly higher in dogs with BPH than in normal dogs, dogs with bacterial prostatitis and prostatic carcinoma.

Canine prostate specific arginine esterase kit was not available in the veterinary market as noted by Gobello *et al.* (2002).

Materials and Methods

3. MATERIALS AND METHODS

The study was conducted in the Department of Clinical Veterinary Medicine, College of Veterinary and Animal Sciences, Mannuthy during the period from 2009 to 2010.

3.1. SELECTION OF ANIMALS

Animals brought to the Veterinary College Hospital Mannuthy and Kokkalai with clinical signs suggestive of prostatic diseases viz, tenesmus, dyschezia, dysuria, arching of back and rare limb weakness, was selected and utilized for the present study. Twenty such cases were selected and based on ultrasonography and transrectal palpation; fifteen cases which seemed to have prostatic involvment were utilized for detailed study.

3.2. OUTLINE OF STUDY

3.2.1. Clinical Examination

Detailed clinical examination of 15 selected dogs was done as per the proforma and significant changes were recorded. The breed, age and detailed history were recorded.

3.3. TRANSRECTAL DIGITAL EXAMINATION OF THE PROSTATE

After the general examination of the animal, suspected cases were subjected to transrectal digital examination of the prostate using a lubricated gloved finger with the assisted caudoabdominal pressure and the following information was recorded.

a. warmness on touching

b. location

c. consistency

d. symmetry

e. mobility

f. presence or absence of pain.

The prostate was massaged for obtaining prostatic fluid.

3.4. ULTRASOUND SCANNING

All the cases suspected to have prostatic enlargements were subjected to ultrasound scanning.

3.4.1. Equipment

Selected dogs were subjected to ultrasound scanning using Mindray DC 6 Vet Ultrasound machine with 3.0, 5.0 and 7.5 MHz transducer.

3.4.2. Scanning Procedure

The hair in the suprapubic area of the dog (between the cranial aspect of the prepuce and pubic bone from the midline to the inguinal fold on each side) was shaved. Ultrasonography was carried out with the patient in dorsal or lateral recumbency using a 3 to 7.5 MHz transabdominal probe after application of acoustic coupling gel to improve contact. For imaging the gland, the probe was placed against the ventral abdominal wall cranial to the pubis. For locating the prostate gland, the bladder was first identified and the transducer then moved caudally to the neck of the bladder and thence to the prostate. A moderately full bladder facilitated localization of the prostate. If the bladder was empty, it was filled with water or saline via a urethral catheter to facilitate identification. In cases in which the prostate was located partly or entirely within the pelvic canal, the transducer was angled caudally under the pubic brim and a gloved finger was inserted in the rectum and used to push the prostate cranially to allow accurate

determination of prostatic size. For visualizing prostate gland completely, the probe was turned to transverse and longitudinal (sagittal) planes. The true sagittal section of the prostate was confirmed by the observation of the hypoechoic urethral tract. The length (L) is defined as the maximum diameter of gland along the urethral axis in sagittal image, height (H) as the maximum diameter perpendicular to the axis of length. The transducer was then rotated 90 degrees to obtain a transverse image of the gland. On transverse images, the height is defined as the diameter of the prostate on a line separating the two lobes of the gland and the width as the maximum diameter perpendicular to the axis of the height. These parameters were measured in centimeters. Prostatic volume was calculated using the formula

$$\text{Volume } (cm^3) = L \times W \times H \times 0.523$$

After visualization of the prostate gland, the following features were specifically recorded

1). Change in echogenecity.

2). Prostatic volume

3.5. COLLECTION OF PROSTATIC FLUID

The prostatic fluid was obtained by following methods.

3.5.1. Prostatic Massage

Prostatic massage combined with urethral catheterization was performed. To perform prostatic massage, the urinary bladder was catheterized in an aseptic manner. All the urine was removed and the bladder was rinsed with saline several times and emptied. A sterile urinary catheter was placed in the prostatic urethra under ultrasound and transrectal guidance. The prostate was then vigorously massaged per rectally, followed by slow injection of 5 ml of saline through the catheter. Continuous aspiration was applied as the catheter advanced through the

prostatic urethra and into the urinary bladder. Aspirated fluid was recovered in a sterile syringe and subjected to cytology and culture and sensitivity. Pre massage and post massage samples were collected to localize the abnormalities of urethra or the prostate.

3.5.2. Ejaculation

To obtain the prostatic fluid by ejaculation, the dog was properly restrained and the penis was gently massaged through the prepuce. When the penis exhibited about 50 percent erection, the prepuce was retracted caudally behind the bulbus glandis, and digital pressure was applied immediately behind the bulbus glandis by encircling the penis using a gloved thumb and index finger. The digital pressure was continued until the animal exhibited pelvic thrusting. The animal was allowed to ejaculate the first and the second fraction of the semen and the third fraction then collected into a separate collection tube. The prostatic fluid so collected was examined for the evidence of presence of blood / pus. Direct smears and centrifuge preparations were subjected to cytological evaluation.

3.5.3. Prostatic Fine Needle Aspiration

Aspiration was performed under transabdominal ultrasound guidance with the sedated dog in lateral or dorsal recumbency. A spinal needle with a stylet was used and suction was applied with a syringe. The fluid collected was evaluated for colour and cytological changes. The tissue collected in the lumen of the needle was expressed on a slide and an impression smear was prepared which then subjected to cytological evaluation.

3.6. EVALUATION OF PROSTATIC FLUID

3.6.1. Cytological Evaluation of Prostatic Fluid

Prostatic smears were prepared from the collected prostatic fluid and stained with leishman's stain and observed under 100 x magnifications.

3.6.2. Culture and Sensitivity Test of Prostatic Fluid

The prostatic fluid were subjected to culture and sensitivity test.

3.6.2.1. Materials

Mueller –Hinton agar (MHA) was used to study the antibiotic sensitivity pattern of the isolates. The following antibiotic discs with known concentration as noted in micrograms (μg) were used (Hi-media).

1. Chloramphenicol	C^{10}	10 μg/disc
2. Enrofloxacin	E^{x}	5 μg/disc
3. Gentamicin	G^{10}	10 μg/disc
4. Oxytetracycline	O^{30}	30 μg/disc

3.6.2.2. Method

In vitro antibiotic sensitivity of the organisms was studied using Disc Diffusion Technique (Barry, 1976)

Five colonies of each pure culture were picked up with sterile nichrome loop and were used as the inoculum in four milliliter of sterile peptone water. It was kept at room temperature for 4 hours to develop turbidity, inoculum was applied uniformly on the surface of MHA, using a sterile cotton swab and the plate kept covered for 15 minutes at room temperature for drying the inoculum. Antibiotic discs were then placed on the surface of the agar 20 mm apart and they were gently pressed on the surface of the agar to ensure contact. The plates were incubated at 37^{0}C for 18 to 24 hours.

3.6.2.3. Interpretation

The zone of inhibition of bacterial growth around each disc was measured and interpreted as sensitive, moderately sensitive or resistant by comparing with the ranges given by manufacturer.

3.7. PROSTATIC BIOPSY

In those cases, which were unresponsive to initial treatment, prostatic biopsy with Tru- cut needle was performed and the collected tissue was cytologicaly evaluated.

3.8. CLINICAL PATHOLOGY

3.8.1. Collection of Clinical Material

Five milliliter of whole blood was collected from saphenous or cephalic vein in dry glass vials with EDTA at the rate of 1-2mg per ml as anticoagulant (Benjamin, 2005).

Ten milliliter of blood was collected in another test tube on the day of admission to separate serum for biochemical analysis. Sera, thus collected were stored at 20°C till further analysis.

Ten ml of urine was also collected into sterile vials from suspected cases of prostatic diseases, by catheterization or cystocentesis.

3.8.2. Hemogram

Hematocrit, haemoglobin, erythrocyte sedimentation rate (ESR), total erythrocyte count (TEC), total leucocyte count (TLC), differential leucocyte count (DLC) and platelet count were estimated as per method described by Schalm *et al.* (1975).

3.8.3. Serum Biochemistry

Total serum protein, albumin, globulin, A:G ratio, serum creatinine, blood urea nitrogen (BUN), calcium, glucose, testosterone, total acid phosphatase and prostatic acid phosphatase were estimated. All the biochemical estimations were carried out in Secomam Basic spectrophotometer using commercially available kits.

Urea was estimated by diacetyl monoxime method using standard kits from Agappe diagnostics (Marsh *et al.,* 1965).

Creatinine concentration was estimated using Jaffe's alkaline picrate method with standard kits supplied by Agappe diagnostics (Slot, 1965).

Serum protein was estimated by direct biuret method using standard kits supplied by Agappe diagnostics (Gornall *et al.,* 1949).

Serum albumin was estimated using bromocresol green methodology with standard kits supplied by Agappe diagnostics (Doumas, 1971)

Serum total acid phosphatase and prostatic acid phosphatase were estimated by alpha-naphthylphosphate method using standard kits supplied by Agappe diagnostics.

Serum testosterone was estimated by chemi luminescent immuno assay method.

Serum calcium was estimated using modified ortho-cresolphthalein complex methodology with standard kits supplied by Agappe diagnostics.

Serum glucose was estimated using glucometer.

3.8.4. Urinalysis

Estimation of urinary pH, detection of blood pigment and specific gravity were done by using uristik strips. Heller's test and heat coagulation test of urine

samples were conducted for detecting the presence of protein (Benjamin, 2005). For examination of urine sediments, the urine samples were mixed well, poured into two ml centrifuge tubes and centrifuged at 3000 rpm for 5 minutes. The supernatant was discarded. The sediment resuspended, transferred to a slide and examined.

3.9. THERAPEUTIC MANAGEMENT AND ASSESSMENT OF RESPONSE TO TREATMENT

On the day of admission, all the cases were treated based on the tentative diagnosis. After confirmation necessary changes were made in the therapeutic regimen. Prostatitis was treated with enrofloxacin @ 10 mg/kg intravenously for one week and then orally for two weeks. Benign prostatic hyperplasia was treated with 5-∝ reductase inhibitors (Finasteride). The concurrent renal failure was treated with fluid therapy and antiemetics. Herniorraphy was performed in perineal hernia associated with prostatomegaly. Prostatic abscess were treated with antibiotics based on culture and sensitivity and drainage of pus under ultrasound guidance. Response to the treatment was evaluated based on clinical improvement.

3.10. HISTOPATHOLOGY

In the event of mortality during the course of clinical investigation and treatment, tissues of prostate were collected at necropsy and examined both grossly and microscopically after staining using hematoxyllin and eosin and Gomori's one step staining method (Luna, 1968). The correlation between haemato- biochemical, ultrasound and histopathology were analyzed.

Results

4. RESULTS

Twenty intact male dogs presented to the Veterinary Hospital, Kokkalai and Mannuthy, with clinical signs suggestive of prostatic diseases viz, tenesmus, dyschezia, dysuria, arching of back and rare limb weakness, were subjected to detailed clinical examination and observations were recorded as per proforma. Suspected cases were subjected to special diagnostic techniques.

4.1. OCCURRENCE

Based on clinical examination and special diagnostic techniques, prostatic diseases were confirmed in 15 dogs (75%). Of these 15 cases, five cases were benign prostatic hyperplasia (33.2%) which included benign prostatic hyperplasia with mild prostatitis (20%) and BPH and associated perineal hernia (13.2%). Seven cases were diagnosed as prostatitis (46.7%) which comprised of acute prostatitis (33%), suppurative prostatitis (6.7%) and prostatitis associated with herpes virus infection (6.7%). One each of prostatic abscess (6.7%), fibroadenoma with paraprostatic pseudocyst and inflammation (6.7%), and papillary adenoma (6.7%) was the other disease (Fig. 1).

4.2. BREED WISE INCIDENCE

The breeds affected with prostatic disorders were seven German shepherds (46.6%) followed by two Rottweilers (13.3%), two Dachshunds (13.3%), one Cocker spaniel (6.7%), one Doberman (6.7%), one Labrador (6.7%) and one Boxer (6.7%). The highest incidence (46.6%) was recorded in German shepherd (Fig. 2).

Fig. 1. Classification of Prostatic Diseases

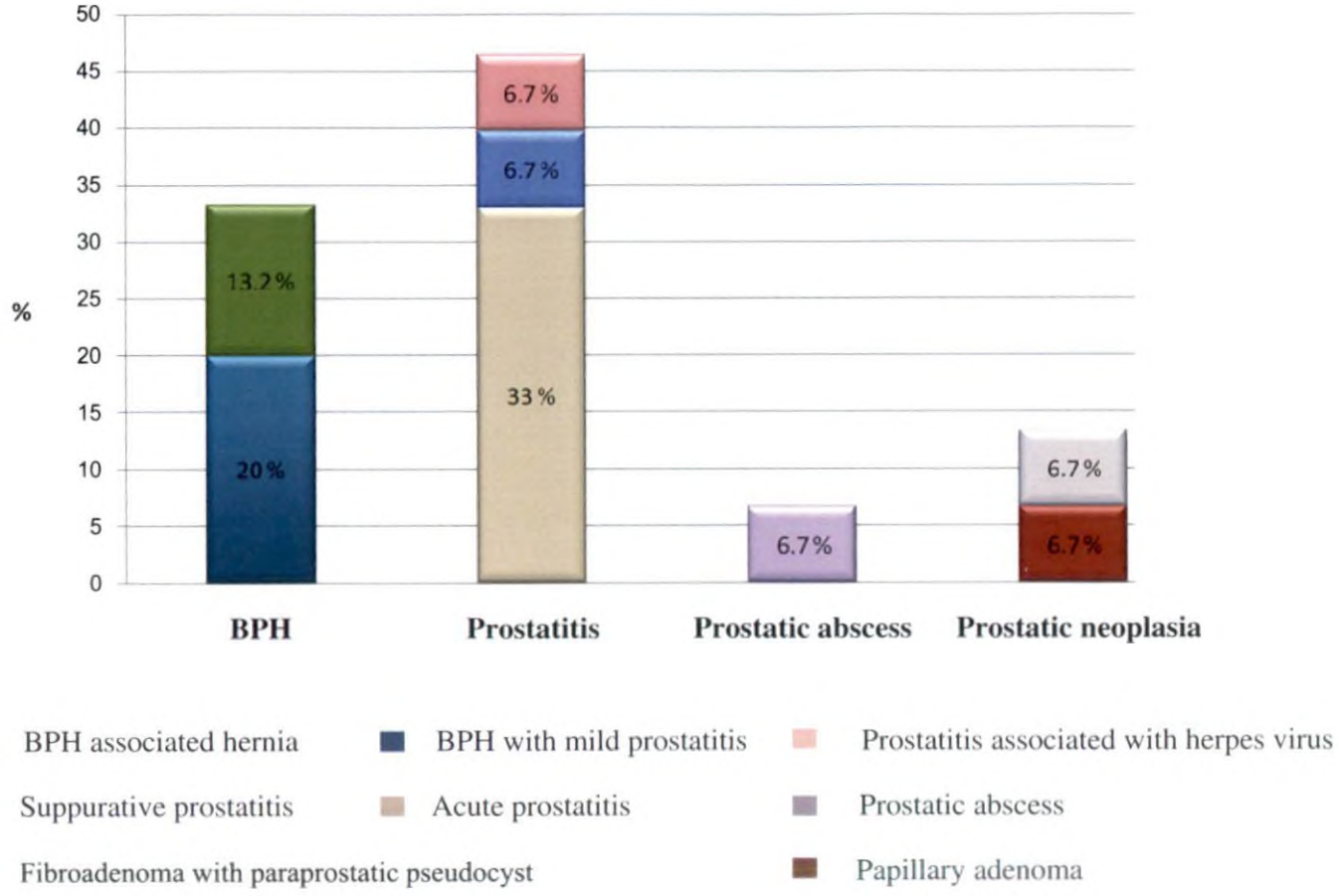
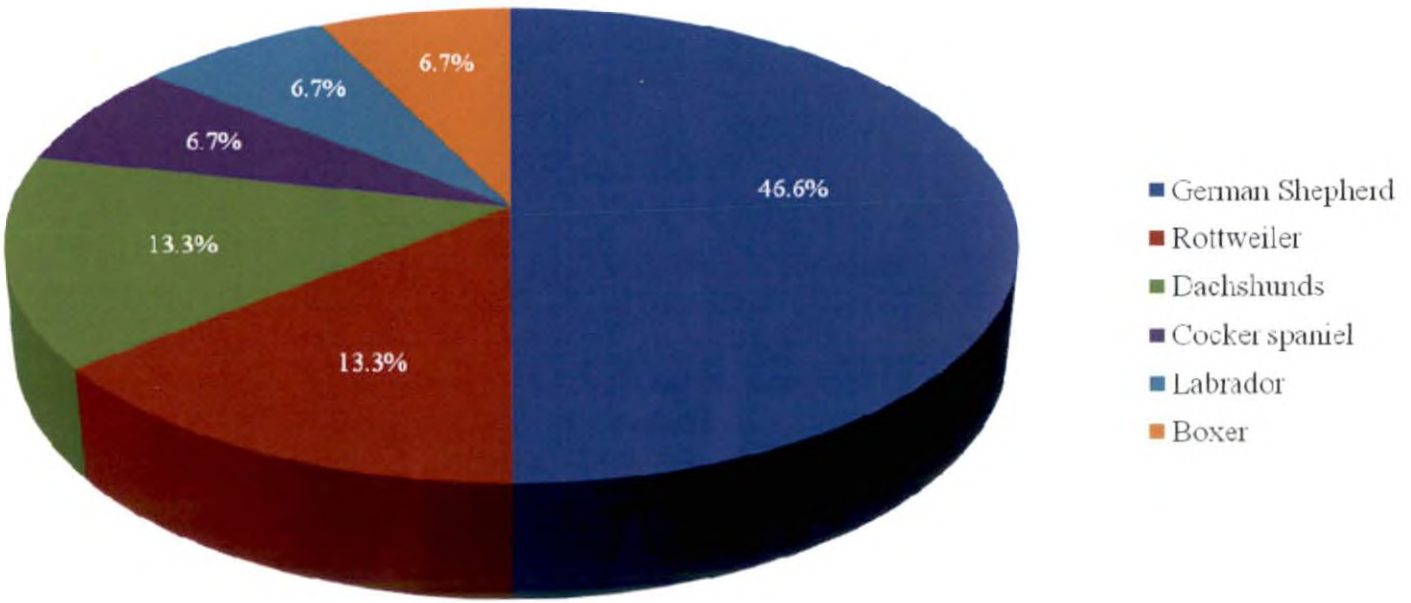


Fig. 2. Breedwise Incidence of prostatic disease



4.3. AGE WISE INCIDENCE

The age of dogs affected with prostatic diseases ranged from 7months to12 years with mean age of 6.7 years. Highest incidence was observed in 5- 10 years age group.

