UTERO-OVARIAN AND ENDOCRINE PATHOLOGY IN GOATS

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Thesis submitted in partial fulfilment of the requirement for the degree of

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DECLARATION

I hereby declare that the thesis entitled **"Utero-ovarian and endocrine pathology in goats"** is a bonafide record of research work done by me during the course of research and that this 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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CERTIFICATE

Certified that this thesis, entitled **"Utero-ovarian and endocrine pathology in goats"** is a record of research work done independently by **Thomas K Thomas,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, associateship or fellowship to him.

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Introduction

1. INTRODUCTION

Economic prosperity of any country is closely related to her animal wealth. One of the greatest requirements of our country today is to increase and improve animal production thus opening up a new vista of freedom from the fear of hunger and malnutrition for our growing population.

India ranks second in world in goat population and has 128 million goats as per 2003 census and consists of 25.64% of total livestock population of India and 20% of total global population. Goats have a high survival rate under critical environmental conditions and can simultaneously produce a variety of products for the family and the market like meat, milk, skin and fibre.

Reproductive efficiency is a major factor in ensuring economic livestock production. Increasing production performance will depend upon the successful measures taken for eliminating infertility brought about by one or more factors. Improving reproductive efficiency begins with an understanding of the normal reproductive process and the problems which can befall it.

The recognition of goat as "poor man's cow" for the landless labour and socially backward members of the society coupled with its capacity to make use of vegetation under diverse ecological situations, make this animal distinctly superior among ruminants in tropical countries. Goat is a multi functional animal that plays a significant role in the economy and nutrition of landless, small and marginal farmers in the country. Since India has the second largest goat population in the world, goats have a significant role to play in the national economy. Goat rearing is an enterprise which has been practiced by a large section of population in rural area in pastoral and agricultural subsistence of the society. The unorganized backyard system which was predominant in Kerala is now changing and organized goat farming, on commercial basis, is gaining more and more importance.

Even though considerable improvement in production potential has been effected by scientific breeding and improved husbandry practices, impaired fertility continues to remain a deterrent to progressive goat farming. Fertile gametes and disease free environment for conception are prerequisites for delivery of healthy young ones. The adoption of new breeding technologies has brought about a significant increase in the productive performance of animals.

Abnormalities of the female genital system may be expressed in the categories such as congenital abnormalities, degenerative alterations, inflammatory processes or neoplastic conditions. Few workers in India have carried out detailed investigations on the reproductive disorders of goat (Prabakaran and Raja, 1972; Singh, 1973; Chand and Chauhan, 1978; Sharma, 1980; Kadu and Kaikini, 1988; Ijapure *et. al.*, 2002 and Khattun *et al.*, 2007). Pathological conditions of the reproductive system could adversely affect the production performance of the goats.

The influence of endocrine glands on the reproductive pathology has not been well correlated in goats. Many diseases of the endocrine glands are characterized by dramatic functional disturbances and characteristic clinicopathological alterations affecting one or several body systems. Although endocrine diseases of animals are frequently encountered in veterinary practice, they are only scarcely reported in literature and therefore they may go undiagnosed by the veterinary practitioner.

Lesions of the endocrine glands are sometimes associated with gonadal changes. Endocrine glands such as pituitary, thyroid and adrenal could influence the reproductive function in various domestic animals. Reddy (1982) reported a significant reduction in the relative weight of ovaries in all hypothyroid goats with the ovaries containing only a few scattered developing primary and secondary follicles. There was also a significant reduction in the relative weight of the uterus with thin muscular layer and few inactive mucosal glands revealing no secretory activity.

A systematic study on various affections/diseases of female genital tract of goats is of paramount importance for proper diagnosis and for adopting suitable preventive and control measures. Hence a detailed investigation is warranted to elucidate the pathology of utero-ovarian disorders seen in goat population to evolve better managemental practices. The present investigation was aimed to:

- Ascertain the incidence, nature and magnitude of utero-ovarian pathology in goats.

- To correlate the utero-ovarian pathology with endocrine lesions.

Review of Literature

2. REVIEW OF LITERATURE

The incidence of various disease conditions of female genital tract of goats recorded by various workers ranged from 1.9-21.2 per cent in goats (Lyngset, 1968; Singh, 1973; Chand and Chauhan, 1978; Sharma, 1980; Kadu and Kaikini, 1988; Sattar and Khan, 1988; Timurkaan and Karadas, 2000; Ijapure *et al.*, 2002; Khatun *et al.*, 2007). In the case of sheep, this varied from 1.6-44 percent (Prabhakaran and Raja, 1972; Rao and Abdullakhan, 1974; Chand and Chauhan, 1978; Long, 1980; Sharma, 1980; Singh *et al.*, 1982; Winter and Dobson, 1992; Smith *et al.*, 1998; Aliasghar and Saad, 2007; Regassa *et al.*, 2008). Pathological lesions or diseases of the reproductive system in sheep and goat were in most cases found to be similar to those in cattle (McEntee, 1990).

2.1 OVARY

Various pathological lesions in the ovaries were found in 32 (3.36%) genitalia out of 950 genitalia examined (Ramachandran, 1980). Kadu and Kaikini (1988) observed 47 (4.42%) cases of ovarian disorders out of the 1057 genital organs examined. Hatipoglu *et al.* (2002) observed 58 (5.4%) cases of ovarian disorders in cows out of the 1113 cases examined. Of the 25.8% of affected ewes in southern Iran, the highest rate of abnormality were in the ovaries (20.1%) and the predominant lesions in ovaries were ovarobursal adhesions (11.5%) and parovarian cysts (7.6%) (Aliasghar and Saad, 2007). Regassa *et al.* (2008) reported that the percentage of ewes with ovarian cysts, ovarobursal adhesions and combination of both were 4.3%, 7.6% and 1.7%, respectively.

2.1.1 Ovarian and Parovarian cysts

2.1.1.1 Ovarian cyst

Incidence of ovarian cysts has been reported to vary from 0.68-2.4 per cent in goats (Lyngset, 1968; Singh, 1973; Chand and Chauhan, 1978; Sharma, 1980; Kadu and Kaikini, 1988; Sattar and Khan, 1988; Timurkaan and Karadas, 2000; Ijapure *et al.*, 2002;

Khatun *et al.*, 2007) and 0.24-6.0 per cent in sheep (Prabhakaran and Raja, 1972; Rao and Abdullakhan, 1974; Chand and Chauhan, 1978; Long, 1980; Sharma, 1980; Singh *et al.*, 1982; Winter and Dobson, 1992; Smith *et al.*, 1999; Regassa *et al.*, 2008).

Singh (1973) found that most of the cysts were on the right side of the ovary in case of goats and these were classified into two types viz. follicular cyst and luteal cyst. Follicular cysts had fibrous connective tissue layer enclosing a few granulosa cell layers along with cellular debris and acidophilic material in the lumen. In the case of the luteal cysts, connective tissue surrounding cystic cavity was chiefly composed of inner loosely arranged connective tissue. Luteal cell layers had variable amounts of connective tissue stroma.

Rao and Abdullakhan (1974) reported that there were 0.24 per cent cases of polycystic ovaries in sheep. Incidence of follicular cysts in the ovaries of sheep and goats was described by Sharma (1980) and these were described as slightly elevated oval structures varying in size from 1-3.5 cm in diameter filled with a clear fluid. Ovaries with such cysts had no functional corpus luteum. Histologically, the cyst wall was composed of thick fibrous tissue capsule and a few granulosa cells in the cyst walls and/or cyst cavities. Few small cystic graffian follicles having varying degrees of degenerative changes were also seen.

Sattar and Khan (1988) classified the ovarian cysts into follicular cysts, luteal cysts and cystic corpora lutea in goats.

Alosta *et al.* (1998) reported that follicular cysts were recorded in 7 (0.8%) cases out of 870 cases of ewes examined, of which 2 (0.2%) were bilateral and 5 (0.6%) were unilateral (3 on the right and 2 on the left). The cysts did not have an ovulation point and were lined by granulosa cells; in some cases the theca layers were partially luteinized. Luteal cysts were diagnosed in 8 (0.9%) ewes: one (10.1%) ewe had a cyst on both the ovaries, while the other seven (0.8%) had unilateral cysts (4 on the right and 3 on the

left). Those cysts, which did not have an ovulation papilla, were lined with a connective tissue layer and had a luteinized theca.

Vanholder *et al.* (2006) reported that cystic ovarian follicles develop when one or more follicles fail to ovulate and subsequently do not regress but maintain growth and steroidogenesis. They are defined as follicle-like structures, present on one or both ovaries, with a diameter of at least 2.5 cm (in case of cows) for a minimum of ten days in the absence of luteal tissue. Macroscopically, cysts can be subdivided into follicular and luteal cysts, which were considered to be different forms of the same disorder. Luteal cysts were believed to be follicular cysts in later stages. Follicular cysts secrete little or no progesterone while luteal cysts clearly do. The many intermediate forms with limited or extensive luteinisation do not allow for a clear identification of the cyst type.

Regassa *et al.* (2008) reported that out of 3275 non-pregnant reproductive tracts of ewes examined, ovarian cysts were observed in 4.3% of the ewes. The prevalence of follicular and luteinized cysts did not differ between the right and left ovaries. The occurrence of bilateral cysts was lower than that of unilateral cysts either on the right or left ovary. Ovaries with follicles of diameter more than 10 mm were considered cystic. Based on the appearance the cysts were classified into follicular and luteinized cysts. Follicular cysts were tense, thin-walled follicles distended with pale yellow fluid while luteinized cysts were thick-walled follicles lined with connective tissue layer containing more amber or dark yellow fluid.

2.1.1.2 Parovarian cysts

Parovarian cysts have been recorded by various workers in goat and sheep. The incidence varied from 0.34-1.79 per cent in goats (Singh, 1973; Chand and Chauhan, 1978; Sharma, 1980; Kadu and Kaikini, 1988) and 0.02 per cent to 17.03 per cent in sheep (Prabhakaran and Raja, 1972; Rao and Abdullakhan, 1974; Chand and Chauhan, 1978; Long, 1980; Sharma, 1980; Biolatti *et al.*, 1984; Kadu and Kaikini, 1988; Winter and Dobson, 1992).

Singh (1973) found that in goat parovarian cysts were located mostly in the mesosalpinx. The cysts were thin walled and filled with clear fluid.

Sharma (1980) described that parovarian cysts were spherical, tense (0.5 to 1.5 cm in diameter), grayish-white fluid filled structures situated at different locations such as mesovarian, mesosalpinx and tips of fimbriae of fallopian tube. Microscopically, the cysts were lined with low cuboidal cells supported by thick fibrous tissue capsule.

Alosta *et al.* (1998) reported that parovarian cysts were found in 1.8% of the ovine genital tracts. They were generally single, but double and triple cysts were also recorded and most of them (68.8%) were on the right side.

2.1.2 Oophoritis

Oophoritis has been rare in occurrence and reported in isolated cases in goats and sheep (Lyngset, 1968; Prabhakaran and Raja, 1972; Singh, 1973; Chand and Chauhan, 1978; Sharma, 1980; Singh *et al.*, 1982; Ahmed *et al.*, 1986; Sattar and Khan, 1988; Kadu and Kaikini; 1988; Winter and Dobson, 1992).

Singh (1973) reported brownish nodular growth on surface of ovary in most cases with one case having greenish white pus in central cavity. Histologically, the nodular growth featured granuloma having central caseocalcified area surrounded by mononuclear cells and occasional giant cells with fibrocellular reaction. Ovaries had pus like material and were infiltrated with neutrophils and mononuclear cells in the vicinity. Organisms resembling streptococci were demonstrated in tissue sections.

2.1.3 Ovarobursal and tubo-ovarian adhesions

Ovarobursal adhesion have been reported by Long (1980); Sattar and Khan (1988) and Winter and Dobson (1992) in goats and sheep in isolated cases. Alosta *et al.*

(1998) observed that 2.3% tracts examined had ovarobursal adhesions, 60% of these were on the right side, 25% were on the left side and 15% were bilateral. The severity of the adhesions varied from a few fibrin strands between the ovary and mesosalpinx to complete adhesion involving the entire ovary and part of the urinary bladder surface.

Incidence of tubo-ovarian adhesions has been described by various workers in goats and sheep (Singh, 1973; Sharma, 1980; Kadu and Kaikini, 1988; Ijapure *et al.*, 2002). Singh (1973) reported chronic oophoritis with tubo-ovarian adhesions in 13 cases.

2.1.4 Sclerosed ovary

Sclerosis of ovaries was observed in 15 cases out of a total of 3791 female genital tracts of goats examined by Singh (1973). Such ovaries were encapsulated grossly and had adhesions with fallopian tube and bursa. Histologically, the ovary had adhesions with the serosal layer of the salpinx in several places.

2.1.5 Rete ovarii

Occurrences of rete ovarii have been reported in goats and sheep (Chand and Chauhan, 1978; Sharma, 1980). Sharma (1980) found rete-tubules in 0.62% cases and described them as a network of anastomosing canals lined by cuboidal to columnar epithelium and separated by thick connective tissue at the ovarian hilus. The author also reported that there was cystic dilatation of the tubules locally.

2.1.6 Epoophoron

Epoophoron have been observed by Singh (1973) in 14 cases upon examination of the female genital tract of 3791 goats. Sharma (1980) reported that these structures were seen in ovaries in 0.64% and 1.70% cases of sheep and goats respectively. Singh (1973) reported epoophoron as short inter- communicating, closely located structure on either

pole of ovary in loose connective tissue of mesovarian attachment. Acini had slit like lumen and were lined by cuboidal to columnar epithelium.

2.1.7 Anovular cord and granulosa cell islands

Anovular cords have been encountered in ovaries in 0.96% and 0.46% of sheep and goats respectively by Sharma (1980). Singh *et al.* (1982) described these cords in 20 out of 250 genital tract of ewes examined and explained these structures to be of variable size and shape, distributed in the cortex as well as in the deep medulla. Most of the cases had dark stained basal nuclei with vacuolated cytoplasm surrounded by thin PAS positive membrane. In a few granulosa cell islands there was a single row of cells and in others, cells were packed irregularly. In the third pattern, two or more layers of granulosa cells were present with a well formed lumen containing acidophilic material.

2.1.8 Folliculoids

Folliculoids have been reported by Singh (1973) in three cases in goats. Grossly, no apparent lesions could be seen but histopathologically, in all cases there were aggregations of acinar structures lined by granulosa cells. Groups of acini were separated from each other by well developed trabecular tissue. The lumen of these tubules did not reveal any PAS positive colloid body. Sharma (1980) encountered folliculoids in the ovaries of 1.85% cases in goats and 3.82% cases in sheep. The author observed pinkish homogenous mass in the centre of granulosa cells resembling Call-Exner bodies.

2.1.9 Ovarian haematoma

Incidence of ovarian haematoma was recorded in 0.45% goats by Das *et al.* (1979). Haematomas appearing as dark reddish brown masses with 1 to 3 cm in diameter could easily be separated from the surrounding ovarian tissue by a slight pressure. Microscopically, a large cavity lined by thick connective tissue membrane having spindle

shaped fibroblasts with many small blood vessels and a few mononuclear inflammatory cells at the periphery of the cavity were seen.

2.2 OVIDUCT

2.2.1 Hydrosalpinx

Incidence of hydrosalpinx has been reported to vary from 0.02-2.9 per cent in sheep and goats (Singh, 1973; Chand and Chauhan, 1978; Sokkar and Kubba, 1980; Badawi *et al.*, 1979; Sharma and Sharma, 1985; Ahmed *et al.*, 1987; Sattar *et al.*, 1988).

Kadu and Kaikini (1988) reported two cases (1.64%) of hydrosalpinx in goats. Grossly, the right fallopian tube was uniformly dilated and distended with a clear fluid. It had a thin wall and transluscent appearance. Microscopically, the oviduct had uniformly dilated lumen with low atrophied tubal rugae lined by low cuboidal cells.

2.2.2 Cystic salpinx

Sharma and Sharma (1985) reported one case of cystic salpinx in goat. The lesion was not appreciable grossly but microscopic examination of the oviduct revealed dilated cystic spaces of variable size and shape filled with a pink homogenous material in mucosa.

Sattar *et al.* (1988) encountered mucosal cysts in the oviduct of three cases upon examination of the female genital tract of 4186 goats. Such cysts have been invariably associated with hyperesterogenism (Chand and Chauhan, 1978).

2.2.3 Salpingitis

Salpingitis have been reported by various workers in sheep and goats. Incidence of salpingitis varies from 0.05 to 19 per cent in sheep (Chand and Chauhan, 1978; Sokkar

and Kubba, 1980; Singh *et al.*, 1982; Dzhurova *et al.*, 1985; Sharma and Sharma, 1985; Ahmed *et al.*, 1987; Rawal *et al.*, 1987) and 0.05 to 4.8 per cent in goats (Singh, 1973; Chand and Chauhan, 1978; Sharma and Sharma, 1985; Kadu and Kaikini, 1988; Sattar *et al.*, 1988).

In goats, most of the cases of salpingitis encountered by Singh (1973) were found to be associated with sub acute endometritis.

Sharma and Sharma (1985) described two types of lesions of salpingitis in goats viz, sub acute suppurative and chronic non suppurative types. In subacute suppurative salpingitis, lesions were confined to the isthmus and a small portion of the ampulla and were characterized by the presence of a pink homogenous material mixed with necrotic cellular debris, disintegrated and few intact neutrophils with some mononuclear cells covering the eroded mucosa. The chronic non suppurative salpingitis revealed desquamated epithelial cells in the lumen and connective tissue proliferation along with lymphocytic infiltration in the lamina propria.

Rawal *et al.* (1987) reported 4.8 per cent incidence of salpingitis and isolated organisms belonging to Proteus sp., Klebsiella sp., Escherichia sp., Staphylococcus sp., Bacillus sp. and Salmonella sp. in goats.

Alosta *et al.* (1998) reported that salpingitis was diagnosed in eight (0.9%) of the ewes. Out of these five cases were bilateral, two cases involved only the right side and one case involved the left side.

2.3. UTERUS

2.3.1 Hydrometra and Mucometra

Hydrometra was reported in two goats by Singh (1973). The gross lesions were enlarged and distended uterine horns containing 1-2 litres of thin, turbid fluid mixed with

flakes. The os-cervix was closed. Histopathologically, endometrium and myometrium had changes of pressure atrophy, with mononuclear cell infiltration in the lamina propria (Emady *et al.*, 1975).

Sharma (1980) identified hydrometra and mucometra in one genitalia each, on examination of genital tracts of 314 sheep and 647 goats. In the case of mucometra, the uterus was grossly enlarged and contained a grayish viscid material. Microscopically, the endometrium was desquamated focally and covered with a pinkish–blue homogenous material mixed with exfoliated epithelial cells. Glands were in various stages of degeneration and at places, mild to moderate periglandular fibrosis was evident.

Kadu and Kaikini (1988) recorded hydrometra and mucometra in 0.09 per cent and 0.28 per cent goats, respectively.

Goats may suffer from hydrometra whether they have been mated or not. The incidence increases in older goats, in goats which have been mated out of season and in goats which have been mated after estrus synchronization with progestagens. Hydrometra in goats is characterized by the accumulation of a varying amount of fluid which accumulates in the non pregnant uterus, presence of one or more corpora lutea on the ovaries and in most cases an enlarged abdomen depending on the amount of fluid (Matthews, 1991).

The histological examination showed that hydrometra led to characteristic histological changes of the uterine wall. In general, the pathological changes described a glandular cystic atrophy of the uterine glands and absence of any lesion in the myometrium or perimetrium. The chemical investigation of the fluid showed that the composition of uterine fluid in hydrometra is completely different from allantoic or amniotic fluid as well as from blood plasma (Wittek *et al.*, 1998).

2.3.2 Cystic endometrial hyperplasia

Bennets *et al.* (1946) reported a severe progressive infertility problem commonly known as Clover's disease in ewes grazing on subterranean pasture in Australia.

Adams (1975) conducted morphological and histochemical studies on the uteri of 34 ewes with permanent clover infertility and found cysts of sizes 2-8 mm scattered through out the endometrium in the phytoestrogen exposed ewes.

In a study on the female genital tract of 2431 sheep and 5404 goats, cystic endometrial hyperplasia was observed in non gravid uteri of 16 sheep and 8 goats (Chand and Chauhan, 1975).

Sharma and Sharma (1987) reported two cases of cystic endometrial hyperplasia in a sheep and a goat. Grossly, the affected uteri were enlarged and flabby. Uterine cavity contained variable amounts of fluid covering the gelatinous mucous membrane. Cystic structures on surface were recorded in one case. Microscopically, the glands revealed varying degrees of dilatation and were lined by cuboidal epithelium. Infiltration of lymphocytes was observed in the lamina propria with varying degrees of fibrocellular reaction.

Cystic endometrial hyperplasia (CEH) is a progressive and pathological endometrial expansion caused by an increase in the size and number of endometrial glands. Endometrial glands may actively secrete fluid into the uterine lumen, and CEH can eventually lead to mucometra, hydrometra, or pyometra. Cystic endometrial hyperplasia can lead to permanent infertility in domestic animals by preventing implantation or fertilization (McEntee, 1990).

Histopathologic examination of the uterine biopsy of a case of endometritis and CEH in a goat revealed multifocal erosions of the superficial epithelium and the endometrium was multifocally infiltrated by widely scattered viable and degenerate neutrophils, lymphocytes, and plasma cells admixed with little cellular debris and extravasated erythrocytes (hemorrhage). The endometrium was markedly expanded by many irregular cystic and hyperplastic glands. Some of these glands contained widely scattered neutrophils, sloughed epithelial cells, cellular debris, and fibrin. The glands

were lined by either normal, hyperplastic or attenuated epithelium. There was mild to moderate multifocal and transmural congestion, edema, and lymphatic ectasia (Radi, 2005).

2.3.3 Endometritis

Incidence of inflammation of uterus varied 0.9-44 per cent in sheep (Prabhakaran and Raja, 1972; Rao and Abdullakhan, 1974; Adam, 1975; Chand and Chauhan, 1978; Sharma, 1980; Sokkar *et al.*, 1980; Biolatti *et al.*, 1984; Turkarslan, 1984; Oshkin, 1989).

Singh (1973) observed endometritis in 1.53 per cent cases in goats and classified it under acute, sub acute and chronic endometritis. Acute endometritis was further categorized into acute mucopurulent and acute necrotic endometritis.

An increase in collagen fibers has been observed in acute and chronic endometritis, and is due to the conversion of reticular fibers to collagen fibers possibly due to the action of bacterial toxins (Chand and Chauhan, 1975).

Endometritis was described as the most common pathological condition (4.49% incidence) of the genitalia in shegoats ranging from 2–6 years of age (Ijapure et al. 2002), whereas a lower incidence of endometritis (3.21% and 1.53% incidence) was reported in local nondescript goats ranging from 1 to 6 years of age (Reddy *et al.*,1997).

Endometritis refers to inflammation limited to the endometrium. Endometritis in goats has been categorized into 3 phases: acute, subacute, or chronic endometritis on the basis of the degree and type of inflammatory changes. The acute phase is epithelial degeneration, necrosis and desquamation, edema and capillary congestion of the lamina propria and submucosa, and neutrophil infiltration into the cotyledonary and intercotyledonary spaces and uterine glands. In the subacute phase, there is epithelial desquamation, diffuse infiltration of lymphocytes, neutrophils, and macrophages into the lamina propria and cotyledonary portions of endometrium along with the presence of

many congested blood vessels. In the chronic phase, uterine glands are dilated, lined by flat epithelial cells and contained eosinophilic material together with mononuclear cell infiltration into the lamina propria and lumen (Singh and Rajya, 1977).

Sharma (1980) classified endometritis as suppurative and non-suppurative endometritis which included the cases of acute, sub acute and chronic endometritis in sheep and goats. The author also described one case of perimetritis.

Sokkar *et al.* (1980) classified endometritis in ewes under acute, acute lymphocytic, chronic non suppurative and suppurative endometritis and a variety of organisms (Escherichia coli, Corynebacterium pyogenes and Staphylococcus aureus) were isolated from these cases.

2.3.4 Metritis

Das *et al.* (1979) reported an incidence of 1.65% metritis in goats. Metritis refers to inflammation involving the entire thickness of the uterine wall. Microscopically, the uterine glands were markedly compressed and obliterated by extensive proliferation of fibrous tissue into the inter-glandular lamina propria. Epithelial cells of uterine glands were cast off at certain places and mononuclear cells were found scattered in the proliferated fibrous tissue.

The genital tract is particularly susceptible to infections after lambing, as bacteria can invade therein (Noakes, 1996). Alosta *et al.* (1998) observed an incidence of 0.5% metritis in ovines.

The host usually can counteract, limit infection and prevent the disease. Nevertheless, in some cases various factors can predispose to the development of acute metritis. These include: dystocia followed by obstetrical assistance, prolapse of the uterus, retained placenta and post-parturient ketosis. In such cases, bacteria can colonize the non-involuting uterus, producing toxins which are absorbed and manifest signs of

toxaemia. Ewes with metritis develop genital (swollen vulva and vagina, vaginal discharge, retention of fetal membranes) or systemic (anorexia, dehydration, fever, toxaemia) symptoms. If the condition remains untreated, it can lead to death. The bacteria implicated more frequently are *Arcanobacterium pyogenes* and *E. coli* (Tzora *et al.*, 2002).

2.3.5 Perimetritis and parametritis

Kadu and Kaikini (1988) observed an incidence of 0.81% perimetritis in goats with nodular plaques. Unlike perimetrial lesions, adhesions were not associated with this condition. Instead, small plaque-like elevations on both carnua and corpus uterus were observed.

Perimetritis is inflammation of the uterine serosal and parametritis is involvement of the tissues surrounding the uterus (McEntee, 1990).

Perimetritis and parametritis usually follow manual manipulation, obstetrical operation and uterine irrigation. The extent of the adhesion may vary from a few fibrous bands to dense connective tissue which obscures the contour of the organs and fixes them to adjacent viscera (Kennedy and Miller, 1993).

Timurkaan and Karadas (2000) reported an incidence of 0.35% for perimetritis-parametritis in goats. Chronic adhesive peritonitis involving the genital tract does not usually result from acute septic metritis because the uterine serosa offers an efficient barrier to the spread of infection.

2.3.6 Pyometra

The microscopical lesions consist of dilated endometrial glands, many of which contain neutrophils, and large, multifocal, periglandular foci of lymphocytes, macrophages and plasma cells. Some glands have prominent periglandular fibrosis. Small

abscesses, some of which are visible on gross examination, may be present in the endometrium. The abscesses arise following destruction of glandular epithelium and extension of the suppurative process into the surrounding stroma (McEntee, 1990).

Pyometra is an acute or chronic suppurative infection of the uterus with accumulation of pus in the uterine cavity and occur as a sequel to uterine infections (Kennedy and Miller, 1993).

Timurkaan and Karadas (2000) observed an incidence of 0.05% pyometra in goats. Aliasghar and Saad (2007) reported an incidence of 0.2% pyometra in ewes and Khatun *et al.* (2007) reported 1.16% incidence of pyometra in sheep wherein the uterus was of irregular shape with enlarged horns and presence of pus in both the horns.

