PATHOLOGY OF UTERO-OVARIAN DISORDERS IN COW

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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 this thesis entitled **"PATHOLOGY OF UTERO-OVARIAN DISORDERS IN COW"** 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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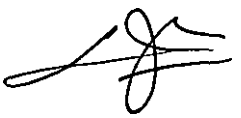
Certified that the thesis entitled **"PATHOLOGY OF UTERO-OVARIAN DISORDERS IN COW"** is a record of research work done independently by **Dr. Shanmugasundaram, K.,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.



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Shanmugasundaram, K.

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Introduction

1. INTRODUCTION

Rearing of cattle had been one of the most ancient agricultural pursuits in India. In fact, from time immemorial Indian farmers had flourished with the cattle as the mainstay of their economy.

As per the provisional figures of 2003 livestock census, the total bovine population in India stands at 283.1 million and among this the cattle population is 187.38 million which accounts for 15 per cent of the world cattle population. Out of the 187.38 million cattle, 22.63 million were crossbred, comprising 12.07 per cent of the total cattle population (F.A.O.2003)

India has become the world's highest milk producing country having produced 95.4 million tonnes in 2005. The annual rate of growth in milk production in India is between 5-6 per cent, against the world's rate of one per cent. Frozen semen technology and genetic up-gradation of cows and buffaloes brought about by progeny testing programs resulted in substantial increase in milk production.

Livestock is an important segment of agricultural sector in India. In 1998-99, it contributed about 23 percent to the agricultural gross domestic product.

Faced with the colossal problem of poverty eradication and improving the standard of living of her people, India immediately after independence launched the Five Year Plans with the primary object of securing a progressive increase in agricultural production.

Cattle production is the main component of livestock production in rural India. There is a great need for raising and maintaining cows for milk, milk products and meat besides supplying the motive power for agriculture and transport

which alone will open up a new vista of freedom from the fear of hunger for our growing population.

Even though considerable improvement in the production of our cattle has been effected by scientific breeding and improved husbandry practices, the genital diseases are prevalent in all species of domestic animals but they occur with higher frequency in dairy animals particularly in cows. Various factors such as congenital defects, anatomical disorders, hormonal disturbances, altered blood biochemistry, infectious diseases and unfavorable environmental conditions cause infertility and /or sterility (Wahid *et al.,* 1991). Reproductive disorders cause infertility ultimately leading to substantial economic losses, increased calving intervals, loss of milk production, maintenance of unproductive animals and culling of valuable breeding animals (Javed and Khan., 1991).

Proper reproductive performance of animals is one of the major contributors to the success of livestock development programmes. In order to have a regular breeding program for animals in pursuit of optimum economic return as well as the maximum production of healthy offspring, the female genital apparatus should be free of ailments /disease. Therefore a systematic study on the disorders of genital organs of cows is of paramount importance for the diagnosis and control of the economically important maladies affecting the reproductive system.

Therefore, the present investigation was therefore conducted with the following objectives.

1. To ascertain the occurrence, nature and magnitude of utero ovarian disorders in cattle.
2. To study the gross and histopathological lesions in the uterus and ovaries.

Review of Literature

2. REVIEW OF LITERATURE

2.1 OVARIAN DISORDERS

Hatipoglu *et al.* (2002) observed 58(5.4 %) cases of ovarian disorders out of the1113 cases examined.

2.1.1 Ovarian hypoplasia

2.1.1.1 Prevalence

Of the 1416 genital organs examined by Summers (1974), bilateral ovarian hypoplasia was present in eight animals (0.12%) comprising five Shorthorns and three Santa Gertrudis.

Of the three cases of ovarian hypoplasia (0.70%) recorded by (Potekar *et al.*, 1982) two were unilateral (0.47%) and one bilateral (0.24%). Herenda (1987) reported six cases (0.11 %) of ovarian hypoplasia.

Of the 13.1 % of the affected Swedish High land breed of cattle, 87.1 % had hypoplasia of the left ovaries, 43% had hypoplasia of the right and 8.6 % in both ovaries (Parkinson, 2001).

Ahmad and Khan (1993) examined reproductive organs of 110 buffaloes and they recorded the incidence of ovarian hypoplasia in 2.56% heifers and in 3.84 % adult buffaloes. Rao *et al.* (1993) examined 1860 Jersey and Holstein cows combinedly for infertility. They recorded 0.7 % of ovarian hypoplasia.

2.1.1.2 Gross pathology

Hypoplastic ovaries were small, smooth, firm and compact with irregular surfaces. There were no graffian follicles or corpus luteum (Roberts, 1971).