4.4. DISEASES OF PROSTATE

4.4.1. Benign Prostatic Hyperplasia

4.4.1.1. Benign Prostatic Hyperplasia with Prostatitis

Three cases were classified as benign prostatic hyperplasia with prostatitis. The breeds affected were German shepherd, Dachshund and Rottweiler. The mean age of affected dogs was 6.3 years.

4.4.1.1.1. Clinical signs

All the three dogs were referred with the complaint of anorexia and constipation for considerable duration. One dog had vomiting. Foul smelling urine was reported in the German shepherd.

4.4.1.1.2 Clinical observation

The mean temperature, pulse and respiration rates of the affected dogs were 102.9° F (101-104.2), 90 /min (70-115) and 21/min (20-24) respectively. Visible mucous membranes were pale in one dog and slightly congested in the other two. The popleteal lymph node was palpable in German shepherd.

4.4.1.1.3 Diagnostic methods

4.4.1.1.3.1. Digital rectal palpation

On digital rectal examination, the prostate of German shepherd was found to be warm and in others, it was isothermic. It was observed that two dogs had intra abdominal/partial intra- abdominal prostate. In the remaining case, it was

intra pelvic. Smooth and firm consistency and enlargement of prostate could be observed in all the dogs. Right lobe was more enlarged in two dogs. The prostate glands of all the dogs were mobile upon digital examination. Digital rectal palpation of prostate elicited pain in one dog.

4.4.1.1.3.2. Ultrasonography

Ultrasonography revealed thickened prostatic capsule in three dogs. Anechoic cavitating cystic areas in both the prostate lobes could be seen in German shepherd (Plate 1. A). Small multifocal anechoic areas in prostatic parenchyma were present in Rottweiler. Homogenous prostatic parenchyma was observed in the third one. Prostatic enlargements were found in all the three dogs. The average prostatic length, depth, width and prostatic volume were 6.56 cm, 4.52 cm, 4.69 cm and 72.7 cm^3 respectively. Splenomegaly was also present in all the cases.

4.4.1.1.3.3. Prostatic fluid evaluation

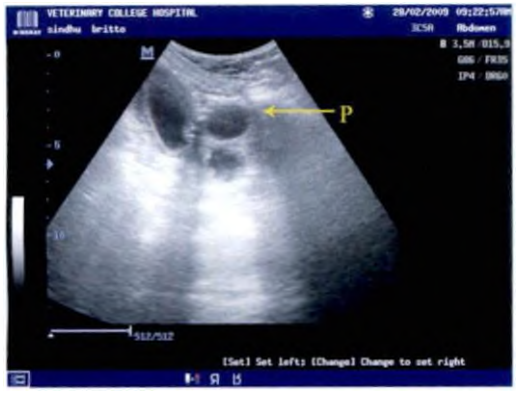
Prostatic fluid was collected from all the cases by prostatic massage combined with catheterization. It was slightly purulent in all the cases. The cytological evaluation revealed hyperplastic cells with mild anisokaryosis and large number of neutrophils in German shepherd (Plate 1. B). Round to oval nuclei with reticulated chromatin, and few neutrophils were present in all the cases. Cultural examinations of prostatic fluid from three dogs revealed the presence of *E. coli.*, which was sensitive to enrofloxacin and chloramphenicol.

4.4.1.1.3.4. Clinical pathology

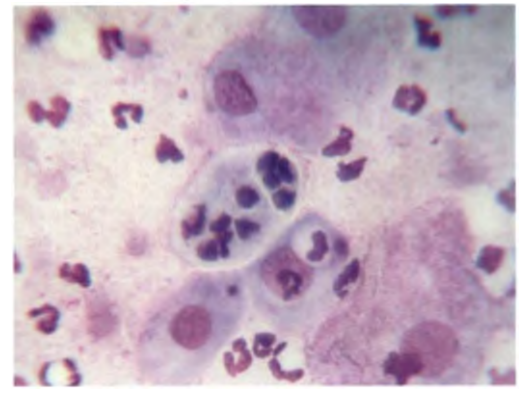
The average hemoglobin concentration (g %), total erythrocyte (count millions/cu. mm) and packed cell volume (%) with range were 10.2 (8-13.3), 4.69 (3.45-5.16) and 26.9 (21.2-32.8) respectively. The average leukocyte count was 28900 /cu. mm (11700-52000). The percentage neutrophil, lymphocyte, eosinophil, and monocyte with range in the parenthesis were 79 (73-84), 10 (10-

Plate: 1. Benign prostatic hyperplasia with prostatitis

A. Prostatomegaly (P) with two cysts

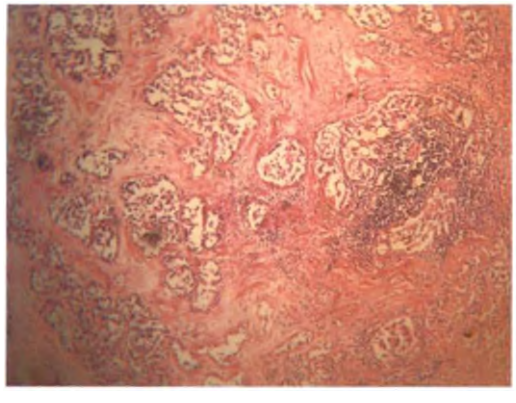


B. Prostatic fluid cytology
Hyperplastic cells, mild anisokaryosis and neutrophils

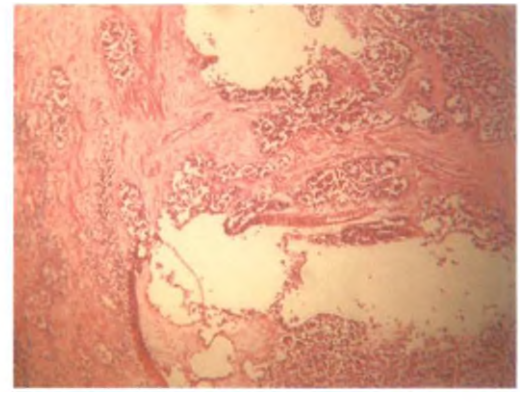


Histopathology of prostate (H&E-10x)

C. Hyperplasia
Infiltration of lymphocytes



D. Papillary intraluminal projections



11), 4 (1-6) and 6 (4-16) respectively. The average platelet count was 141,006/cu.mm with the range of 100,200-214,000/ cu. mm.

The average values of total protein, albumin, globulin and AG ratio were 6.63 g/dl, 2.27 g/dl, 4.57 g/dl and 0.53 respectively. Corresponding range values were (5.7-7.8), (1-3.8), (4-4.7) and (0.2- 0.95) respectively. The average serum creatinine level was 9.6 mg/dl with the range of (1.1 – 26). Average blood urea nitrogen level of affected dogs was 93 mg/dl (14-248). The average value of serum glucose and calcium were 63 mg/dl (38-92) and 7.5 mg/dl (6.8-8.7) respectively. The mean serum acid phosphatase level was 6.6 U/L (4.2-9) with prostatic fraction was 2.2 U/L (1.2-3.9). The average serum testosterone of affected dogs was 1.60 ng /ml (0.61- 3.4).

The urinary pH was acidic in two dogs and alkaline in one dog. The average urine specific gravity was 1.022 (1.012- 1.023). The urine sample of only one dog was positive for protein (3+). Blood pigments were detected in two dogs with an average of 2+. Pus cells were present in the urine sample of two dogs. Culture of urine samples was negative for bacterial growth.

4.4.1.1.4. Treatment and response

Two dogs were treated with 5-α reductase inhibitor (Finast) @ 1 mg/kg orally, enrofloxacin @ 10 mg/kg IV for 7 days followed by symptomatic therapy. Out of these two dogs, the dog with renal failure was additionally treated with fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC) and proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV). But that dog with renal failure died on 2nd day and autopsy could not be conducted. The condition of other dog started improving by 5th day, but not to a satisfactory level. It was advised to continue the administration of enrofloxacin as well as finasteride orally for one month. The third dog was treated with enrofloxacin @ 10 mg/kg IV alone for five days. The condition of that dog improved by 5th day and treatment was continued with enrofloxacin @ 10 mg/kg orally for 14 days. Though the clinical condition of the dog improved considerably by this treatment but, the case was presented

again with the same complaint after 2 months and the dog was not responding to treatment and succumbed after 5 days.

4.4.1.1.5. Autopsy and histopathology

Postmortem examination of only one animal could be conducted. Both the lobes of prostate were enlarged, rough and the prostatic tissues of both lobes were replaced by a cavity containing slightly purulent fluid. Irregular whitish grey bands had lined the cavity. Slight splenomegaly was observed. Histopathological observations of prostate revealed hyperplasia of the glands, which were lined by columnar cells, some area showed papillary infoldings. Stroma showed hypertrophied fibrocollagenous tissue with diffuse infiltration by lymphocytes and plasma cells. Dense collections of inflammatory cells were seen around the acini (Plate 1. C and D).

4.4.1.2. Benign Prostatic Hyperplasia Associated Hernia

Benign prostatic hyperplasia with cyst was reported in one dog, with perineal hernia and in another dog with both perineal and inguinal hernia (Plate 2. A and B).

The dog with perineal hernia was an eight year old Boxer and the other one was a nine-year old Dachshund.

4.4.1.2.1. Clinical signs

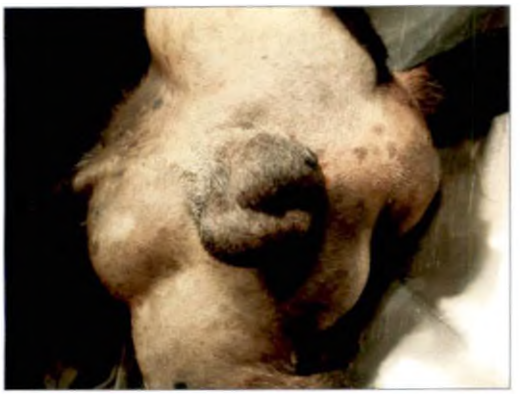
The Boxer was presented with the history of swelling adjacent to the anal region for one-year duration. The animal was active and had normal appetite and urination. The Dachshund was presented with the complaint of inguinal hernia and perineal hernia for six months and the dog had inappetance and dyschezia.

4.4.1.2.2. Clinical observation

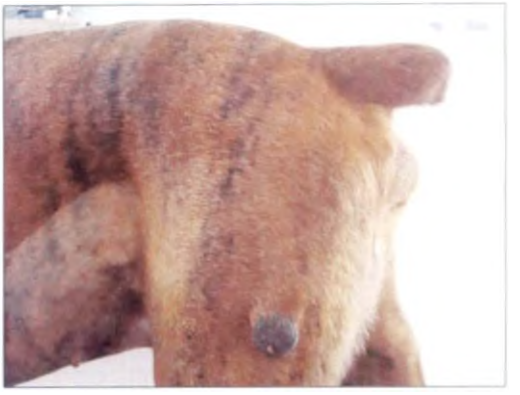
A swelling was present on the right perineal area of the Boxer and the size of swelling was found to increase while barking. No pain was felt at the region

Plate: 2. Benign prostatic hyperplasia associated hernia

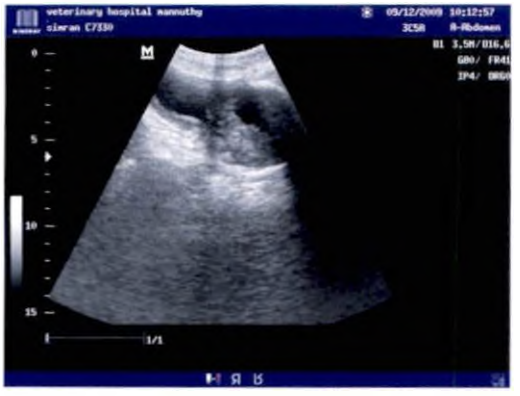
A. Dachshund dog with inguinal and perineal hernia



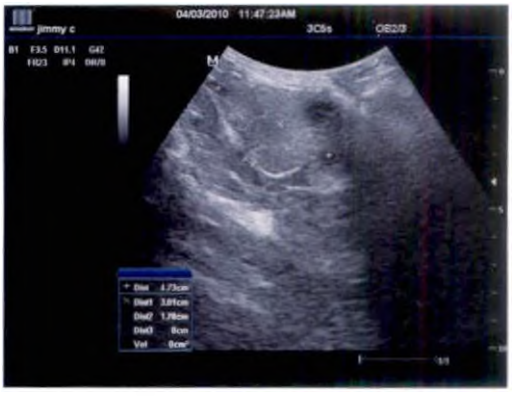
B. Boxer with perineal hernia



C. Prostatomegaly with cyst and thickened capsule



D. Prostatomegaly



and the Boxer was a cryptorchid. The temperature, pulse and respiration were 103.2° F, 116/min and 22/min respectively. The visible mucous membranes were congested.

A swelling, on the inguinal as well as perineal region was observed in the Dachshund and the animal showed no signs of pain on palpation. Temperature, pulse, respiration were 102.6° F, 89/min and 19/min respectively. Visible mucous membranes were congested.

4.4.1.2.3. Diagnostic methods

4.4.1.2.3.1. Digital rectal palpation

On digital rectal examination, the prostate gland was found to be isothermic in both the dogs. The prostate gland of the Boxer had intrapelvic position, smooth and soft, while that of Dachshund occupied partly intrapelvic and partly intraabdominal position with smooth and some soft areas. Prostate of both the dogs were highly enlarged and slightly movable upon rectal digital palpation.

4.4.1.2.3.2. Ultrasonography

Ultrasonographic examination of Boxer revealed thickened, slightly irregular capsule and anechoic area (cyst) in the prostatic parenchyma (Plate 2. C). Mild hyperechoic changes were also present. The prostatic length, depth, width and volume were 6.25 cm, 5.63 cm, 2.63 cm and 48 cm^3 respectively. Ultrasonographic examination of Dachshund also revealed anehoic area in one of the prostate lobe (Plate 2. D). Prostatic length, depth, width and volume of the case were 5.8 cm, 4.8 cm, 2.2 cm, 32.03 cm^3 respectively.

4.4.1.2.3.3. Prostatic fluid evaluation

Prostatic fluid cytology revealed clusters of prostatic epithelial cells with indistinct cytoplasmic borders and abundant basophilic cytoplasm. The nuclei

were round to oval with finely reticulated chromatin pattern and the nucleus cytoplasm ratio was preserved in both the animals.

4.4.1.2.3.4. Clinical pathology

The hemoglobin concentration, total erythrocyte count and packed cell volume of both dogs were 13.7 g % and 11.8 g %, 4.36 and 4 millions/cu. mm and 41.5 % and 35 % respectively. The leukocyte count with the differential count of the dogs were 8500/cu.mm and 10200/cu.mm, neutrophils 39 % and 69 %, lymphocytes 54 % and 30%, eosinophils 4 % and 1 % and monocytes 3% for Boxer only. The platelet counts were 261000/cu.mm and 272000/cu.mm respectively.

Total protein, albumin, globulin and AG ratio were 8.8 g % and 6.8 g % 2.5 g % and 3 g %, 6.3 g % and 3.8 g %, 0.4 and 0.7 respectively. Serum creatinine levels were 1.9 mg/dl and 1.1 mg/dl respectively. The blood urea nitrogen levels of the dogs were 22 mg/dl and 12 mg/dl respectively. Serum calcium levels were 8 mg/dl and 9.4 mg/dl respectively. Serum glucose levels were 72 mg/dl and 68 mg/dl respectively. Serum acid phosphatase levels of the two dogs were 13 U/L and 4 U/L respectively. The corresponding values for prostatic fraction were (4.5 U/L) and (2.1U/L) respectively. Serum testosterone levels were 3.5 ng/ml and 5.9 ng/ml respectively.

The urinary pH was acidic in both dogs and the average urine specific gravity was 1.023 (1.024-1.022). Urine sample of both the dogs were negative for protein, blood pigment and pus cells. On cultural examination of urine samples were negative for bacterial growth.

Both the animals were referred to surgery department for herniorrhaphy.

4.4.1.2.4. Treatment and response

Boxer was subjected to perineal herniorrhaphy. Inguinal herniorrhaphy and castration was conducted in Dachshund. Urinary bladder and prostate gland

had herniated in both the dogs. The boxer was treated with 5α reductase inhibitor (Finast) @ 1 mg/kg/day for 4 weeks. But another hernia developed adjacent to the previous one after 45 days.

In Dachshund, there was no recurrence of the condition during three months of observation.

4.4.2. Prostatitis

4.4.2.1. Acute Prostatitis

A total of five dogs (33.3%) were affected with prostatitis. The average age of dogs affected with prostatitis was 5.3 year with a range of 3.6 – 9 years. Out of five dogs affected with prostatitis, three were German shepherd and one each was Labrador and Doberman.