2.4 CERVIX

2.4.1 Cervicitis

Studies on the female genital tract of 3431 Sheep and 5404 goats revealed acute cervicitis in 2 sheep and 1 goat, chronic cervicitis in seven sheep and three goats and acute pericervicitis in one sheep (Chand and Chauhan, 1978).

Sharma (1980) reported cervicitis in 1.91 percent cases in sheep and 0.62 percent cases in goats. Suppurative cervicitis was observed in one sheep associated with macerated foetus. Histologically, the epithelium showed marked squamous metaplasia along with other changes. Non-suppurative cervicitis included acute, subacute and chronic cervicitis respectively.

Biolatti *et al.* (1984) conducted studies on the female genital tract of 1107 sheep of Piedmont and Sard breed and recorded cervicitis in 27 cases.

Cervicitis has generally been reported in association with vaginitis and/or endometritis. All animals with endometritis have some degree of cervicitis but the inflammatory reaction is less severe in the cervix than in the endometrium (McEntee, 1990).

Timurkaan and Karadas (2000) observed that 1% of 4000 goats examined had cervical lesions and the major lesions of the cervix were acute catarrhal cervicitis (0.25%), acute purulent cervicitis (0.10%) and chronic non purulent cervicitis (0.42%).

2.5 TUMORS

Terlecki and Watson (1967) reported a case of endometrial adenocarcinoma in an ewe. Histopathologically, the neoplasm consisted of solid group of variably defined acini of neoplastic epithelial cells scattered through out the tissues.

Anderson and Sandison (1969) examined 4.5 million sheep in Great Britain and found only two tumors in the female genital tract of sheep. Both of these tumors were found in the wall of the uterus and were described as leiomyoma and fibroma.

Chand and Chauhan (1978) observed a case of cavernous haemangioma on the serous surface of vagina in one goat out of 5404 female genital tracts of goat examined.

Long (1980) described a case of cystadenoma in the ovary of an ewe. Histologically, the case exhibited the characteristic multi branched papillae and fluid filled cysts.

Ryan (1980) reported a uterine leiomyosarcoma from a seven year old Saanen doe grossly characterized by 1.5 cm circular rent in the uterine wall surrounded by a broad based multi lobulated, rubbery white mass originating from the dorsum of the uterine body and extending into the lumen of the greatly enlarged uterus.

Sharma (1980) found cystadenoma in the ovaries of one sheep and two goats. He also reported a case of mesothelioma in the uterus of a goat. Grossly, the serous surface of the uterus had grayish-white diffusely disseminated firm cauliflower like growths.

A case of dysgerminoma has been described in a goat which revealed the arrangement of neoplastic cells in sheets, cords or alveoli (Smith, 1980).

Hawkins *et al.* (1981) conducted a survey on 80 farms to study the incidence of squamous cell carcinoma in ewes and found a mean prevalence of 2.29 per cent that increased with age.

Squamous cell carcinoma of vulva has been reported in cattle, sheep and goat, associated with a high level of solar irradiation. Papilloma virus is suspected to play a key role in the induction of carcinoma (Vanselow and Spradbrow, 1983).

Lofstedt and Williams (1986) reported a case of granulosa cell tumor in a 3 year old Toggenburg goat that had short estrus cycles and infertility followed by male like behaviour.

The histopathologic diagnosis from a biopsy specimen of a mature nulliparous Saanen-type goat examined for episodes of vulvar hemorrhage with coalescing nodular vaginal masses was leiomyofibromatosis. Removal of the cystic ovaries caused regression of the vaginal tumors, although transvaginal adhesions formed because of the extensive superficial necrosis originally present (Haibel *et al.*, 1990).

Reports of neoplasms of the ruminant female reproductive tract are rare. An estimated 10–50% of such tumors were of smooth muscle origin, of which approximately 10% were considered malignant. Leiomyosarcomas in these species were reported to display a growth pattern of low-grade malignancy, with slow invasion and rare metastasis. Case reports of genital smooth muscle neoplasms in goats have been sporadic (Whitney *et al.*, 2000).

2.6 CONGENITAL ANOMALIES

Singh and Rajya (1977) reported seven cases of hermaphroditism in adult female goats. Five of these cases had well marked uterus, vagina, bladder and two well developed testes on either side adhering to the fallopian tube in place of ovaries. In the remaining two cases ovotestis were found. Accessory ovary has been reported by Chand and Chauhan (1978) in goats. Dennis (1979) found three male pseudohermaphrodites in a series of 401 malformed lambs. Krishna and Paliwal (1981) recorded a similar case in a Jamnapari goat. Histopathological examination of materials collected from a buck revealed functional male genital tract and replacement of fibrous tissue elements in female organs. Examination of 4186 ovaries of goats slaughtered at Faisalabad abattoir revealed single case of hermaphroditism (Sattar and Khan, 1988). Congenital anomalies in female genital tract of sheep and goats were limited except for those pertaining to hermaphroditism (intersexes). The majority of intersex goats were male pseudohermaphrodite with a female karyotype (Mc Entee, 1990).

O'Shea *et al.* (1974) reported normal estrus cycle in five ewes in which left uterine horn was congenitally absent. Uterus unicornis is predominant congenital malformation that has been reported in ovine uterus and is characterized by congenital absence of one uterine horn. Uterus bicorporbicollis (uterus didelphys) has been reported by Singh and Rajya (1977). In this case two external os instead of one were present with independent cervical canals. Ansari (1978) found five cases of uterus unicornis during examination of reproductive tract from 3590 slaughtered ewes. The left uterine horn was affected in four cases. Nasseri and Prasad (1986) and Rawal *et al.* (1987) reported similar cases in sheep.

Smith *et al.* (1998) reported that in a 15 month survey of 33506 ovine reproductive tracts undertaken in slaughterhouses in southwest England, twenty cases of uterus unicornis, six of uterus didelphys and 11 of segmental aplasia were encountered, such that partial aplasia of the paramesonephric ducts accounted for 3.3% of all abnormalities. Although developmental abnormalities of the ovine female genital system.

were relatively uncommon, a substantial proportion of these can be accounted for by development defects of the paramesonephric ducts.

Adenomyosis has been reported in two cases in sheep by Chand and Chauhan (1978). Sharma and Sharma (1986) reported one case of adenomyosis in sheep and four cases in goats out of 314 sheep and 647 goats examined for pathological abnormalities of female genital tract. In most cases the endometrial tissue was enclosed within a fibrous tissue capsule.

Badawi *et al.* (1979) reported three cases of bent cervix in 683 Barberi ewes examined. Sharma and Sharma (1986) also reported bent cervix in one sheep and three goats. Grossly, the cervix appeared hard cord like and had two curves in posterior third portion. The external os was closed and cervical canals were narrow with ill developed cervical rings. Khatun *et al.* (2007) reported an incidence of 29% bent cervix in sheep and 2% in goat.

Gustaffson and Holmberg (1966) examined reproductive tract of 502 slaughtered ewes and found one genital tract with duplication of cervix and vagina and four tracts with duplication of caudal portion of vagina. Maxwell (1977) reported about the occurrence of 11% constriction of vagina with a range of 2-27% in 12 flocks of Merino ewes examined. The defect was located at junction of cranial and middle third of the vagina. Median vertical band at the level of vulvovaginal orifice in two goats has been reported by Singh and Rajya (1977). The vertical bands extended from the floor of vagina to the roof at the hymenal border immediately anterior to uterine meatus resulting in the division of vagina into two independent canals. Mc Entee (1990) reported a case of rectovaginal fistula in goats. The fistula connected the rectum with the vestibule and opened at junction between vestibule and vagina and faecal matter was partly discharged through the vagina.

2.7 ENDOCRINE PATHOLOGY IN REPRODUCTIVE DISORDERS

2.7.1 General

The process of growth in animals is under endocrine control for most part of their life and reproductive processes are primarily under endocrine control (MacDonald, 1969).

McClain (1994) reported that the development of proliferative lesions (usually hyperplasia and benign tumors) in the endocrine organs and hormone- responsive tissues are common findings in chronic studies with high doses of many nongenotoxic xenobiotic chemicals administered to sensitive rodent species and might have limited significance for human safety assessment.

Capen (2001) reported that the subnormal function of the fetal endocrine system, especially in ruminants, might disrupt normal fetal development and result in the prolongation of gestation period. Prolongation of the gestation period also occured in ewes that ingested the plant *Veratrum californicum* early in gestation. Toxins in the plant caused extensive malformations of the central nervous system (CNS) and hypothalamus. A genetically determined failure of development (aplasia) of the adenohypophysis resulted in a lack of fetal pituitary trophic hormone secretion during the last trimester and hypoplastic development of target endocrine organs (eg, adrenal cortex, thyroid follicular cells and gonads).

Gonadotropin releasing hormone (GnRH) is a hypothalamic neuronal secretory decapeptide that played a pivotal role in mammalian reproduction. GnRH and its analogues are used extensively in the treatment of hormone dependent diseases and assisted reproductive technology. Although hypothalamus and pituitary are the principal source and target sites for GnRH, there are extra-hypothalamic GnRH and GnRH

receptors in various reproductive tissues such as ovaries, placenta, endometrium, oviducts, testes, prostrate, and mammary glands (Ramakrishnappa *et al.*, 2005).

Hormones mediate a major part of our essential physiological functions. Both endogenous and exogenous compounds and their metabolites are known to act through hormone receptors leading to regulation of endocrine function. The exogenous compounds that are structurally and functionally similar gain entry into animals including humans through the diet or by occupational exposures, causing endocrine disruption and are known as "endocrine disruptors" (Roy and Pereira, 2005).

Maturation of the hypothalamo-pituitary-gonadal axis was effected by the interaction between environmental and genetic factors and depends on the coordinated functions of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Thus, the gonadotroph population was the morphological setting for the endocrine mechanism that prepares the reproductive system for puberty and then for sexual maturity in the mammal (Wa'nkowska and Polkowska, 2006).

2.7.2 Pituitary gland

Pituitary dwarfism in German shepherd dogs usually are associated with a failure of the oropharyngeal ectoderm of Rathke's pouch to differentiate into trophic hormone–secreting cells of the pars distalis. This resulted in a progressively enlarging, multiloculated cyst in the sella turcica and a partial to complete absence of the adenohypophysis. The cyst was lined by pseudostratified, often ciliated, columnar epithelium with interspersed mucin-secreting goblet cells. The mucin filled cysts eventually occupy the entire pituitary area in the sella turcica and severely compress the pars nervosa and infundibular stalk. Cysts associated with pituitary dwarfism morphologically were distinct from the cysts that develop following the abnormal accumulation of colloid in the residual lumen of Rathke's pouch. In the latter, the normally developed pars distalis and pars nervosa were compressed to varying degrees by

the abnormal accumulation of colloid in a preformed normal cavity of the pituitary gland (Alexander, 1962).

Hyperfunction of an endocrine organ also can be the result of hormonal imbalances induced by xenobiotic chemicals. Hyperactivity of the pituitary gland in rodents during chronic toxicity testing often results in an increased development of tumors in the gonads or mammary glands. An excess production of luteinizing hormone, usually due to disruption of negative feedback control by estrogen or testosterone, increases the incidence of tubulostromal adenomas and granulosa cell tumors in the ovary of mice and Leydig interstitial cell adenomas of the testes in rats (Murphy and Beamer, 1973).

Zaki and Liu (1973) reported that nonfunctional (endocrinologically inactive) pituitary tumors are most common in dogs, cats, and certain strains of laboratory rats and are uncommon in other species. Although these adenomas are endocrinologically inactive, they may result in significant functional disturbances by virtue of compression atrophy of the pars nervosa and pars distalis or extension into the overlying brain and optic nerves. Animals with nonfunctional pituitary adenomas usually have clinical disturbances related to dysfunction of the central nervous and neurohypophyseal systems as well as a lack of secretion of pituitary trophic hormones with diminished end-organ function (eg, thyroid follicular cells, adrenal cortex, and gonads).

Panhypopituitarism in German shepherd dogs often occurs in littermates and related litters, suggesting a simple autosomal recessive mode of inheritance (Nicholas, 1978).

Sikdar and Bhowmik (1993) reported hyperplastic lesions (9%) in the pituitary glands of 300 goats examined and the highest prevalence was in the age group of more than 5 years.

Busato *et al.* (1995) reported that the differences between the pituicytes of cystic group, diestrous group and periestrous group cows indicate that the endocrine condition of the cyst-bearing animals was dissimilar from that of diestrous group or periestrous group animals. The LH gonadotropes of the cystic group compared with those of the diestrous group and periestrous groups were atrophic and contained lower hormone concentrations, suggesting reduced hormone synthesis, impaired hormone release and, perhaps, intracellular hormone-degradation.

Chapin (1995) reported that the dietary exposure of sheep, acute injection of rats, and dietary exposure of women to phytoestrogens alter pituitary secretion of LH. The effect seems to resolve around diminished responsivity of the pituitary to the stimulatory actions of gonadotropin-releasing hormone rather than suppression of "basal" secretion of gonadotropins. The implication is that phytoestrogen exposure appears to reversibly diminish response of the pituitary to gonadotropin hormone-releasing hormone stimulation.

Mudge (1995) reported that the pituitary-gonadal axis dysfunction was associated with gonadal dysfunction and various types of sexual precocity.

Smith (1996) demonstrated that the entire reproductive neuroendocrine system of freemartin ewes has undergone a significant masculinisation with the responses of the hypothalamus to steroids and, to a lesser extent, inhibin, also resembling that of the male more closely than that of the female.

Kaneko *et al.* (1997) demonstrated that inhibin neutralization during the early luteal phase produces hypersecretion of FSH with a coincident stimulation of follicular development, indicating that inhibin is an important factor for the negative regulation of FSH secretion during the early luteal phase when secretion of estradiol and progesterone are normally high.

Peters *et al.* (2000) concluded that the seminomas were not endocrine-logically active although both sertoli cell tumours and leydig cell tumours could cause increased estrogen production leading to signs of feminization. However sertoli cell tumours produced a reduction in FSH concentrations.

Capen (2001) reported that a destructive lesion in the pituitary gland interferes with the secretion of trophic hormones and results in subnormal function of target endocrine glands in secondary hypofunction. Large, endocrinologically inactive neoplasms may interfere with the secretion of multiple pituitary trophic hormones and result in clinically significant hypofunction of the adrenal cortex, follicular cells of the thyroid and gonads.

Emmakeeble (2001) reported that pituitary hyperplasia and adenomas are extremely common in older rats (70% incidence in older Sprague-Dawley rats)

Vanholder *et al.* (2006) reported that the most widely accepted hypothesis explaining the formation of a cyst is that LH release from the hypothalamus-pituitary is altered: the pre-ovulatory LH-surge is either absent, insufficient in magnitude or occurs at the wrong time during dominant follicle maturation, which leads to cyst formation. This aberrant LH release does not seem to be caused by a lower GnRH content of the hypothalamus, nor by reduced GnRH receptor numbers or LH content in the pituitary. It is the altered feedback mechanism of oestrogens on the hypothalamus- pituitary which results in an aberrant GnRH/LH release and cyst formation.

Wa´nkowska and Polkowska (2006) suggested that the requirement for ovine sexual maturation is a change in the histomorphological feature of pituitary gland, dependent upon the gradual increase in the subpopulation of endocrine cells containing LH, lasting to the end of juvenile period.

2.7.3 Thyroid gland

Sreekumaran (1976) while studying the pathological features of experimentally induced clinical hypothyroidism in kids indicated that the reproductive organs were also affected in the hypothyroid state. Histologically the thyroid gland showed formation of colloid depleted microfollicles and the pituitary was hypertrophied with hyperplasia of basophil cells. Both male and female gonads showed degenerative changes.

DeFesi *et al.* (1979) recorded maximum increase in the pituitary weight after thyroidectomy in rats. Increase in both pituitary DNA content and total pituitary cell number was observed in thyroidectomised rats when compared to euthyroid animals.

Reddy (1982) reported that there was a significant reduction in the relative weight of ovaries in all hypothyroid goats and the ovaries contained only a few scattered developing primary and secondary follicles. There was also a significant reduction in the relative weight of the uterus with thin muscular layer and the mucosal glands were few and inactive and revealed no secretory activity.

Peterson (1984) described a syndrome of hyperthyroidism in aged animals associated with multinodular goiter, adenomas, and occasionally adeno-carcinomas derived from follicular cells of the thyroid.

Longcope (1991) reported that hypothyroidism was closely associated with the changes in folliculogenesis and the formation of corpus luteum in rats, and thyroid dysfunction causes disorders in ovarian functions in women. When mature female rats were thyroidectomised, estrous cycles become irregular and their ovaries became atrophic.

Thyroid hormone receptors and/or their messenger ribonucleic acid (mRNA) had been detected in porcine pre ovulatory antral follicle granulosa cells (Maruo *et al.*, 1992).

Capen (1993) reported that abnormalities in reproduction were a common functional disturbance in hypothyroidism. Lack of libido and reduction in sperm count might occur in males, whereas abnormal or absent estrous cycles with reduced conception rate occured in females.

A study using non-lactating cows showed that estrus behavior was unaffected by hypothyroidism induced by thyroidectomy (Stewart *et al.*, 1993).

The effects of thyroidectomy and thyroxine on the reactivity of rat uterine muscle to electrical stimulation in vitro was studied in adult albino Wistar-strain rats. Thyroidectomy decreased the frequency of uterine smooth-muscle contraction. Thyroxine (6–8 μg/100 g body wt. per day) for 35 days caused a potentiation of acetylcholine and ergometrin-induced contractile responses of the uterine smooth muscle. Those findings suggested that the thyroid hormone affected the intrinsic contractile state of the uterine smooth muscle (Adeniyi *et al.*, 1994).

Fitko *et al.* (1995) reported that there was an increase in ovarian iodide uptake during hypothyroidism induced by thiouracil. The hypothyroid state was characterized by an absolute or relative depletion of thyroid hormones, altered sensitivity and ovarian response to gonadotropins leading to a rise in the content of mucopolysaccharides, followed by a tendency in some species towards the development of polycystic ovaries. In fact, ovarian cyst formation was greatly intensified in women with primary hypothyroidism and in experimentally hypothyroid animals exposed to hyperstimulation with gonadotropins.

Fitko *et al.* (1996) reported that the level of GnRH in the hypothalamus, and LH and FSH in the pituitary showed a tendency to parallel with thyroid function which may indicate a role of this gland in their production or secretion. In hypothyroid animals an increase of LH and PRL and a slight decrease of secretory function of the ovaries were noted.

The hypothyroid cows had greater numbers of luteinized follicles and on day 7 after ovulation, the ratio of luteal to serum progesterone was greater in the hypothyroid than in euthyroid cows (Bernal *et al.*, 1999).

Hypothyroidism does not result in sterility, but does interfere with gestation, usually during the first half of pregnancy resulting in increased resorption of embryos, reduced litter sizes and increased stillbirths. Hyperthyroidism induced by administration of T4 was associated with long periods of diestrus with few mature follicles or corpora lutea. An increased quantity of thyroid hormones caused abortion and neonatal death (Longcope, 2000).

Immune-mediated injury is an important mechanism resulting in hypofunction of endocrine glands in animals, including the thyroid gland. The immunologic basis of the development of chronic lymphocytic thyroiditis in animals appears to be through production of autoantibodies usually directed against thyroglobulin or a microsomal antigen (thyroperoxidase) and infrequently against the TSH receptor protein, nuclear antigen, or a second colloid antigen from thyroid follicular cells. Microscopic lesions consist of multifocal to diffuse infiltrates of lymphocytes, plasma cells, and macrophages and, sometimes, lymphoid nodules. Thyroid follicles were small and lined by columnar epithelial cells; lymphocytes, macrophages, and degenerated follicular cells are often seen in vacuolated colloid (Capen, 2001).

The study of Spicer *et al.* (2001) provided supportive evidence for a role of T3 and T4 in regulating steroidogenesis of bovine follicles.

Huszenicza *et al.* (2002) reported that there was doubtless evidence concerning the involvement of thyroid hormones in the process of resumption of cyclic ovarian function in ruminants.

The effects of Carbimazole (CZ)-induced hypothyroidism were investigated in goats during summer season. CZ treatment at a daily dose of 3mg/kg body weight for 11 days inhibited ovulation and corpus luteum formation. This suggested that heat stress, which usually caused a decline in thyroid hormones to reduce basal metabolism, may produce its effect on fertility in a similar manner to CZ-induced hypothyroidism (Khaled, 2004).

Todini (2007) reported that changes of blood thyroid hormone concentrations are an indirect measure of the changes in thyroid gland and extrathyroidal deiodination activity. Besides endogenous and environmental climatic factors, nutrition plays a primary role on thyroid gland activity and blood thyroid hormone concentrations.

2.7.4 Adrenal gland

Wilkie and Krook (1970) revealed a mass 16 to 18 cm in diameter adjacent to the anterior pole of the right kidney that apparently replaced the right adrenal. It was soft, spongy, and on cut surface, dark brown with numerous blood vessels containing thrombi. The tumour was histopathologically confirmed as pheochromocytoma.

One of the abnormalities of the adrenal glands is displacement of the accessory adrenal nodules. These nodules are usually found near the adrenal glands, kidneys, and gonads. Both intracapsular and extracapsular accessory adrenal cortical nodules were found in the adrenals of goats and sheep, whereas accessory nodules were absent in pigs and fowl (Prasad and Sinha, 1980).

Wentzel (1982) suggested that adrenal hyperfunction in Angora goats caused high maternal blood corticosteroid levels which created an unfavourable foetal environment resulting in foetal death and expulsion of an oedematous foetus. Normal levels of progesterone, low levels of urinary oestrogen and elevated levels of maternal blood corticosteroid were found in aborting goats compared to goats with a normal pregnancy.

The disorders of the adrenal cortex can be viewed from the dual perspectives of hyperfunction and hypofunction. Clinical expressions of hyperfunctional adrenocortical syndromes include Cushing's syndrome, primary hyperaldosteronism, and the adrenogenital syndrome. The expressions of hypofunctional syndromes include Addison's disease and selective hypoaldosteronism (Kannan, 1988).

An experiment conducted to study the relationships between adrenals and reproductive cycle revealed that dexamethasone caused prolonged luteal function either by the suppression of prostaglandin $F_2\alpha$ synthesis or by the suppression of pituitary stimulation of follicular growth (Alam *et al.*, 1989).

McDonald and Pineda (1989) reported that adrenal cortices normally produce androgens, and adrenal androgen secretion may be greatly increased in a variety of pathological condition, including adrenal hyperplasia or neoplasia, nymphomania, and adrenal virilism

A study designed to examine the basal ACTH and cortisol in ewes of four different reproductive statuses: ovariectomized, nonpregnant cycling, nonpregnant noncycling, and pregnant, demonstrated that there is an increase in basal ACTH and cortisol in ovine pregnancy (Bell *et al.*, 1991).

Capen (1993) described that pheochromocytoma was the most common neoplasm in the adrenal medulla of animals, occurring most often in cattle and dogs and infrequently in other species. The pheochromocytomas developed concurrently with calcitonin secreting C-cell neoplasms of the thyroid gland.

Women with congenital adrenal hyperplasia due to 21-hydroxylase deficiency often have a polycystic ovary-like syndrome, consisting of hyperandrogynism, infertility, menstrual irregularities, and elevated LH levels. Congenital adrenal virilization programs the hypothalamic-pituitary axis for hypersecretion of LH at puberty. This is postulated to frequently cause ovarian hyperandrogenism even when adrenal androgen excess

was subsequently controlled by glucocorticoid therapy (Barnes *et al.*, 1994).

Lavoie and Lacroix (1995) reported that the studies of the function of adrenal "incidentalomas" have revealed that a proportion of those tumors secrete cortisol insufficiently to produce overt clinical Cushing's syndrome, but that their autonomous cortisol production can suppress the hypothalamo-pituitary-adrenal (HPA) axis to various degrees; this needs to be recognized to avoid acute adrenal insufficiency after adrenalectomy.

To investigate the causes and mechanisms of foetal loss in Norwegian dairy goats, blood parameters in 40 goats that lost foetuses were compared with those in 40 goats that experienced a normal pregnancy. The normal level of progesterone and cortisol in goats with foetal loss indicated that the function of the corpus luteum and adrenal glands, respectively, were not disturbed. The rapid decline in progesterone level associated with foetal loss may therefore be a result, rather than the cause of foetal death. The lowered level of oestrone sulphate and elevated level of 15-ketodihydro-PGF2a in goats with foetal loss clearly indicated that the endocrine foetal–placental function was disturbed (Engeland *et al.*, 1999).

Studies by Piffer and Pereira (2004) indicated that the use of hydrocortisone at a dose that apparently does not endanger the neonate led to undesirable effects in the adult reproductive phase, resulting in later deleterious alteration of the reproductive physiology in female rats.

Usta *et al.* (2006) reported a case of an ectopic adrenal tissue within the wall of an ovarian serous cystadenoma of a 21-year-old woman as a peculiar site. Microscopically the inner surface of the fibrotic cyst wall was lined by serous, low cuboidal epithelium, and the cyst was diagnosed as "serous cystadenoma of the ovary" and the above mentioned nodule was diagnosed as "ectopic adrenal tissue with well defined borders". Two distinct regions of the adrenal, namely cortex and medulla were identified. Female

genital tract is a common site for ectopic adrenal tissue but ovarian localization is very rare.

Virilization due to androgen-secreting neoplasms in women is a result of androgen overproduction from benign or malignant tumors that are found in the ovaries or rarely in the adrenal glands. Virilizing tumors that arise from ectopic adrenal tissue are extremely rare. An ectopic mass was detected behind the left iliopsoas muscle of a patient operated upon and an oblong-shaped lesion, weighing 6 g, was removed. Histologically, the tissue was identified to be of adrenal origin. Postoperatively the androgen levels decreased to normal levels (Mavroudis *et al.*, 2007).

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy to ascertain the incidence, nature and magnitude of utero-ovarian pathology in goats and to correlate the utero-ovarian pathology with endocrine lesions.

3.1 MATERIALS

3.1.1 Sample collection

A total of 1000 genital organs of female goats collected from the Naval butchery, Ernakulam and Corporation slaughter house, Thrissur were utilized for the study. From among these genitalia, uterus and ovaries with lesions were subjected to gross and histopathological examination. Pituitary, adrenal and thyroid glands from animals with gross utero-ovarian lesions were subjected to detailed gross and histopathological examination.

3.2 METHODS

3.2.1 Gross examination

The ovaries were incised longitudinally extending from free border to the attached border. The ovarian bursa was spread out to detect the presence of gross lesions. The tubular genital tracts were opened by dorsal incision commencing from the body of uterus to cornua. The exposed mucous membrane was examined for gross changes in colour, nature of fluid and alterations. Observations were recorded in detail. The fallopian tubes were examined by palpation and incision. The presence of any abnormalities in the ovary, oviduct and uterus were recorded and classified. The pituitary, adrenal and thyroid glands were examined for gross pathological changes like hypertrophy, hypoplasia, cyst, haemorrhage and tumour which were subsequently recorded and correlated.