Hypoplastic ovaries were smaller than normal, lacked any visible follicles and had characteristic superficial longitudinal grooves (Summers, 1974, Potekar *et al.*, 1982. Sujata *et al.*, 2003).

2.1.1.3 Histopathology

Histological examination of hypoplastic ovaries showed absence of ovarian follicles of any type and lack of previous ovarian activity as indicated by the absence of corpora atretica and corpora albicantia (Summers, 1974).

Thick tunica albugenia and extensive dense stromal connective tissue without any evidence of follicles or corpus luteum were the histopathological lesions observed by Potekar *et al.*, (1982) and Sujata *et al.*, (2003).

2.1.2 Follicular cysts

2.1.2.1 Prevalence

Mylera (1962) recorded an incidence of 0.6% follicular cysts in the reproductive tracts of a random sample of 333 cows which included beef cows.

Goswami and Choudhary (1963) reported one case of follicular cyst while 77 female genitalia collected from a Calcutta slaughterhouse. Derannan and Mac Pherson (1966) reported 2.5 % incidence of follicular cysts.

Out of the 483 cases of follicular cysts 203 were described as miliary, 115 as single or multiple follicular cysts of intermediate size and 50 as large or giant follicular cysts (Boitor *et al.*, 1975).

During the course of an abattoir survey, 486 cases (3.8 %) of the ovaries were found to be cystic (Al-Dahash and David, 1977).

Galvan *et al.* (1982) observed an incidence of 6 % follicular cyst in 2015 ovaries examined.

Kavani *et al.* (1986) reported follicular cysts of small and large sizes in eight cases. Herenda (1987) examined the genital organs of 5800 beef heifers among which bilateral cystic ovaries were found in 807 (14.7%) non pregnant beef heifers. Multiple cysts were observed in 92 cases (11%).

The ovaries of 214 cows examined by Wahid *et al.* (1991) follicular cysts were present in 4.21 % cases.

Ahmad and Khan (1993) examined reproductive organs of 110 buffaloes. They recorded an incidence of cystic follicles as 2.56 % in heifers, 3.84% in adults and 2.22% in aged buffaloes. Rao *et al.* (1993) recorded 1.4 % incidence of follicular cysts. From 5.6 to 18.8 % of dairy cows developed this condition; the mean percentage probably ranges between 10 and 13 % (Garverick, 1997).

Reproductive organs from 402 cows were examined out of which two cases of ovarian follicular cyst were observed, one unilateral and one bilateral multiple follicular cyst (Assey *et al.*, 1998).

Lawton *et al.* (2000) examined reproductive organs from 1134 dairy cows out of which the incidence of follicular cysts was 0.88 %.

Hatipoglu *et al.* (2002) conducted an abattoir study in which the genital tracts of 1113 cows and heifers from different breeds were examined for disorders of the female genital tract. Follicular cyst was found in 27 cases (1.88%), one case was bilateral, while 20 cases were unilateral.

2.1.2.2 Gross pathology

Roberts (1971) described follicular cyst as a thin walled cyst with the wall being more tense and distended and bearing a smooth convex surface. The follicular cyst contained a pale yellow clear fluid and is more common than luteal cyst.

The cyst has a tense convex surface with a thin transparent wall and contained a pale yellowish clear fluid (Wahid *et al.*, 1991).

George (1994) observed that follicular cysts were thin walled with no evidence of luteal tissue lining the inner wall.

Grossly, the follicular cysts varied in size from 2 to 4 cm, and the cyst contained clear a fluid (Hatipoglu *et al.*, 2002).

2.1.2.3 Histopathology

The granulosa layer of cells and ovum are often missing, but scant portions of granulosa cells could be found in the basal portions of the cysts and in some cases these appeared luteinized. The theca interna was likely to be flocculent and edematous showing degenerative changes. Some hyaline changes were occasionally observed. The degenerative changes were more in the peripheral portions of the cyst (Roberts, 1971).

The wall of the follicular cyst was thin and fibrous. The granular cells were completely missing and pressure atrophy of the adjacent ovarian stroma was evident (Rao *et al.*, 1975).

Follicular cysts had fibrous walls enclosing a few degenerated granulosa cell layers along with cellular debris and acidophilic material in their lumen (Singh and Rajya, 1976).

Kennedy and Miller (1991) described follicular cyst degeneration of the granulosa cells and sloughing of these cells into the cyst lumen. The oocyte also underwent degeneration. The theca was partially luteinized in some cases, whereas in others it was replaced by fibrous tissue. Occasionally the luteal tissue occurred in patches or in varying thickness.