4.4.2.1.1. Clinical signs

Anorexia, purulent urethral discharge and dyschezia were the most common presenting signs in dogs affected with prostatitis (Plate 3. A). All the dogs in this group were showing signs of lethargy. Four dogs (80%) had dyschezia and vomiting. Haematuria was seen in two (40%) cases.

4.4.2.1.2. Clinical observation

The mean temperature, pulse and respiration rates of affected dogs were 103.4° F (102-104), 94/ min (73-108) and 20/min (17-24) respectively. Three dogs of this group showed pain on palpation of caudal abdominal areas.

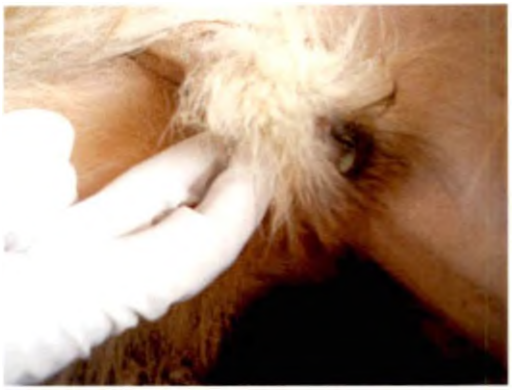
4.4.2.1.3. Diagnostic methods

4.4.2.1.3.1. Digital rectal palpation

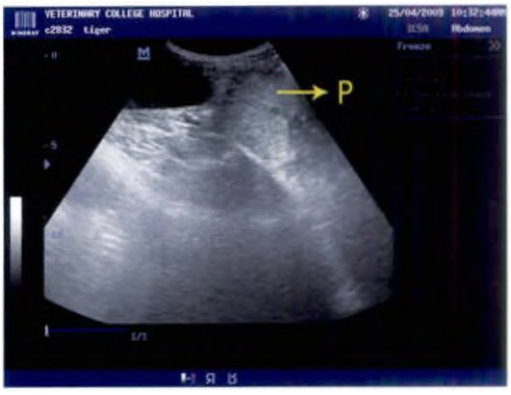
On digital rectal palpation, prostate gland of all the dogs with prostatitis were warm to touch and 40 % of the animal had intra pelvic prostate, another 40

Plate: 3. Acute prostatitis

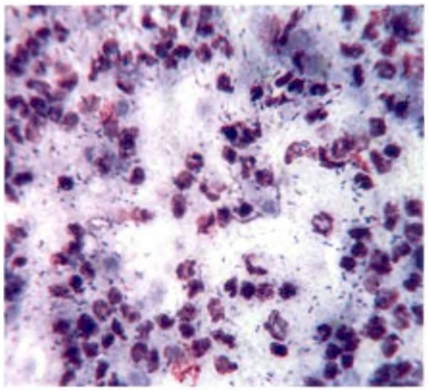
A. Purulent urethral discharge



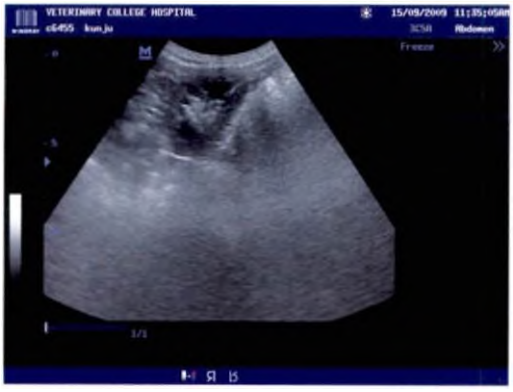
B. Diffused increase in echogenisity of prostate (P)



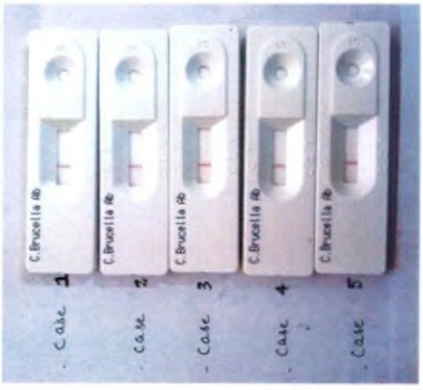
C. Prostatic fluid cytology
Bacteria laden neutrophils



D. Kidney
Indistinct corticomedullary junction



E. Brucella Ab test negative



% had intra- abdominal prostate and in remaining 20 %, the prostate were positioned partially in the pelvic cavity and partially in the abdominal cavity. The consistency of the prostate was smooth and firm in all cases. Forty percent of the dogs with prostatitis had asymmetrical prostate and symmetrical prostate was observed in the remaining cases. On rectal digital palpation, the prostate of all the dogs with prostatitis was found to be slightly enlarged. The prostate gland was mobile upon digital examination in 60 % of cases and fixed in remaining cases. Digital rectal palpation of the prostate gland evinced pain in all the five animals.

4.4.2.1.3.2. Ultrasonography

Ultrasonographic examination of all the dogs with prostatitis revealed diffused increase in echogenisity of prostatic parenchyma (Plate 3. B). Two dogs had multifocal hyperehoic areas in prostatic parenchyma. Normal, echogenic capsule was visualized in four dogs. Thickened irregular capsule was noticed in one dog. One dog had small kidneys with indistinct corticomedullary junction (Plate 3. D).

Urinary bladder of three dogs contained hyperechoic particles. Splenomegaly was detected in four cases. The mean length, depth, width and prostatic volume were 5.77 cm, 4.66 cm, 4.95 cm and 68.76 cm^3 respectively.

4.4.2.1.3. 3. Prostatic fluid evaluation

Prostatic fluid was collected by prostatic massage combined with urethral catheterization in 3 cases and the 2 dogs were subjected to digital manipulation of the penis to obtain the 3^{rd} fraction of semen. Large numbers of neutrophills were presented in all cases. Presence of bacteria- laden neutrophils were observed in 40 % of cases (Plate 3. C).

Cultural examination of prostatic fluid of five dogs with prostatitis revealed *E. coli* that was sensitive to enrofloxacin and tetracycline.

4.4.2.1.3.4. Clinical pathology

The average values of haemoglobin, total erythrocyte count, volume of packed red cells of the affected dogs with range in the parenthesis were 11.08 g % (9-16.5), 4.95 millions/ cu. mm (3.45-7.02) and 30.14 % (21.2-43.8) respectively. The average values of the total leukocyte count, percentage of neutrophils, lymphocytes, eosinophils, monocytes, basophils and band forms of the affected dogs with range in the parenthesis were 14880 / cu. mm (10100-52000), 83 (73-92), 11.4 (5-13), 2.8 (1-8), 0.8 (1-16), 0 and 2 respectively. The average platelet count of the dogs with prostatitis was 202,000/ cu. mm.

The average values of total protein, albumin, globulin and A: G ratio were 6.8 g/dl (6-7.6), 2.16 g/dl (1.6-2.6), 4.64 g/dl (3.9-5.7) and 0.44 (0.3-0.53) respectively. The mean value with range in parenthesis of serum creatinine, blood urea nitrogen, serum glucose and calcium were 4.42 mg/dl (1.1-12.4) 39.6 mg/dl (14-110), 63 mg/dl (43-90) and 7.6 mg/dl (6.3-9) respectively. The average serum acid phosphatase value of dogs with prostatitis was 6.32 U/L (5.6-7) with prostatic fraction was 2.9 U/L (2.8-3). The average serum testosterone level was 0.9 ng / ml (0.8-1).

The serum samples of all the dogs were negative for brucella antibody test (Plate 3. E).

The pH of urine, specific gravity, presence of blood pigments and protein were measured using uristix dipstick test. Urine protein was detected on an average of 2+. The urinary pH was acidic in three dogs and alkaline in two dogs. The mean specific gravity was found to be 1.022. Blood pigment was present in two cases. Culture of two urine samples revealed *E. coli* organism that was sensitive to enrofloxacin, tetracycline, chloramphenicol and gentamicin. In the rest of the cases, urine was found to be sterile.

4.4.2.1.4. Treatment and response

All the animals were treated with the antibiotics Enrofloxacin @ 10 mg/kg IV for one week and other symptomatic therapy as the condition warranted. It was advised to continue the antibiotics orally for 2 more weeks. Dogs with renal failure were additionally treated with fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC), proton pump inhibitors (Inj Pantaprazole @ 1 mg/kg BW IV once daily). In three cases, clinical improvement was observed within 3 days of treatment and complete recovery was observed by 14^{th} day of treatment. Out of these three cases, one dog had renal failure with creatinine 5.6 mg/dl, which then decreased to 2.4 mg/dl after 4 days of treatment. In the remaining two cases, one dog, which had serum creatinine of 12.4 mg/dl on the first day, did not respond to the treatment and the condition worsened and later died on 5^{th} day and autopsy could not be carried out since owner was not willing. The other dog did not respond favorably to the treatment.

4.4.2.2. Suppurative Prostatitis

An Eight year old male German shepherd was presented with inappetance, vomiting, dyschezia, ribbon like stools, dribbling of urine and foul odor from mouth for 6 days (Plate 4. A).

4.4.2.2.1. Clinical observation

Animal was less active. Uremic smell from mouth and purulent urethral discharge was present. Caudal abdominal palpation elicited pain. The temperature was 104 °F, pulse 80/min, respiration 23/ min and visible mucous membranes were pale roseate.

4.4.2.2.2. Diagnostic methods

4.4.2.2.2.1. Digital rectal palpation

Warmness could be felt on digital rectal palpation of the prostate gland, which was located in pelvic cavity. The gland was enlarged, smooth and firm in consistency and movable on rectal examination. Rectal palpation of prostate elicited pain.

4.4.2.2.2.2. Ultrasonography

Ultrasonographic examination of prostate revealed multifocal anechoic and hyperechoic areas in the prostatic parenchyma (Plate 4. B). Prostatic capsule was irregular. The measured prostatic length, depth, width and prostatic volume were 6.14 cm, 4.30 cm, 2.30 cm and 31.76 cm^3 respectively. Splenomegaly was observed and both the kidneys were small, irregular with indistinct corticomedullary junction.

4.4.2.2.2.3. Prostatic fluid evaluation

The prostatic fluid collected by prostatic massage combined with urethral catheterization was purulent in nature. The cytological examination revealed large number of toxic neutrophils and few squamous cells. The cultural examination revealed the presence of *E.coli* which was sensitive to enrofloxacin only (Plate. 4 C).

4.4.2.2.2.4. Clinical pathology

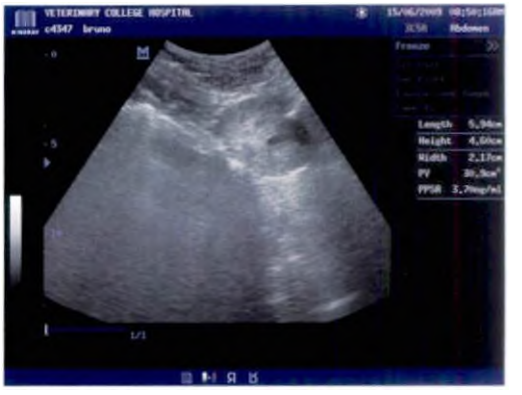
The hemoglobin, erythrocyte count, PCV were 12.7 g /dl, 5.85 millions/cu mm and 32 % respectively. Total leucocyte count was 26900/cu.mm with differential count of neutrophils 79 %, lymphocytes 13 % and band forms 8 %. The platelet count was 1,35,000/ cu mm.

The total protein, albumin, globulin and AG ratio were 7.9 g/dl, 2.3 g/dl, 5.6 g/dl and 0.41 respectively. The serum creatinine was 8.5 mg/dl. It was

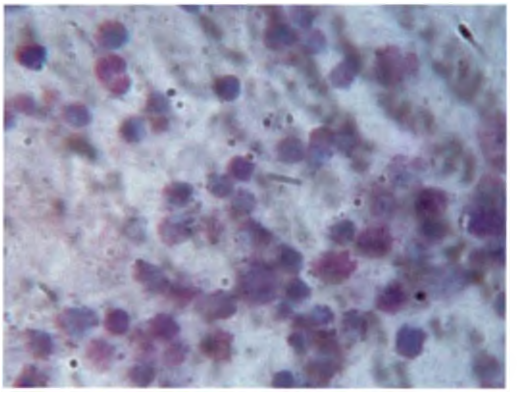
Plate: 4. Suppurative prostatitis

A. German shepherd dog showing dyschezia

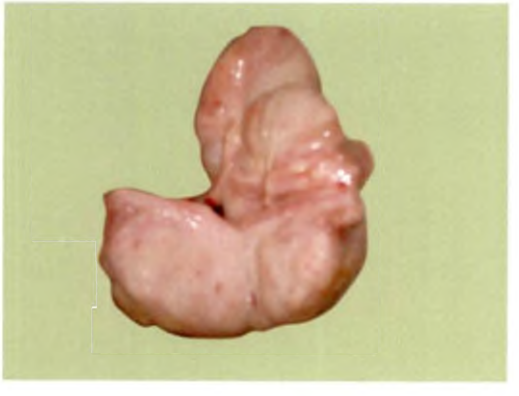
B. Multifocal anechoic and hyperechoic areas in prostate



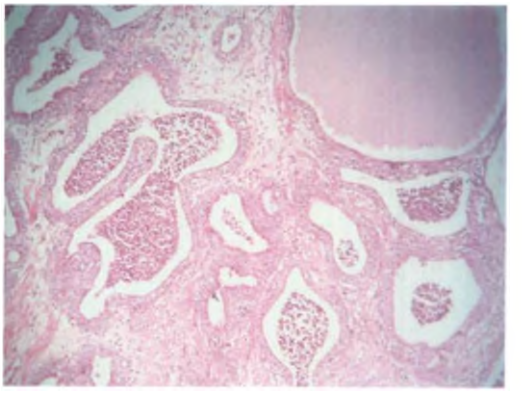
C. Prostatic fluid cytology - toxic neutrophils



D. Prostate gross



E. Histopathology of prostate (H&E-10x) Acini filled with neutrophils and accumulated secretion



increased to 15.5 mg/dl on 18th day. Blood urea nitrogen was 78 mg/dl. The serum values of glucose and calcium were 72 mg/dl and 6.5 mg/dl respectively. Serum total acid phosphatase was 9.8 U/L with the prostatic fraction as 4.5 U/L. Serum testosterone level was 1 ng/ml.

The urine sample had pH of 6.5, specific gravity 1.002, protein 2+ and pus cells of 2+. Blood pigment was absent in the urine sample. On cultural examination, urine was negative for bacterial growth.

4.4.2.2.3. Treatment and response

The dog was treated with enrofloxacin @ 10 mg/kg IV, fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC) and proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV) for 20 days. Slight improvement was noted within 4 days of treatment. But later the condition worsened. The dog succumbed after 20 days.

4.4.2.2.4. Autopsy and histopathology

Both the prostate lobes were enlarged and purulent fluid slightly oozed out on sectioning (Plate 4. D). Both the kidneys were smaller and irregular with necrotic areas. Histopathology of prostate revealed that, almost all acini were filled with neutrophils and some cystic with accumulated secretion. Lining cells of glandular acini were not clearly visible and moderate interstial fibrosis was also observed (Plate 4. E).

4.4.2.3. Prostatitis Associated with Herpes Virus Infection

A German shepherd male dog aged 7 ½ month was presented with the history of anorexia, vomiting, straining while defecation and difficulty in urination since 10 days (Plate 5. A).

4.4.2.3.1. Clinical observation

The animal was less active. The temperature was 103.2 °F, pulse 79/min, respiration 24/min and visible mucous membranes were slightly congested. Small vesicles were noted in the conjuctival mucous membrane and hyperemic vesicles could be observed on bulbus glandis while collecting prostatic fluid (Plate 5. B and C).

4.4.2.3.2. Diagnostic methods

4.4.2.3.2.1. Digital rectal palpation

The prostate gland was found to be warm to touch on rectal digital examination and positioned intrapelvically. The prostate was smooth, firm and slight enlargement could be observed on digital rectal palpation. The prostate gland was movable and the animal exhibited slight pain while doing rectal examination.

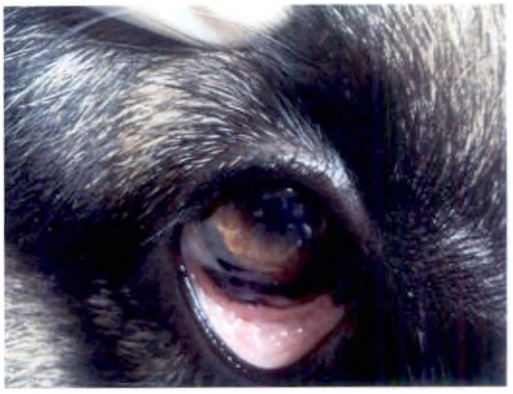
4.4.2.3.2.2. Ultrasonography

On ultrasonography, the prostatic capsule was thickened and mixed echogenic changes were observed in the prostatic parenchyma. The prostatic length, depth, width and prostatic volume were 3.02 cm, 3.65 cm, 1.54 cm and 8.88 cm^3 respectively. Both kidneys had focal hyperechoic areas. The right kidney was enlarged (Plate 5. D and E).