3.2.2 Histopathology

Representative samples of ovary, oviduct and uterus from the grossly affected and from those suspected to be affected were immediately fixed in 10% formalin solution. The tissues were processed by routine paraffin embedding techniques (Sheehan and Hrapchak, 1980). Sections were cut at 4 micron thickness and stained with routine Haematoxylin and Eosin (Bancroft and Cook, 1995) for histopathological studies. The stained sections were examined in detail under light microscope and histopathological lesions of these organs were recorded and classified.

Representative samples of pituitary and adrenal gland were preserved in Bouin's fluid and the tissues were processed by routine histological techniques (Luna, 1968). The thyroid gland tissues were fixed in 10% neutral buffered formalin and the tissues were processed by conventional method (Reddy and Rajan, 1985). Paraffin wax sections were cut at 3-4 microns and stained with routine Haematoxylin and Eosin. Special stains such as PAS, Trichrome and Alizarin red were used wherever necessary as per the methods described by Luna (1968). The stained sections were examined in detail under light microscope and histopathological lesions of these organs were recorded and correlated.

Results

4. RESULTS

The present study was conducted at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy to ascertain the incidence, nature and magnitude of utero-ovarian pathology in goats and to correlate the utero-ovarian pathology with endocrine lesions. A total of 1000 genital organs collected from the Naval butchery, Ernakulam and corporation slaughter house, Thrissur were utilized for the study. From among these genitalia, uterus and ovaries with lesions were subjected to gross and histopathological examination. Pituitary, adrenal and thyroid glands from animals with gross utero-ovarian lesions were subjected to detailed gross and histopathological examination. Out of the 1000 genital tracts examined, 84 cases showed one or more lesions in the ovary, oviduct and uterus. The prevalence of the utero-ovarian disorders in goats is shown in table 1 and Fig.1. Lesions of varying frequency in the pituitary, thyroid and adrenal glands were recorded in 23 out of 84 cases examined and are shown in Fig.5.

4.1 OVARIES

Out of the 1000 genital tracts examined, 54 cases showed lesions of varying frequency in the ovaries. The conditions encountered were ovarian hypoplasia, follicular cyst, luteal cyst, perioophoritis, atretic follicles, epoophoron, serous inclusion cyst, embedded corpus luteum, persistent corpus luteum, ovarian haematoma, ovarian sclerosis, senile atrophy and parovarian cyst (Table 2 and Fig. 2). The pathological features observed in each case are as follows.

4.1.1 Ovarian hypoplasia

4.1.1.1 Incidenc

The incidence ovarian hypoplasia was found in 6 goats (0.6%). All the cases were unilateral involving the left ovaries in four cases and the right in two cases.

4.1.1.2 Gross pathology

The ovaries were flat, small and firm. The surface was smooth and did not reveal any follicle, developing or degenerating corpus luteum or luteal scars. The development of the tubular genitalia was normal (Fig. 6).

4.1.1.3 Histopathology

The affected ovaries were composed mostly of medullary connective tissue and blood vessels with a thin investment of cortical tissue. Organization of germinal epithelium into secondary graffian follicles was absent. Most of the cortical follicles were atretic with degenerating oocyte.

4.1.2 Follicular cyst

4.1.2.1 Incidence

Follicular cysts were noticed in 14 cases (1.4 per cent). All were unilateral. Among the unilateral cases, the left ovary was involved in 06 cases and right ovary in 08 cases. Multiple follicular cysts were found in two cases. Among these, the left ovary was involved in two cases and right ovary in three cases.

4.1.2.2 Gross pathology

Grossly, the cysts appeared as slightly elevated structures, containing clear watery fluid with thick and tense wall and were situated mostly on the right side. Size of the cysts varied from 1.0-2.0 cm (Fig. 7).

4.1.2.3 Histopathology

Microscopically, the cyst wall showed thick connective tissue, surrounding the degenerating granulosa cell layers. Basement membrane was absent in all the cases. Degenerated and necrotic cumulus oophorus with remnants of ova were seen in some of the cysts examined. The two layers of theca interna and theca externa could not be demarcated (Fig. 27).

4.1.3 Luteal cyst

4.1.3.1 Incidence

Luteal cysts were recorded in three (0.3 per cent) out of the 1000 cases examined. All the cysts were unilateral. Right ovary was affected in all the three cases.

4.1.3.2 Gross Pathology

Luteal cysts contained darker yellow or brown cloudy fluid circumscribed by thick walls. The diameter of the cyst varied from 0.5 to 1.0 centimeter (Fig. 8).

4.1.3.3 Histopathology

Microscopically, the cyst wall revealed inner loosely arranged layer of luteal cells intermixed with connective tissue. The theca layer had thick connective tissue with some blood capillaries. The inner layer consisted of a thin band of loose connective tissue that separated adjacent luteal tissue from the cystic contents. The middle layer was composed of lutein cells of varying thickness. The outer wall contained concentrically arranged dense connective tissue stroma of the ovary (Fig. 28).

4.1.4 Parovarian cyst

4.1.4.1 Incidence

Parovarian cysts were recorded in 06 cases (0.6 per cent), two cases being bilateral and the remaining four cases unilateral. Among the unilateral cases, the left side was involved in three cases and right side in one case. The cysts were seen on the mesovarium, mesosalpnix and the ovary.

4.1.4.2 Gross Pathology

The cysts were small, soft and thin walled and contained colorless watery fluid with the shape of the cysts varied from circular to oval (Fig. 9).

4.1.4.3 Histopathology

The cyst wall comprised of a layer of fibrous connective tissue and muscle fibers. The cyst wall was lined by a single layer of cuboidal or low columnar epithelium. The lumen did not reveal any stainable material (Fig. 29).

4.1.5. Embedded corpus luteum

4.1.5.1 Incidence

Embedded corpus luteum was recorded in 03 cases (0.3%). All the cases were unilateral in which two cases were in the right ovary and one case in the left ovary.

4.1.5.2 Gross pathology

Grossly there were no lesions but cut surfaces of the ovaries revealed encapsulated, yellowish brown coloured corpus luteum of approximately 0.1-0.2

centimeter in diameter within the ovary. These corpora lutea were solid and slightly larger than normal in size. The corpus luteum appeared hard in consistency.

4.1.5.3 Histopathology

The embedded corpus luteum had a fibrous connective tissue capsule which separated the corpus luteum from the surrounding ovarian stroma. These connective tissue fibers penetrated into the lutein tissue giving a lobular pattern. The luteal cells had extreme vacuolation due to regressive changes. There was no evidence of any developing follicles in the ovarian cortex but some of the existing developed follicles underwent cystic transformation (Fig. 30).

4.1.6 Persistent corpus luteum

4.1.6.1 Incidence

Persistent corpus luteum was recorded in two cases (0.2%) and both the cases were seen in the right ovary.

4.1.6.2 Gross pathology

The persistent corpus luteum was solid and larger in size than that of the normal corpus luteum. The crown was projected on the ovarian surface. The corpus luteum was pale and hard in consistency (Fig. 10).

4.1.6.3 Histopathology

The persistent corpus luteum showed fibrous connective tissue capsule which divided the luteal cells into many lobes. There was reduced vascularisation in the parenchyma which led to regressive changes in the luteal cells. In the cortex, developing

follicles were seen but some of them underwent atresia. Some of the follicles transformed into cystic follicles.

4.1.7 Ovarian haematoma

4.1.7.1 Incidence

Ovarian haematoma was recorded in two cases out of the 1000 cases examined.

4.1.7.2 Gross pathology

The swelling was grossly visible as a blood clot within the ovary. The surface was smooth and the contents fluctuated on palpation. The clotted blood presented a non-adherent laminated appearance on sectioning. The blood clots were easily removed from the ovary (Fig. 11).

4.1.7.3 Histopathology

The haematoma was well organized and were found to be completely encapsulated by the ovarian stroma. The content was homogenous in one case but was granular in another case. Fibrous tissues septa traversed the haematoma and formed a laminated appearance. Few leukocytes were seen in the haematoma (Fig. 31).

4.1.8 Serous inclusion cyst

4.1.8.1 Incidence

Serous inclusion cyst was recorded in two cases (0.2%) out of the 1000 cases examined.

4.1.8.2 Gross pathology

Grossly no changes could be detected.

4.1.8.3 Histopathology

Microscopically, these were seen close to the ovarian surface. The cyst wall was lined by a layer of cuboidal epithelium. The cyst lumen was devoid of any contents (Fig. 32).

4.1.9 Epoophoron

4.1.9.1 Incidence

Incidence of epoophoron was recorded in two cases (0.2%) out of the 1000 cases examined.

.4.1.9.2 Gross pathology

Grossly no changes could be detected.

4.1.9.3 Histopathology

Microscopically, acini like structures were noticed in the medulla. The acini had slit-like lumen and were lined by cuboidal epithelium. Some of the acini showed degenerative changes, without cellular infiltrations. Large dilated lymphatic ducts were seen in the medulla (Fig. 33).

4.1.10 Atretic follicles

4.1.10.1 Incidence

Incidence of atretic follicles was recorded in four cases (0.4%).

4.1.10.2 Gross pathology

Grossly no changes could be detected.

4.1.10.3. Histopathology

In the cortex, multiple follicles of various types showed follicular atresia. Degenerated ovum surrounded by collapsed zona pellucida was seen. In the small follicles, the granulosa cells showed nuclear pyknosis and cytoplasmic vacuolation and these desquamated into the cavity which later underwent degeneration. In the larger follicles, degenerated ovum and granulosa cells were seen. This was enclosed by the remnants of the zona pellucida (Fig. 34).

4.1.11 Ovarian sclerosis

4.1.11.1 Incidence

Ovarian sclerosis was recorded in four cases (0.4%). All the cases were unilateral, one case in right ovary and three in the left ovary.

4.1.11.2 Gross pathology

Sclerosed ovaries were small and hard. Neither corpus luteum nor follicles were evident on the surface. The cut surface revealed a dense stroma.

4.1.11.3 Histopathology

The surface epithelium was absent in a few places. Tunica albugenia was thickened. Stroma of the cortex had dense fibrous connective tissue. There were no developing follicles in the ovarian cortex (Fig. 35).

4.1.12. Senile atrophy

4.1.12.1 Incidence

Senile atrophic ovaries were recorded in three cases (0.3%) and all were bilateral.

4.1.12.2 Gross pathology

The senile ovaries were pale and waxy with a rough pitted surface. The ovarian cortex had numerous pin head sized follicles, red bodies and corpus albicans (Fig. 12).

4.1.12.3 Histopathology

The cortical areas were reduced. There were only a few primary follicles in the cortex, wherein the squamous cells were transformed into a single layer of columnar granulosa cells. Tunica albugenia was thickened. The surface epithelium was almost completely missing and these cells had lost their cuboidal character and became flattened bearing pyknotic nuclei.

4.1.13. Perioophoritis

4.1.13.1 Incidence

Perioophoritis was observed in three (0.3%) out of the 1000 cases examined.

4.1.13.2 Gross pathology

Grossly the ovarian surface was shaggy in appearance. The total ovario-bursal adhesion was seen in the affected ovary resulting in the encapsulation of the ovary.

4.1.13.3 Histopathology

Periovarian connective tissue was seen to be infiltrated by lymphoid cells, plasma cells and a few mononuclear cells. The surface epithelium was absent focally and the tunica albugenia was infiltrated with lymphocytes and few macrophages (Fig. 36).

4.2 OVIDUCT

Out of the 1000 genital tracts examined, seven cases showed lesions of varying frequency in the oviduct. The conditions encountered were hydrosalpinx, hyperplasia of the salpingeal epithelium and ovario-bursal adhesion (Table 3 and Fig. 4). The pathological features observed in each case were as follows.

4.2.1 Hydrosalpinx

4.2.1.1 Incidence

Hydrosalpinx was recorded in two cases (0.2%) out of 1000 genital tracts. Both were seen in the right oviduct.

4.2.1.2 Gross pathology

The fallopian tube was distended with a clear amber coloured fluid. The distension was more towards the ampulla. The wall of the tube was thin and translucent (Fig 13).

4.2.1.3 Histopathology

Microscopically, the mucosal epithelial folds were considerably flattened and there was dilation of the lumen of the oviduct. The mucosa was lined by low cuboidal to columnar epithelium without cilia. Atrophy of the muscular coat and lamina propria were observed (Fig. 37).

4.2.2 Hyperplasia of the salpingeal epithelium

4.2.2.1 Incidence

Hyperplasia of the salpingeal epithelium was recorded in two cases (0.2%).

4.2.2.2 Gross pathology

Grossly thickening of the mucosa was prominent.

4.2.2.3 Histopathology

Microscopically, large proliferating epithelial cells resting on the papillary projections of mucosa were observed. Papillary hyperplasia giving rise to projections resulting in obliteration of the tubular lumen was characteristic (Fig. 38).

4.2.3 Ovario-bursal adhesion

4.2.3.1 Incidence

The condition was seen in 3 (0.3%) genitalia examined. Unilateral bursitis was observed in two genitalia and bilateral bursitis in one genitalia.

4.2.3.2 Gross pathology

Grossly, there is total ovario-bursal adhesion of the affected ovary resulting in the encapsulation of the ovary (Fig. 14).

4.2.3.3 Histopathology

Microscopically, there was heavy infiltration of the oviductal tissue by eosinophils and presence of intraepithelial cysts in the ampulla with fibrous adhesions on the ovary.

4.3 UTERUS

Out of the 1000 genital tracts examined, 36 cases showed lesions of varying frequency in the uterus. The conditions encountered were cystic endometrial hyperplasia, adenomyosis, mucometra, perimetrial cyst, various forms of endometritis and acute suppurative metritis (Table 4 and Fig. 3). The pathological features observed in each case were as follows.

4.3.1 Cystic endometrial hyperplasia

4.3.1.1 Incidence

Cystic endometrial hyperplasia was recorded in six cases (0.6%).

4.3.1.2 Gross pathology

The affected uterus revealed no gross lesions except moderate thickening and uterine horns were slightly enlarged containing small amount of watery fluid.

4.3.1.3 Histopathology

Microscopically, endometrium showed focal areas of hyperplastic changes. Cysts containing acidophilic material with some debris were observed just below the epithelial lining. Glands were of variable sizes. Some showed cystic dilatation and in others hyperplastic changes were seen. Mononuclear cells infiltration and fibrocellular reaction was discernible in the lamina propria. Blood vessels in submucosa were congested. In both cases, cystic endometrial hyperplasia was associated with subacute endometritis. There were diffuse infiltration of neutrophils, monocular cells and occasional desquamation of the epithelium (Fig. 39).

4.3.2 Adenomyosis

4.3.2.1 Incidence

Adenomyosis was recorded in four cases (0.4%).

4.3.2.2 Gross pathology

Grossly, no changes were evident in the affected uterus.

4.3.2.3 Histopathology

Microscopically, affected uterine horns revealed glands along with endometrial stroma in the myometrium. The glands were surrounded by connective tissue and were seen lying deep in the myometrium. In one case degeneration of the endometrial glands was noticed (Fig. 40).

4.3.3 Mucometra

4.3.3.1 Incidence

Mucometra was recorded in four cases (0.4%).

4.3.3.2 Gross pathology

Grossly, both uterine horns were enlarged, asymmetrically distended, thin walled and containing 250-300 ml of turbid, whitish, viscid fluid. The condition was associated with luteal cyst in the ovary. The cotyledons were small flattened and reduced in size (Fig. 15).

4.3.3.3 Histopathology

Microscopically, the endometrium was very thin. The epithelial cells of endometrium were desquamated at places and covered with homogenous pinkish material. The lamina propria also showed mononuclear cell infiltration and mild periglandular fibrosis. At places the endometrium and myometrium were degenerated, atrophied and compressed (Fig. 41).

4.3.4 Perimetrial cyst

4.3.4.1 Incidence

Perimetrial cyst was observed six cases (0.6%)

4.3.4.2 Gross pathology

The cyst was oval in shape and contained clear watery fluid. Cyst was noticed on the serosal surface (Fig. 16).

4.3.4.3 Histopathology

The cyst wall consisted of smooth muscle fibers and lined by a layer of flattened epithelium.

4.3.5 Acute non-suppurative endometritis

4.3.5.1 Incidence

Acute non-suppurative endometritis was encountered in seven goats.

4.3.5.2 Gross pathology

Grossly, the uterine horns were enlarged and oedematous. On cutting, the horns revealed varying quantity of mucus in the lumen. Uterine wall was corrugated and cotyledons appeared enlarged and swollen. Mucosa was congested and os cervix was open (Fig. 17).

4.3.5.3 Histopathology

Microscopically, there was focal desquamation of the endometrial epithelium and oedema in the submucosa and diffuse infiltration with inflammatory cells like lymphocytes and neutrophils. Blood vessels in submucosa and muscularis were engorged with blood. Endometrial glands were proliferated at places (Fig. 42).

4.3.6 Sub acute non-suppurative endometritis

4.3.6.1 Incidence

Sub acute non-suppurative endometritis was recorded in five cases (0.5%).

4.3.6.2 Gross pathology

Grossly, the uterine horns were enlarged with thick walls. Lumen of uterine horns contained thick brownish sticky fluid. Cotyledons were yellowish-brown with eroded surface. Mucous membrane was rough. The endometrium was congested (Fig. 18).

4.3.6.3 Histopathology

Microscopically, there were focal areas of desquamation of the mucosa and the submucosa was diffusely infiltrated with lymphocytes, neutrophils, macrophages and plasma cells. Mild fibro-cellular reaction was discernible around glands. There was periglandular fibrosis and one to three layers of fibrous tissue was seen around the glands. The glandular epithelium showed vacuolation in their cytoplasm (Fig. 43).

4.3.7 Chronic non-suppurative endometritis

4.3.7.1 Incidence

Chronic non-suppurative endometritis was recorded in 2 cases.

4.3.7.2 Gross pathology

Grossly endometrium was dry, devoid of mucus, wrinkled in focal areas and showed grayish discoloration of the uterine mucosa (Fig. 19).

4.3.7.3 Histopathology

Microscopically, there was extreme periglandular fibrosis. All the glands appeared atrophic and slit like. The lining cells showed degeneration and desquamation and some appeared as clumps within the lumen (Fig. 44).

4.3.8 Acute suppurative metritis

4.3.8.1 Incidence

Acute suppurative metritis was recorded in two cases (0.2%).

4.3.8.2 Gross pathology

Grossly, the uterine horns were enlarged, edematous and soft, containing chocolate colored lochia, which was slightly tenacious and without any foul odour. The endometrium was congested and hemorrhagic and the endometrial surface appeared dark red and roughened (Fig. 20).

4.3.8.3 Histopathology

Microscopically, the lumen of uterine horns was filled with homogenous material containing cellular debris with dead and disintegrated neutrophils. Endometrial lining was eroded at many places with frank necrosis. Lamina propria was congested and diffusely infiltrated with neutrophils and a few mononuclear cells. Uterine glands were dilated at places and lined by cuboidal epithelium. Lumen of some of the glands was filled with neutrophils. Moderate perivascular and periductular fibrosis were evident (Fig. 45).

4.4 ENDOCRINE DISORDERS

4.4.1 Pituitary gland

4.4.1.1 Pituitary abscess

4.4.1.1.1 Incidence

Pituitary abscess was recorded in a single case out of the 84 cases examined.

4.4.1.1.2 Gross pathology

Viscous green exudate was noted within the pituitary fossa surrounding the pituitary gland and extending along the caudal fossa of the skull.

4.4.1.1.3 Histopathology

Extensive areas of necrosis accompanied by variable neutrophilic infiltrate effaced predominantly the pars distalis of the pituitary gland (Fig. 46).

4.4.1.2 Pituitary cyst

4.4.1.2.1 Incidence

Pituitary cysts were seen in six cases out of the 84 cases examined.

4.4.1.2.2 Gross pathology

The cysts were seen both in the adenohypophysis and neurohypophysis. Cyst was either single or multiple and the pituitary gland was grossly enlarged with presence of straw coloured turbid fluid in the cystic cavity (Fig. 21).

4.4.1.2.3 Histopathology

The cystic cavity was either empty or filled with homogenous pink staining exudate. The cyst wall was lined by flattened cells. Degeneration, necrosis and hyalinization of acidophils and basophils were seen in the vicinity of the cysts. Widespread haemorrhage was seen adjacent to the cystic spaces (Fig. 47).

4.4.1.3 Pituitary apoplexy

4.4.1.3.1 Incidence

Pituitary apoplexy was seen in two cases out of the 84 cases examined.

4.4.1.3.2 Gross pathology

Grossly there was oedema, hyperemia and severe congestion of the adenohypophysis and the cut section was haemorrhagic (Fig. 22).

4.4.1.3.3 Histopathology

Haemorrhage was seen within the pars distalis. Homogenous pink staining fluid separated the pituicytes within the parenchyma. There was diffuse hypertrophy of the acidophils and basophils in the pars distalis. Acidophils were proportionately much less (Fig. 48).

4.4.2 Thyroid gland

4.4.2.1 Thyroid hypoplasia

4.4.2.1.1 Incidence

Thyroid hypoplasia was seen in seven cases and in all the cases it was bilateral.

4.4.2.1.2 Gross pathology

The thyroid glands were reduced in size, elongated and pale in colour. There was a loss of glistening appearance and was hard in consistency indicating moderate fibrosis (Fig. 23).

4.4.2.1.3 Histopathology

Microscopically, the connective tissue stroma appeared prominent and there was presence of many microfollicles. The lumen was filled with pale staining colloid in some follicles whereas majority of follicles were devoid of colloid. The follicular epithelium was lined by flattened cells (Figs. 49, 50 and 51).

4.4.2.2 Thyroid hyperplasia

4.4.2.2.1 Incidence

Diffuse hyperplastic colloid goitre was seen in five cases and both lateral lobes of the thyroid gland was uniformly affected in all the cases.

4.4.2.2.2 Gross pathology

The thyroid gland was increased in size with dark brown colour. They were soft in consistency with the presence of pale yellow gelatinous material on the cut surface.

4.4.2.2.3 Histopathology

Microscopically, there was presence of progressively distended macrofollicles with pale staining colloid inside the follicles. Follicular cells lining the macrofollicles were flattened and atrophic. Some follicles were found collapsed because of the lack of colloid (Fig. 52).

4.4.2.3 Multifocal nodular hyperplasia

4.4.2.3.1 Incidence

Nodular goitre was observed in two cases and both the lobes were affected.

4.4.2.3.2 Gross pathology

The affected lobes had a moderately enlarged and irregular contour with presence of nodules of varying sizes (Fig. 24).

4.4.2.3.3 Histopathology

Multiple foci of hyperplastic follicular cells and coalescing of some of the follicles to form nodules were seen. The follicles were lined by one or more layers of columnar cells that formed papillary projections into the lumen (Fig. 53).

4.4.3 Adrenal gland

4.4.3.1 Adrenal cortical hypoplasia

4.4.3.1.1 Incidence

Adrenal cortical hypoplasia was noticed in two cases.

4.4.3.1.2 Gross pathology

Decreased size of the adrenal gland with pale brown colour and soft in consistency was noticed in adrenal cortical hypoplasia (Fig. 25).

4.4.3.1.3 Histopathology

Microscopically, there was great reduction in the various zones of the cortex which appeared narrow. There was no clear demarcation of the different cortical zones and vacuolation of the cells were seen (Fig. 54).

4.4.3.2 Adrenal Hyperplasia

4.4.3.2.1 Incidence

Adrenal cortical hyperplasia was seen in five cases and adrenal medullary hyperplasia noticed in eight cases.

4.4.3.2.2 Gross pathology

Grossly, the adrenal gland was enlarged and increased in size. They were hard in consistency and dark brown in colour (Fig. 26).

4.4.3.2.3 Histopathology

Microscopically, in cortical hyperplasia increase in width of all zones of cortex and narrowing of the medullary zone was evident (Fig. 55). In medullary hyperplasia, the medullary zone was very much distended and there was hyperplasia of the cells reducing the cortical segment to a narrow rim under the capsule. Dilatation of the vessels, sinusoids and haemorrhage were seen in the medulla (Fig. 56).

4.4.4 Concurrent lesions in endocrine glands with utero-ovarian disorders

The most prominent gross and histopathological findings of pituitary, thyroid, adrenal glands associated with various utero-ovarian disorders are given in Table 5. The pituitary lesions included pituitary abscess, pituitary cyst and pituitary apoplexy along

with varying degrees of congestion and oedema. The thyroid gland lesions recorded were hypoplasia, diffuse hyperplastic colloid goitre and multifocal nodular hyperplasia. The lesions in adrenal gland noticed were cortical hypoplasia, cortical and medullary hyperplasia.

Lesions of pituitary glands were recorded in nine cases. Pituitary abscess was recorded in a single case in association with acute suppurative metritis. Pituitary gland cysts were seen in six cases in association with cystic ovarian lesions which included follicular and luteal cysts and a case of follicular atresia. Pituitary apoplexy along with congestion and oedema was seen in association with a case of both follicular cyst and senile atrophy.

Lesions of thyroid glands were recorded in 14 cases. These were hypoplasia in seven cases, colloid goiter in five cases and nodular goiter in two cases. The hypoplasia was recorded in the ovarian hypoplasia, follicular cyst, senile atrophy and cystic endometrial hyperplasia. The colloid goitre was seen in ovarian hypoplasia, follicular cyst, atretic follicle and senile atrophy. The nodular goitre was recorded in ovarian hypoplasia and atretic follicles.

Lesions of adrenal gland were seen in 15 cases which included cortical hypoplasia in two cases, cortical hyperplasia in five cases and medullary hyperplasia in eight cases. The cortical hypoplasia was seen in ovarian hypoplasia. The cortical and medullary hyperplasia was seen in ovarian hypoplasia, follicular cyst, luteal cyst, atretic follicle and cystic endometrial hyperplasia.