Granulosa cells were either completely detached or had few layers bearing wide intercellular spaces with darkly stained pyknotic nuclei. The external limiting membrane was absent. The theca was markedly thickened and could not be differentiated into internal or external partners. Some blood vessels were seen at the periphery (Wahid *et al.*, 1991).

In the follicular cyst, membrana granulosa layer was extremely thin with two or three layers at places and only a single layer of flattened epithelial cells in a few places. There were a few desquamated granulosa cells in the lumen of the cyst. Even though the theca interna and externa could be distinguished histologically it was difficult to distinguish the two layers due to destruction of the interna cells and connective tissue fibers (George, 1994).

The follicular cysts were lined by 1-3 layers of granulosa cells. In some cases, degeneration was seen in the granulosa cells. No ova could be seen in the cystic lumen in all the cases examined (Hatipoglu *et al.*, 2002).

2.1.3 Luteal cyst

2.1.3.1 Prevalence

Derannan and Mac Pherson (1966) reported 0.6% incidence of luteal cyst.

Galvan *et al.* (1982) examined 2015 genital organs out of which 6% had luteal cysts. Kavani et al. (1986) reported luteal cysts in three cases.

Wahid *et al.* (1991) reported a 2.34 % incidence of luteal cysts and they were mostly single and unilateral and commonly prevalent in the left ovaries.

2.1.3.2 Gross pathology

The luteal cyst was thick walled and fluid in the cyst was usually more amber or dark yellow or brown in colour (Roberts, 1971).

Luteal cyst had a soft convex surface with a relatively thick wall lined by orange coloured layers, containing amber yellow colour fluid or even thick dark brown fluid. Major parts of the affected ovaries were completely degenerated. However, the ovaries were significantly heavier and larger than normal ones (Wahid *et al.*, 1991).

2.1.3.3 Histopathology

The wall of the luteal cyst was fibrous and chiefly composed of inner fibrous tissue. The luteal cell layer had variable amounts of connective tissue stroma. The outer thecal cell layer had some interlacing blood capillaries and connective tissue (Singh and Rajya, 1976).

Granulosa cells were completely absent in the luteinised cyst and the deepest parts of the thecal layers were transformed into typical lutein tissue with irregular cell boundaries, vacuolated cytoplasm and large spherical and lightly staining nuclei. The luteinized layer appeared as a continuous layer below the superficial parts of the cavity lining the theca (Wahid *et al.*, 1991).

The luteal cyst wall consisted of an inner thin layer of connective tissue capsule lining the cavity and a fairly thick and luteinized theca interna layer and an outer theca externa. The membrana granulosa layer was absent and even a single layer of flat epithelium on the basement membrane was lacking. There was intense proliferation of collagen laying fibroblasts. The lumen of the cyst contained neither degenerated ovum nor the degenerated and desquamated granulosa cells (George, 1994).

2.1.4 Cystic corpus luteum

2.1.4.1 Prevalence

Mylera (1962) recorded a 36.9 % incidence of cystic corpora lutea. Cystic corpus lutea is the most common cystic condition affecting bovine ovary with the incidence being two and half times greater than the cystic follicles (Roberts, 1971).

Cubic (1972) recorded 77 cases of cystic corpora lutea out of a total of 444 reproductive organs of cows examined. While right ovary was found to be affected in 60 % cases, the frequency of occurrence in left ovary was only 40 %.

Herenda (1987) recorded 19.2 % of cystic corpus luteum.

Corpus lutein cyst accounted for 4.12 % of the cases studied. The conditions were prevalent in the right ovaries (Wahid *et al.*, 1991).

The incidence of cystic corpus luteum was 0.19 % in heifers, 0.17 % in adults and 1.60 % in aged buffaloes (Ahmad and Khan, 1993).

George (1994) recorded 2.78 % incidence of cystic corpora lutea by examining seven cases, of which five were in the right ovary and remaining two in the left.

The incidence of cystic corpus luteum was 2% (Fathallal *et al.*, 2000). Hatipoglu *et al.* (2002) encountered 28 cases of unilateral cystic changes (2.51%) (17 on the right and 11on the left).

Sujata *et al.* (2003) recorded three cases of cystic corpus luteum (1.0%).

2.1.4.2 Gross pathology

George (1994) described that cystic corpora lutea were soft and fluctuating. The size of each cystic corpus luteum appeared slightly larger than the normal corpus luteum with the diameter of central cavity varying from 0.6 to 1.3 cm. The cavity contained a light straw coloured fluid.