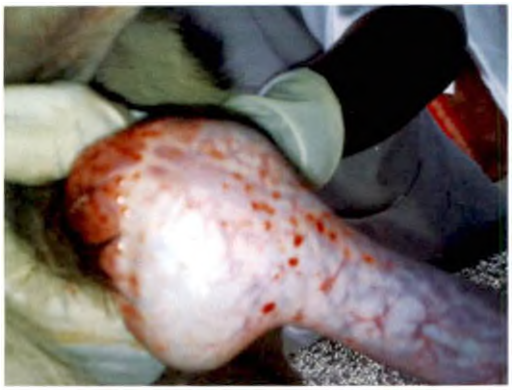
Plate: 5. Prostatitis associated herpes virus infection

A. German shepherd dog showing dyschezia

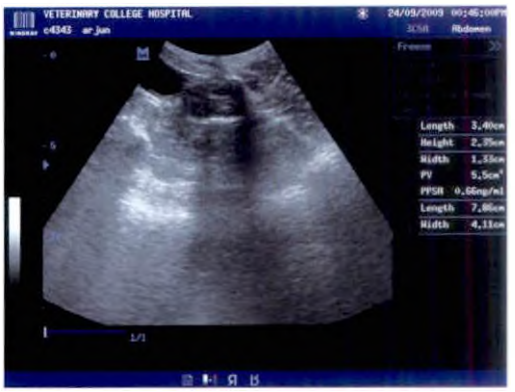
B. Vesicular lesions in conjuctival mucous membrane



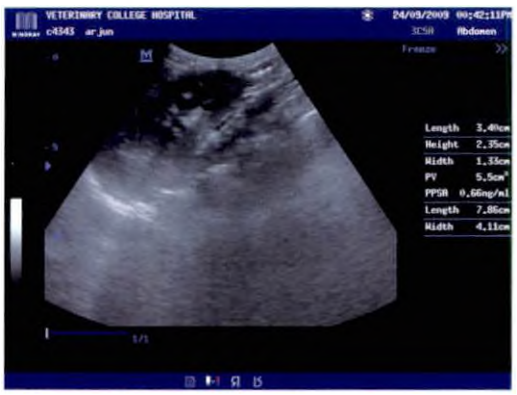
C. Hyperemic vesicles on bulbus glandis



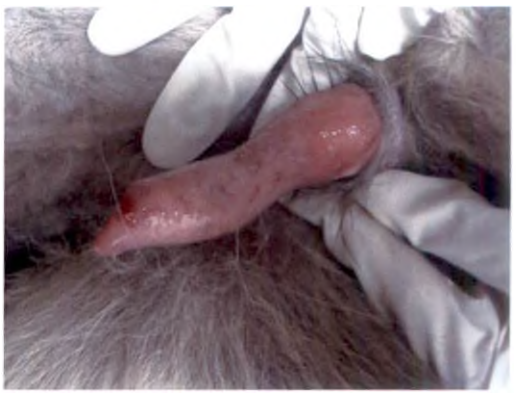
D. Thickened capsule and mixed echogenisity in prostate



E. Enlarged right kidney



F. After treatment



4.4.2.3.2.3. Prostatic fluid evaluation

Prostatic fluid collected by ejaculation was slightly purulent in nature. The cytological evaluation revealed clumps of neutrophils and occasional cuboidal cells indicating inflammatory process.

The culture and sensitivity of prostatic secretion revealed the presence of staphylococcus. The organism was sensitive to enrofloxacin, resistant to chloramphenicol, tetracycline and gentamicin.

4.4.2.3.2.4. Clinical pathology

Haemoglobin, erythrocyte count and PCV were 9.9 g/dl, 4.33 millions/cu mm and 25.5 % respectively. Total leukocyte count, percentage of neutrophils, lymphocytes and eosinophils were 14400 /cu mm, 60, 30 and 10 respectively. The platelet count was 279000/ cu mm.

Total protein, albumin, globulin and AG ratio were 3.90 g/dl, 1.00 g/dl, 2.90 g/dl and 0.30 respectively. The serum creatinine on the day of admission was 3.80 mg/dl and the subsequent value on seventh day was 5 mg/dl. Blood urea nitrogen was 32 mg/dl. The serum glucose and calcium were 68 mg/dl and 7mg/dl respectively. The serum acid phosphatase and prostatic fraction values were 12 U/L and 3.5 U/L respectively. The serum testosterone level was 0.9 ng/ml

The urine pH and specific gravity were 7.5 and 1.004 respectively. Proteinuria (4+) was present whereas, blood pigments were negative. The culture and sensitivity result of urine sample was negative for any microbial growth.

4.4.2.3.3. Treatment and response

The case was treated with enrofloxacin @ 10 mg/kg IV, fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC) and proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV) for 10 days. Additionally, the dog was treated with tablet Althrocin 250 mg (Erythromycin), tablet Zinnetac 150

mg and Herperax (aciclovir) ointment. The vesicular lesions of the eye and bulbus glandis were cured (Plate 5. F). Food intake also improved and there was slight reduction in constipation but the serum creatinine level further increased to 5 mg/dl. Since the owner was not interested in continuing the treatment, further observations could not be made.

4.4.3. Prostatic Abscess

A six year old intact male Rottweiler dog weighing 25 kg was presented with a history of anorexia and vomiting for 10 days. Melena and dyschezia were also present.

4.4.3.1. Clinical Observations

The animal was dehydrated, having difficulty to stand up. The respiration was shallow and uremic smell from mouth was present. The temperature was 102° F, pulse 120/min, respiration 17/ min and the visible mucous membranes were congested.

4.4.3.2. Diagnostic Methods

4.4.3.2.1. Digital rectal palpation

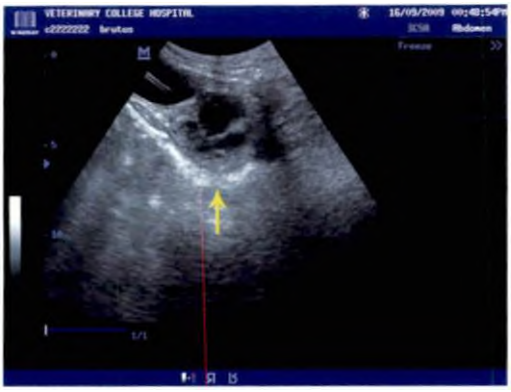
On digital rectal palpation, the prostate gland was found to be isothermic. Position of the prostate gland was partly in the pelvic and abdominal cavities. Prostate was found to be moderately smooth and soft in consistency. The prostate gland was enlarged asymmetrically and movable upon rectal examination. The animal exhibited slight pain during rectal examination.

4.4.3.2.2. Ultrasonography

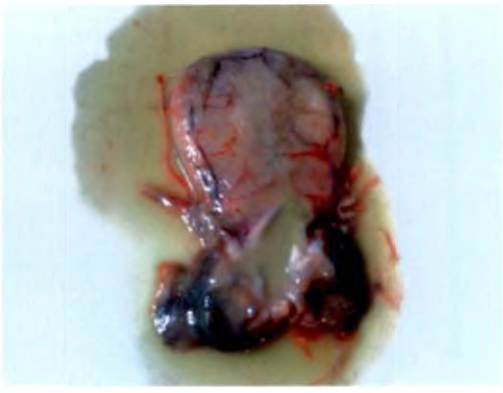
On ultrasonography, multiple hypoechoic cavitary lesions with irregular borders were visualized in the prostate gland. The fluid within the lesion had mixed echogenisity/ flocculent appearance (Plate 5. A). The prostatic length, depth, width and volume were 5.79 cm, 6.28 cm, 5.02 cm and 95.46 cm^3

Plate: 6. Prostatic abscess

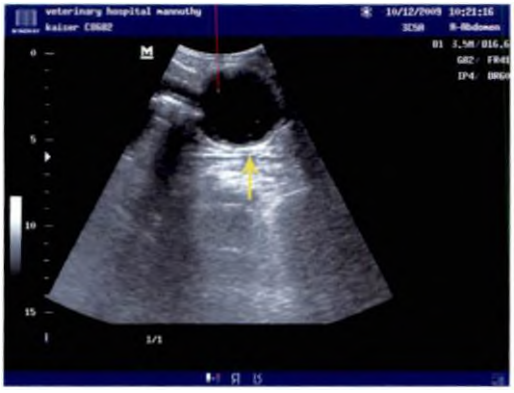
A. Multiple hypoechoic areas with irregular border



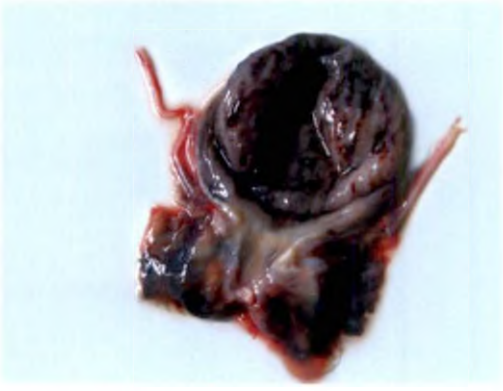
B. Gross
Prostate and urinary bladder with pus



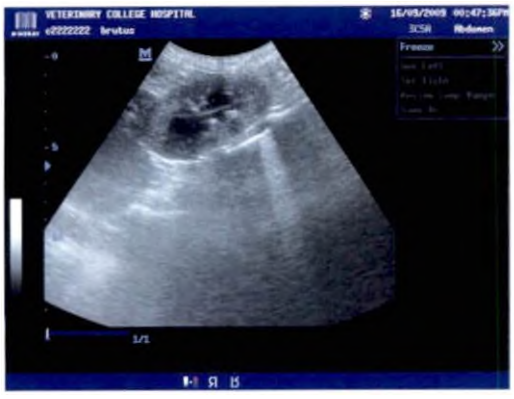
C. Thickened urinary bladder wall



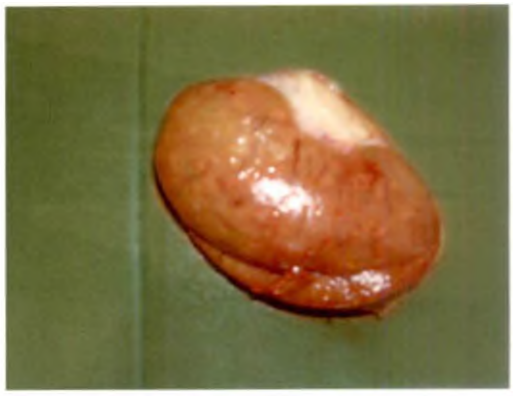
D. Gross
Echymotic patches on urinary bladder wall



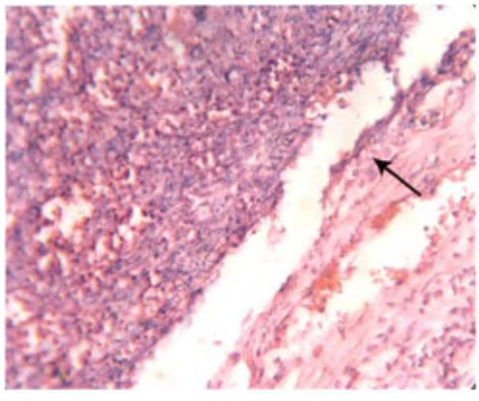
E. Kidney - Thickened cortex



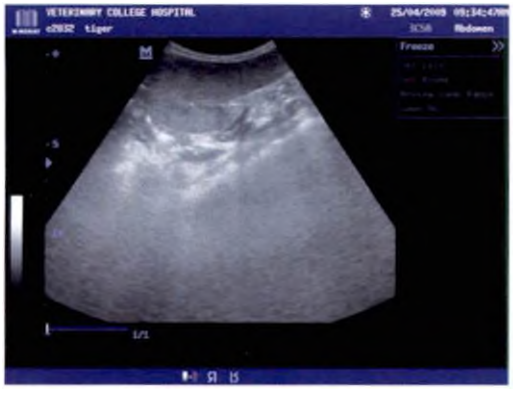
F. Gross - Kidney



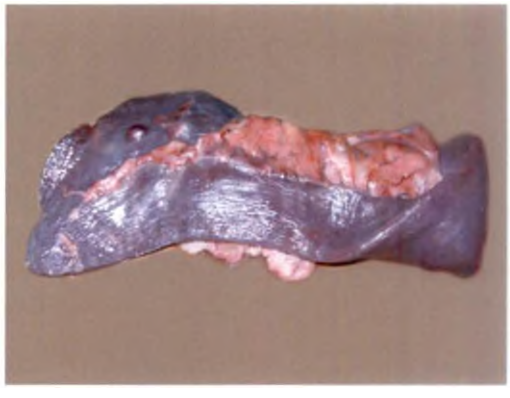
G. Histopathology of prostate -H&E (10x)
Abscess wall formed by
inflammatory granulation tissue



H. Splenomegaly



I. Spleen-gross



respectively. Both the kidneys were enlarged with thickened cortex and the corticomedullary distinction was absent. Urinary bladder with thickened wall containing mixed echogenic substances was observed. Splenomegaly was also present (Plate 5. C, E and H).

4.4.3.2.3. Prostatic fluid evaluation

Prostatic fluid collected by prostatic massage combined with urethral catheterization, was purulent and the cytological examination revealed large number of degenerated neutrophils and bacteriae.

Culture and sensitivity test of prostatic fluid revealed the presence of *E. coli* organism, which was sensitive to tetracycline, enrofloxacin and chloramphenicol.

4.4.3.2.4. Clinical pathology

Haemoglobin, erythrocyte count and PCV were 9.7 g/dl, 4.26 millions / cu. mm and 26.6 % respectively. The total leucocyte count was 25500/ cu. mm with differential count of neutrophils 88 % and lymphocytes 12 %. The platelet count of the animal was 415000/ cu. mm.

Serum total protein, albumin, globulin and the AG ratio were 7.9 g/dl, 2.5 g/dl, 5.4 g/dl and 0.46 respectively. Serum creatinine was 28 mg/dl and BUN was 675 mg/dl. Serum calcium and glucose were 9.1 mg/dl and 57 mg/dl respectively. The serum acid phosphatase was 8.5 U/L with prostatic fraction of 3.6 U/L. The serum testosterone level was 1.2 ng/ml.

The urine sample was acidic in nature and had the specific gravity of 1.008. Blood pigment was absent while proteinuria was (3+). The cultural examination of urine sample isolated *E. coli* organism, which was sensitive to tetracycline, enrofloxacin, amoxicillin and chloramphenicol.

4.4.3.3. Treatment and response

The treatment was started with enrofloxacin @ 10 mg/kg b.wt. IV fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg b. wt SC), proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV). But the animal died on the 2nd day of presentation.

4.4.3.4. Autopsy and histopathology

Postmortem examination revealed gastroenteritis, irregular kidneys with inflammatory changes and necrotic areas, splenomegaly with nodular changes. Both the lobes of prostate were enlarged and filled with pus. The right lobe was bluish to black in colour. The urinary bladder was also filled with purulent fluid and echymotic patches were present in bladder mucosa (Plate 5. B, D, F and I).

The prostatic weight, length and width measured during autopsy were 25 g, 5.5 cm, and 4.0 cm respectively.

Histopathology of prostate gland revealed an abscess wall that was formed by inflammatory granulation tissue. Some acini were distended with necrotic material inside. Stroma showed dense infiltration by lymphocytes, plasma cells and polymorphs. Some areas had hyperplastic glands (Plate 5. G).

4.4.4. Prostatic Neoplasia

4.4.4.1. Fibroadenoma with Paraprostatic Pseudocyst

A German shepherd male intact dog aged 3 ½ year was presented with the history of pyrexia, anorexia, intermittent urination, haematuria and dyschezia for 10 days (Plate 7. A).

4.4.4.2. Clinical observation

The animal was less active. Purulent urethral discharge was present. The animal exhibited pain while palpation of caudal abdominal area. The temperature

was 106° F, pulse 98/min, respiration 21/min and the visible mucous membranes were slightly congested.

4.4.4.3. Diagnostic methods

4.4.4.3.1. Digital rectal palpation

The prostate was found to be warm, slightly irregular, firm, immobile, painful and severely enlarged. The prostate was positioned partly intrapelvic and partly intrabdominal.

4.4.4.3.2. Ultrasonography

On ultrasonography, two anechoic cavities with irregular thickened borders were found lateral and cranial to the prostate. Diffused hyperechoic changes were observed in the remaining prostatic parenchyma. The anechoic cavity contained mixed echogenic substances. The length, depth, width and volume of anechoic cavity were 5.66 cm, 4.6 cm, 2.62 cm and 35.67 cm^3 respectively. Indistinct cortico medullary junction of kidney was observed (Plate 7. B and F). Splenomegaly was also observed.

4.4.4.3.3. Prostatic fluid evaluation

The prostatic fluid was aspirated under ultrasound guidance using spinal needle. Grossly, the fluid aspirated appeared as serosanguinous to brown coloured. Cytological evaluation revealed distorted and damaged cells resembling neutrophils. Anaplastic prostatic cells with anisocytosis was also found (Plate 7. C and D). Cultural examination of prostatic fluid revealed pseudomonas organism, sensitive only to chloramphenicol.