TABLE 1. PREVALENCE OF UTERO-OVARIAN LESIONS

S.No	Type of organ	No of eases examined	No of cases with lesions
1	Ovary	1000	54
2	Oviduct	1000	7
3	Uterus	1000	36

TABLE 2. PREVALENCE OF OVARIAN LESIONS

S.No.	Type of ovarian abnormality	Left ovary	Right ovary	Bilate-ral	Total	Per cent
1	Ovarian hypoplasia	4	2	-	6	0.6
2	Follicular cyst	6	8	-	14	1.4
3	Luteal cyst	-	3	-	3	0.3
4	Cystic corpus luteum	1	2	-	3	0.3
5	Parovarian cyst	3	1	2	6	0.6
6	Embedded corpus luteum	1	2	-	3	0.3
7	Persistent corpus luteum	-	2	-		0.2
8	Ovarian haematoma	1	1	-	2	0.2
9	Serous inclusion cyst	1	1	-	2	0.2
10	Epoophoron	2	-	-	2	0.2
11	Atretic follicles	1	1	2	4	0.4
12	Ovarian sclerosis	3	1	-	4	0.4
13	Senile atrophy	-	-	3	3	0.3
14	Perioophoritis	2	1	-	3	0.3

TABLE 3. PREVALENCE OF OVIDUCT LESIONS

S.No	Type of oviductal abnormality	Left	Right	Bilat-eral	Total	Per cent
1	Hydrosalpinx	-	2	-	2	0.2
2	Salpingeal hyperplasia	-	1	1	2	0.2
3	Ovario-bursal adhesion	1	1	1	3	0.3

TABLE 4. PREVALENCE OF UTERINE LESIONS

S.No	Type of uterine abnormality	No of cases	Per cent
1	Cystic endometrial hyperplasia	6	0.6
2	Adenomyosis	4	0.4
3	Mucometra	4	0.4
4	Perimetrial cyst	6	0.6
5	Acute non-suppurative endometritis	7	0.7
6	Sub acute non-suppurative endometritis	5	0.5
7	Chronic non-suppurative endometritis	2	0.2
8	Acute suppurative metritis	2	0.2

TABLE 5. PREVALENCE OF ENDOCRINE GLAND LESIONS IN UTERO – OVARIAN DISORDERS

S.No.	Utero-Ovarian Disorders	Endocrine gland lesions								
		Pituitary			Thyroid			Adrenal		
		Abscess	Cyst	Apo-plexy	Hypo-plasia	Colloid goitre	Nodular goitre	Cortical hypo-plasia	Cortical hyper-plasia	Medullary hyper-plasia
1	Ovarian hypoplasia				1	2	1	2		1
2	Follicular cyst		3	1	3	1			3	3
3	Luteal cyst		2		1				2	2
4	Atretic follicles		1			1	1			1
5	Senile atrophy			1	1	1				
7	Cystic endometrial hyperplasia				1					1
8	Acute suppurative metritis	1								
	Total	**1**	**6**	**2**	**7**	**5**	**2**	**2**	**5**	**8**

Fig.1.Distribution of different utero - ovarian lesions

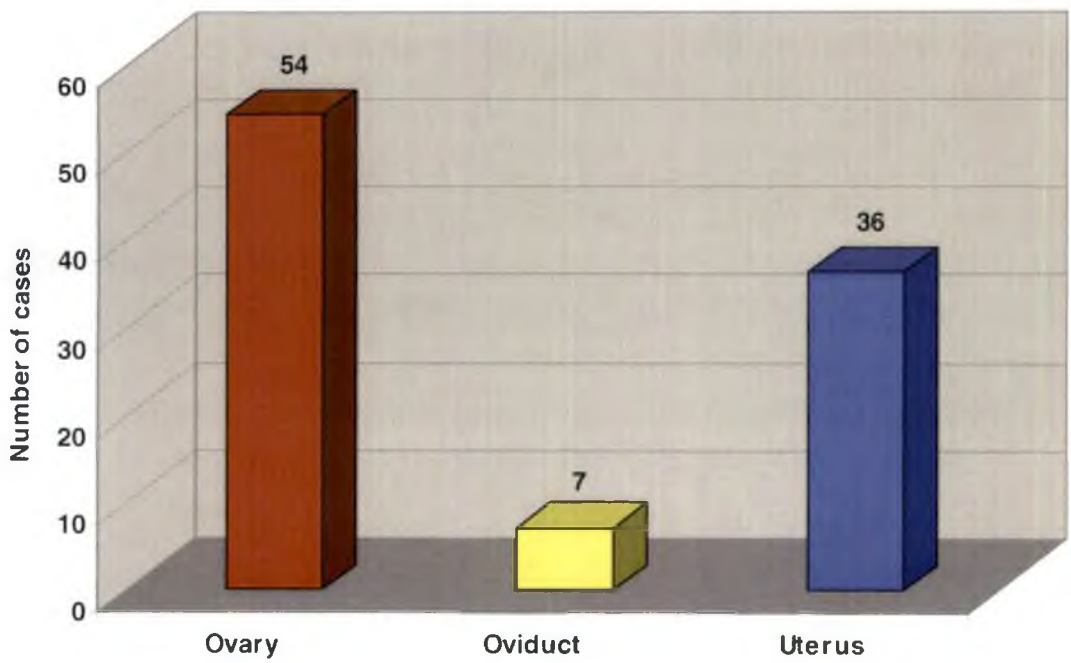


Fig.2. Distribution of different ovarian lesions

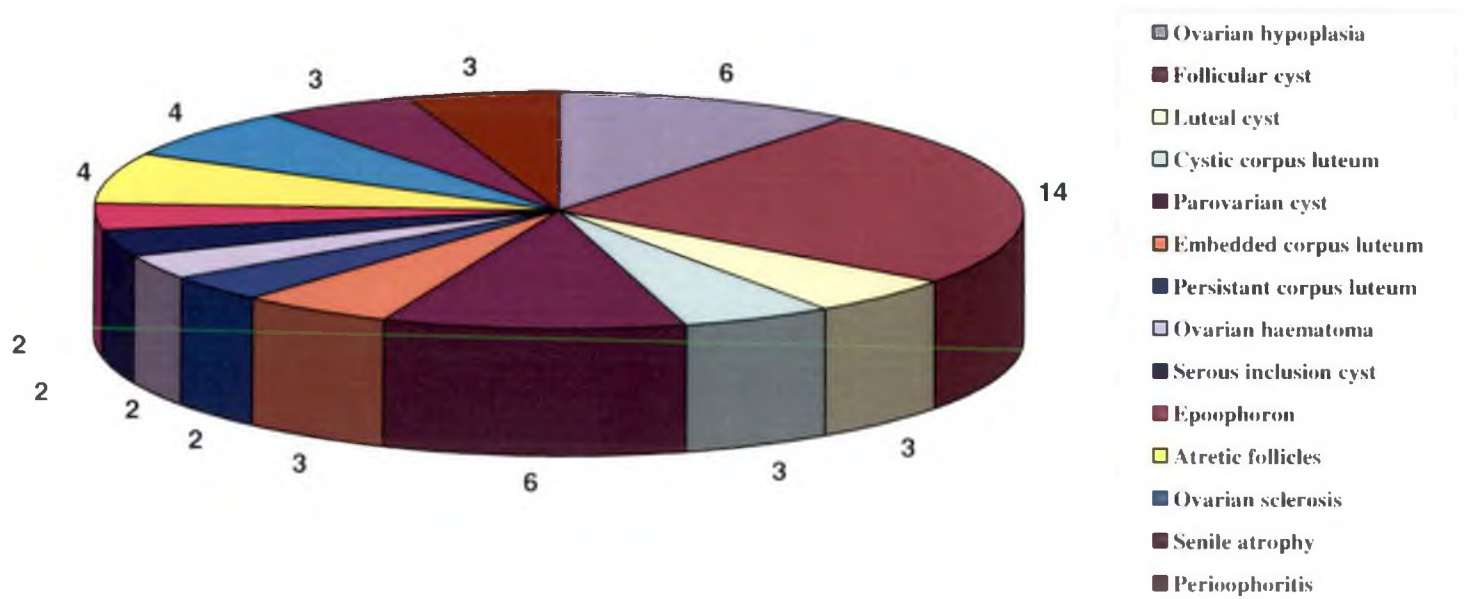


Fig.3. Distribution of uterine lesions

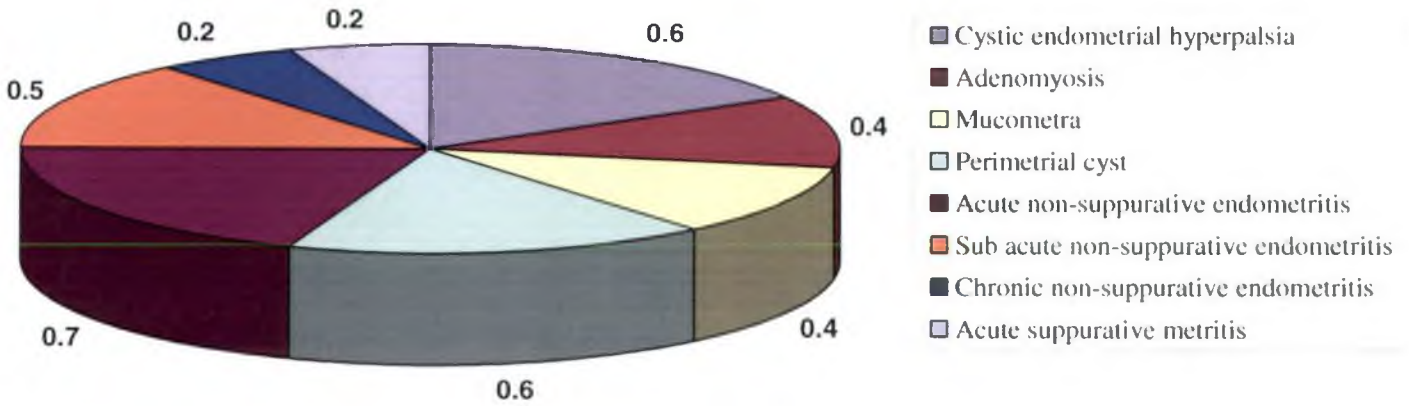


Fig.4. Distribution of oviduct lesions

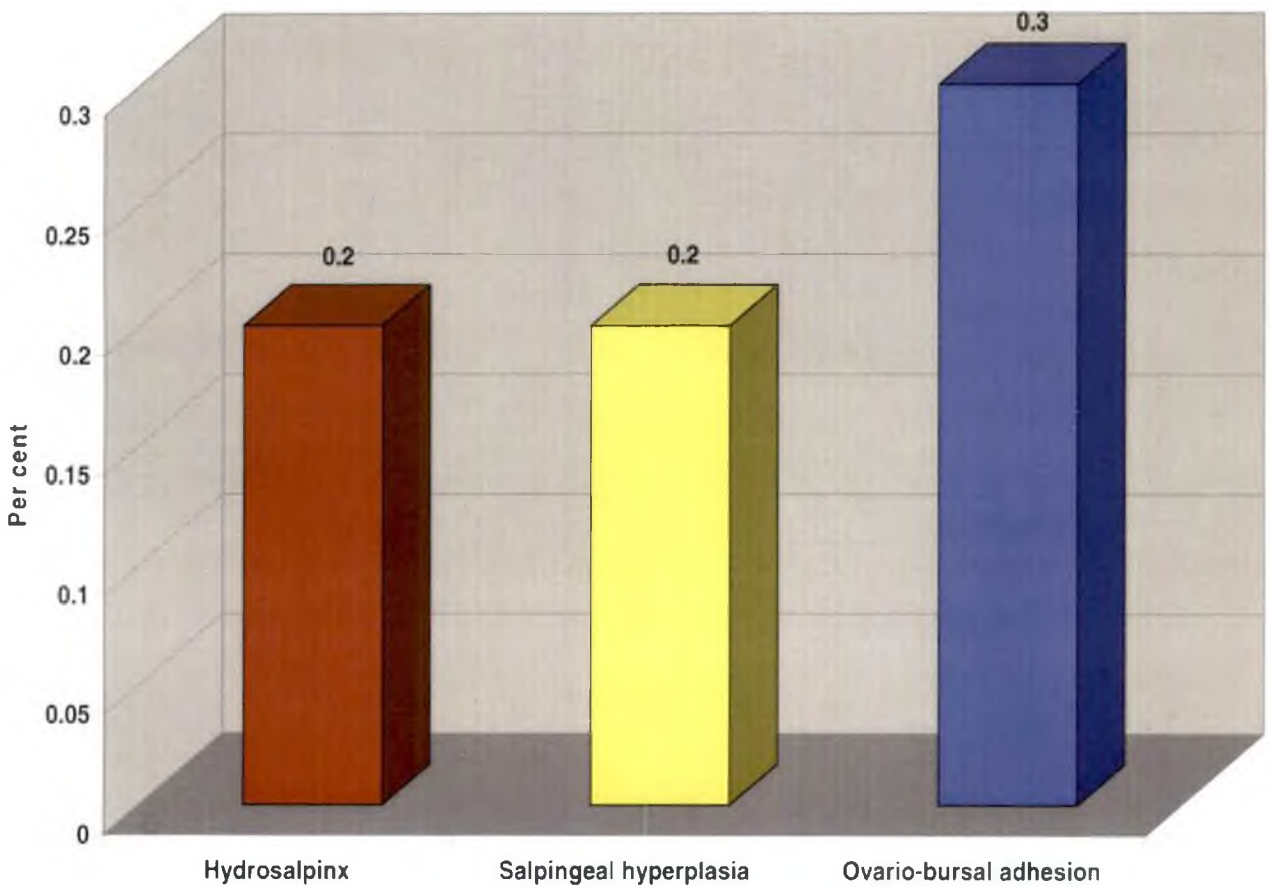
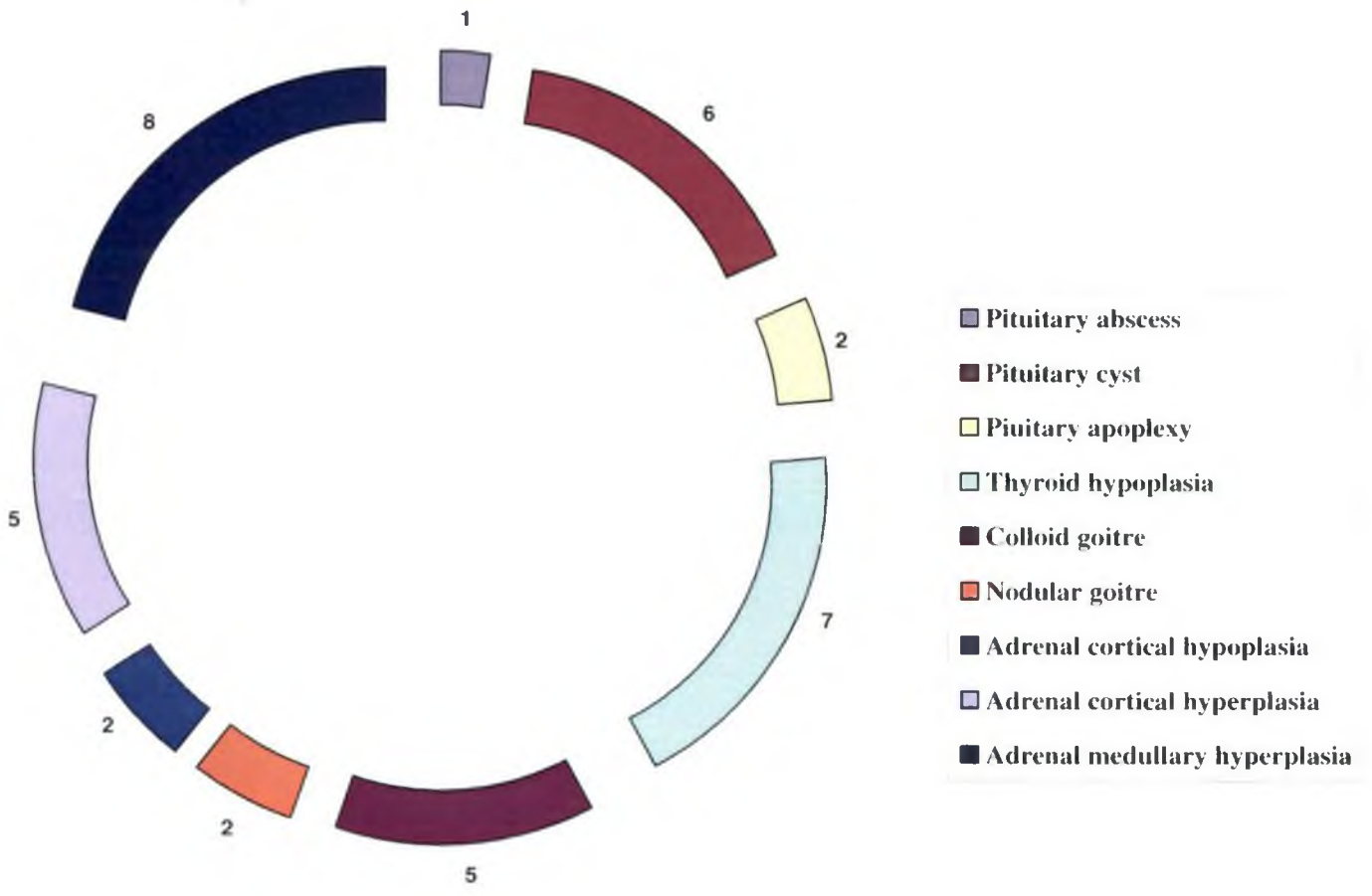


Fig.5. Distribution of lesions in endocrine glands



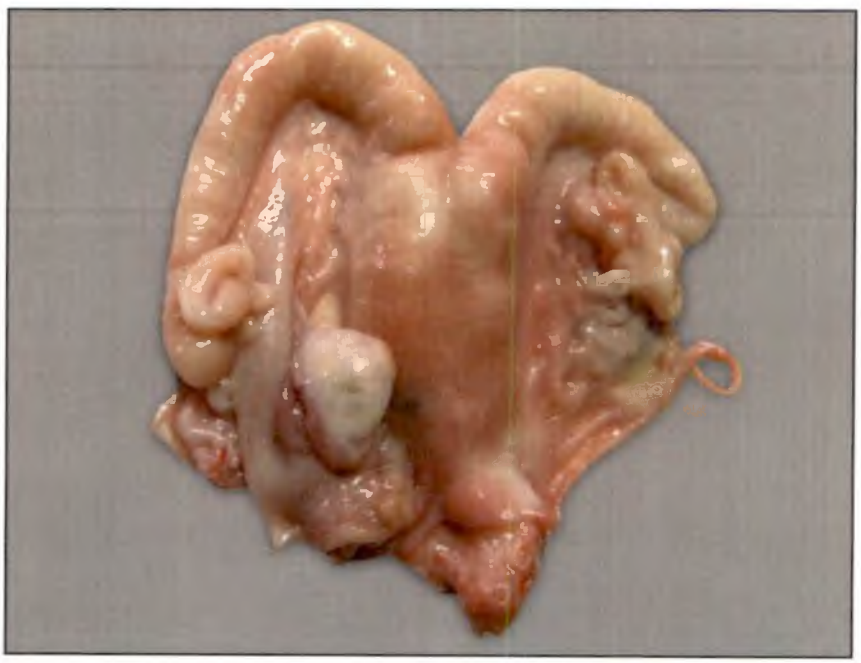


Fig. 6.

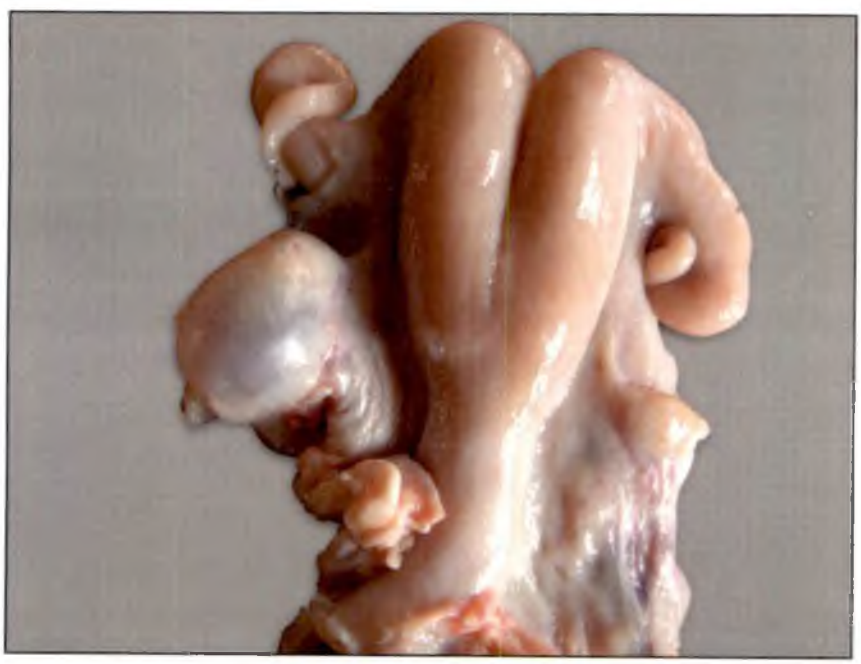
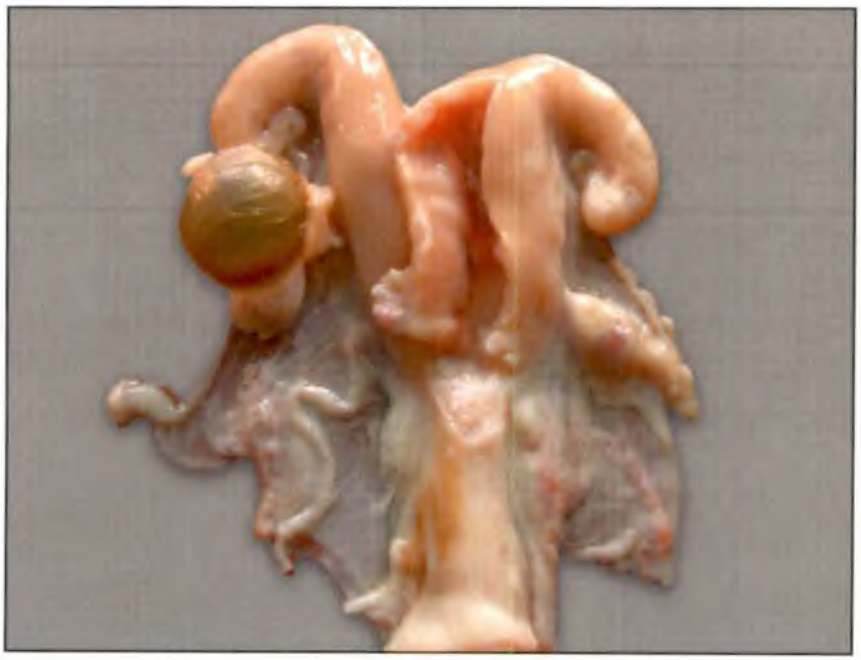


Fig. 7.

Fig. 6. Hypoplasia of the right ovary

Fig. 7. Follicular cyst in the left ovary



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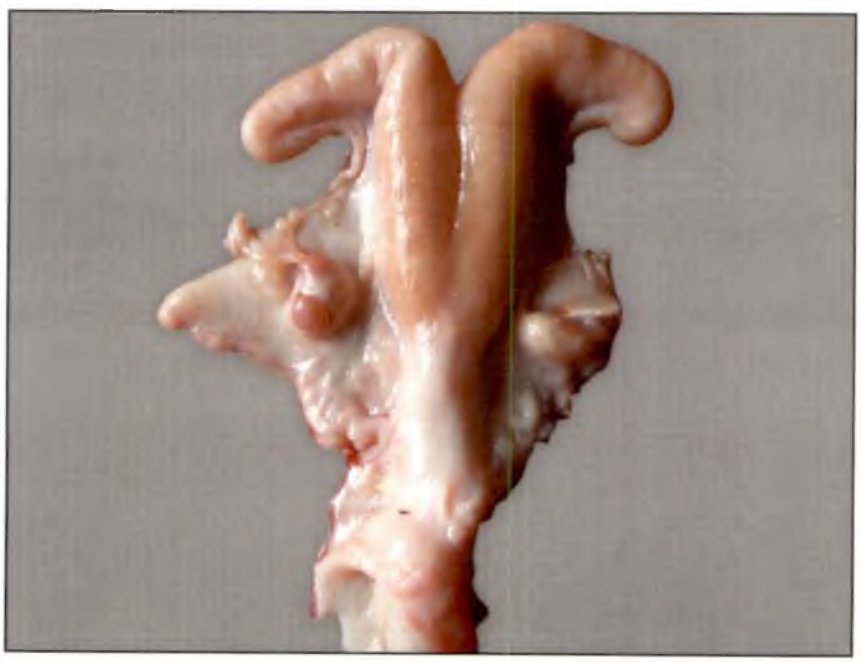


Fig. 9.

Fig. 8. Luteal cyst in the left ovary

Fig. 9. Parovarian cyst in the right side

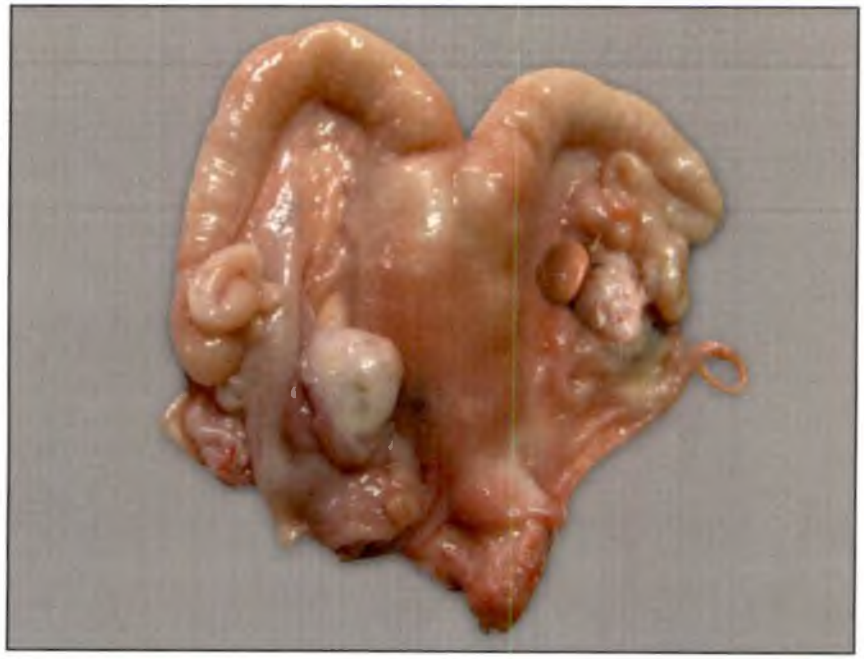


Fig. 10.

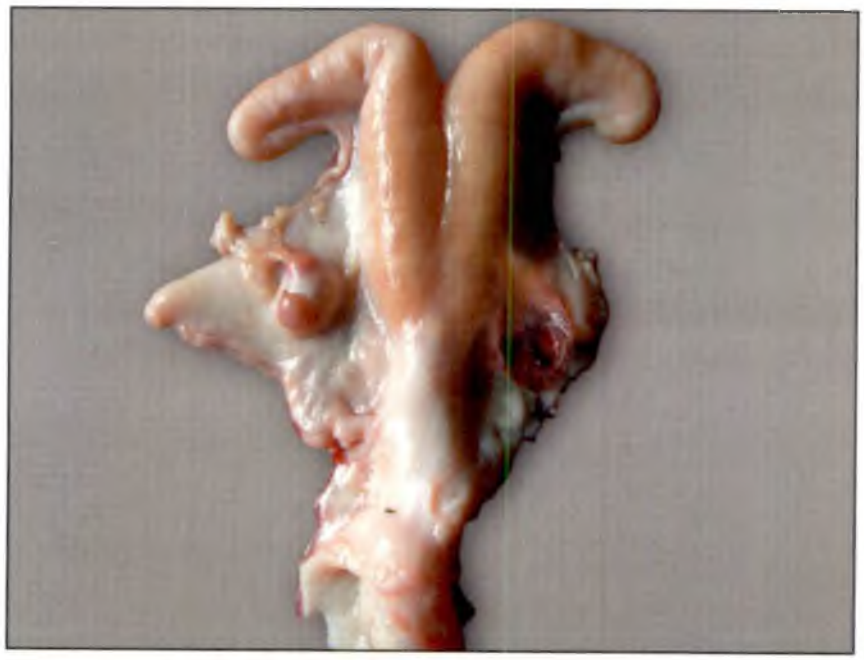


Fig. 11.