The corpora lutea had an average diameter of 1.5-3.5 cm. The diameter of the cystic cavity in the center of corpora lutea varied considerably from about 0.5 to 3.2 cm (Hatipoglu *et al.*, 2002).

Ovaries bearing cystic corpus lutea felt softer and upon sectioning revealed fluid containing central lacunae or cavities ranging from 0.5-0.8 cm in diameter (Sujata *et al.*, 2003 and Fathallal *et al.*, 2000).

2.1.4.3 Histopathology

Microscopically, a cystic corpus luteum is characterized by a large circumscribed cavity lined by thick layers of collagenous fibers joining the capsular trabeculae. Moreover, the luteal cells appeared normal without any special peculiarities (Wahid *et al.*, 1991).

George (1994) described that the cystic cavity was lined by a fairly thick layer of connective tissue without any epithelial lining. The thick luteal tissue did not show the normal lobulation and was least vascular. There was extreme vacuolation in the luteal cells. The nuclei of luteal cells were lightly stained and cell outlines were

indistinct. There was an outer investment of connective tissue which separated the corpus luteum from the ovarian cortex.

The cystic corpora lutea had a zone of fibrous connective tissue between the luteal tissue and the cystic cavity (Hatipoglu *et al.*, 2002).

Histologically, the corpus luteum revealed prominent blood vessels in the external connective tissue. The thecal fibrous tissue showed a tendency of invasion into the body of the corpus luteum dividing it into a number of lobes (Sujata *et al.*, 2003).

2.1.5 Embedded corpus luteum

2.1.5.1 Prevalence

Rahman *et al.* (1977) recorded a single case of persistent corpus luteum. The incidence of the condition was 0.79 %.

Embedded corpus luteum was found in seven cases (0.13%) out of the 5140 cases examined. All the cases were unilateral with four being in right ovaries and three in left (Ghora, 1995).

Sujata *et al.* (2003) recorded eight cases (4%) of embedded corpus luteum in buffalo cows out of the 200 cases examined.

2.1.5.2 Gross pathology

These corpora lutea were solid and slightly larger than normal size. The corpus luteum felt slightly hard on palpation and was pale in appearance (Nair, 1974).

Grossly, there was no lesion but cut surface revealed small, encapsulated yellowish brown lutein tissue of 0.5 to 1.25 cm. in diameter within the cortical stroma (Ghora, 1995).

Sujata *et al.* (2003) described the presence of small, encapsulated dark brown corpora lutea in the ovaries embedded in the thick fibrous stroma of the cortex.

2.1.5.3 Histopathology

Microscopically, the embedded corpus luteum had considerable amount of fibrous connective tissue breaking up the lutein tissue into irregular cell masses. There was intense proliferation of fibroblasts along with laying of collagen fibers. There was reduced vascularisation of the parenchyma. The outer connective tissue capsule of the corpus luteum was considerably thickened. There was clear demarcation of corpus luteum from the surrounding ovarian stroma (Nair, 1974).

Microscopically, the embedded corpus luteum had normal lutein cells. The fibroblastic proliferation forming irregular masses was evident. The lutein cells were large and contained abundant vacuolated cytoplasm (Ghora, 1995).

Histologically embedded corpus luteum showed an outer stromal tissue and an inner thecal layer with infiltrating blood vessels. The luteinized tissue was divided into several lobules by connective tissue septa. Large polyhedral cells were seen possessing vacuolated cytoplasm and centrally placed nuclei (Sujata *et al.*, 2003).

2.1.6 Ovarian haematoma

2.1.6.1 Prevalence

A 0.5 % incidence of ovarian haematoma was recorded by Nair (1974) and Fathallal *et al* (2000).

2.1.6.2 Gross pathology

Haematomas appearing as dark reddish brown masses with 2 to 10 cm diameter could easily be separated from the surrounding ovarian tissue by slight pressure. The clotted blood presented a laminated appearance. In the unorganized haematoma, there were nearly 15 to 20 areas of haemorrhage spread throughout the ovarian cortex (Nair, 1974; George, 1994).

Ovarian haematomas were bilateral and their sizes ranged from 10 to 15 cm. The swelling was grossly visible as a blood clot within a thin-shelled ovarian integument involving the oviducts. The ovarian surface felt smooth and fluctuating on digital manipulation (Fathallal *et al.*, 2000).

2.1.6.3 Histopathology

Microscopically, a large cavity lined by thick connective tissue membrane having spindle shaped fibroblasts. Besides, many small blood vessels and a few mononuclear inflammatory cells were seen at the periphery of the cavity (Wahid *et al.*, 1991).