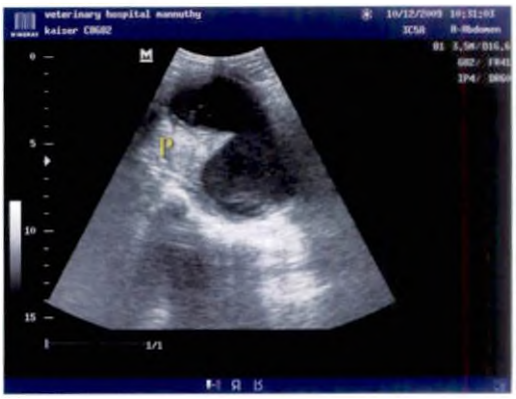
4.4.4.3.4. Clinical pathology

Hemoglobin, erythrocyte count and PCV were 9.6 g/dl, 4.1 millions/cu mm and 26.8 % respectively. Total leucocyte count with differential leucocyte counts of neutrophils, lymphocytes, eosinophils and monocytes were 50,900 /cu

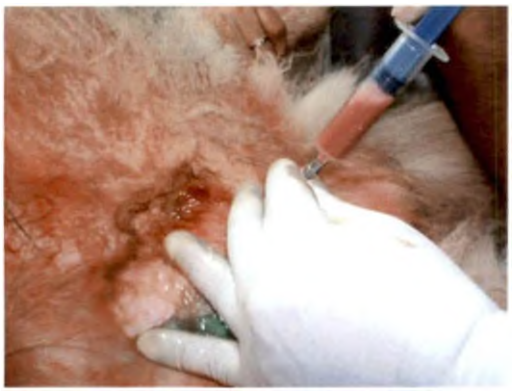
Plate: 7. Fibroadenoma with paraprostatic pseudocyst

A. Haematuria

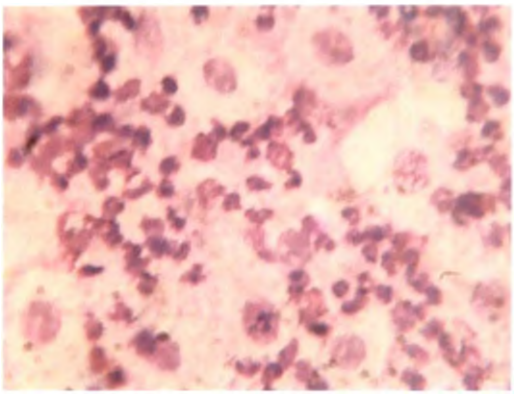
B. Two anechoic cavities adjacent to prostate (P)



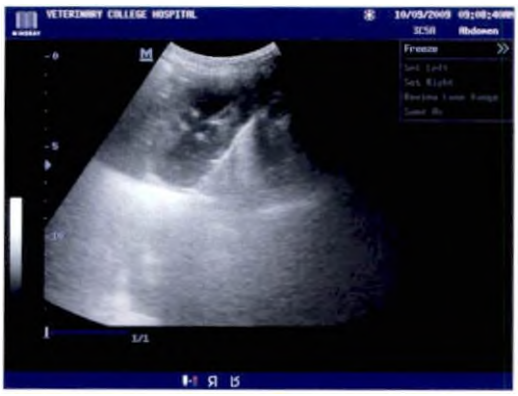
C. Aspiration of prostatic fluid



D. Prostatic fluid cytology
Anisocytosis, damaged neutrophils

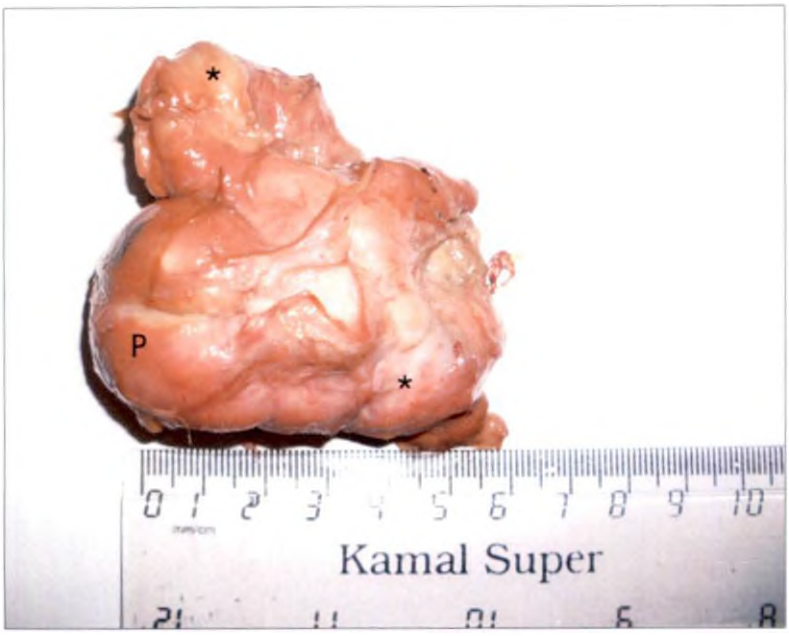


E. Kidney
Indistinct corticomedullary junction



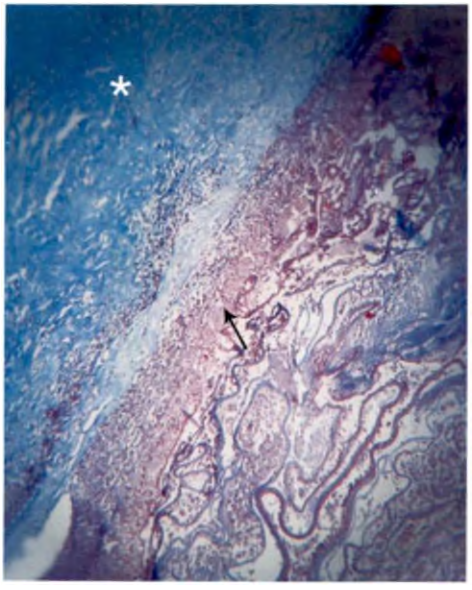
F. Gross kidney

G. Gross - prostate (P)
Paraprostatic pseudocyst (*)

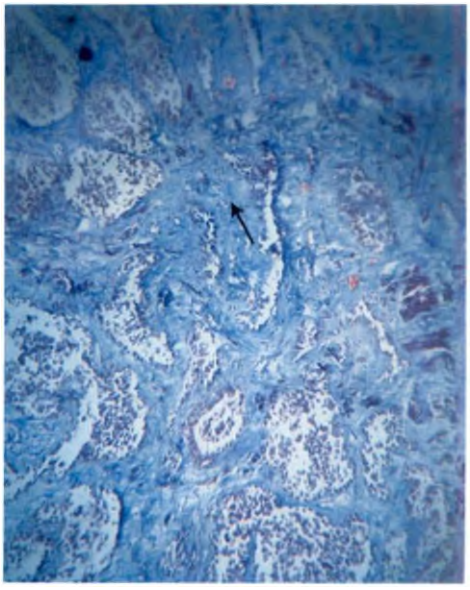


Histopathology of prostate -
gomori's one step trichrome method (10x)

H. Cystic wall with fibrous tissue (*)
and granulation tissue proliferation (↑)



I. Adenomatous proliferation of
fibrous tissue



mm, 51 %, 46 %, 1 % and 2 % respectively. The platelet count was 173150/cu mm.

Total protein, albumin, globulin and AG ratio were 7.2 g/dl, 1.9 g/dl, 5.3 g/dl and 0.4 respectively. Serum creatinine and blood urea nitrogen were 1.8 mg/dl and 25 mg/dl respectively. Subsequent value of creatinine on 14th day was 5.4 mg/dl. Serum glucose and calcium were 50 mg/dl and 7 mg/dl respectively. The total acid phosphatase was 1 U/L with prostatic fraction value of 0.4 U/L. Serum testosterone value was 1.4 ng/ml.

The urine sample collected had the pH of 4, specific gravity of 1.010, protein of 2+, pus cells (2+) and blood pigments were absent. The culture and sensitivity of urine revealed the presence of pseudomonas organism, which was sensitive only to chloramphenicol.

4.4.4.4. Treatment and response

The case was initially treated with enrofloxacin @ 10 mg/kg IV. The condition improved by 5th day. But later there was discontinuity in the treatment and the condition worsened and progressed to renal failure. Then, the treatment was changed to chloramphenicol @ 25 mg/kg IV twice daily based on culture and sensitivity result. Additionally, fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC) proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV) were also given as the condition progressed to renal failure. The dog succumbed after 23 days.

4.4.4.5. Postmortem examination

On postmortem examination, both the lobes of prostate were found enlarged and two cavitating structures containing slightly purulent fluid, cranio lateral to the prostate were present. There was no direct communication between the cyst and prostatic parenchyma. The urinary bladder was filled with pus. Both

the kidneys were irregular and necrotic. The prostate had weight of 50 g and length and width were 5.7 cm and 5 cm respectively (Plate 7. F and G).

Histopathological studies of prostate section stained with Gomori's one step trichrome method revealed adenomatous proliferation of fibrous tissue and diffuse infiltration of lymphocytes and plasma cells. The inner lining of the cyst was seldom lined by epithelium and had granulation tissue proliferation. The wall was chiefly composed of fibrous tissue with the outer lining of mesothelium. The condition was diagnosed as fibroadenoma with paraprostatic pseudocyst (Plate 7. H and I).

4.4.4.2. Papillary Adenoma

A Cocker spaniel aged 8 year was referred with the history of anorexia, vomiting and dyschezia for 5 days. Melena and oliguria were reported.

4.4.4.2.1. Clinical observation

Animal was very weak and melena was noted. Abdominal thudding was present. The temperature, pulse and respiration were 100.2° F, 85/min and 16/min respectively and the visible mucous membranes were slightly congested.

4.4.4.2.2. Diagnostic methods

4.4.4.2.2.1. Digital rectal palpation

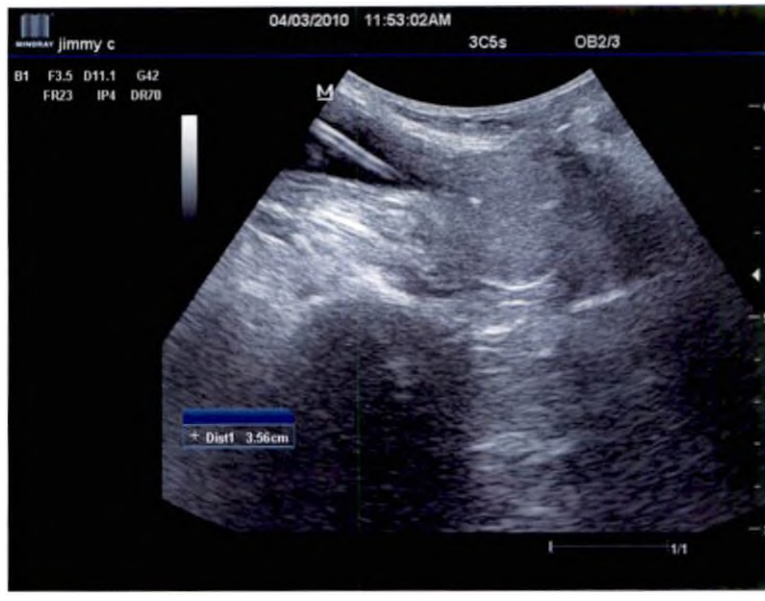
The prostate gland was found to be isothermic, intrabdominal, smooth, firm, symmetrically enlarged, movable and painful on rectal examination.

4.4.4.2.2.2. Ultrasonography

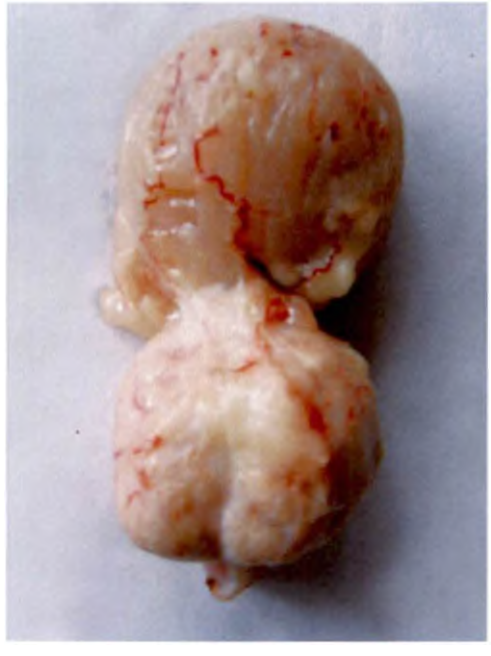
On ultrasonography, prostate lobes were symmetrically enlarged and focal hyperechoic areas were observed in one lobe of prostate (Plate A). The prostatic length, depth, width and prostatic volume were 5.73 cm, 4.56 cm, 3.78 cm and

Plate: 8. Papillary adenoma

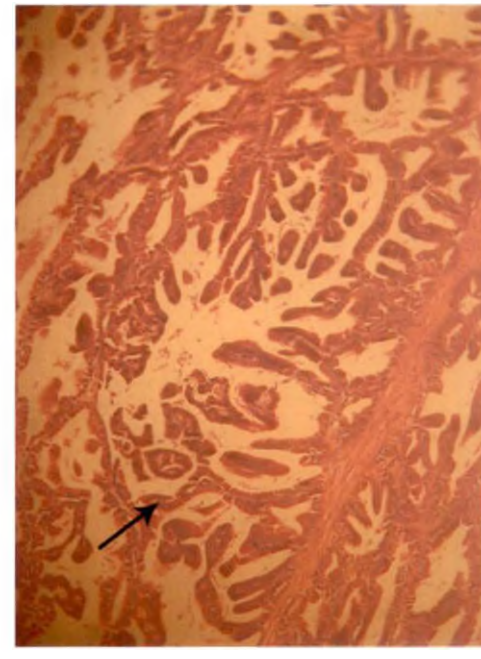
A. Symmetrically enlarged prostate



B. Gross - Prostatomegaly



C. Histopathology of prostate - H&E (10x)
Adenomatous papillary folds



51.65 cm^3 respectively. Hyperechoic changes and indistinct cortico medullary junction were observed in left kidney.

4.4.4.2.2.3. Prostatic fluid evaluation

Prostatic massage combined with urethral catheterization was performed to collect prostatic fluid. The prostatic fluid was slight creamy in colour. Cytological evaluation revealed clumbs of cuboidal cells. Cultural examination of prostatic fluid was negative for bacterial growth.

4.4.4.2.2.4. Clinical pathology

Hemoglobin, erythrocyte count, and PCV were 5.29 gm %, 2.93 millions/ cu. mm and 19.4 % respectively. The total leucocyte count was 6820/ cu. mm with differential count of neutrophils 85 % (band forms 6 %) and eosinophils 9 %. Platelet count was 392000/cu. mm.

The serum total protein, albumin, globulin and the AG ratio were 6.5 g/dl, 3 g/dl, 3.5 g/dl and 0.66 respectively. Serum creatinine and blood urea nitrogen were 24.4 mg/dl and 597 mg/dl respectively. The serum calcium and glucose were 7.1 mg/dl and 68 mg/dl respectively. Level of serum total acid phosphatase and testosterone were 10.6 U/L (prostatic fraction 3.8 U/L) and 1.5 ng/ml respectively.

The urine sample had the pH of 5 and the specific gravity was 1.006. Erythrocytes were present (12-15/HPF). Pus cells were also present. The cultural examination revealed no bacterial growth.

4.4.4.2.3. Treatment and response

Treatment was started with fluids, antiemetics (Inj Metoclopramide @ 0.2 mg/kg BW SC) proton pump inhibitors (Inj Pantoprazole @ 1 mg/kg BW IV) and enrofloxacin @ 10 mg/kg IV. The animal showed temporary improvement but succumbed to death on the next day.

4.4.4.2.4. Post mortem examination

Both the lobes of prostate were symmetrically enlarged and the prostate had round shape. The surface was smooth and firm. Histopathology revealed adenomatous proliferation of the lining cells that formed papillary projections into lumen. Hence it was confirmed - a case of papillary adenoma (Plate B and C).

Discussion

5. DISCUSSION

The present study was undertaken to study the clinico – biochemical and ultrasonographic findings associated with diseases of prostate and to evaluate the response to the treatment adopted. The information gathered suggested that hematobiochemical parameters utilized in this study were unrewarding to diagnose prostatic diseases. However, ultrasonography could be used as the most important diagnostic tool for prostatic diseases.

Prostatic disorders observed in this study were classified into four groups namely, 1). Benign prostatic hyperplasia (benign prostatic hyperplasia with concurrent prostatitis, benign prostatic hyperplasia and associated hernia). 2). Prostatitis (acute prostatitis, suppurative prostatitis and prostatitis associated with herpes virus infection) 3). Prostatic abscess 4). Prostatic neoplasia (fibro adenoma with paraprostatic pseudocyst and papillary adenoma). The occurrence, signalment, clinical signs, clinical observation, findings of digital rectal palpation, ultrasonography, prostatic fluid evaluation, clinical pathology, treatment and autopsy findings of these diseases were discussed in detail.

5.1. OCCURRENCE

A total of 20 cases with clinical signs suggestive of prostatic disorders were selected. Based on clinical examination and special diagnostic techniques, prostatic diseases were confirmed in 15 cases. The occurrence of 75 % of prostatic disorders obtained in the present study was very high when compared with those reported by earlier workers. The occurrence of 2.5 % was observed in the total number of males seen during the five-year study of Krawiec and Heflin (1992). This difference is due to the difference in selection criteria adopted for the present study. However, histopathological studies by Amorim *et al.* (2004a)

revealed that 81.3 % prostate gland had some kind of prostatic hyperplasia upon screening prostate glands from dogs between five and fifteen years of age.

The most common prostatic disorder recorded in the present study was prostatitis (46.7 %). Krawiec and Heflin (1992) also reported similar findings. This is in contrary to the observations of Dhanya (2004) who reported benign prostatic hyperplasia (73.47 %) as the most common prostatic disease during postmortem examination and histopathologic studies. This difference in the present study might be due to masking of mild BPH with inflammation, which was the limitation of cytologic diagnosis for canine prostatic disorder as reported by Powe *et al.* (2004). The inflammatory process of prostate gland is not an uncommon urologic disorder in older intact male dogs, as lower urinary tract infection generally comes together with prostatic infection.

Acute prostatitis (33 %) was the most common prostatitis reported in the present study where as, chronic prostatitis was the most common prostatitis observed in the study conducted by Dhanya (2004).

5.2. SIGNALMENT

5.2.1. Breed

Although all the common breeds presented to the hospital were affected, increased incidence was observed in German shepherd (46.6 %). Dhanya (2004) also reported that German shepherd was most frequently affected with prostatic diseases. The other breeds were Rottweiler, Dachshund, Cocker spaniel, Doberman, Labrador and Boxer. These findings regarding the breeds in the present study was in agreement with that of Johnston *et al.* (2001) who reported that there was no breed predilection for prostate disease and large breed dogs such as German shepherd and Doberman had increased prevalence. Causal relationship had not been established.