Fig. 10. Persistent corpus luteum on the right ovary

Fig. 11. Haematoma in the right ovary

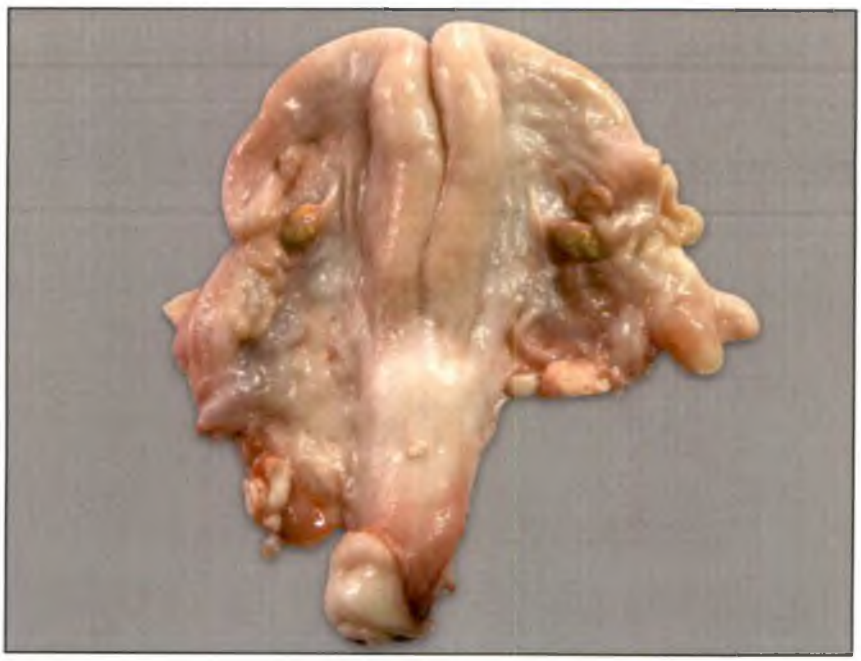
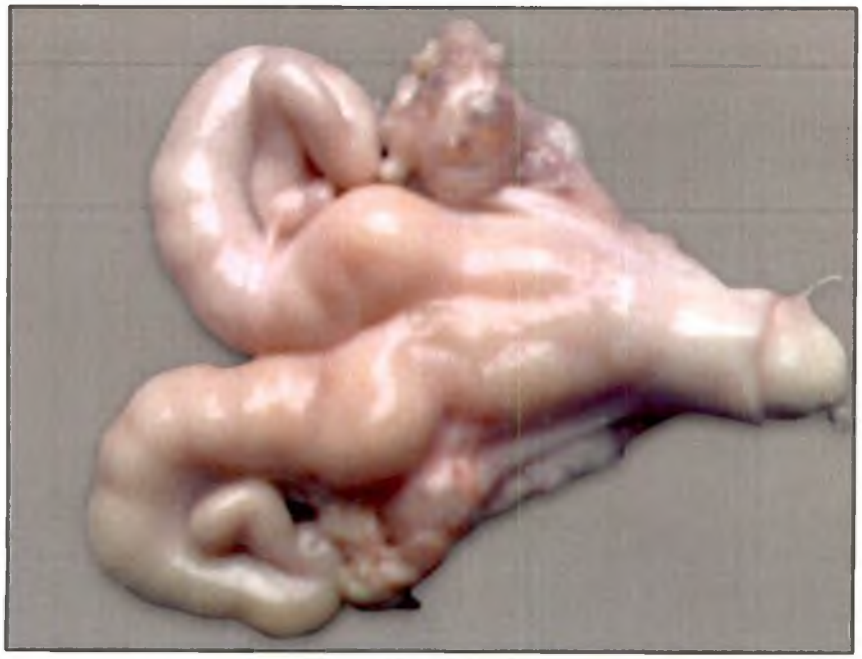


Fig. 12.

Fig. 13.

Fig. 12. Senile ovaries

Fig. 13. Hydrosalpinx of the left side



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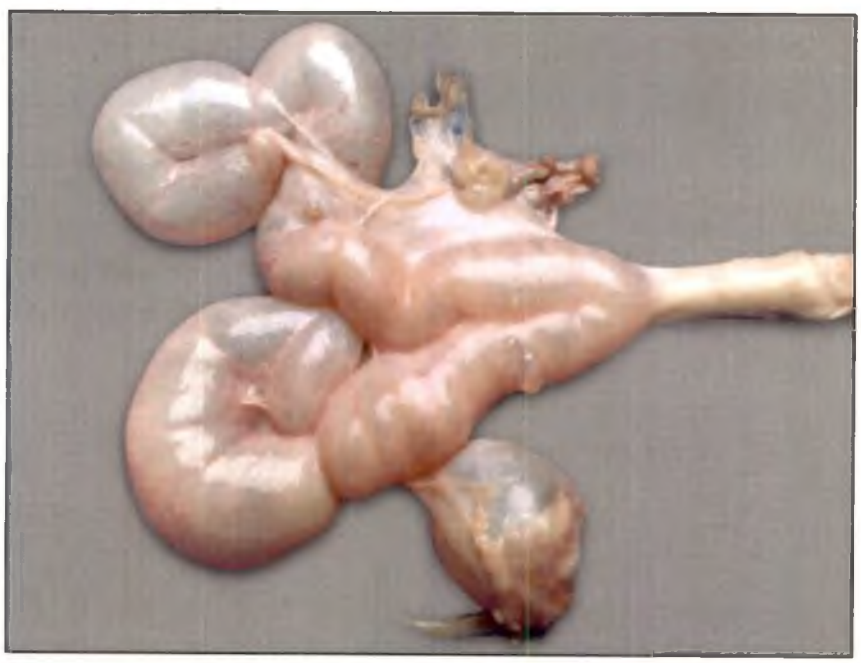

Fig. 15.

Fig. 14. Bilateral ovario-bursal adhesion

Fig. 15. Mucometra

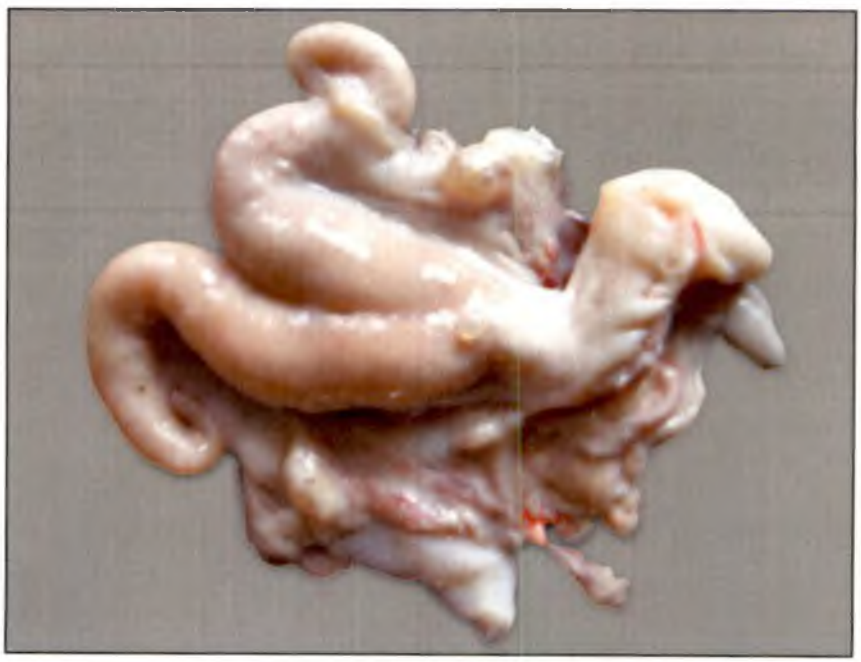

Fig. 16.

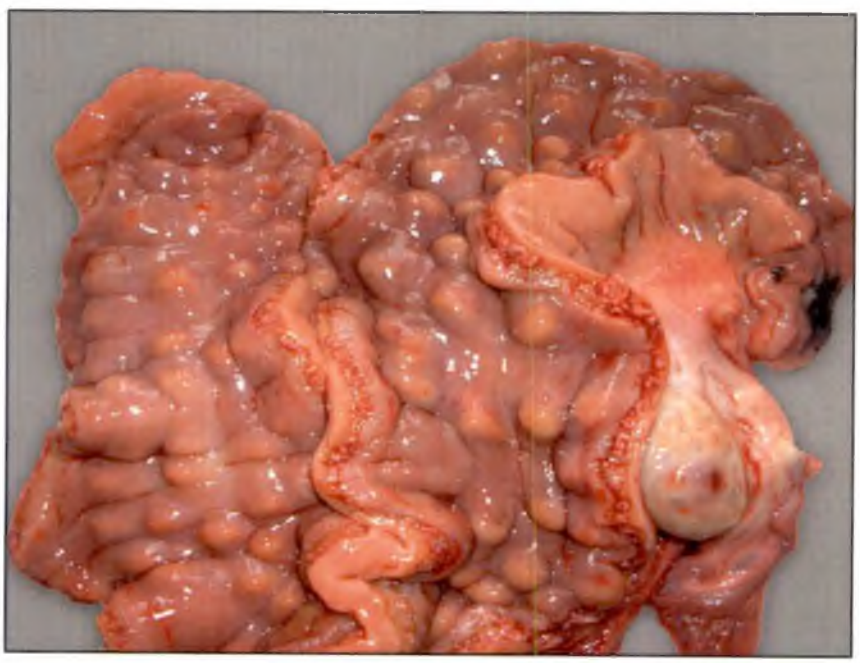

Fig. 17.

Fig. 16. Perimetrial cyst

Fig. 17. Acute non-suppurative endometritis

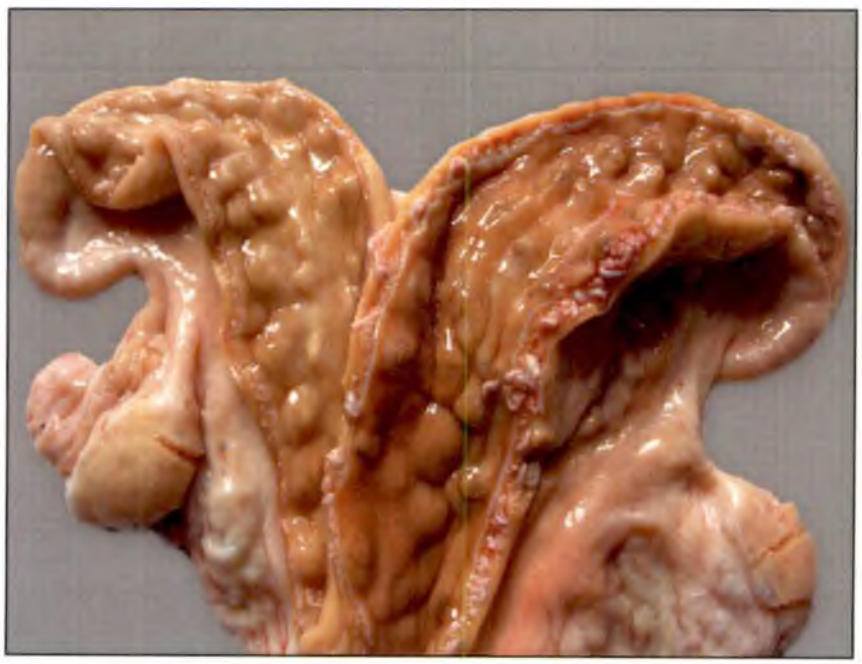

Fig. 18.

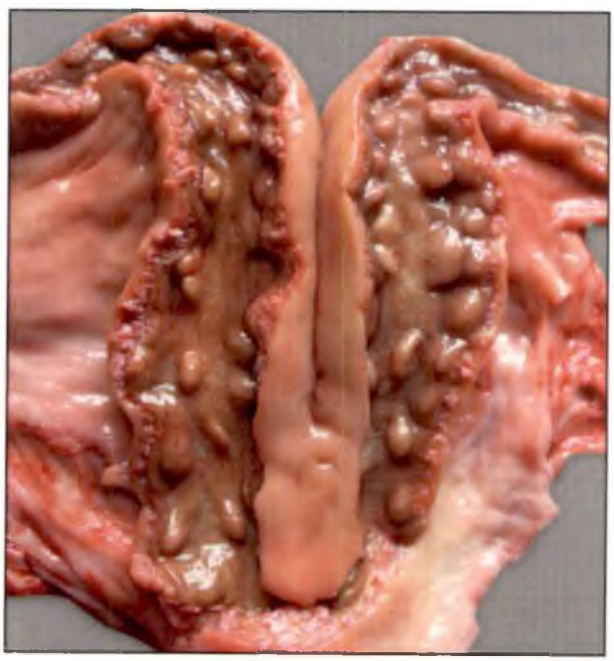

Fig. 19.

Fig. 18. Sub acute non-suppurative endometritis

Fig. 19. Chronic non-suppurative endometritis

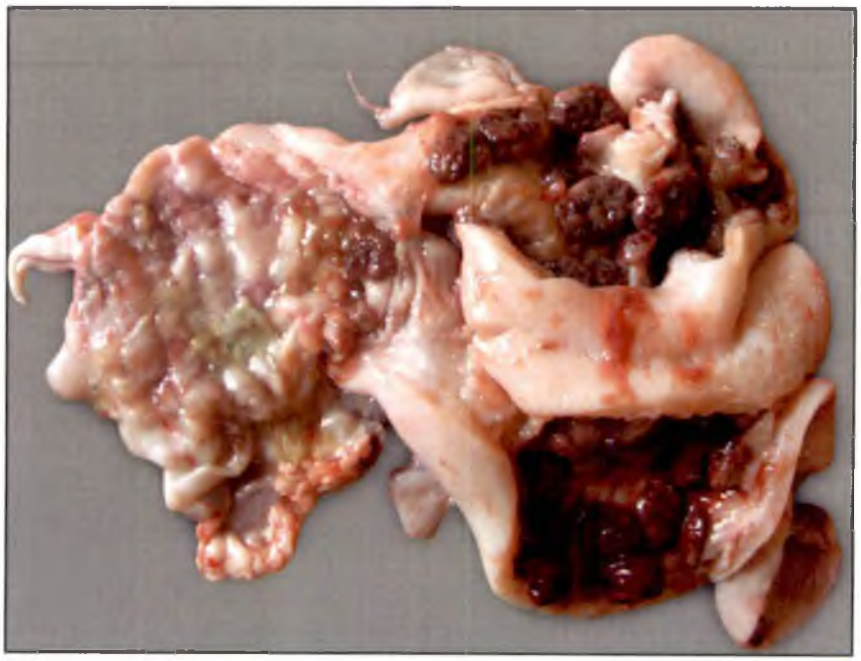

Fig. 20.

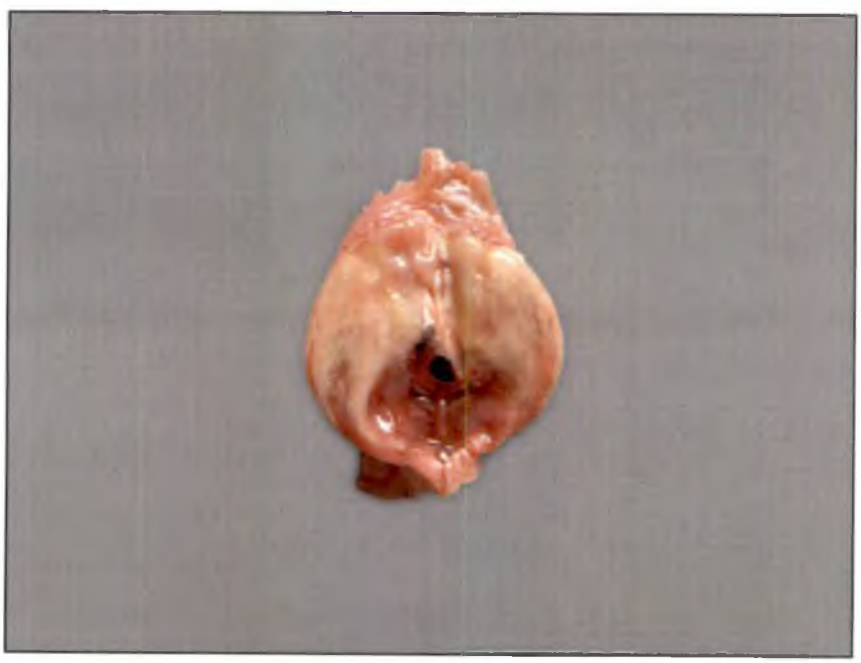

Fig. 21.

Fig. 20. Acute suppurative metritis

Fig. 21. Pituitary cyst

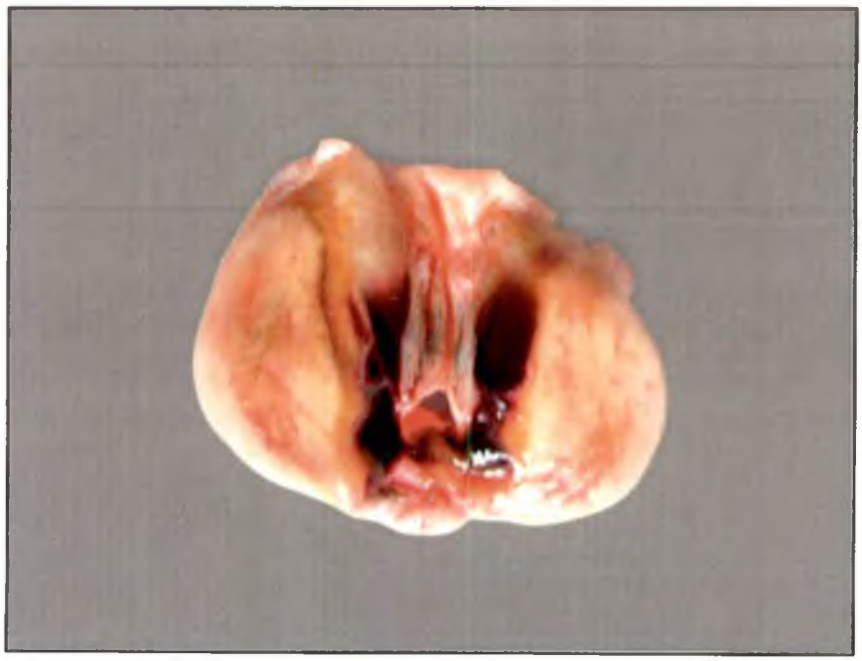

Fig. 22.

Fig. 23.

Fig. 22. Pituitary apoplexy

Fig. 23. Thyroid hypoplasia (left-normal thyroid)

Fig. 24.

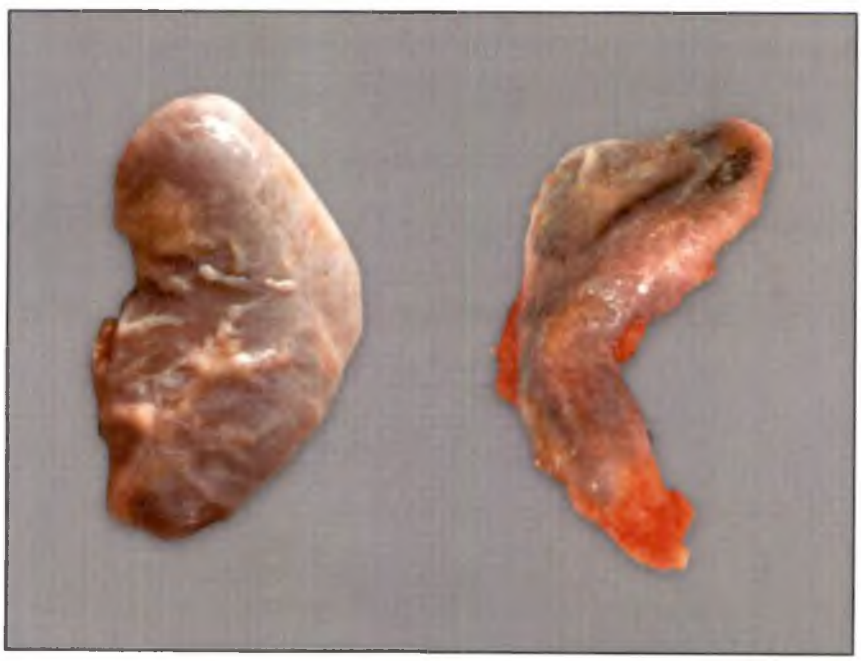

Fig. 25.

Fig. 24. Nodular goitre

Fig. 25. Adrenal hypoplasia (left - normal adrenal)

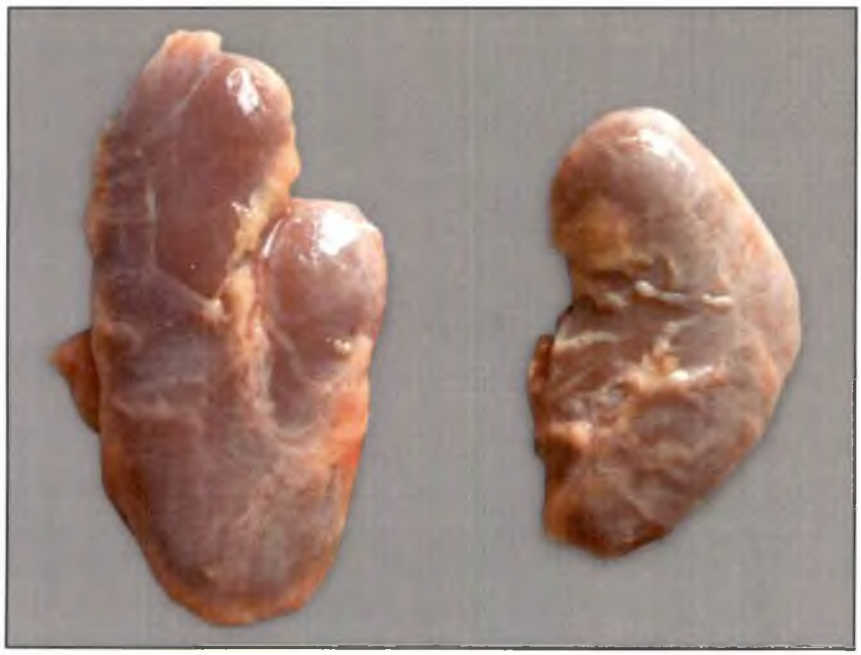

Fig. 26.

Fig. 26.Adrenal hyperplasia (right - normal adrenal)

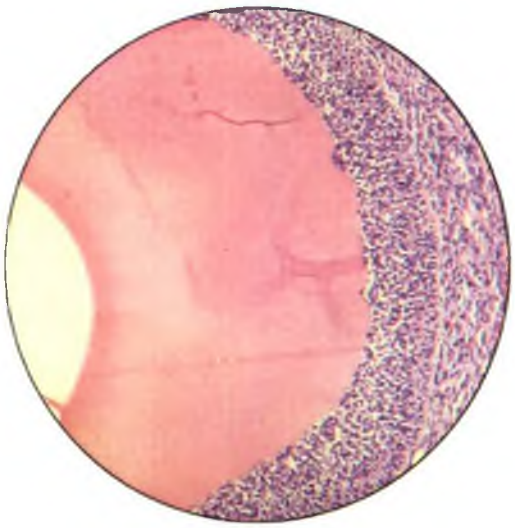

Fig.27.

Figure. 27. Follicular cyst.
H&Ex100

Multiple rows of granulosa cells with acidophilic material in the lumen

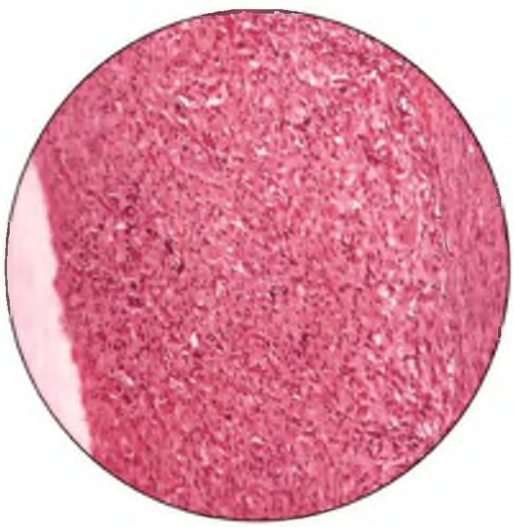

Fig. 28.

Figure. 28. Luteal cyst.
H&Ex100

Inner layer of connective tissue seperating the luteal tissue from the cystic contents

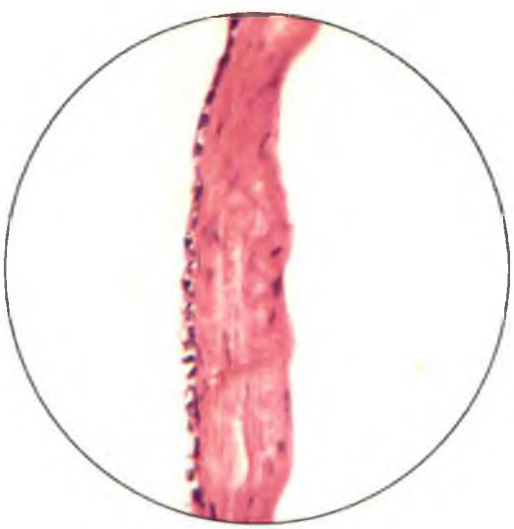

Fig. 29.

Figure. 29. Parovarian cyst.
H&Ex100

Cyst wall comprising of muscle and connective tissue and lined by a layer of cuboidal epithelium

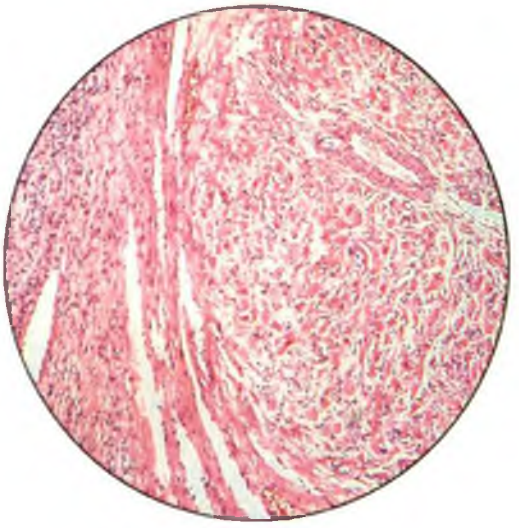

Fig. 30.