5.2.2. Age

The mean age of dogs affected with prostatic diseases in the present study was 6.7 year, which closely relates to the report by Lowseth *et al.* (1990) who reported that prostatic diseases were more common in dogs over six years. The highest incidence was recorded in the age group of 5-10 years, which emphasizes the fact that the onset of prostatic disease was more in aged dogs and the incidence increases with age. This had been previously reported by Amorim *et al.* (2004a) who revealed that 81.3 % of the dogs had prostatic hyperplasia upon screening them between five and fifteen years of age.

5.3. DISEASES OF PROSTATE GLAND

5.3.1. Benign Prostatic Hyperplasia

5.3.1.1. Benign Prostatic Hyperplasia with Prostatitis

Benign prostatic hyperplasia with prostatitis was accounted for 20 % of prostatic diseases in the present study. Benign prostatic hyperplasia (BPH) is a spontaneous and age related condition in human and intact male dogs (Johnston *et al.* 2000). Leads and Leav (1969) reported a case of hyperplasia of the prostate with and without concurrent inflammation.

Wallace (2001) reported that infection of the prostate gland could occur in male dogs of any age, but it was more common in older dogs with BPH. It was rare or nonexistent in castrated dogs due to atrophy of the prostate gland. The glandular changes and disruption of normal urine flow and/or prostatic fluid flow associated with BPH predisposes the gland to infection. The urethra communicates with the prostate gland via the prostatic ducts, and ascending infection is therefore the most common etiology.

Mean age of affected dogs in this study was 6.3 year. More than 80 % of intact male dogs over 5 year exhibited BPH as reported by Johnston *et al.* (2000).

5.3.1.1.1. Clinical signs

The clinical signs in dogs affected with BPH with concurrent prostatitis were anorexia, constipation and dripping of prostatic fluid from the tip of penis. Similar symptoms were reported by Krawiec and Heflin (1992), Baker and Lumsden (2000), Parry (2007) and Smith (2008). Urine with foul smell reported in one dog might be due to the urine retention caused by the pressure of enlarged prostate on the prostatic urethra. Dhanya (2004) reported that prostatic urethra became elongated and compressed in hyperplasia, leading to defective emptying and thus accumulation of urine in the bladder. The residual urine may readily get infected and act as a source of infection. The most common complication of BPH in dogs is secondary bacterial infection of gland leading to prostatitis. In prostatic hyperplasia, the acini may be dilated with accumulated secretion. This might also provide a favorable environment for the growth of pathogens and act as a source of infection. Vomiting reported in one dog might be due to the renal failure, which developed, secondary to prostatitis as reported by Kanaran (2009).

5.3.1.1.2. Clinical observation

The mildly elevated temperature and congested mucous membranes were due to septicemia associated with prostatitis. Smooth, movable, enlarged, isothermic and painless prostate could be found on perectal digital palpation. Johnston *et al.* (2000) and Davidson (2003) described similar findings on rectal palpation of prostate of dog with BPH. Warm and painful prostate observed in one case may be due to severe concurrent inflammation.

5.3.1.1.3. Ultrasonography

The ultrasonographic picture of prostatic parenchyma in BPH has been described as homogenous with or without cavitating cystic lesions (Johnston *et al.* 2000). In the present investigation also, ultrasonography revealed anechoic cystic areas in two dogs and homogenous parenchyma in one dog. The anechoic areas were typically well defined and smoothly marginated.

The measured prostatic volume was 72.7 cm^3, which was higher than the normal volume 18.9 ± 15.5 cm^3 as reported by Ruel *et al.* (1998). The Prostatic volume of affected dogs may be 2 to 6.5 times greater than that of normal dogs of similar weight. The present finding was in accordance with that of Kamolpatana (1998). Splenomegaly was observed in all the cases.

5.3.1.1.4. Prostatic fluid cytology

Even though the prostatic fluid was slightly purulent in all the cases, cytological evaluation identified few leucocytes in two cases. Cytological examination of other case revealed hyperplastic cells with mild anisokaryosis, unaltered nucleus cytoplasm ratio and large number of neutrophils. Baker and Lumsden (2000) reported increase in cell size and mild anisokaryosis with preserved nucleus: cytoplasm ratio in BPH. Prostatic epithelial cells with round to oval nuclei with reticulated chromatin were observed in all the cases, which closely agrees with the findings of Zinkl (1999).

Prostatic fluid culture from the entire dog revealed the presence of *E. coli*, which was sensitive to enrofloxacin and chloramphenicol. The glandular changes and disruption of normal urine flow and/or prostatic fluid flow associated with BPH predisposes the gland to infection (Wallace, 2001).

5.3.1.1.5. Clinical pathology

The mild anemia, leucocytosis with neutrophilia observed in the present investigation is in contrary to the reports of Gobello and Corrada (2002) and Parry (2007) as they reported normal hemogram and serum biochemistry in BPH. The leucocytosis and neutrophilia might be due to concurrent prostatitis.

Normal level of total protein, albumin, globulin and mild hypoglycemia and hypocalcaemia were observed in the present study. The increased serum creatinine and blood urea nitrogen in the present study is in agreement with Kanaran (2009) who reported concurrent renal failure in BPH affected dogs.

Elevated levels of serum acid phosphatase with prostatic fraction obtained in the present study were in accordance with the report by Corazza *et al.* (1994). The normal serum testosterone value obtained in the present investigation emphasize the fact opined by Lowseth *et al.* (1990) that the measured level of serum testosterone could not be used to predict the degree of BPH present.

Proteinuria observed in the dog with renal failure might be due to damaged nephrons. Haematuria and pyuria were present in two samples. Similar observations were also made by Gobello and Corrada (2002). Cultural examination of urine samples was negative for bacterial growth, which is in accordance with the findings of Parry (2007).

5.3.1.1.6. Treatment and response

Two dogs were treated with 5∝ reductase inhibitor @ 1mg/kg orally for 3 weeks and enrofloxacin @ 10mg/kg IV for 7 days. One of these had renal failure and died on 2nd day. Sirinarumitr *et al.* (2001) found that the most common medical treatment for BPH was finasteride. The objective of treatment in dogs with BPH is to decrease prostatic size, which alleviates the signs related with BPH. Finasteride is a synthetic steroid that inhibits type Π 5 α reductase, therefore blocks conversion of testosterone to dihydrotestosterone (Span *et al.*, 1998), which is essential for prostatic growth. Hence, the administration of finasteride helps in reducing the size of prostate in BPH by apoptosis. The Dachshund had advanced renal failure and died on 2nd day and so the effectiveness of treatment regimen could not be evaluated. The Rottweiler treated with 5∝ reductase inhibitor @ 1mg/kg responded only moderately. This might be due to inadequate dosage. Laroque *et al.* (1994, 1995) reported that finasteride at doses of 1 to 5 mg/kg per day caused atrophy of glandular and stromal compartments of prostate and a decrease in prostatic weight and volume. However, treatment with a lower dose of finasteride (0.1-0.5 mg/kg/day) is being investigated and appears to be effective (Kustrtz and Klausner, 2000).

The other dog was not treated with 5∝ reductase inhibitor but treated with enrofloxacin alone since the case was first diagnosed as a case of prostatitis only, due to masking of BPH with inflammation as opined by Powe *et al.* (2004). The condition improved but recurrence was noted after 2 months and later the condition worsened and the dog succumbed and conducted autopsy.

5.3.1.1.7. Autopsy and histopathology

Grossly, the prostate was enlarged, rough and both the lobes were replaced by cavity and contained purulent fluid. Bloom (1954) noticed that in canine prostate hyperplasia, the gland was severely enlarged with the outer surface smooth or irregularly nodular and on sectioning, the lobules were outlined by wide irregular whitish grayish bands of stromal tissue, along with cysts containing clear or cloudy fluid, irregularly distributed through out the parenchyma.

Histological appearance of prostate was hyperplasia of glands with papillary infoldings. Stroma was hypertrophied with fibrocollagenous tissue and diffusely infiltrated with lymphocytes and plasma cells. Similar findings were noted by Bloom (1954) and Leeds and Leav (1969).

5.3.1.2. Benign Prostatic Hyperplasia and Associated Hernia

Benign prostatic hyperplasia with hernia was reported in two dogs. Kumar *et al.* (2008) reported a case of cystic type of hyperplasia along with perineal hernia in a Pomeranian dog. Hosgood *et al.* (1995) reported that 4 of 32 dogs with perineal hernia had prostate gland as the hernial content. In the present study, perineal hernia was observed in an 8-year-old Boxer and perineal and inguinal hernia in an 9-year-old Dachshund.

The Boxer had normal appetite but had dyschezia. Inappetance and dyschezia were the clinical signs of Dachshund. Kumar *et al.* (2008) observed difficulty in defecation and urination in a dog with perineal hernia, which was

progressive in nature. Dysuria was less common in dogs with BPH as the gland expands uniformly and away from the urethra, in contrast to BPH in man (Parry, 2007).

The prostate was isothermic, smooth, highly enlarged, and movable upon rectal digital examination. These observations were similar to the previous recordings of Barsanti and Finco (1989), Johnston *et al.* (2000) and Davidson (2003).

5.3.1.2.1. Ultrasonography

Prostatomegaly with intraparenchymal cystic lesions in the present cases were supported by the findings of Johnston *et al.* (2000) and Smith (2008).

5.3.1.2.2. Prostatic fluid evaluation

The clusters of prostatic epithelial cells with indistinct cytoplasmic borders, the round to oval nuclei with finely reticulated chromatin pattern and unaltered nucleus cytoplasmic ratio observed in the prostatic fluid cytology were similar to the report by Zinkl (1999). Unaltered nucleus cytoplasmic ratio helped to rule out prostatic neoplasms.

5.3.1.2.3. Clinical pathology

Hematobiochemical results were unrewarding as suggested by Parry (2007).

Elevation of serum prostatic acid phosphatase and testosterone was observed in the present study. This findings support the fact opined by Corazza *et al.* (1994) that the prostatic acid phosphatase was androgen dependent and its increase in serum probably due to degeneration of prostatic secretory cells induced by increased dihydrotestosterone concentrations within the gland. Urinalysis of both the dog was normal as reported by Gobello and Corrada (2002).

5.3.1.2.4. Treatment and response

The Boxer was treated with 5 ∝ reductase inhibitor @ 1 mg/kg orally for 3 weeks and perineal herniorrhaphy was carried out. But the response with 5 ∝ reductase inhibitor @ 1 mg/kg was very slow. After one month, another small swelling developed in the region adjacent to the previous one. Laroque *et al.* (1994) reported that the decrease in prostatic size was positively correlated with dose and duration of treatment. Laroque *et al.* (1994, 1995) reported that finasteride at doses of 1 to 5 mg/kg per day caused atrophy of glandular and stromal compartments of prostate and decrease in prostatic weight and volume. Hence, the dose and duration of finasteride therapy used in this particular case might not have been completely effective.

The Dachshund was treated surgically (inguinal herniorrhaphy and castration) and there was no report of recurrence of the condition during the 3 months of post surgical period. White (2000) suggested that castration was the most effective treatment for removing the hormonal influence in dogs with BPH.

5.3.2. Prostatitis

5.3.2.1. Acute Prostatitis

The occurrence of prostatitis observed in the present study was 33 % was in accordance with the findings of Cowan and Barsanti (1991) who reported the prevalence rate of prostatitis as 20-70 %. Age of the affected dogs ranged from 3.6 – 9 years with a mean of 5.3 year.

5.3.2.1.1. Clinical signs

Dogs presented with prostatitis had symptoms such as anorexia, urethral discharge, dyschezia and hematuria that were also reported by Krawiec and Heflin (1992), Dorfman and Barsanti (1995) Johnston *et al.* (2000) and Smith (2008). Clinical signs such as straining to defecate and constipation may result secondary to displacement and narrowing of large intestine due to prostatomegaly

as opined by Hoffer *et al.* (1977). Haematuria might be due to irritation of the bladder by ammonia released from retained urine (Kiren, 2008). Urine retention or incontinence or dysuria may occur from impingement upon the bladder and urethra as reported by Hoffer *et al.* (1977).

Fever (103.4° F) was observed in dogs with prostatitis in the present study. The same observation was also made by Krawiec and Heflin (1992), Dorfman and Barsanti (1995). Three dogs with prostatitis had caudal abdominal pain, which, agrees with the findings of Smith (2008).

5.3.2.1.2. Digital rectal palpation

The warmness and pain on palpation of the prostate gland of all the dogs indicated prostatitis. The variation in position of the prostate gland might be due to variation in degree of prostatomegaly with regard to inflammation. These observations were similar to the report of Davidson (2003) and Smith (2008).

5.3.2.1.3. Ultrasonography

Ultrasonographic examination revealed diffused increase in echogenisity of prostatic parenchyma and 40 % of dogs had multifocal hyperechoic areas in prostatic parenchyma. The mean prostatic length, depth, width and volume in the present study were 5.77 cm, 4.66 cm, 4.95 cm and 68.76 cm^3 respectively. Duque *et al.* (2009) also observed diffused increase in prostatic echogenisity and enlarged prostate with 5.28 cm length and 4.9 cm width in prostatitis. The occurrence of multi focal hyperechoic areas in 40 % dogs in the present study might be due to more severe localized infection.

Hyperechoic particles in urinary bladder of three dogs might be due to spread of inflammation from prostate. In the absence of micturition or ejaculation, urethral pressure moved the prostatic fluid cranially into the bladder by prostatic fluid reflux as opined by Romagnoli (2007).

Small kidneys with indistinct corticomedullary junction might be due to ascending grade of infection from prostate (Jayathangaraj *et al.* 1993).

5.3.2.1.4. Prostatic fluid evaluation

Prostatic fluid cytology of the all the cases revealed large number of neutrophils. Bacteria-laden neutrophils in prostatic fluid could be observed in 40 % of the cases. Ling *et al.* (1983) opined that bacteria-laden white blood cells in canine prostatic fluid were indicative of active infection. Cultural examination of prostatic fluid from the five dogs revealed *E. coli.* Ling *et al.* (1983), Barsanti and Finco (1986) Johnston *et al.* (2000) and Dhanya (2004) reported that *E. coli* was the most common bacterial organism identified in dogs with bacterial prostatitis.

5.3.2.1.5. Clinical pathology

Mild anemia with hemoglobin of 11.08 g %, RBC of 4.95 million /cu.mm and PCV of 30.14 %, leucocytosis (14880/cu.mm) and neutrophilia (83 %) with left shift (2 %) observed in the present study is in agreement with the findings of Johnston *et al.* (2000), Davidson (2003), Smith (2008) and Duque *et al.* (2009). The mean platelet count was within normal range. There is no report regarding the platelet count in dogs with prostatitis.

Normal total protein (6.8 g/dl), mild hypoalbuminemia (2.16g/dl) and hyperglobulinemia (4.64g/dl) were observed in the present study. There is paucity of literature regarding this. The elevation of mean creatinine (4.42 mg/dl) and BUN (39.6 mg/dl) value were observed in the present study which has also been reported by Jayathangaraj *et al.* (1993) and Duque *et al.* (2009). Most of the dogs with prostatic infection may also have bladder infection and renal failure (Barsanti *et al.* 1983) and this was confirmed with the present case by ultrasonogram of bladder and increased serum creatinine. The mild hypoglycemia and hypocalcemia observed in the study may be due to anorexia associated with prostatitis.

Barsanti and Finco (1979) suggested that most of the prostatic infections were secondary to migration of bacteria from the urethra although spread through blood, semen and rectal flora was also possible.

The level of serum total acid phosphatase (6.32 U/L) and prostatic acid phosphatase (2.9 U/L) were elevated in dogs with prostatitis. Elevated human serum PAP concentrations were present in benign prostatic hypertrophy, prostatitis, following urethral catheterization and prostatic massage as suggested by Wadstrom *et al.* (1984), Collier and Pain (1986) and Heller (1987), in prostatic adenocarcinoma (Babaian and Orlando, 1986) and gastrointestinal tract tumors (Sorbin *et al.* 1986). Prostatic acid phosphatase is androgen dependent and its increase in serum was probably due to degeneration of prostatic secretory cells induced by increased dihydrotestosterone concentration within the gland (Corazza *et al.*,1994).

No correlation was found between serum testosterone level and prostatitis as the value was within normal range. Lowseth *et al. (1990)* estimated serum testosterone in healthy dog as 92-2550 pg/ml.

The serum samples from all the dogs were negative for Brucella antibody test. *Brucella canis, Brucella suis* and *Brucella abortus* are all capable of causing prostatitis in dogs as opined by Barr *et al.* (1986). Haematuria and proteinuria were observed in two cases. Similar observations were also made by Davidson (2003), Smith (2008) and Duque *et al.* (2009). Prostate gland is in close proximity to the microflora of distal urethra. This may be the reason for prostatitis being mostly coexistent with nephritis and cystitis as observed in the present study.

Culture of urine sample of two dogs revealed *E. coli.* This may be due to possible contamination of the fluid from the urinary bladder or urethra, while in the rest of the two cases, urine was found sterile, and the prostatic fluid revealed a pathogen. This evidence emphasizes the fact that the isolated pathogen originates certainly from the prostate gland.

5.3.2.1.6. Treatment and response

Three animals responded successfully to the treatment with enrofloxacin, which is in agreement with Duque *et al.* (2009). Enrofloxacin could easily diffuse through blood- prostate barrier and achieved therapeutic concentration in prostatic fluid.