Figure. 30. Embedded corpus luteum. H&Ex100

Outer connective tissue capsule seperating corpus luteum from surrounding ovarian stroma

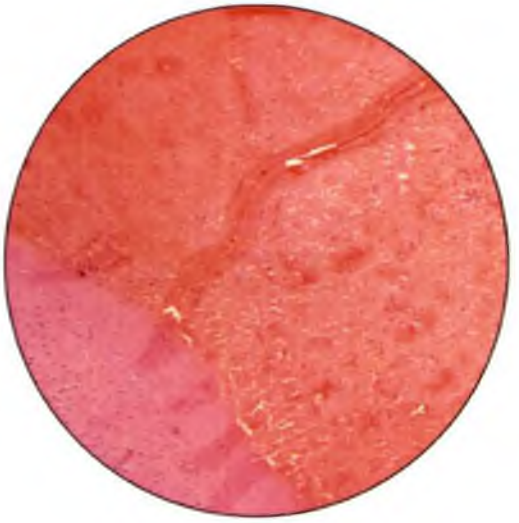

Fig. 31.

Figure. 31. Ovarian haematoma. H&Ex100

Well organised haematoma encapsulated by ovarian stroma

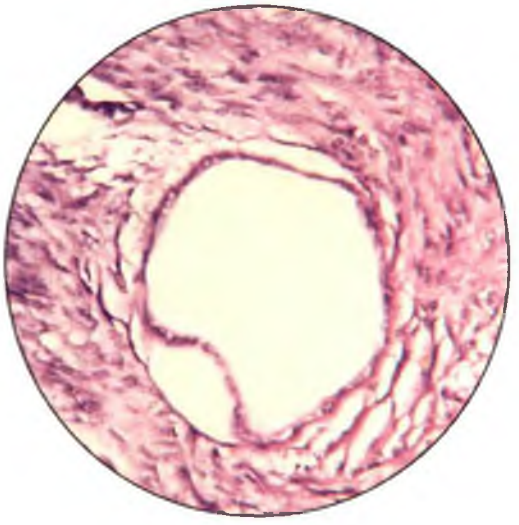

Fig. 32.

Figure. 32. Serous inclusion cyst. H&Ex400

Cystic lumen without any content and wall lined a layer of cuboidal epithelium

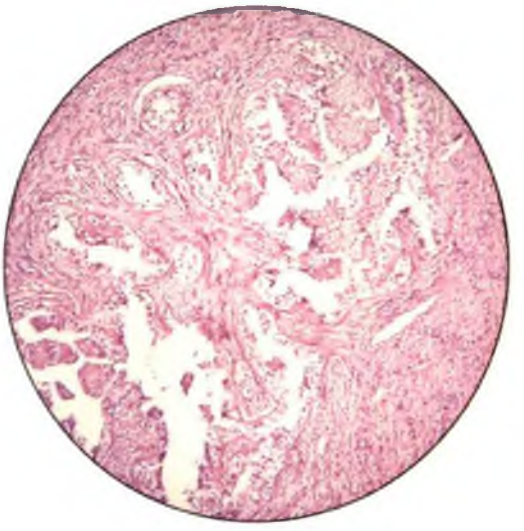

Fig. 33.

Figure. 33. Epoophoron.
H&Ex100

Group of acini like structures in the ovarian medulla

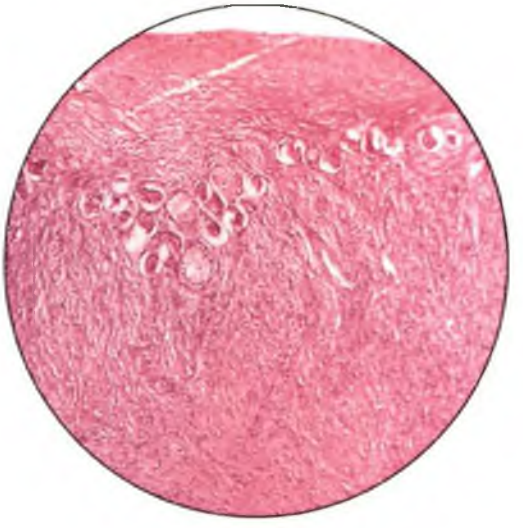

Fig. 34.

Figure. 34. Atretic follicles.
H&Ex100

Group of atretic follicles in the ovarian cortex

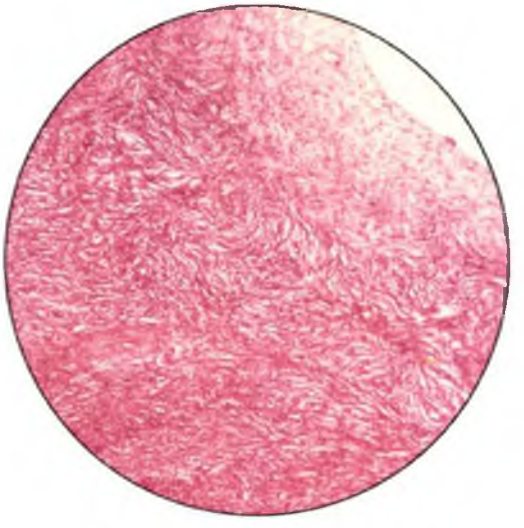

Fig. 35.

Figure. 35. Ovarian sclerosis.
H&Ex100

Fibrous connective tissue stroma devoid of developing follicles

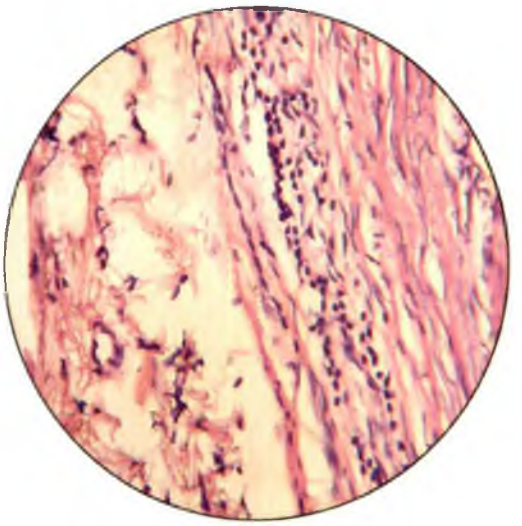

Fig. 36.

Figure. 36. Perioophoritis.
H&Ex400

Infiltration of lymphocytes and few macrophages

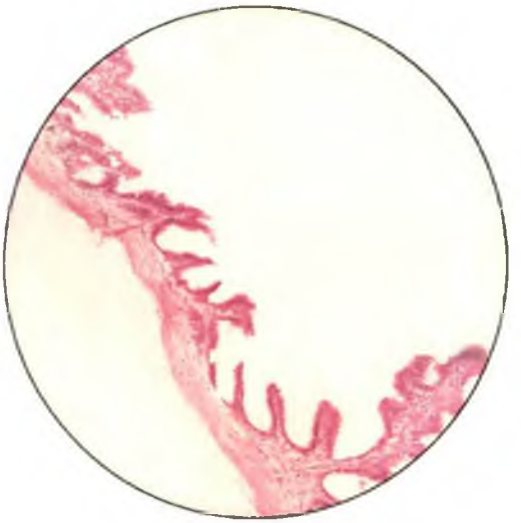

Fig. 37.

Figure. 37. Hydrosalpinx.
H&Ex100

Dilatation of the lumen and flattened mucosal epithelial folds

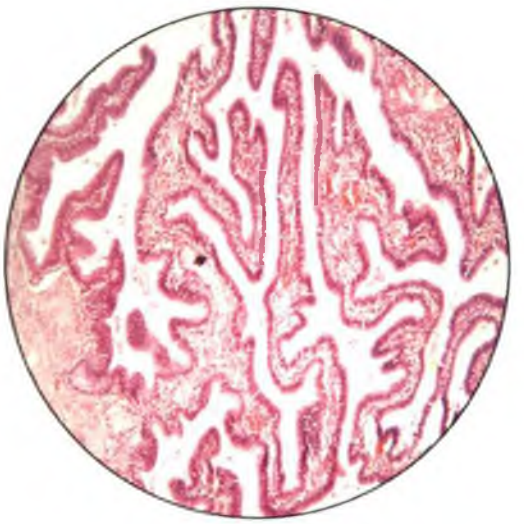

Fig. 38.

Figure. 38. Salpingeal hyperplasia.
H&Ex100

Vascular congestion and hyperplasia of epithelial folds

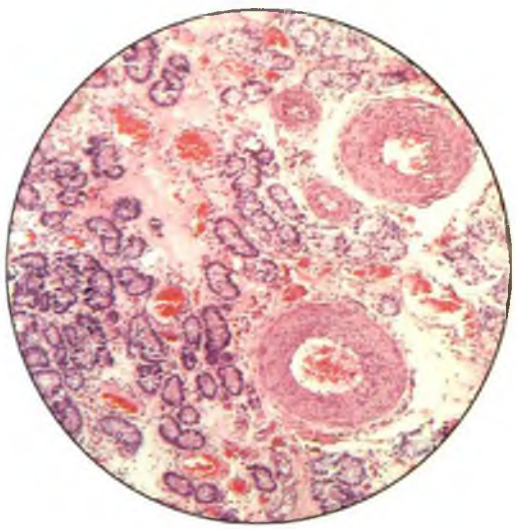

Fig. 39.

Figure. 39. Cystic endometrial hyperplasia. H&Ex100

Glandular hyperplasia and vascular congestion

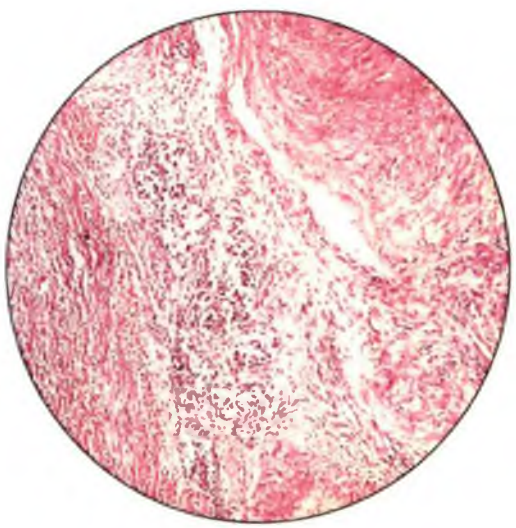

Fig. 40.

Figure. 40. Adenomyosis. H&Ex100

Presence of endometrial glands in the myometrium

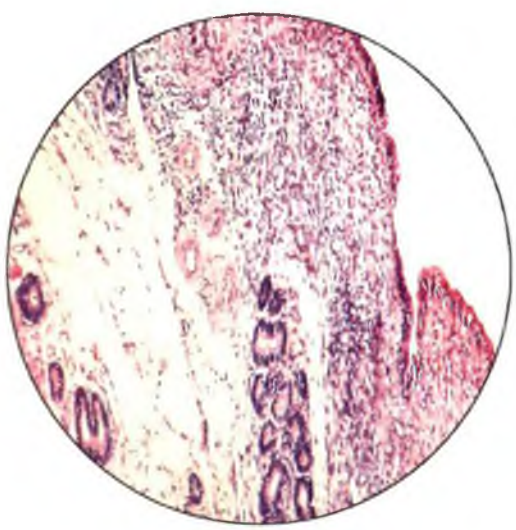

Fig. 41.

Figure. 41. Mucometra. H&Ex100

Thin endometrium with reduced number of uterine glands

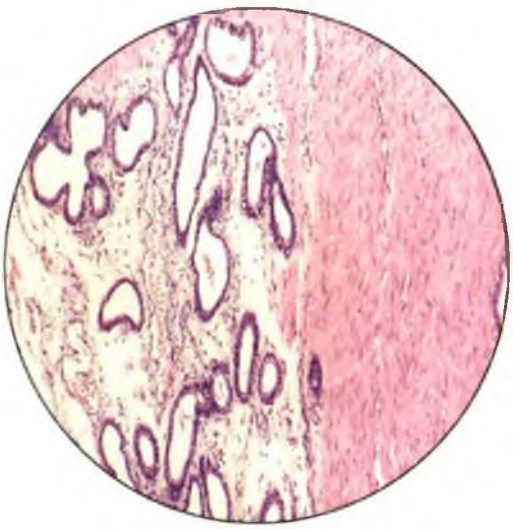

Fig. 42.

Figure. 42. Acute non-suppurative endometritis.
H&Ex100

Proliferation of endometrial glands with infiltration of neutrophils

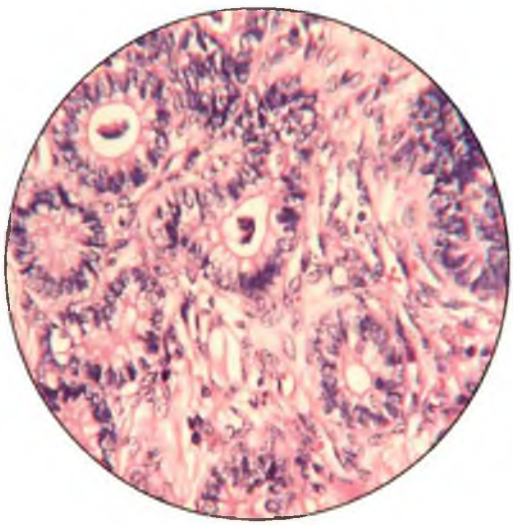

Fig. 43.

Figure. 43. Sub acute non-suppurative endometritis.
H&Ex400

Diffuse inflammatory cell infiltration and periglandular fibrosis

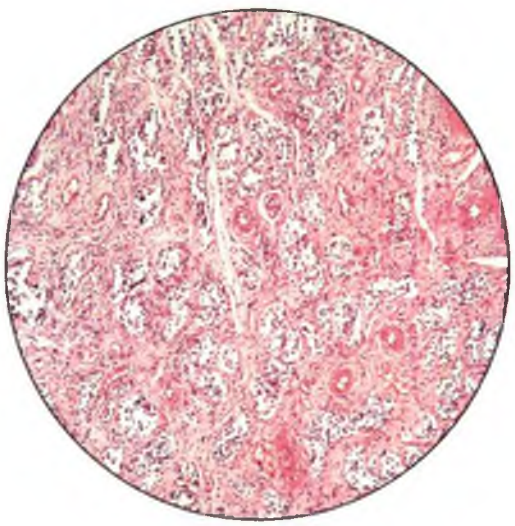

Fig. 44.

Figure. 44. Chronic non-suppurative endometritis.
H&Ex100

Atrophy of glands and extreme periglandular fibrosis

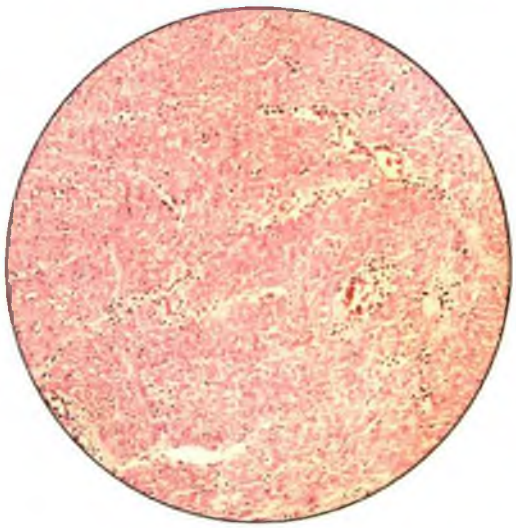

Fig. 45.

Figure. 45. Acute suppurative metritis. H&Ex100

Congestion of lamina propria and diffuse infiltration of neutrophils and mononuclear cells

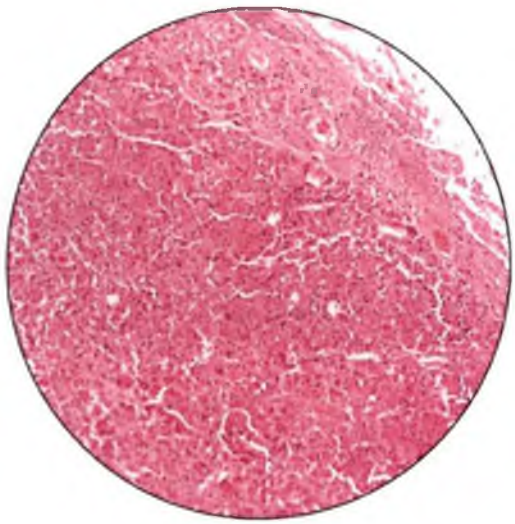

Fig. 46.

Figure. 46. Pituitary abscess, H&Ex100

Areas of necrosis, congestion and infiltration of neutrophils

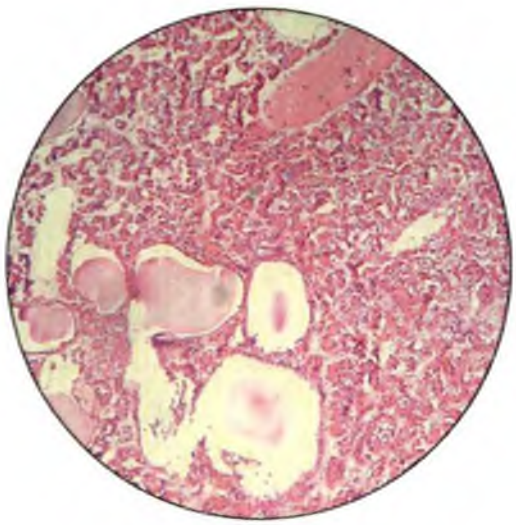

Fig. 47.

Figure. 47. Pituitary cyst. H&Ex100

Cystic cavity filled with homogenous pink staining material and haemorrhage adjacent to cysts

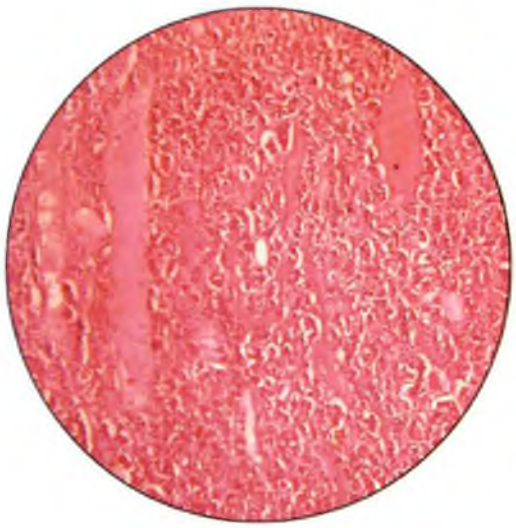

Fig. 48.

Figure. 48. Pituitary apoplexy. H&Ex100

Presence of haemorrhage and oedema

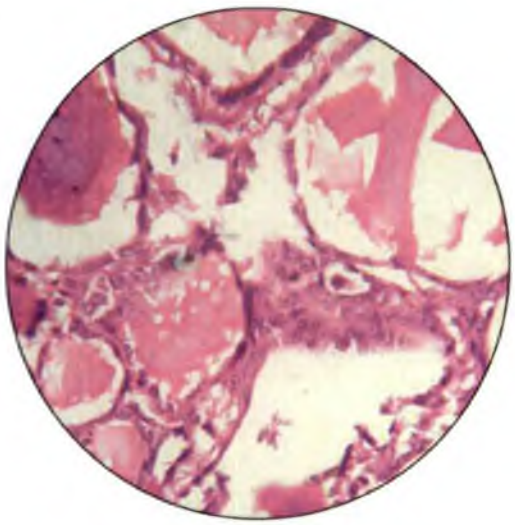

Fig. 49.

Figure. 49. Thyroid hypoplasia. H&Ex400

Collapse of follicle due to lack of colloid and presence of distended macrofollicles

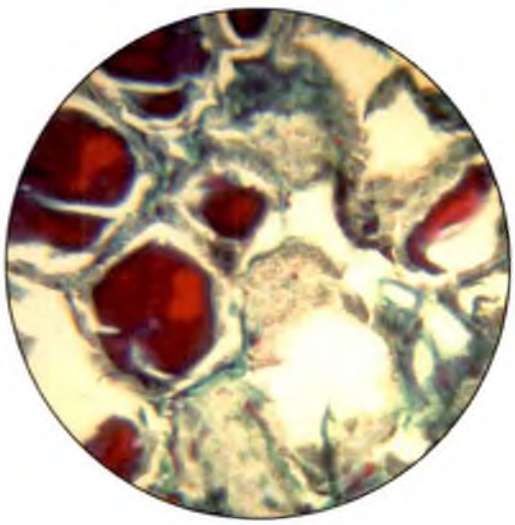

Fig. 50.

Figure. 50. Thyroid hypoplasia. Trichrome x 400

Colloid seen red and fibrous tissue seen green

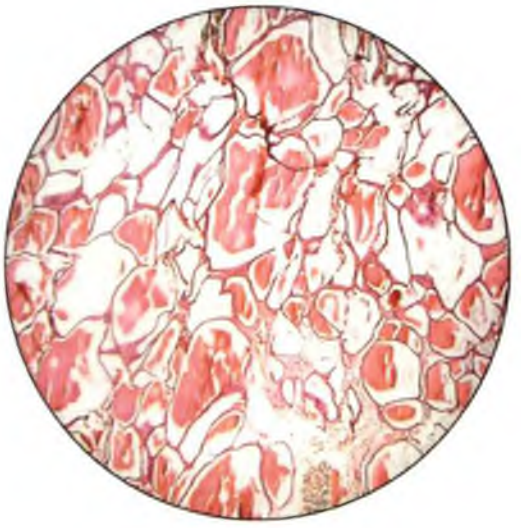

Fig. 51.

Figure. 51. Thyroid hypoplasia.
PAS x 400

Follicles with scanty or no colloid

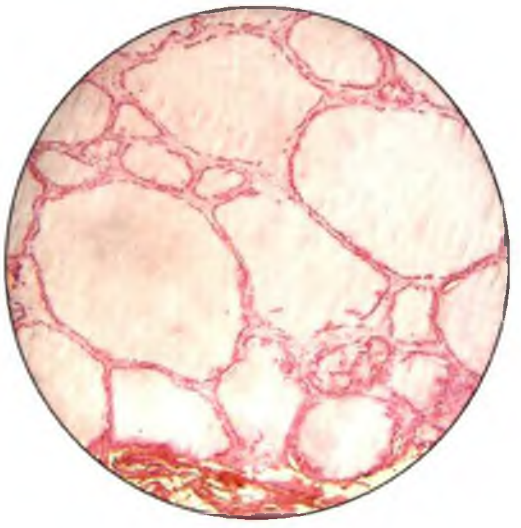

Fig. 52.

Figure. 52. Colloid goitre.
H&Ex400

Distended follicles lined by flattened cells

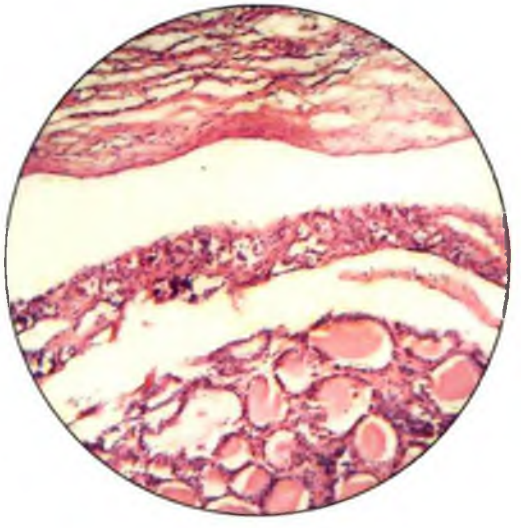

Fig. 53.

Figure. 53. Nodular goitre.
H&Ex400
Follicles lined by multiple layer of cells and compression of adjacent follicles

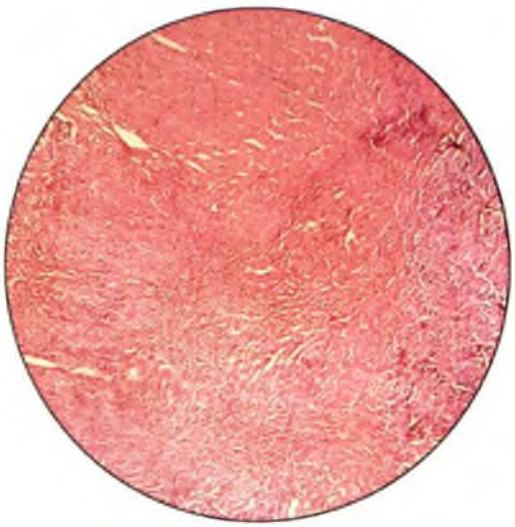

Fig. 54.

Figure. 54. Adrenal cortical hypoplasia. H&Ex40

Narrow cortical zone

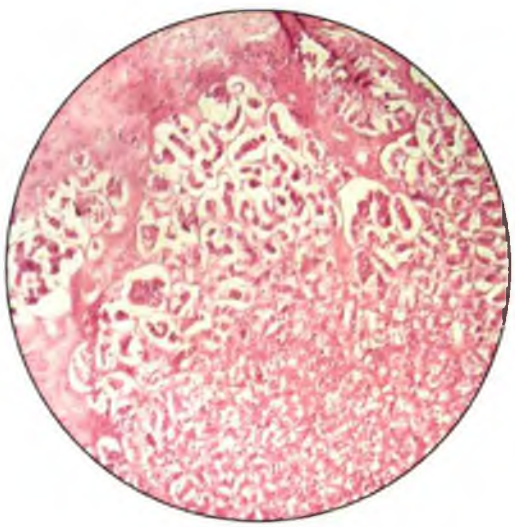

Fig. 55.

Figure. 55. Adrenal cortical hyperplasia. H&Ex400

Accessory cortical nodule seperated by fibrous tissue

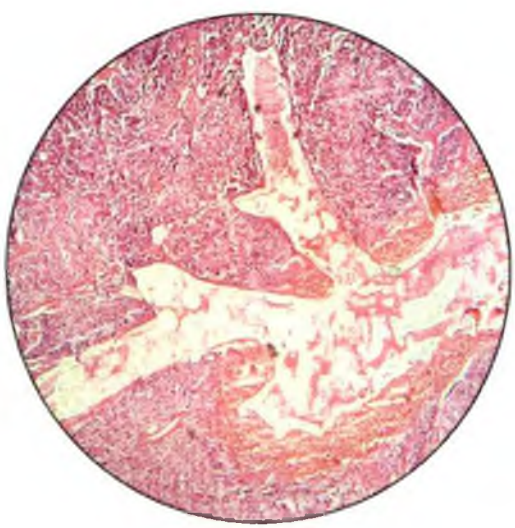

Fig. 56.

Figure. 56 Adrenal medullary hyperplasia. H&Ex100

Dilatation of vessels, sinusoids and haemorrhage in the medulla

Discussion

5. DISCUSSION

The present investigation was aimed to ascertain the incidence, nature and magnitude of utero-ovarian pathology in goats and to correlate the utero-ovarian pathology with endocrine lesions.

A total number of 1000 genital tracts were screened for utero-ovarian disorders. Out of these, 84 genitalia showed lesions in the ovary, oviduct and uterus. Among these 54 cases showed lesions of varying frequency in the ovaries, seven cases showed lesions in the oviduct and 36 cases showed lesions in the uterus. Detailed gross and histopathological examinations were carried out on cases of ovary and uterus with grossly visible lesions. Pituitary, adrenal and thyroid glands from animals with gross utero-ovarian lesions were subjected to detailed gross and histopathological examination. Lesions of varying frequency in pituitary, thyroid and adrenal glands were recorded in 23 out of 84 cases examined.

Six cases (0.6%) of unilateral hypoplasia of the ovaries were recorded in the present study. This incidence is more than that recorded by Ijapure *et al.* (2002) but lower than that reported by Regassa *et al.* (2008). The surface of the ovaries were smooth and did not reveal any follicle and were composed more of medullary connective tissue and blood vessels with a thin investment of cortical tissue and absence of secondary graffian follicles. These gross and histopathological findings were in agreement with the reports of Das *et al.* (1979).

The follicular cysts recorded in 14 cases (1.4%) in the present study were in agreement with the reports of the previous workers (Kadu and Kaikini, 1988; Sattar and Khan, 1988; Timurkaan and Karadas, 2000; Ijapure *et al.*, 2002). A higher incidence of 3.5 to 4.3 per cent was observed by Khatun *et al.* (2007) and Regassa *et al.* (2008). The causes of development of follicular cysts were

manifold. The cysts developed as a result of a failure of coordination of various endocrine mechanisms. Significantly lower luteinizing hormone concentration during proestrus and oestrus in goats with follicular cysts was reported by Vanholder *et al.* (2006). He also reported that stress, mediated by the adrenocortical hormone was a contributing factor in the development of follicular cysts. In the present study, in certain cases of follicular cysts, conditions like salpingeal hyperplasia, cystic endometrial hyperplasia and adenomyosis were present which could be attributed to the prolonged estrogenic phase.

Luteal cyst was observed in 0.3 % of cases which is in consonance with the observations made by Alosta *et al.* (1998) and Ijapure *et al.* (2002). The luteal cyst had a thick wall and the fluid in the cyst was more amber or darker yellow or brown in colour. The characteristic microscopic lesions were the cystic cavity surrounded by an inner layer of thin band of connective tissue, middle layer of lutein cells of varying thickness and an outer wall with concentrically arranged connective tissue from the ovary. These gross and histopathological findings were in agreement with the report of Alosta *et al.* (1998). The reason for the development of the cyst could be a failure of hypophysis to release an adequate surge of luteinizing hormone (Vanholder *et al.*, 2006).

Incidence of parovarian cyst was observed in six cases (0.6 %). This was in line with those reported by Singh (1973), Chand and Chauhan (1978), Sharma (1980) and Kadu and Kaikini (1988). The gross and histological findings were in agreement with those of Sharma (1980) and Alosta *et al.* (1998). The cyst wall comprised of a layer of fibrous connective tissue and muscle fibers. The cyst wall was lined by a single layer of cuboidal or low columnar epithelium. Roberts (1971) reported that these cysts were vestiges of the mesonephric duct system or the mullerian duct system. Parovarian cyst does not usually interfere with the

reproductive performance of the animal. However, larger parovarian cysts causing stenosis of the oviductal lumen by compression, could affect fertility.

The incidence of embedded corpus luteum in the present study was 0.3 %. Gross and microscopical features were in agreement with the finding of Ghora (1995) and Sujata *et al.* (2003) for bovines. The cut surfaces of the ovaries revealed encapsulated, yellowish brown coloured corpus luteum of approximately 0.5 centimeter in diameter within the ovary. This defect will interfere with the reproductive performance as continuous production of progesterone from such animals could enhance the anestrous condition.

The incidence of ovarian haematoma in the present study was 0.2 % which coincided with that reported by Das *et al.* (1979). Severe trauma during manual enucleation of the corpus luteum or rupture of the follicular cysts might be the etiological factors. Hemorrhage into the ovary could occur on account of toxic infectious diseases (Roberts, 1971; Parkinson, 2001).

Persistent corpus luteum was recorded in two cases. Gross and microscopical features were in agreement with those reported by George (1994). Any factor which interferes with the production or release of PGF2 alpha will result in the development of persistent corpus luteum. Uterine infections could interfere with the production and release of PGF2 alpha (Parkinson, 2001).

Serous inclusion cysts were recorded in two cases. Microscopically multiple cysts were noticed close to the ovaries. Cyst wall was lined by a layer of cuboidal epithelium. The etiological factor is unknown. It does not affect the fertility unless the cysts are multiple and large enough to block ovulation (Kennedy and Miller, 1993).

Incidence of epoophoron was recorded in a single case. Microscopically, acini like structures were noticed in the medulla. The acini had slit-like lumen and were lined with cuboidal epithelium. These findings were in agreement with that of Singh (1973) and Sharma (1980). Kennedy and Miller (1993) reported that this developed from the caudal portion of the mesonephros. Significance of these structures is not known and needs further elucidation.

Follicular atresia was recorded in four cases (0.4%). A lesser incidence of 0.1% was reported by Ramachandran (1980). Microscopically, in the cortex, multiple follicles of various types showed follicular atresia. Degenerated ova surrounded by collapsed zona pellucida were seen. These findings were in agreement with those of Kennedy and Miller (1993).

Sclerosed ovaries were recorded in four cases. Neither corpus luteum nor follicles were evident on the surface of the sclerosed ovaries. The cut surface revealed a dense stroma. Microscopically, the surface epithelium was absent in a few places. The tunica albugenia was thickened. The stroma of the cortex had a dense fibrous connective tissue. There was no evidence of any developing follicles in the ovarian cortex. The same observation was made by Singh (1973) and Das *et al.* (1979). Lack of hormonal effect and imbalance due to malnutrition and endocrine lesions in pituitary, thyroid and adrenal glands could have played a significant role in the onset of such conditions for which the nutritional and hormonal status of the animals need to be assessed (Elsawaf and Schmidt, 1963).

Senile atrophic ovaries were recorded in three cases. The senile ovaries were pale and waxy with rough pitted surfaces. The ovarian cortex bore numerous pin head sized follicles, red bodies and corpus albicans. The cortical areas were reduced. There were only a few primary follicles in the cortex with thickened

tunica albugenia. These findings were in agreement with those reported for cows by Nair (1974) and George (1994).

Perioophoritis was observed in three cases (0.3%). The total ovario-bursal adhesion was seen in the affected ovary resulting in the encapsulation of the ovary. Periovarian connective tissue was seen to be infiltrated by lymphoid cells, plasma cells and a few mononuclear cells. These findings were in agreement with those of Ramachandran (1980) and Timurkaan and Karadas (2000).

The incidence of hydrosalpinx was recorded in two cases (0.2%) which agree with those reported by Badawi *et al.* (1979), Sharma and Sharma (1985), Ahmed *et al.* (1987) and Aliasghar and Saad (2007). Both gross and microscopical features in this present study were in agreement with those of Kadu and Kaikini (1988). The etiological factors that could lead to the obstruction of the oviduct are ascending uterine infections, ovarobursal adhesions, and uterine irrigations with irritant solutions, manual enucleation of the corpus luteum and the presence of parovarian cysts. Congenital anomalies like freemartins and segmental aplasia of the uterine horns also could promote the development of hydrosalpinx (Kennedy and Miller, 1993).

Hyperplasia of the salpingeal epithelium was recorded in two cases in the present study. The affected salpinx revealed moderate thickening. Microscopically, the mucosal epithelial folds were considerably flattened and the lumen of the oviduct was dilated. The mucosa was lined by low cuboidal to columnar epithelium without cilia. Atrophy of the muscular coat and lamina propria were seen. This has the potential to promote infertility as the passage of ovum through such a duct is liable to be obstructed. The lesions suggested the chronic nature of the disease process which indicated persistent exposure of a stimulatory agent as the etiological

factor. The sub clinical infections may be an attributing factor. Ghora (1995) discussed the incidence of salpingeal hyperplasia as a result of hyperestrogenism.

Ovario-bursal adhesion was recorded in three cases (0.3%) which did not agree with those reported by Kadu and Kaikini (1988), Ijapure *et al.* (2002), Alosta *et al.* (1998) and Aliasghar and Saad (2007) who observed a higher incidence varying from 2.3% to 11.5%. Grossly, there is total ovario-bursal adhesion of the affected ovary resulting in the encapsulation of the ovary. Ovario-bursal adhesion observed can be considered as the effect of the perioophoritis since the incidence is almost similar.

Cystic endometrial hyperplasia was recorded in six cases. The affected uterus revealed moderate thickening. Microscopically, there was an increase in the size and number of glands that appeared irregular in their distribution and course. The glandular epithelium in all cases were tall columnar and the lumen contained secretions. These histological findings are in close agreement with those of Sharma and Sharma (1987) and Kennedy and Miller (1993). In these cases the hyperplasia was associated with follicular cysts. Cystic endometrial hyperplasia in goats can be associated with ovarian follicular cysts or granulosa cell tumors (Radi, 2005). Both these conditions could bring about prolonged hyperestrogenism leading to endometrial hyperplasia and clinically manifesting as anoestrus or nymphomania. The possibility of phytoestrogen from the forages as a source cannot be ruled out and has to be investigated further.

Adenomyosis was recorded in four cases. Microscopically, endometrial glands were located in the myometrium. This could be due to the result of prolonged hyperestrogenism or developmental disturbances (Acland, 2001).

Mucometra was encountered in four cases. Microscopically, endometrium was thin and lined by a single layer of columnar epithelium and contained reduced number of active glands in the endometrial stroma. These histological findings were in agreement with that of Duquesnel *et al.* (1992) and Wittek *et al.* (1998). Factors such as congenital or acquired obstructions to the outflow of the normally produced mucus resulted in the development of mucometra (Acland, 2001). The possibility of subclinical infections which act as an irritant to the mucosa may be a factor causing increased secretion of mucus resulting in mucometra.

Perimetrial cyst was observed on the uterine surface in six cases in the present study. Ghora (1995) suggested that this cyst could have originated from the obliterated portions of the oviducts which later became cystic.

Incidence of endometritis was recorded in 14 cases (1.4%) which were in agreement with that of Timurkaan and Karadas (2000). Endometritis was described as the most common pathological condition (4.49% incidence) of the genitalia in shegoats ranging from 2–6 years of age (Ijapure *et al.*, 2002); whereas a lower incidence of endometritis (3.21% and 1.53% incidence) was reported in local nondescript goats ranging from 1 to 6 years of age (Reddy *et al.*1997). The gross and histological findings were in agreement with those of Singh and Rajya (1977) and Radi (2005). Endometritis might follow abnormal parturitions, traumatic lesions and unhygienic artificial inseminations. These factors were associated with delayed uterine involution and conception rate (Reddy *et al.*, 1997).

Acute suppurative metritis was recorded in two cases in the present study. The uterine lumen contained chocolate colored lochia, which was slightly tenacious and without any foul odour. The endometrium was congested and hemorrhagic.

Microscopically, the mucosa, muscular layer and serosa were infiltrated with neutrophils and a few mononuclear cells. These findings were in agreement with those of Das *et al.* (1979). The genital tract is particularly susceptible to infections after lambing, as bacteria can invade therein (Noakes, 1996).

Localized or systemic diseases of the animal may have contributed to the various utero-ovarian and endocrine gland disorders observed in the present study. Karagiannis and Harsoulis (2005) reported that the gonadal function was significantly affected in many acute and chronic diseases determined through the integrity of the hypothalamic-pituitary-gonadal axis. In acute stress reactions, the ovarian function was harmed indirectly by way of gonadotrophin suppression and directly by the action of cytokines upon the ovary. In chronic stress reactions, the ovarian dysfunction could be due to primary ovarian failure with reduced production of oestrogen and elevated gonadotrophin levels.

In the present study various lesions in pituitary, thyroid and adrenal glands were encountered along with different types of utero-ovarian disorders in goats. The ovarian hypoplasia, follicular cyst, luteal cyst and atretic follicles were seen in the goats affected by pituitary gland cysts. This indicated that the cyst in the pituitary gland interfered in the gonadotrophin hormone secretion leading to the degeneration of germinal epithelium. Capen (1993) described that pituitary cyst may develop from remnants of the craniopharyngeal duct, which normally disappears by birth in most of the animal species. The cyst leads to hypofunction of the adenohypophysis resulting in gonadal atrophy. These cysts, which caused compression of the adjacent pituitary cells, might have contributed to gonadal dysfunction. Jubb *et al.* (1991) reported that the structures adjacent to the cysts showed atrophy of varying degrees owing to compression and interference with the blood supply. Capen (1993) stated that the disruption of a large cyst with escape of

the proteinaceous contents into the adjacent tissues might incite an intense local inflammation with subsequent fibrosis that may interfere with pituitary function leading to gonadal atrophy. Due to functional abnormalities of the pituitary gland gonadotrophin secretion was decreased resulting in the gonadal dysfunction. Mudge (1995) reported that the pituitary-gonadal axis dysfunction was associated with gonadal dysfunction and various types of sexual precocity.

A solitary case of pituitary abscess was recorded in association with a case of acute suppurative metritis. The pathogenesis of the pituitary abscess is uncertain. Perdizet and Dinsmore (1986) reported that the extensive capillary network constituted by a complex mesh of interwined arteries and capillary beds known as the rostral epidural rete mirabile in ruminants makes the pituitary gland susceptible to bacterial seeding from other sources of infection such as mastitis, arthritis and lung abscess or pneumonia. In this case, the abscess could have been caused by a direct extension of infection from acute suppurative metritis.

In one case of follicular cyst, pituitary gland revealed congestion and haemorrhagic changes along with hyperplastic changes of the adrenal and thyroid glands. This indicated that the pituitary haemorrhage might cause secondary lesions in the ovary, thyroid and adrenal gland. Though the exact cause for the lesions in the pituitary gland cannot be pinpointed, generalized vascular disturbances could lead to haemorrhagic lesions in the pituitary gland and also with a cascading effect on the thyroid and adrenal glands. Schmidt and Wallace (1998) reported pituitary apoplexy following sudden enlargement of the pituitary gland in human beings. Other factors responsible for haemorrhages are pituitary adenomas, anticoagulants, arterial or intra cranial hypertension as well as traumatic injury. Veldhuis and Hammond (1980) reported an incidence of Growth Hormone (GH) deficiency of 88%, secondary adrenal deficiency of 66%, secondary hypothyroidism of 42% and

hypogonadotrophic hypogonadism of 67% in human patients as a result of pituitary apoplexy.

In majority of the ovarian disorders like hypoplasia, follicular cyst, luteal cyst, atretic follicles, senile atrophy and cystic endometrial hyperplasia of the uterus, lesions of varying degree could be detected in the thyroid gland. The hypoplasia, colloid and nodular goitre observed in the various utero-ovarian disorders clearly indicated that there was correlation between them (Sreekumaran, 1976; Reddy, 1982; Sharma and Ramkumar, 2001). Jubb *et al.* (1991) reported that hypofunction of the thyroid gland in long standing cases could lead to disorders in reproduction manifested by abnormal oestrous or anoestrous with reduced conception rates in females. The primary function of the thyroid hormone is regulation of cellular oxidation and stimulation of oxygen consumption for normal growth and development. Fitko *et al.* (1995) reported that there was an increase in the ovarian iodide uptake during hypothyroidism induced by thiouracil. The hypothyroid state is characterized by an absolute or relative depletion of thyroid hormones, altered sensitivity and ovarian response to gonadotropins leading to a rise in the content of mucopolysaccharides, followed by a tendency in some species towards the development of polycystic ovaries. In fact, ovarian cyst formation is greatly intensified in women with primary hypothyroidism and in experimentally hypothyroid animals exposed to hyperstimulation with gonadotropins. The results of the present study prove that the above fact may be applicable to goats also, since the alterations of the thyroid gland were almost consistent in ovarian disorders.

Reddy (1982) reported that there is a significant reduction in the relative weight of ovaries in all hypothyroid goats with the ovaries containing only a few scattered developing primary and secondary follicles. There was also a significant reduction in the relative weight of the uterus with thin muscular layer and the

mucosal glands were few and inactive and revealed no secretory activity. In the present study, five cases of hyperplastic conditions of thyroid glands could be associated with various ovarian lesions like hypoplasia, senile atrophy, cystic and atretic follicles. Sreekumaran (1976) recorded testicular degeneration along with hyperplasia of the thyroid gland in cases of experimental hypothyroidism in goats. Reddy and Rajan (1985) also reported similar pathological findings in experimental hypothyroidism in goats. Abraham *et al.* (1987) reported an increase of relative weight of the adrenal glands in experimentally induced hypothyroidism in male calves.

In the present study, the lesions encountered in adrenal gland were cortical hypoplasia, cortical hyperplasia and medullary hyperplasia. Cortical hypoplasia was encountered in ovarian hypoplasia where depletion of fat in the cells of zona fasiculata was recorded. In this study, five cases of cystic condition of the ovaries were associated with cortical hyperplasia of the adrenal gland. The medullary hyperplasia of the adrenal glands was associated with ovarian hypoplasia, follicular and luteal cysts, atretic follicles and cystic endometrial hyperplasia of the uterus. The cortical and medullary hyperplasia might be due to various stress factors concurrently affecting the adrenal gland. Adrenal cortices normally produce androgens, and adrenal androgens secretion might be greatly increased in a variety of pathological condition including adrenal hyperplasia (McDonald and Pineda, 1989). Mudge (1955) attributed adrenal hyperplasia to a metabolic block in the normal synthesis of adrenal steroids, with resultant accumulation of androgenic steroids leading to the development of testicular disorders via pituitary–gonadal axis.

A number of other environmental chemicals have been implicated as endocrine disruptors. The mechanism of action of these compounds or substances

A number of other environmental chemicals have been implicated as endocrine disruptors. The mechanism of action of these compounds or substances are not well known but they may involve in agonistic or antagonistic activity against sex hormones especially oestrogen; modulatory effects on enzymes controlling sex hormone metabolism; or direct influence on the hormone-producing organs such as the thyroid gland, pituitary gland, and adrenal glands. These compounds may also affect oestrogen levels through indirect feedback mechanisms. Research on possible reproductive effects in human and animal were limited (Roy and Pereira, 2005). The goats being a voracious grazing animal, the exposure to plants with phytoestrogen is a possibility to be ruled out.

Various chemicals, toxic plants, insecticides, pesticide and other environmental pollutants also could have played a vital role in the onset of endocrine and utero-ovarian disorders observed in the present study. Jubb *et al.* (1991) reported that long-term perturbations of the pituitary-thyroid axis by various xenobiotics predispose the animal to a higher incidence of proliferative lesions in the gonads than in human beings.

The investigation carried out, has helped to document various utero-ovarian disorders encountered in goats. It has also been possible to assess and study the pathomorphological affections. Among these affections, follicular cyst was the most frequently encountered lesions in the ovaries followed by different types of endometritis. The result of the study especially of the reproductive organs does not reveal much deviation from the patterns observed by the earlier workers. The pathological changes associated with various utero-ovarian disorders encountered were suggestive of endocrine imbalances. The present investigation undertaken has made it possible to identify the various endocrine lesions associated with utero-ovarian disorders in goats. It was observed that in many cases there was a co-

existence of lesions in the ovary along with the pituitary, thyroid or adrenal glands. Though lesions of varying degrees were observed in the pituitary, thyroid and adrenal glands histologically, not all functional disturbances need be reflected in structural alterations. But ultra structural lesions of the endocrine elements can cause alterations of the hormone secretion, resulting in hormonal imbalances which may have adverse effect on the reproductive performances. Future studies should be aimed at identifying the causative factors that contribute to the development of these disorders. Multiple nutritional deficiencies, infection and managemental factors were also suspected in some of the cases. Therefore the results of the study also highlighted the need to monitor the hormone profile and micronutrient levels in the blood, besides better management practices. This investigation had inherent deficiencies like the absence of reproductive history, limited sampling size and collection of samples from the same places where apparently healthy animals were normally slaughtered. A comprehensive study taking into consideration all these deficiencies is worthwhile.

Summary

6. SUMMARY

The present investigation was carried out, to study the pathological features of spontaneously occurring utero-ovarian disorders in goats. One thousand specimens of genitalia collected formed the material for the study which was subjected to detailed gross and histopathological examinations. Of these, 84 genitalia showed one or more lesions in the ovary, oviduct and uterus. Pituitary, adrenal and thyroid glands from animals with gross utero-ovarian lesions were subjected to detailed gross and histopathological examination and lesions of varying frequency were recorded in 23 out of 84 cases examined.

The conditions encountered in the ovaries were ovarian hypoplasia, follicular cyst, luteal cyst, embedded corpus luteum, persistent corpus luteum, ovarian haematoma, ovarian sclerosis, senile atrophy, serous inclusion cyst, epoophoron, atretic follicles, parovarian cyst and perioophoritis. Lesions of the oviduct were recorded in seven cases and the conditions encountered were hydrosalpinx, hyperplasia of the salpingeal epithelium and ovario-bursal adhesion. Lesions of the uterus were recorded in 36 cases and the conditions encountered were cystic endometrial hyperplasia, adenomyosis, mucometra, perimetrial cyst, varying degrees of endometritis and metritis.

Among the ovarian lesions, follicular cyst was the most common condition encountered. It was observed in 14 cases (1.4 %). The right ovary (8 cases) was found to be more frequently involved than the left ovary (6 cases). Follicular cyst was thin walled and distended with a pale yellow clear fluid. Ovarian hypoplasia was recorded in six cases (0.6% per cent) and all were unilateral. The ovaries were flat, small and firm. The surface was smooth and did not reveal any follicle, developing or degenerating corpus luteum or luteal scars.

Luteal cysts were recorded in three cases (0.3 per cent) and the luteal cyst had a thick wall and contained a darker yellow or brown coloured fluid. Parovarian cysts were recorded in six cases (0.6 per cent), two cases being bilateral while the remaining four cases were unilateral. Larger parovarian cysts causing stenosis of the oviductal lumen by compression, could affect fertility. Embedded corpus luteum was recorded in three cases (0.3 %); two were in the right ovary and one in the left. The embedded corpus luteum had a fibrous connective tissue capsule from which connective tissue fibers traversed into the lutein tissue dividing it into few lobes. The persistent corpus luteum was observed in two cases. The persistent corpus luteum had fibrous connective tissue stroma that divided the luteal tissue into several lobes. There was reduced vascularisation in the parenchyma with regressive changes in the luteal cells.

Ovarian haematoma was recorded in two cases. Severe trauma during manual enucleation of the corpus luteum or rupture of the follicular cysts could be the etiological factors. Hemorrhage into the ovary could occur on account of toxic infectious diseases. Serous inclusion cyst was recorded in two cases (0.2 %). The cyst wall was lined by a layer of cuboidal epithelium. It does not affect the fertility unless the cysts are multiple and large enough to block ovulation. Incidence of epoophoron was recorded in two cases. Microscopically, acini like structures were noticed in the medulla. The acini had slit-like lumen and were lined by cuboidal epithelium. Incidence of atretic follicles was recorded in four cases (0.4 %). In the cortex, multiple follicles of various types showed follicular atresia. Ovarian sclerosis was recorded in four cases (0.4 %), one in the right ovary and three in the left. Sclerosed ovaries were small and hard .Neither corpus luteum nor follicle was evident on the surface. Senile atrophic ovaries were recorded in three cases (0.3%) and all were bilateral. The senile ovaries were pale and waxy with a rough pitted

surface. There were only a few primary follicles in the cortex. Perioophoritis were recorded in three cases (0.3%). Periovarian connective tissue was seen to be infiltrated by lymphoid cells, plasma cells and a few mononuclear cells.

Hydrosalpinx was recorded in two cases (0.2 %). The fallopian tube was distended with a clear amber coloured fluid. Microscopically, the mucosal epithelial folds were flattened and the lumen of the oviduct was dilated. Hyperplasia of the salpingeal epithelium was recorded in two cases (0.2 %). Thickening of the mucosa was prominent. Microscopically, papillary hyperplasia was observed. Ovario-bursal adhesion was recorded in three cases (0.3 %). There was total ovario-bursal adhesion of the affected ovary resulting in the encapsulation of the ovary.

Six recorded cases of cystic endometrial hyperplasia showed microscopic features like increased number and size of the glands with columnar epithelium. Factors like prolonged hyperestrogenism and developmental disturbances resulted in the invasion of the myometrium by the endometrial glands. This condition called adenomyosis was observed in four cases. In four cases of mucometra resulting from the congenital or acquired obstructions to the mucous outflow, the uterine horns were distended with an opaque and viscid fluid. Six cases of clear watery fluid containing oval perimetrial cysts were observed which was lined by a layer of flattened epithelium.

Endometritis was observed in fourteen cases and were graded into three types based on the degree of fibrosis of the uterine glands and the extent of infiltration of inflammatory cells in the uterine mucosa. The changes observed in the uterine glands varied from straight tubular to tortuous and lined by cuboidal epithelium without periglandular fibrosis to extreme periglandular fibrosis and all the glands appeared atrophic. In two cases of acute suppurative metritis, the uterine lumen contained

chocolate colored lochia and without any foul odor. Microscopically the mucosal, muscular and serosal layers were infiltrated with neutrophils and a few mononuclear cells.

In the present study various lesions in pituitary, thyroid and adrenal glands were encountered along with different types of utero-ovarian disorders in goats. Pituitary abscess was recorded in a case of acute suppurative metritis. The follicular cyst, luteal cyst and atretic follicles were seen in goats affected by pituitary gland cysts. Apoplexy along with congestion and oedema of the pituitary gland was seen in follicular cyst and senile atrophy of the ovary. In such cases, hyperplasia of thyroid gland and adrenal gland were also noticed.

The various lesions in the thyroid gland observed in utero-ovarian disorders were hypoplasia, colloid and nodular goitre. Thyroid hypoplasia was recorded in cases with ovarian hypoplasia, follicular cyst, senile atrophy and cystic endometrial hyperplasia. The hyperplastic conditions of thyroid glands revealed various ovarian lesions like hypoplasia, senile atrophy, cystic and atretic follicles.

The lesions encountered in the adrenal gland were cortical hypoplasia, cortical hyperplasia and medullary hyperplasia. Ovarian hypoplasia was encountered in cortical hypoplasia of adrenal gland while a few cystic conditions of the ovaries were associated with cortical hyperplasia of the adrenal gland. The medullary hyperplasia of the adrenal glands was associated with ovarian hypoplasia, follicular and luteal cysts, atretic follicles and cystic endometrial hyperplasia of the uterus.

The present investigation undertaken has made it possible to ascertain the incidence, nature and magnitude of utero-ovarian pathology in goats and to correlate the utero-ovarian pathology with the various endocrine lesions.