The case with renal failure, which developed secondary to prostatitis, was effectively treated with enrofloxacin, fluid and anti ulcer therapy. The serum creatinine value of 5.6 mg/dl was reduced to 2.4 mg/dl with the above treatment. Whereas the one with highly elevated creatinine 12.4 mg/dl did not respond to the treatment and succumbed, this might be due to advanced renal failure.

The unsatisfactory response showed by the other dog might be due to mild BPH, which was masked by inflammation.

5.3.2.2. Supurative Prostatitis

Suppurative prostatitis was reported in an eight years old German shepherd.

Klausner *et al.* (1995) reported that suppurative and chronic prostatitis were the most common types of prostatitis. Where as acute, suppurative and chronic types of prostatitis were seen in the study conducted by Dhanya (2004) with the chronic type being the most common and one case of suppurative prostatitis was observed in a total of 20 prostatitis cases.

5.3.2.2.1. Clinical signs

Anorexia, vomiting, dyschezia, ribbon like stools, dribbling of urine, prostatic shuffle, pyrexia, uremic smell from mouth, purulent urethral discharge and pain on caudal abdominal palpation were the major clinical signs. Wallace (2001) reported that some dogs with prostatic disease may exhibit a wide-based

gait in the hind limbs, called the 'prostatic shuffle', which is an attempt to ease discomfort while walking.

Johnston *et al.* (2000) reported that when the inflammatory response is suppurative, the prostate would be uniformly enlarged. It was common for dogs with an enlarged prostate to have ribbon or tapered stools due to compression of the rectum by the enlarged prostate (Smith, 2008).

Vomiting, inappetance, constipation and pyrexia in a dog with prostatitis might be due to secondary acute renal failure as reported by Jayathangaraj *et al.* (1993). The pain on caudal abdominal palpation and warm painful prostate gland on per rectal examination might be due to suppurative process of the prostate.

5.3.2.2. 2. Ultrasonography

The ultrasonographic picture of the prostate gland revealed multifocal anechoic and hyperechoic areas. The change in sonogram of kidney could be due to secondary renal failure developed from prostatitis either by post urethral obstruction or ascending grade of infection. Grauer (2005) had enlisted prostatic diseases as a cause of glomerular disease. Another factor related to prostatic enlargement causing renal failure was obstructive nephropathy, which causes the functional changes in kidney that resulted from obstruction to the flow of urine (Gloor and Torres, 2007).

5.3.2.2. 3. Prostatic fluid cytology

Toxic neutrophils in prostatic fluid cytology indicated suppuration of prostate gland. Dhanya (2004) also observed similar changes in suppurative prostatitis.

5.3.2.2. 4. Clinical pathology

Leucocytosis of 26900/cu.mm and neutrophilia with shift to left observed in this case was similar to the observation made by Parker (1975). The

hyperglobulinemia and azotemia observed in the present case was also reported by Davidson (2003). The elevated level of serum acid phosphatase is in agreement with the report of Wadstrom *et al.* (1984). Serum sample was negative for brucella antibody test. Low specific gravity and proteinuria identified in the present case correlated with the elevated serum creatinine level and renal ultrasonography.

5.3.2.2. 5. Treatment and response

Even though the dog was treated with enrofloxacin and treatment for renal failure was done, the condition was not cured as the dog was in advanced stage of renal failure and succumbed after 20 days.

5.3.2.2. 6. Autopsy and histopathology

Both the prostate lobe was enlarged and purulent fluid oozed out on sectioning. Both the kidneys were smaller and irregular with necrotic areas. Histopathology of prostate revealed that, almost all acini were filled with neutrophils, some of acini cystic with accumulated secretion. Lining cells of glandular acini were not clearly visible, moderate interstial fibrosis was present. These findings were similar to the observations made by Dhanya (2004).

5.3.2.3. Prostatitis Associated with Herpes Virus Infection

Canine herpes viral infection is well known, to be capable of producing generalized infection leading to death in neonatal and foetal pup. The mortality in neonatal or infant puppies is virtually 100% in most of the outbreaks of canine herpes viral infection. (Carmichael *et al.* 1965). Where as, Sigel (1962) reported that the age of an animal at the time of infection with many viruses often determines the outcome of disease. Anvik (1991) reported that herpes could cause both systemic and localized disease and had predilection for upper respiratory, genital and neurologic tissues. The author also reported that oropharyngeal and genital forms were milder than other forms.

The case was reported in a 7-½ month old German shepherd male dog. The dog might have exposed to canine herpes virus in utero, during passage through the birth canal, or as newborns from the dam, infected littermates, or formites (Anvik, 1991). Immunosuppressive factors were facilitating the viral reactivation.

5.3.2.3.1. Clinical signs

The case was reported with the history of soft stools with straining while defecation. Other clinical signs were anorexia, vomiting, difficulty in urination and malaise. Hashimoto *et al.* (1978) reported greenish yellow diarrhea, anorexia, vomiting, serous nasal discharge and dyspnoea in naturally occurring cases of canine herpes viral infection. The difficulty in urination might be due to renal failure that developed from the necrosis and hemorrhage in kidney produced by the herpes viral infection as reported by Hashimoto and Hirai (1984). Neonates infected with canine herpes viral infection could show severe ocular inflammation characterized by cataracts, keratitis and retinitis within five days of inflammation (Anvik, 1991).

5.3.2.3.2. Clinical observation and ultrasonography

Pyrexia, hyperemic vesicular lesions in the conjuctival mucous membranes and petichae, vesicular and roughened surface on bulbus glandis were the clinical observations after four days of presentation. Similar observations were reported by Anvik (1991). Warmness and pain on per rectal examination of prostate revealed presence of prostatitis.

Ultrasonographic appearance of prostate suggested prostatitis, which could be correlated with the results of cytological evaluation of prostatic fluid and the culture report of prostatic fluid, which was positive for staphylococcus infection. The latent herpes viral infection might have either emerged into active infection due to the immunosuppression by staphylococcal infection or the prostatitis could develop from herpes viral infection as the virus could cause

genital lesions and staphylococcal infection occurred secondary to herpes infection. Anvik (1991) reported that the stress of concurrent disease could play a role in the activation of latent infection.

5.3.2.3.3. Clinical pathology

The hemoglobin value 9.9g/dl, RBC of 4.43 millions/.cu.mm and PCV of 25.5% suggestive of mild anemia might be due to focal hemorrhage in kidneys, liver, lungs and intestines and hyperemia on penis due to herpes virus infection. The mild leucocytosis with eosinophilia may be due to infectious process. Eosinophilia might occur with the inflammation of gastro intestinal, urogenital, respiratory tracts, or the skin (Allen *et al*, 2005). Hypoalbuminemia might be due to liver and kidney disorder by herpes infection and also from increased stress condition such as fever and infection. The elevated level of serum creatinine (5 mg/dl) and BUN (32 mg/dl) correlated with the ultrasonogram of kidney indicating renal failure, which occurred due to herpes viral infection. Hypocalcemia (7 mg/dl) might be due to renal insufficiency. The serum glucose level was within normal range. The increased level of serum prostatic acid phosphatase (4.5U/L) was in accordance with report of Wadstrom *et al.* (1984). The reason for that was probably due to degeneration of prostatic secretory cells induced by prostatitis. The low specific gravity and proteinuria correlates well with the renal failure.

5.3.2.3.4. Treatment

The treatment with the enrofloxacin alleviated the prostatitis only moderately. The vesicular lesions of eye and bulbus glandis were cured completely by althrocin tablets and acyclovir ointment. Anvik (1991) reported that the treatment regimens for CHV infection have not been documented, but King (1984) reported that drugs such as acyclovir were used to lessen the severity of herpes virus infections. Further clinical evaluation could not be conducted as the animal was not presented afterwards.

5.3.3. Prostatic Abscess

Prostatic abscesses developed as a result of bacterial contamination spreading from another part of the urinary tract, by hematogenous route, or from cysts that become secondarily infected as opined by Barsanti and Finco (1986) and Basinger *et al.* (1993).

According to Baker and Lumsden (1999), prostatic abscesses developed either after fusion of small areas of infection within the gland or after infection of prostatic cysts.

Prostatic abscess was reported in 6.7 % (one case) of the total prostatic diseases diagnosed in the present study. Krawiec and Heflin (1992) reported that out of 33 dogs diagnosed with prostatitis, four (12.12 per cent) had abscesses. Kiren (2008) recorded 6.7 % of dogs affected with prostatic disorder as prostatic abscess. In the present study, the age of affected animal was 6 year. Parker (1975) reported a case of prostatic abscess in a 4 year old springer spaniel.

5.3.3. 1. Clinical Signs

Major clinical signs such as anorexia, vomiting, dyschezia and melena were the characteristic sign of the disease while, Kutzler and Yeager (2005) and Smith (2008) reported clinical signs of prostatic abscess as anorexia, fever, depression, gait abnormalities, tenesmus, dysuria and caudal abdominal pain. Uremic smell from mouth observed in this case might be due to concurrent renal failure, which is ascertained by increased serum creatinine, ultrasonographic and autopsy findings.

Clinical signs were often vary depending on the size of the abscess and whether the infection became systemic or not as suggested by Smith (2008).

In the present case, the prostate was asymmetrically enlarged, movable, soft and painful on palpation. Boland *et al.* (2003) reported that the rectal examination of dog with prostatic abscess revealed asymmetrical enlargement of

prostate and pain on palpation. Palpable fluctuant areas were present in prostatic abscess as reported by Davidson (2003).

5.3.3. 2. Ultrasonography

Multiple hypoechoic cavitary lesions with irregular borders, containing mixed echogenic or flocculent fluid were visualized in the prostatic parenchyma. Irregular border with blackish necrotic areas on surface of prostate with purulent fluid inside were observed during autopsy. The typical prostatic abscess appeared as hypoehoic cavitary lesions with irregularly defined margins. The fluid within the lesion might have mixed echogenisity or a flocculent appearance as opined by Boland *et al.* (2003). Ultrasonography of abscess revealed focal hypoechoic to anechoic areas in prostatic parenchyma and prostatic abscess often had irregular borders and the surrounding parenchyma had decreased echogenicity (Davidson, 2003). The prostatic length, width, depth and volume in the present case were higher than the normal value, 3.4 ± 1.1 cm, 2.8 ± 0.8 cm, 3.3 ± 0.9 cm and 18.9 ± 15.5 cm^3, which was reported by Ruel *et al.* (1998). These variations may be due to variations in degree of severity of case under the study.

Enlarged kidneys with indistinct corticomedullary junction and thickened cortex were in correlation with the increased serum creatinine level and irregular kidney with necrotic area observed during autopsy.

Splenomegaly observed in ultrasonography, was confirmed during autopsy. Earlier workers did not report splenomegaly in prostatic diseases. Splenomegaly could be due to septicemia or localized peritonitis. Jones (1997) reported that infectious process such as septicemia causes splenic enlargment due to congestion as well as an influx of neutrophils. Chronic splenitis was seen in many infectious diseases and might lead to diffuse enlargment / irregular (nodular) enlargment depending on the offending organisms. Wallace (2001) reported that a subset of dogs with bacterial prostatitis would develop bacterial abscesses of the prostate gland. These infections could be life threatening, because the abscess may result in septicemia, endotoxemia, and localized

peritonitis. The condition had a high mortality rate, because septicemia, disseminated intravascular coagulation, and hypoalbuminemia from peritonitis could happen.

5.3.3. 3. Prostatic Fluid Evaluation

The fluid aspirated from the abscess was purulent in nature. Cytopathological studies revealed large number of degenerated neutrophils and bacteriae as reported by Boland *et al.* (2003). The culture of prostatic fluid revealed presence of *E. coli,* which was sensitive to tetracycline, enrofloxacin and chloramphenicol.

Boland *et al.* (2003) reported that 62.5 % of dogs diagnosed with prostatic abscess were positive for *E. coli.*

5.3.3. 4. Clinical Pathology

Leucocytosis (25500/cu.mm) with neutrophilia (88%) and lymphopenia (12%) was observed in this case whereas Boland *et al.* (2003) reported that hematology and serum biochemical result were variable in prostatic abscess and the most common abnormalities included mild to moderate leucocytosis, lymphopenia or both. Mild anemia was also reported in hematology. Hyperglobulinemia, hypoglycemia and azotemia were observed in the present study. Davidson (2003) also reported similar findings.

Serum total acid phosphatase and prostatic fraction were elevated. The reason for this might be due to degenerative process of the prostatic tissue by the abscess formed as opined by Corazza *et al.* (1994) who reported that prostatic acid phosphatase was liberated during degeneration which was induced by dihydrotestosterone. The serum testosterone was within the normal range.

Proteinuria and low specific gravity might be due to renal failure associated with prostatic abscess, and this was confirmed by increased serum creatinine value, ultrasonographic and autopsy findings.

E. coli was isolated from both prostatic fluid and urine. Antibiotics such as tetracycline, enrofloxacin and chloramphenicol were sensitive to both samples whereas, amoxicillin in the case of urine sample only.

5.3.3. 5. Treatment and Response

The dog was treated with enrofloxacin @ 10 mg/kg b.wt. IV, fluids, antiemetics and proton pump inhibitor. But the animal died on the second day of admission due to severe septicemia and so the effectiveness of therapy could not be evaluated. All the cases of prostatic abscess may not respond well and more elaborate procedures such as abscess drainage and prostatectomy may be necessary to save the life of patient.

5.3.3. 6. Autopsy

Both the lobes of prostate were enlarged and filled with pus, which was in correlation with ultrasonographic findings. The purulent fluid in urinary bladder and echymotic patches in bladder mucosa were suggestive of cystitis, which could be correlated with culture of urine.

In the present case, the measured prostatic weight during autopsy was higher than that of normal prostatic weight for that particular body weight. Bloom (1954) opined that weight and size of the prostate gland was variable depending on the age and breed. The normal ratio recorded varied from 0.1 to 0.7 g with an average of 0.4 g of prostate to 1 kg of body weight. Where as Lowseth *et al.* (1990) reported that prostatic diseases were more common in dogs over six years and found that the weight and volume of the prostate increased with age as 1.08 ± 0.22g/kg body weight at 3 years of age increasing to 2.64 ± 0.37g/kg at 14 years of age.

Kidneys were irregular with inflammatory and necrotic areas. Spleen was enlarged and had one nodular lesion, which might be due to chronic splenitis as reported by Jones (1997).

Histopathological examination of prostate gland revealed abscess wall formed by inflammatory granulation tissue, acini with necrotic material, lymphocytes, plasma cells and polymorphs infiltration into the stroma that were all could be correlated with ultrasonographic findings and prostatic fluid cytology.

5.3.4. Prostatic Neoplasia

In the present study, fibroadenoma with paraprostatic pseudocyst in a German shepherd intact male dog aged 3.5 years and papillary adenoma in an eight-year-old cocker spaniel dog were diagnosed.

The reported prostatic neoplasia included squamous cell carcinoma (Leib *et al.*, 1986), transitional cell carcinoma of ductal epithelium, prostatic adenocarcinoma (Barsanti and Finco, 1989), prostatic adenomas, leiomyomas, fibromas and sarcomas represented less than 10% of all neoplasms as reported by Turrel (1989), leiomyosarcoma and lymphoma (Mainwaring, 1990).

Environmental chemicals that have hormonal activity (environmental estrogens) and those which act as endocrine disrupters are theorized to cause preneoplastic or overly neoplastic changes in many tissues, including reproductive tissues (Johnston *et al.* 2000) and this might be responsible for increased incidence of neoplasm in dogs.

5.3.4.1. Fibroadenoma with Paraprostatic Pseudocyst

A case of fibroadenoma with paraprostatic pseudocyst was reported in a 3 .5 year old German shepherd.

Paraprostatic cyst was one of the rarest prostatic diseases in dogs as reported by Krawiec (1994). The present case was a fibroadenoma with paraprostatic pseudocyst and the data from the literature regarding fibroadenoma with paraprostatic pseudocyst is scarce. Hoffer *et al.* (1977) reported that the most common cyst biopsied and submitted was the paraprostatic pseudocyst. The term

pseudocyst was used since the inner lining was seldom lined by epithelium and therefore impossible to determine their origin. The present case was reported at 3.5 years of age. Whereas, Krawiec and Heflin (1992) reported that paraprostatic cyst was usually seen in middle and old aged dogs.

5.3.4.1.1. Clinical signs

Clinical signs were anorexia, pyrexia, intermittent urination, haematuria, purulent urethral discharge and dyschezia. Davidson (2003) reported similar observations in prostatic neoplasia. The purulent urethral discharge and elevated temperature in the present case might be due to secondary infection of the prostate which developed due to prevention of normal defense mechanism such as normal urine flow during micturition, by the prarprostatic cyst as reported by (Dorfman and Barsanti, 1995). Girard and Despots (1995) reported signs of urinary tract disease (dysuria and hematuria) and fecal tenesmus in a Doberman with mineralized paraprostatic cyst.

The prostate was warm, firm, slightly irregular, immobile, severely enlarged and painful on palpation. Johnston *et al.* (2000) also found similar observation in prostatic neoplasia.

5.3.4.1.2.. Ultrasonography

On ultrasonography, two anechoic cavities containing mixed echogenic substances with irregular thickened borders were seen dorsolateral to the prostate, which was then diagnosed as paraprostatic cyst in the autopsy. Histologically, with gomori's trichrome one step staining method it was confirmed as a combination of fibroadenoma of prostate and paraprostatic pseudocyst with inflammation.

Diffused hyperechoic changes were observed in the remaining prostatic parenchyma. Feeney *et al.* (1987) reported either focal or diffused hyperechoic

pattern in prostatic adenocarcinoma, similar to that observed in bacterial prostatitis or benign hypertrophy.

The mixed echogenic substances in the cavities were confirmed as purulent fluid during autopsy. The ultrasonographic dimensions of prostate revealed prostatomegaly. Finn and Wrigley (1989), Bell *et al.* (1991) and Klaunsner *et al.* (1995) reported prostatomegaly, focal to diffuse hyperechoic areas and irregular prostatic contour in prostatic neoplasia.

5.3.4.1.3. Prostatic fluid evaluation

Prostatic fluid aspirated was serosanguinous to brown coloured and cytological evaluation revealed distorted, damaged cells resembling neutrophils and prostatic cells with anisocytosis. Pseudomonas organism was identified on cultural examination of prostatic fluid. The same pathogen could be cultured from urine also. Hence, it could not be proved that the pathogen isolated from prostatic fluid originated certainly from the prostate gland.

5.3.4.1.4. Clinical pathology

Mild anemia with severe leucocytosis and neutropenia was found in the present study. Davidson (2003) reported leucocytosis with neutrophilia and regenerative or nonregenertive anemia in prostatic neoplasia. The severe neutropenia might be due to initial stage of an acute bacterial infection or due to reduced mature granulocyte survival due to excessive destruction or utilization which is seen in overwhelming bacterial infection and hypersplenism (Benjamin, 2005). The above changes could be correlated with ultrasonographic findings and autopsy.

Hypoalbuminemia with hyperglobulinemia, azotemia, hypocalcemia and hypoglycemia could be observed in the present study. Davidson (2003) reported increased alkaline phosphatase, varying levels of serum calcium,

hypoalbuminemia and azotemia in prostatic neoplasia. Hyperglobulinemia might be due to acute inflammatory process (Benjamin, 2005).

Normal level of serum total acid phosphatase with prostatic fraction was found in the present study. Corazza *et al.* (1994) opined that low serum concentrations of total acid phoshatase and prostatic acid phosphatase did not rule out prostatic adenocarcinoma in the dog, but elevated concentrations in prostatic adenocarcinoma compared to BPH could be the useful criteria for differentiating the tumor from BPH. Normal serum testosterone level was observed.

The low specific gravity and proteinuria might be due to renal failure, which occurred after obstruction of ureters or urethra by tumor mass. Leucocyturia was present.

5.3.4.1.5. Treatment and response

The dog was treated with enrofloxacin since it was initially diagnosed as prostatitis. But, there was only a temporary improvement. The treatment with chloramphenicol was then started based on culture and sensitivity. Treatment for renal failure was also given. The dog did not respond to treatment and succumbed to death as it was progressed to advanced renal failure, which was confirmed during autopsy.

Treatment of canine prostate cancer was generally unrewarding. Castration resulted in involution of the non-neoplastic portion of the prostate, but did not affect progression of the neoplastic disease. Surgical resection usually was not recommended, because the disease was not usually diagnosed at an early stage, and prostatic surgery often resulted in urinary incontinence (Johnston *et al.*, 2000).

5.3.4.1.6. Autopsy

The two moderately thick walled cystic structures located dorsal and craniolateral to the prostate was confirmed as paraprostatic pseudocyst by

histopathological study. Adenomatous proliferation of fibrous tissue and diffuse infiltration of lymphocytes and plasma cells could be identified in the prostate as observed by Hall *et al.* (1976). The innerlining of the cyst was seldom lined by epithelium and had granulation tissue proliferation. The wall was chiefly composed of fibrous tissue with the outer lining of mesothelium. Similar observations were made by Hoffer *et al.* (1977) in dog. Since the inner wall was devoid of continuous epithelium, its origin could not be found out and hence the cyst was termed as pseudocyst as opined by Hoffer *et al.* (1977). The presence of fibrous tissue proliferation was confirmed by gomori's trichrome one step method (Luna, 1968).

The paraprostatic cyst observed in this case, probably resulted from anomalous development and this might be remanant of uterus masculinus or of prostatic origin as reported by Barsanti and Finco (1995). Both the prostate and paraprostatic cyst had infiltration of lymphocytes and plasma cells indicating concurrent chronic inflammation as opined by Kustritz and Klausner (2000).

5.3.4.2. Pappillary Adenoma

The case was reported in an eight year old Cocker spaniel. Bell *et al.* (1991) and Krawiec and Heflin (1992) reported that the average age of dogs with prostatic neoplasia was about 9 year.

Durham and Dietze (1986) classified prostatic adenocarcinoma into alveolar papillary, acinar and organod (rosette) types. Alveolar papillary pattern was the most frequently observed histologic type and consisted of papillary ribbons of epithelial cells that projected into various sized alveolar like spaces surrounded by connective tissue. The individual cells had large cytoplasmic vacuoles with proteinacious droplets and mucoid material that displaced the nucleus and resembled a signet ring.

5.3.4.2.1. Clinical signs

Clinical signs such as anorexia and dyschezia reported in this case were also reported by Parry (2007). Vomiting, oliguria and melena observed in this case can be correlated with increased serum creatinine level, renal ultrasonography and autopsy findings of kidney. Benign prostatic hypertrophy and prostatic tumors were the two diseases causing obstructive nephropathy in human beings (Gloor and Torres, 2007).

Abdominal thudding observed might be due to anemia associated with renal failure.

5.3.4.2.2. Digital rectal palpation

The prostate gland was isothermic, intra abdominal, smooth, firm, symmetrically enlarged, movable and painful on per rectal palpation. While, the prostate was firm, irregular, immobile, asymmetrical, and painful on rectal examination in dogs with prostatic neoplasia as reported by Johnston *et al.* (2000) and the very large the prostate might be palpable in the caudal abdomen. (Davidson, 2003).

5.3.4.2. 3.Ultrasonography

Ultrasonographically, prostate lobes were symmetrically enlarged with focal hyperechoic areas. Ultrasonographic appearance of the neoplastic canine prostate included prostatomegaly, mineralization of the parenchyma, presence of focal to diffuse hyperechoic areas, and irregular/ discontinuous prostatic contour (Finn and Wrigley, 1989; Bell *et al.*, 1991 and Klaunsner *et al.*, 1995), Where as Feeney *et al.* (1987) reported that changes in canine prostatic ultrasonography were common to variety of prostatic diseases as prostatic adenocarcinoma produced either a focal or diffuse hyperechoic pattern similar to that observed with bacterial prostatitis or benign hypertrophy. Hyperechoic changes in kidneys could be correlated with increased serum creatinine level and autopsy finding.

5.3.4.2.4. Prostatic fluid evaluation

The exfoliated clumps of cuboidal cells with hyper chromatin observed in the prostatic fluid cytology could be correlated with the histopathology of the prostate, which revealed adenomatous proliferation of lining cells.

5.3.4.2.5. Clinical pathology

The relevant observations in hemogram were reduced hemoglobin percentage, reduced erythrocytes and PCV. Davidson (2003) reported regenerative or nonregenerative anemia in prostatic neoplasia. Elevated serum creatinine, blood urea nitrogen and hypocalcemia were reported in this case. Davidson (2003) reported varying levels of serum calcium and azotemia in prostatic neoplasia. Serum acid phosphatase with prostatic fraction was 10.6 U/L and 3.8 U/L. Corazza *et al.* (1994) reported the serum concentration of total acid phosphatase and prostatic acid phosphatase in prostatic adenocarcinoma as 21.4 ± 18.9 and11.9 ± 12.8 U/L respectively and the author also opined that prostatic acid phosphatase should not be considered as a specific marker for prostatic disease, since its levels were higher in gastrointestinal tract cancer, BPH, inflammation of the prostate and after prostatic massage. Serum testosterone was within normal range. Haematuria might be due to urethral obstruction and the low specific gravity might be due to renal failure.

5.3.4.2.6. Treatment and response

The case was treated with fluids and enrofloxacin but the animal succumbed on the next day.

5.3.4.2.7. Autopsy

On autopsy, both the lobes of prostate were symmetrically enlarged with round shape. Histopathology revealed adenomatous proliferation of the lining cells that forms papillary projections into lumen which is similar to the observation by Durham and Dietz (1986).

The present study suggests that disorders of prostate are more than visually expected because of many of the disease may not be manifested unless it interferes with normal functioning of various systems. It was documented that sexually intact dogs of larger breeds like German shepherd and Doberman had a higher than expected prevalence. Most common prostatic disease was bacterial prostatitis followed by benign prostatic hyperplasia. In human beings by age of 51-60, 50% have pathologic BPH and incidence of BPH approaches 100% in dog population by 7 to 8 years of age. So the dog has been used as a model to study the development of this disease in human beings. Canine prostate cancer has also served as an important model for studying human prostatic cancer. The poor prognosis of prostatic cancer in dogs can be due to its late diagnosis as there are no serum biochemical markers such as PSA of human beings. However biomarkers such as TAP and PAP in serum could be useful criteria for the diagnosis of prostatic neoplasms. Ultrasonography and prostatic fluid cytology are the major diagnostic tools for other prostatic diseases such as BPH and prostatitis and the treatment response is dependent on the dose, duration of treatment and the concurrent renal failure.

Summary

6. SUMMARY

Dogs presented to the Veterinary hospitals, Kokkalai and Mannuthy with clinical signs suggestive of prostatic diseases such as constipation, dysuria, tenesmus and urethral discharge were subjected to detailed investigations. Based on various diagnostic techniques prostatic diseases were confirmed in15 cases. The average age of affected animals was 6.7 years with a range of 7½ month – 10 years. The occurrence of prostatic diseases in this study was 75%. The incidence of disease was more in German shepherd (46.6%).

The major clinical signs noted in the study were constipation, inappetance, purulent urethral discharge, dysuria, vomiting, pyuria, hematuria, pyrexia and caudal abdominal pain.

After detailed investigation using various diagnostic techniques such as, transrectal digital palpation, abdominal ultrasonography, prostatic fluid evaluation and clinico pathology the cases were classified as benign prostatic hyperplasia with prostatitis (n=3), benign prostatic hyperplasia and associated hernia (n=2), acute prostatitis (n=5), suppurative prostatitis (n=1), prostatitis associated with herpes viral infection (n=1), prostatic abscess (n=1), fibroadenoma with paraprostatic pseudocyst (n=1) and papillary adenoma (n=1).

Elevated rectal temperature and congested mucous membranes were observed in acute prostatitis, suppurative prostatitis, prostatitis associated herpes viral infection, BPH with prostatitis and fibroadenoma with paraprostatic pseudocyst, which might be due to septicemia associated with prostatitis.

Smooth, movable, enlarged, isothermic and painless prostate could be found on perectal digital palpation of dogs suffering from BPH with prostatitis and BPH with hernia. Warm and painful prostate in the other cases may be due to severe concurrent inflammation.

The ultrasonographic pattern of prostate gland was different for different diseases. Prostatomegaly with intraparenchymal cystic areas were the major ultrasonographic image of BPH. Whereas, diffused increase in echogenisity of prostatic parenchyma was noted in prostatitis. Multiple hypoechoic cavitary lesions with irregular borders were the characteristics of prostatic abscess. In fibroadenoma with paraprostatic pseudocyst, two anechoic cavities with irregular thickened borders were found lateral to the prostate. Symmetrical enlargement with focal hyperechoic areas was observed in pappilary adenoma. Splenomegaly was observed in most of the cases. Indistinct corticomedullary junction of kidney and increased serum creatinine was observed in 8 cases.

Prostatic fluid cytology of prostatitis revealed large number of neutrophils including bacteria- laden neutrophils. Large number of degenerated neutrophils and bacteria was present in prostatic abscess. Hyperplastic round to oval prostatic epithelial cells with reticulated chromatin and unaltered nucleus cytoplasmic ratio was observed in BPH. Whereas, damaged and distorted cells in fibroadenoma with paraprostatic pseudocyst and clumbs of columnar cells in papillary adenoma was observed. Prostatic fluid culture from dogs with acute prostatitis, suppurative prostatitis and prostatic abscess revealed *E. coli* organism. Staphylococcal organism was cultured from the prostatic fluid of dog with prostatitis associated herpes viral infection and pseudomonas from fibroadenoma with paraprostatic pseudocyst.

Hematobiochemical values were unrewarding to diagnose prostatic diseases. However, leucocytosis with neutrophilia was observed in prostatitis and left shift in acute prostatitis. Serum creatinine and BUN was elevated in cases which had renal failure developed secondary to prostatic disorders. Even though, serum prostatic acid phosphatase and total acid phosphatase was elevated in prostatic diseases; it could not be used as marker for prostatic diseases since it was elevated in other disease condition also. There was no significant difference between acid phosphatase values of different prostatic diseases. The serum

testosterone was elevated only in BPH associated hernia. The serum samples of all dogs with prostatitis were negative for brucella antibody test.

Urinalysis revealed pyuria, proteinuria and presence of blood pigments in varying degrees in different cases. The specific gravity of urine was low in dogs with secondary renal failure. Culture of urine sample in some cases of prostatitis revealed the presence of similar organism as that of prostatic fluid culture. This might be due to ascending grade infection of the prostate from the lower urinary tract.

Treatment regimen was different for different prostatic disease. The BPH could not be successfully treated with 5-∝ reductase inhibitor @ 1mg/kg. Acute prostatitis without advanced renal failure could be treated successfully with enrofloxacin @ 10 mg/kg. The prostatic abscess and suppurative prostatitis did not respond to the antibiotic treatment and supportive fluid therapy. This might be due to advanced renal failure that developed secondary to prostatic inflammation. The prostatic neoplasia could not be cured. Since the most of the above mentioned parameters was not specific for prostatic diseases, the role of canine prostate specific arginine esterase may be studied in detail for further investigation.

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* Originals not considered

CLINICO-BIOCHEMICAL AND ULTRASONOGRAPHIC EVALUATION OF DISEASES OF PROSTATE IN DOGS

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ABSTRACT

The study on "Clinico - biochemical and ultrasonographic evaluation of diseases of prostate in dogs" was conducted in fifteen dogs. The parameters studied were signalment, history, clinical signs, clinical observations, perectal digital palpation of prostate, ultrasonography of prostate gland, prostatic fluid cytology, hematology, serum biochemistry, urinalysis, and response to treatment, autopsy and hisopathology.

The prostatic disorders studied were classified into benign prostatic hyperplasia with prostatitis, benign prostatic hyperplasia and associated hernia, acute prostatitis, suppurative prostatitis, prostatitis associated with herpes viral infection, prostatic abscess, fibroadenoma with paraprostatic pseudocyst and papillary adenoma.

The major clinical signs noted in the study were constipation, inappetance, purulent urethral discharge, dysuria, vomiting, pyuria, hematuria, pyrexia and caudal abdominal pain. Perectal palpation revealed enlarged, smooth and isothermic prostate in BPH and prostatic neoplasia where as, warmness could be felt in prostatitis. Ultrasonographically, prostatomegaly was the most common feature. Intraparenchymal cyst formation in BPH, diffused increase in echogenisity in prostatitis, focal hyperechoic areas in prostatic neoplasia, multiple hypoechoic cavitary lesions with irregular borders in prostatic abscess were also noted. Prostatic fluid cytology revealed large number of neutrophils including bacteria- laden neutrophils in prostatitis, degenerated neutrophils and bacteria in prostatic abscess, hyperplastic round to oval prostatic epithelial cells with reticulated chromatin and unaltered nucleus cytoplasmic ratio in BPH.

Hematological changes such as neutrophilia was observed in prostatitis and left shift in acute prostatitis. The serum creatinine and BUN was elevated in dogs with secondary renal failure. The level of acid phosphatase was elevated but there was no significant difference between different prostatic diseases. Serum testosterone was within normal range except in BPH where it was slightly elevated. The common abnormalities observed on urinalysis were pyuria, low specific gravity, proteinuria and presence of blood pigments.

It was concluded from this study that prostatic disease was a major problem in old aged male dogs and was one of the major etiology for renal failure in them. Specific therapy with accurate dosage will cure prostatitis and BPH provided there is no advanced renal failure. This study also revealed the fact that ultrasonography is an excellent diagnostic tool for diagnosing prostatic disorders.

Appendix

PROFORMA

Case No. / Sl No. **Date**

1. Name And Address Of The Owner :
2. Detatils Of The Animal :
Breed :
Age :
Colour :

3. Clinical History

Date	Diseases encountered in the past	Treatment adopted

4. General Clinical Examination :

5. Examination Of Urinary System :

6. Clinical Observation

a) Clinical Data

1. Respiration rate (per minute) :
2. Pulse (rate per minute) :
3. Temperature :
4. Mucous membrane : (pale / congested/ icteric)
5. Lymph nodes :
6. **Transrectal digital examination** :

b) Clinical Signs

(Present / Absent)

1. Lethargy :
2. Vomiting :
3. Anorexia :
4. Haematuria :
5. Dysuria :
6. Stranguria :
7. Tenesmus :
8. Dyschezia :
9. Rear limb weakness :
10. Urethral discharge :

7. Results of Special Examination

1. Ultrasonography Findings

i) Prostate :

ii) Kidney :

2. Haemato-biochemical Findings

Sl no	Parameters	Result
		Day 1
1.	Hb (gm/dl)	
2.	VPRC %	
3.	TLC(10^3/cu.mm)	
4.	DLC	
5.	Neutrophils (%)	
6.	Lymphocytes (%)	
	Eosnophils (%)	
	Monocytes (%)	
	Basophils (%)	
7.	**Serum Analysis**	
	Acid Phosphatase (IU/L)	
	Glucose (mg/dl)	
	Calcium (mg/dl)	
	Total Protein (g/dl)	
	Albumin (g/dl)	
	A:G Ratio	
	Serum Creatinine (mg %)	
	Testosterone (ng/ml)	

3. Urinalysis

pH :

Specific gravity :

Blood pigments :

Protein :

Sediments :

4. Prostatic Fluid Cytology :

5. Fine Needle Aspiration Biopsy results (if necessary) :

6. Diagnosis :

7. Treatment :

8. Response to treatment :

SIGNATURE OF THE CHAIRMAN **SIGNATURE OF THE STUDENT**