References

REFERENCES

Abraham, M.J., Valasala, K.V. and Rajan, A. 1987. Pathology of the testes and endocrine glands in experimental hypothyroidism in cattle. *Cheiron.*16 : 45-53

Acland, M.H. 2001. Reproductive system: Female. *Thomson's Special Veterinary Pathology*. Third Edition. (eds McGavin, M.D., Carlton, W.W. and Zachary, J.F.). Mosby Company, London. pp 601-634

Adams, N.R. 1975. A Pathological and bacteriological abattoir survey of reproductive tract of Merino ewes on W. Australia. *Aust. Vet. J.* 51: 351

Adeniyi, K.O., Ogunkeye, O.O., Senok, S.S. and Udoh, F.V. 1994. The effects of thyroidectomy and thyroxine on the reactivity of rat uterine muscle to electrical stimulation *in vitro*. *Pathophysiol.* 1:151-154

*Ahmed, R., Khan, M. Z. and Chisti, M. A. 1987. Incidence and pathology of genital abnormalities of adult sheep. III. Pathology of oviducts. *Pak. Vet. J.* 7: 77

*Ahmed, R., Khan, M.Z. and Samad. H.A. 1986. Incidence and pathology of genital abnormalities of adult sheep. I. Pathology of ovaries. *Pak. Vet. J.* 9:175

Alam, M. G., Ahmed, J. U. and Jahan, S. 1989. Effect of dexamethasone on the estrous cycle length in Black Bengal goats (*Capra hircus*). *Theriogenology.* 31: 935-941

Alexander, J.E. 1962. Anomaly of craniopharyngeal duct and hypophysis. *Can. Vet. J.* 3: 83.

Aliasghar, M. and Saad, G. 2007. Abattoir survey of gross abnormalities of the ovine genital tracts in Iran. *Small Rumin. Res. Vol.* 73(1-3) : 259-261

Alosta, R.A., Vaughan, L. and Collins, J.D. 1998. An abattoir survey of ovine reproductive tracts in Ireland. *Theriogenology* 50 : 457—464

Anderson, J. and Sandison, A.T. 1969. Tumours of the female genitalia in cattle, sheep and pigs found in a British abattoir survey. *J. Comp. Path.*79 : 53

*Ansari, H. 1978. Functional and Pathological conditions of genital organs of ewes in Thehran. *Revue de Medicine Veterinaire*. 129 (2) : 285

*Badawi, A.B.A., Habib, A.M. and Zaki, K. 1979. Studies on abnormalities of organs of genital organs of Libyan ewes. *Assiut Vet. Med. J.* 6 (11/12) : 255

Bancroft, J .D and Cook, H.C. 1995. *Manual of histological Techniques*. Second edition. Churchill Livingston, Edinburg, 761 p.

Barnes, R. B., Rosenfield, R. L., Ehrmann, D. A., Cara, J. F., Cuttler, L., Levitsky, L.L. and Rosenthal, I. M. 1994. Ovarian hyperandrogynism as a result of congenital adrenal virilizing disorders: evidence for perinatal masculinization of neuroendocrine function in women. *J. Clin. Endocrinol. Metabol.* 79 : 1328-1333

Bell, M. E., Wood, C. E., Keller-Wood, M., Kane, C., Kluwe, C., Manlove, E., Taranovich, C. and Johnson, J. 1991. Influence of reproductive state on pituitary-adrenal activity in the ewe. *Domest. Anim. Endocrinol.* 8 (2) : 245-254

Bennetts, H.W., Underwood, E.J. and Shier, F.L. 1946. A specific breeding problem of sheep on subterrainean clover pastures in Western Australia. *Aust. Vet. J.* 22 : 2.

Bernal A., De'Moraes G.V., Thrift T.A., Willard, C.C. and Randel, R.D. 1999. Effects of induced hypothyroidism on ovarian response to superovulation in Brahman (*Bos indicus*) cows. *J. Anim. Sci.* 77 : 2749–2756.

*Biolatti, B., Guarda, F. and Pau, S. 1984. Female genital diseases of routinely slaughtered sheep. *Summa.* 1 : 310

Busato, A., Romagnoli, S., Kupfer, U., Rossi, G.L. and Bestettilb, G.E. 1995. LH, FSH, PRL AND ACTH cells in pituitary glands of cows with ovarian cysts. *Theriogenology.* 44 : 233-246

Capen, C.C. 1993. The endocrine glands. *Pathology of domestic animals.* (eds. Jubb, K.V.F., Kennedy, P.C. and Palmer, N.). Fourth edition. Vol.3. Academic Press, USA. pp.267-347

Capen, C. C. 2001. Overview of Structural and Functional Lesions in Endocrine Organs of Animals. *Toxicol. Pathol.* 29 : 8-33

Chand, S. and Chauhan, H.V.S. 1975. Cystic endometrial hyperplasia in sheep and goats. *Indian J. Anim . Sci.* 45 : 71

Chand, S. and Chauhan, H. V. S. 1978. Pathology of female genital organs of sheep and goats in Haryana state. *Indian J. Vet. Path.* 3 : 23.

Chapin, R. E., Stevens, J. T., Hughes, C. L., Kelce, W. R., Hess, R. A. and Daston, G. P. 1995. Endocrine modulation of reproduction. *Fund. Appl. Toxicol.* 29 : 1-17

Das, K.K., Borogohain, B.N. and Rajkonwar, C.K. 1979. Notes on the incidence of pathological conditions and the histo-pathological changes in the female reproductive organs of local goats of Assam. *Indian J. Anim. Sci.* 49 : 1099-1101

DeFesi, C.R., Astier, H.S. and Surks, M.I. 1979. Kinetics of thyrotrops and somatotrops during development of hypothyroidism and L-triiodo thyronine treatment of hypothyroid rats. *Endocrinol.* 104 :1172-1180

Dennis, S. M. 1979. Urogenital defects in sheep. *Vet. Rec.* 105 : 344

*Duquesnel, R., Parisot, D., Pirot, G., Mialot, J.P., Saboureau, L., Etienne, P., Delaval, J., Guemraud, J.M., Prengere, E., Montigny, G., de Guerrault, G., Perrin, G., Humblot, P., Fontaubert, Y. and de Chemineau, P. 1992. La pseudogestation chez la cheivre. *Ann. Zootech.* 41 : 407-415

*Dzhurova, I., Marinov, M., Marinov, P. and Tashev, S. 1985. Pathological changes in genital organs of culled ewes. *Veterinarno meditsinki Nauki.* 22 (5) : 43

Elsawaf, S.A. and Schmidt, K. 1963. Morphological changes in normal and abnormal ovaries of buffaloes with special reference to their function. *Vet. Med. J.* 8 : 249

Emady, M., Noakes, D. E. and Arthur, G. H. 1975. Analysis of reproductive function of the ewe based on post-mortem examination. *Vet. Rec.* 96 : 261.

Emmakeeble. 2001. Endocrine diseases in small animals. *In Practice* 23 : 11-13

Engeland, I. V., Ropstad, E., Kindahl, H., Andresen, O., Waldeland, H. and Tverdald, A. 1999. Foetal loss in dairy goats: function of the adrenal glands, corpus luteum and the foetal–placental unit. *Anim. Reprod. Sci.* 55 : 205–222

Fitko, R., Kucharski, J. and Szlezyngier, B. 1995. The importance of thyroid hormone in experimental ovarian cyst formation in gilts. *Anim. Reprod. Sci.* 29 : 159–68.

Fitko, R., Kucharski, J., Szlezyngier, B. and Jana, B. 1996. The concentration of GnRH in hypothalamus, LH and FSH in pituitary, LH, PRL and sex steroids in peripheral and ovarian venous plasma of hypo- and hyperthyroid, cysts-bearing gilts. *Anim. Reprod. Sci.* 45 : 123- 138

George, V. 1994. Pathology of ovary and ovarian bursa in cross breed cattle. M.V. Sc. Thesis. Kerala Agricultural University, Thrissur. p 95

Ghora, T.K. 1995. Pathology of female genital tract of buffaloes. M.V.Sc. Thesis, Deemed University, IVRI, Bareilly. p 75

*Gustaffson, B. and Holberg, O. 1966. Postmortem examination of genital tract in ewes with special reference to occurrence of developmental abnormalities. *Svensik. Vet.-Tidn.* 18 : 432

Haibel, G.K., Constable, P.D. and Rojko, J.L. 1990. Vaginal leiomyofibromatosis and goiter in a goat. *J. Am. Vet. Med. Assoc.* 196 : 627–629

Hatipoglu, F., Kiran, M.M., Ortatatli, M., Erer, H. and Ciftci, M.K. 2002. An abattoir study of genital pathology in cows: I. Ovary and oviduct. *Revue. Med. Vet.* 153 : 29-33

Hawkins, C.D., Swan, R.A. and Chapman, H.M. 1981. The epidemiology of squamous cell carcinoma of the perineal region of sheep. *Aust. Vet. J.* 57 : 455

Huszenicza, G., Kulcsar, M. and Rudas, P. 2002. Clinical endocrinology of thyroid gland function in ruminants. *Vet. Med. Czech.* 47 : 199–210

Ijapure, K.P., Singh, B.K. and Chauhan, R.A.S. 2002. Incidence of pathological affections of genitalia in she goats. *Indian Vet. J.* 79 : 298-299

Jubb, K.V.F., Kennedy, P.C. and Palmer, N. 1991. Female reproductive system. *Pathology of Domestic Animal.* Fourth edition. Academic press, London. pp 349-470

Kadu, M.S. and Kaikini, A.S. 1988. Pathological conditions in the female genital organs of the goat. *Indian J. Anim. Sci.* 58 : 795-798

Kaneko, H., Taya, K., Watanabe, G., Noguchi, J., Kikuchi, K., Shimada, A. and Hasegawa, Y. 1997. Inhibin is involved in the suppression of FSH secretion in the growth phase of the dominant follicle during the early luteal phase in cows. *Domest. Anim. Endocrinol.* 14 (4) : 263-271

Kannan, C. R. 1988. Diseases of the adrenal cortex. *Dis. Month.* 34 (10) : 601

Karagiannis, A. and Harsoulis, F. 2005. Gonadal dysfunction in systemic diseases. *Eur. J .Endocrinol.* 152 : 501-513

Kennedy, P.C. and Miller, R.B.. 1993. Female reproductive system. *Pathology of Domestic Animal.* Vol 3. Fourth edition. (eds Jubb, K.V.F., Kennedy, P.C. and Palmer, N.). Academic press. London. pp 374-385

Khaled, A.B. 2004. The Effect of Induced Hypothyroidism on Fertility of Goats During Summer Season. *Scientific Journal of King Faisal University.* 5 (1) : 1425

Khatun, A., Wani, G.M., Choudhary, A.R. and Pandit, B.A. 2007. Incidence of reproductive abnormalities in small ruminants. *Indian J. Anim. Reprod.* 28 : 53-56

Krishna, L. and Paliwal, O.P. 1982. Hepatic ossification and hermaphroditism in goats. *Indian J. Vet. Path.* 6 : 37

Lavoie, L. and Lacroix, A. 1995. Partially autonomous cortisol secretion by incidentally discovered adrenal adenomas. *Trends. Endocrinol. Metabol.* 6 (6) : 191-197

Lofstedt, R. M. and William, R. 1986. Granulosa cell tumor in a goat. *J. Am. Vet. Med. Assoc.* 189 : 206.

Long, S.E. 1980. Some pathological conditions of the reproductive tract of the ewe. *Vet. Rec.* 106 : 175-177

Longcope, C. 1991. The male and female reproductive systems in hypothyroidism. *The Thyroid. A Fundamental and Clinical Text.* (eds Braverman, R. D., Utiger, S. C., Werner, L.B. and Inber, S. H.). Philadelphia. pp. 1052-1055.

Longcope, C. 2000. The male and female reproductive systems in thyrotoxicosis. *The Thyroid.* Eighth edition. Lippincott Williams &Wilkins. Philadelphia. pp 652–659.

Luna, L.G. 1968. *Manual of Histology Staining Methods of the Armed Forces Institute of Pathology.* Third edition. McGraw Hill Company, New York, 258p.

*Lyngset, 0. 1968. Studies on reproduction in goats. V. Pathological conditions and malformations of genital organs of the goats. *Acta. Vet. Scand.* 9 : 364

MacDonald, L.E. 1969. *Textbook of Veterinary Endocrinology and Reproduction.* Lea and Febiger, Philadelpia, 345 p.

McClain, R.M. 1994. Mechanistic considerations in the regulation and classification of chemical carcinogens. *Nutritional Toxicology (eds* Kotsonis, F. N., Mackey, M. and Hjelle, J.). pp 273–304.

McDonald, L.E. and Pineda, M.H. 1989. *Veterinary Endocrinology and Reproduction.* Fourth edition. Lea and Febiger, Philadelpia, 272 p.

Maruo, T., Hiramatsu, S., Otani, T., Hayashi, M. and Mochizuki, M. 1992. Increase in the expression of thyroid hormone receptors in porcine granulosa cells in follicu--lar maturation. *Acta. Endocrinol.* 127 : 152–160.

Matthews, J.G. 1991. *Outline of Clinical Diagnosis in the Goat.* Wright-Butterworth-Heinemann, Oxford, England. pp. 6-7.

Mavroudis, K., Aloumanis, K., Papapetrou, P. D., Voros, D. and Spanos, I. 2007. Virilization caused by an ectopic adrenal tumor located behind the iliopoas muscle. *Fertility and Sterility.* 87: 1468.e13-1468.e16

Maxwell, J.A.L. 1977. The occurrence and apparent effect on reproduction of construction of the vagina in Merino ewe. *Aust. Vet J.* 53 : 181

McEntee, K. 1990. *Reproductive pathology of domestic animals.* Academic Press, Ic., San Diego, California. 401p.

Mudge, G.H. 1995. Pituitary-gonadal relationship. *Am. J. Med.* 24 : 123-134

Murphy, E. D. and Beamer, W.G. 1973. Plasma gonadotrophin levels during early stages of ovarian tumor genesis in mice of the Wx/Wv genotype. *Cancer Res.* 33 : 721–723.

Nair, T.P. 1974. Various pathological affections of female genitalia of cows. M.V.Sc. Thesis, Kerala Agricultural University, Thrissur, 127p.

Nasseri, A.A. and Prasad, M.C. 1986. Uterus unicornis in sheep - A case report. *Indian Vet. J.* 63: 681.

Nicholas, F. 1978. Pituitary dwarfism in German shepherd dogs: A genetic analysis of some Australian data. *J. Small Anim. Pract.* 19 : 167–174

Noakes, D. 1996. The puerperium and the care of the newborn. *Veterinary Reproduction and Obstetrics.* 7th edition. (eds Noakes, G.H., Pearson, D.E. and Parkinson, T.J.) Saunders, London. pp. 171–182.

O'Shea, J.D., Lee, C.S. and Cumming, I.A. 1974. Normal duration of the oestrus cycle in ewes with congenital absence of one uterine horn. *J. Reprod. Fertil.* 38 : 201

*Oshkin, D.I. (1989). Placental retention and endometritis in ewes. *Veterinariya (Moskva).* 12 : 43.

Parkinson, T. 2001. Infertility: Cow. *Arthur's Veterinary Reproduction and Obstetrics.* Eighth edition (eds Noakes, D.K., Parkinson, T., England, W.W. and Arthur, W.B.). Saunders company. London. pp 383-510

Prabhakraran, N.K. and Raja, C.K.S.V. 1972. Investigations on the pathological conditions in the female genital organs of goats. *Kerala J. Vet. Sci.* 3 : 106

Perdizet, J.A. and Dinsmore, P. 1986. Pituitary abscess syndrome. *Comp. Cont. Educ.* 8 (5) : 311-318

Peters, M.A., deJong, F.H., Teerds, K.J., deRooij, D.G., Dieleman, S.J. and VanSluijs, F.J. 2000. Ageing testicular tumours and the pituitary–testis axis in dogs. *J. Endocrinol.* 166 : 153-161

Peterson, M.E. 1984. Feline hyperthyroidism. *Vet. Clin. North. Am.* 14 : 809 –826.

Piffer, R. C. and Pereira, O. C. M. 2004. Reproductive aspects in female rats exposed prenatally to hydrocortisone . *Comp. Biochem. Physiol.* 139 : 11-16

Prasad, G. and Sinha, R.D. 1980. Occurrence of accessory adrenal cortical nodule in the domestic animals. *Indian. J. Anim. Sci.* 50 : 1060-1063.

Radi, A.Z. 2005. Endometritis and cystic endometrial hyperplasia in a goat. *J. Vet. Diagon. Invest.* 17 : 393–395

Ramakrishnappa, N., Rajamahendrana, R., Lin, Y. M. and Leung, P. C. K. 2005. GnRH in non-hypothalamic reproductive tissues. *Anim. Reprod. Sci.* 88 : 95–113

Rao, L.R. and Abdullakhan, C.K. 1974. A survey of pathological conditions in the genital organs of ewes. *Ceylon Vet. J.* 22 : 66.

Ramachandran, K. 1980. Investigation on the pathological conditions in the genitalia of female goats. M.V.Sc. Thesis, Kerala Agricultural University, Thrissur.

Rawal, C.V.S., Parihar, N.S. and Luktuke, S.N. 1987. Pathological conditions affecting oviduct of sheep. *Indian J. Vet. Path.* 11 : 64

Reddy, M.N. 1982. Pathology of the reproductive organs in experimental hypothyroidism in goats. Ph.D. Thesis. Kerala Agricultural University, Thrissur.

Reddy, N.M. and Rajan, A. 1985. Hypothyroidism in male goats. *Indian Vet. J.* 62 : 837-842

Reddy, K.C.S., Reddy, V.S.C. and Rao, A.S. 1997. Studies on the incidence of reproductive abnormalities in local non-descript female goats. *Indian J. Anim. Reprod.* 18 : 51–53.

Regassa, F., Mengesha, D., Dargie, M. and Tolosa, T. 2008. Abattoir evidence on association between uterine and ovarian abnormalities in Ethiopian highland ewes. *Anim. Reprod. Sci.* doi:10.1016/j.anireprosci.2008.03.020

Roberts, S.J. 1971. Infertility in cow. *Veterinary Obstetrics and Genital Diseases*. Second edition. pp 376-522

Roy, P. and Pereira, B. M. 2005. A treatise on hazards of endocrine disruptors and tool to evaluate them. *Indian J. Exp. Biol.* 43 (11) : 975-992

Ryan, M.J. 1980. Leiomyosarcoma of the uterus in a goat. *Vet. Path.* 17 : 389.

Sattar, A. and Khan, M.Z. 1988. Incidence and pathology of ovarian diseases of goats. *Pak. Vet. J.* 8 : 18

Sattar, A.K., Khan, M.Z. and Siddique, M. 1988. Incidence, pathology and bacteriology of abnormalities of fallopian tubes in goats. *Pak. Vet. J.* 8 (1): 14

Schmidt, D.N. and Wallace, K. 1998. How to diagnose hypopituitarism? *J. Postgraduate Med.* 104 : 7-11

Sheehan, D.C. and Hrapchak, B.B. 1980. *Theory and practices of histotechnology.* Second edition. Mosby Company Ltd, London, 481p.

Sharma, A.K. 1980. Affections of female genital tract of sheep and goats – occurrence and pathomorphological study. M.V.Sc. Thesis, C.S.A. University of Agriculture and Technology, Mathura, India.

Sharma, A.K. and Sharma, D.N. 1985. Affections of fallopian tubes in sheep and goats. *Indian Vet. Path.* 9 : 58.

Sharma, A.K. and Sharma, D.N. 1986. Bent cervix and adenomyosis in sheep and goats. *Indian J. Vet. Path.* 10 : 85

Sharma, A.K. and Sharma, D.N. 1987. Cystic conditions affecting female genital organs of sheep and goats. *Indian J. Vet. Path.* 11 : 57

Sharma, A.K. and Ramkumar, P. 2001. Pathology and effects of iodine supplementation in clinically goitrous goats. *Indian J. Vet. Pathol.* 25 : 41-43

Sikdar, M.K. and Bhowmik, K. 1993. Pathology of spontaneous lesions occurring in pituitary gland of goats. *Indian J. Vet. Pathol.* 17 : 44-47

Singh, N. 1973. Disorders of female genital system in goats in pathoanatomical and histochemical studies. Ph.D. Thesis, Agra University, UP, India.

Singh, N., Kwatra, M.S. and Singh, B. 1982. Observations on genital tract of ewes collected from an abattoir. *Indian J. Vet. Path.* 6 :1.

Singh., N. and Rajya, B.S. 1977. Pathology of female reproductive system in goats. *Indian J. Anim. Sci.* 47: 22

Smith, K. C. 1996. Reproductive abnormalities in ewes. Ph.D. Thesis, University of Bristol. U.K.

Smith, M. C. 1980. Caprine reproduction. *Current Therapy in Theriogenology.* Saundess, Philadelphia, Pennysylvvania. p 987

Smith, K.C., Long, S.E. and Parkinson, T..J. 1998. Abattoir survey of congenital reproductive abnormalities in ewes. *Vet. Rec.* 143 : 679-685

*Sokkar, S.M. and Kubba, M.A. 1980. Pathological studies on the fallopian tube of ewes. *Zentralblatt Fur Veterinarmezidin.* 27 : 118

Sokkar, S. M., Kubba, M. A. and Al-Augaidy, F. 1980. Studies on natural and experimental endometritis in ewes. *Vet. Pathol.* 17 : 693

Spicer, L.J., Alonso, J. and Chamberlain, C. S. 2001. Effects of thyroid hormones on bovine granulosa and thecal cell function *in vitro*: dependence on insulin and gonadotropins. *J. Dairy Sci.* 84 : 1069–1076.

Sreekumaran, T. 1976. Pathology of experimental hypothyroidism in goats. M.V.Sc. thesis, Kerala Agricultural University, Trichur. 155p

Stewart, R.E., Stevenson, J.S., Mee, M.O. and Rettmer, I. 1993. Induction of estrus after thyroidectomy in non lactating Holstein cows. *J. Dairy Sci.* 76 : 2619–2623.

Sujata, R., Rao, D.G.K., Suresh, S. and Mundas, S. 2003. Pathology of the ovarian abnormalities in buffaloes. *Indian Vet. J.* 80 : 412-415

Terlecki, S. and Watson, W.A. 1967. Adenocarcinoma of the uterus of a ewe. *Vet. Rec.* 80 : 516

Timurkaan, N. and Karadas, E. 2000. Morphological Investigations on the Pathological Changes of the Female Reproductive Organs in Goats. *Saglık Bilimleri Dergisi Fırat Universitesi.* 14 (1) : 209-220

Todini, L. 2007. Thyroid hormones in small ruminants: effects of endogenous, environmental and nutritional factors. *Animal* 1 (7) : 997–1008

*Turkarslan, M.T. 1984. Evaluation of cases of in fertility in the pregnancy rate in uterine horns of ewes slaughtered at abattoirs in Istanbul. *Veterineri Fakulteri Desegist Istanbul Universitiesi* 10 (1): 67

Tzora, A., Leontides, L. S., Amiridis, G. S., Manos, G. and Fthenakis, G. C. 2002. Bacteriological and epidemiological findings during examination of the uterine content of ewes with retention of fetal membranes. *Theriogenology.* 57: 1809–1817

Usta, U., Turkmen, E. and Aydin, E.N. 2006. Adrenal Ectopia Within the Wall of an Ovarian Serous Cystadenoma. *Trakya Univ. Tip. Fak. Derg.*. 23 (2) : 95-98

Vanholder, T., Opsomer, G. and Kruif, A.D. 2006. Aetiology and pathogenesis of cystic ovarian follicles in dairy cattle: a review. *Reprod. Nutr. Dev.* 46 : 105–119

Vanselow, B.A. and Spradbrow, P.B. 1983. Squamous cell carcinoma of the vulva, hyperkeratosis and papilloma virus in a ewe. *Aust. Vet. J.* 60 : 194

Veldhuis, J.D. and Hammond, J.M. 1980. Endocrine function after spontaneous infarction of human pituitary. *Endor. Rev.* 1:100-107

Wa´nkowska, M. and Polkowska, J. 2006. The postnatal ontogeny of gonadotroph cells in the female sheep. Developmental patterns of synthesis, storage and release of gonadotrophic hormones. *J. Chem. Neuroanat.* 31: 130–138

Wentzel, D. 1982. Non-infectious abortion in Angora goats. Third International Conference of Goat Production and Disease. 10–15 January, AZ. pp 155–161.

Whitney, K. M., Valentine, B. A. and Schlafer, D. H. 2000. Caprine Genital Leiomyosarcoma. *Vet. Pathol.* 37 : 89–94

Wilkie, B.N. and Krook, L. 1970. Ultimobranchial tumour of the thyroid and pheochromocytoma in the bull. *Vet. Pathol.* 7: 126-134

Winter, A.C. and Dobson, H. 1992. Observations on the genital tract of cull ewes. *Vet. Rec.* 130 : 68-70

Wittek, T. J., Erices, J. and Elze, K. 1998. Histology of the endometrium, clinical and chemical parameters of the uterine fluid and blood plasma concentrations of progesterone, estradiol-17 and prolactin during hydrometra in goats. *Small. Rumin. Res.* 30 : 105-112

Zaki, F.A. and Liu, S.K. 1973. Pituitary chromophobe adenoma in a cat. *Vet. Pathol.* 10 : 232–237.

* Originals not consulted

UTERO-OVARIAN AND ENDOCRINE PATHOLOGY IN GOATS

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ABSTRACT

The present study was aimed at finding out the various utero ovarian disorders affecting goats and correlating with the lesions of pituitary, thyroid and adrenal glands. A total number of 1000 genital tracts were examined for utero-ovarian disorders. Out of which 84 genitalia showed one or more lesions in the ovary, oviduct and uterus. A total of 54 cases out of 84 showed lesions in the ovaries. The follicular cysts were the most common condition and were recorded in 14 cases (1.4%). The other conditions encountered were ovarian hypoplasia (0.6%), luteal cyst (0.3 %), embedded corpus luteum (0.3 %), persistent corpus luteum (0.2 %), ovarian haematoma (0.2 %), ovarian sclerosis(0.4 %), senile atrophy (0.3 %), follicular atresia (0.4 %), epoophoron (0.2 %), serous inclusion cyst (0.2 %), parovarian cyst (0.6 %) and perioophoritis (0.3 %). Seven cases showed lesions in the oviduct. The conditions encountered were hydrosalpinx (0.2 %), hyperplasia of the salpingeal epithelium (0.2 %) and ovario-bursal adhesion (0.3%).Uterine disorders were recorded in 36 cases. The conditions encountered were cystic endometrial hyperplasia (0.6%), adenomyosis (0.4 %), mucometra (0.4 %), perimetrial cyst (0.6%), endometritis (1.4 %) and metritis (0.2%). The histopathological changes of all these conditions have been studied in detail and the possible pathogenesis described. The various pathological changes associated with reproductive disorders are suggestive of endocrine imbalances, multiple nutritional deficiencies, infectious nature, managemental factors and developmental defects. The results of the study highlighted the need to monitor the hormonal levels in the blood, micronutrients and managemental factors.

Lesions of varying frequency in pituitary, thyroid and adrenal glands were recorded in 23 out of 84 cases with utero-ovarian lesions examined. Lesions observed in the pituitary glands were pituitary abscess (1), Pituitary cyst (6) and

pituitary apoplexy (2). In the thyroid gland, lesions recorded were hypoplasia (7), colloid goitre (5) and nodular goitre (2). Lesions observed in the adrenal glands were cortical hypoplasia (2), cortical hyperplasia (5) and medullary hyperplasia (8). Correlation study has highlighted that in many of the utero-ovarian disorders there were concurrent affections of the endocrine glands particularly pituitary, adrenal and thyroid glands which is of major significance.