2.1.7 Parovarian cyst

2.1.7.1 Prevalence

Singh and Rajya (1976) recorded 21 cases of parovarian cysts in goats.

Parovarian cysts were encountered in four cases (0.94%) in buffaloes (Potekar *et al.*, 1982).

Incidence of parovarian cysts were seen in 2.56 % heifers and 3.84 % in adult buffaloes (Ahmad and Khan, 1993). This condition was recorded in four cases (0.32%) (Nair, 1974). Assey *et al.* (1998) recorded two cases of parovarian cysts.

Parovarian cyst was frequently observed on the mesovarium and mesosalpinx. The prevalence rate was 2 % (Fathallal *et al.*, 2000). Lawton *et al.* (2000) recorded 0.26 %. incidence of parovarian cysts.

Parovarian cysts were recorded in eight cases (0.72%) out of which five were on the right side and three on the left (Hatipoglu *et al.*, 2002).

2.1.7.2 Gross pathology

A thin walled parovarian cyst containing a clear fluid was located in the mesosalpinx (Singh and Rajya, 1976).

The cysts were small, soft to feel and measured from 5 to 10 mm.in diameter. The cystic cavity contained a light yellow fluid (Nair, 1994).

Grossly the thin walled parovarian cysts located on the ovaries were circular with the diameter ranging from 1.0 to 1.5 cm and contained a colorless watery fluid (Potekar *et al.,* 1982).

Parovarian cysts were grossly visible as translucent vesicles on the broad ligament (Fathallal *et al.*, 2000).

2.1.7.3 Histopathology

Histologically, the cyst wall had a few muscle fibers and was lined by a single layer of cuboidal or columnar epithelium (Singh and Rajya, 1976).

The cyst wall was made up of a layer of fibrous connective tissue and muscle fibers and it was lined by a single layer of cuboidal or low columnar epithelium. The lining epithelium showed pseudociliary protoplasmic prolongations (Nair, 1994).

The wall of the parovarian cyst had smooth muscle fibers and was lined by a single layer of cuboidal epithelium (Hatipoglu *et al.,* 2002).

2.1.8 Smooth inactive ovaries

2.1.8.1 Prevalence

Nair (1974) recorded 0.8 % incidence of smooth inactive ovaries while Wahid *et al.*, (1991) recorded 3.27 % incidence of smooth inactive ovaries.

2.1.8.2 Gross pathology

The ovaries appeared waxy and yellowish with a pitted surface. There was no evidence of any developing follicles or corpus luteum. The cut surface of the ovaries revealed a firm partially fibrotic interior interspersed with numerous bands of fibrous tissue. There was visible demarcation of the cortical and medullary regions with the cortical region somewhat reduced. Moreover, there were numerous red bodies representing old regressed corpus luteum (Nair, 1974).

The ovaries appeared smooth with no visible follicular growth or corpora lutea on the external surface both grossly and after longitudinal section and these were significantly smaller than normal ovaries (Wahid *et al.*, 1991).

2.1.8.3 Histopathology

The germinal epithelium was virtually absent. In areas where it existed, the cells had become flattened and pyknotic. The tunica albugenia was thickened. The cortex was thinner than that in active ovaries. A few primary follicles were present, but a great majority of them were atretic. There were considerable number of corpora lutea atretica and corpus albicans (Nair, 1974).

Histopathology revealed marked thickening of tunica albugenia ovarii. The cortical area was mostly occupied by collagenous and reticular fibers, numerous fibroblasts, few follicles and unovular follicles. Medullary regions were characteristically wide with clear rete ovarii (Wahid *et al.*, 1991).

2.1.9 Oophoritis

2.1.9.1 Prevalence

Takushi *et al.* (1981) carried out post mortem studies of 10703 female genital organs at abattoirs and recorded oophoritis in 100 cases.

Ghora (1995) recorded eight cases (0.11%) of oophoritis. The right ovary was affected in six cases and the left in two cases.

2.1.9.2 Histopathology

Rao *et al.* (1975) described oophoritis as characterized by infiltration of lymphocytes into the ovarian stroma with a tendency to form aggregates.

Microscopically, the surface epithelium was missing at places. Tunica albuginea was thickened with fibrous connective tissue. The ovarian stroma showed mononuclear infiltration along with a few plasma cells (Ghora, 1995).

2.1.10 Granulosa cell tumor

2.1.10.1 Prevalence

Anderson and Sandison (1969) in a survey on the incidence of genital tumors in Great Britain reported two cases of granulosa cell tumor.

In a study on bovine ovarian tumors, Ito and Fujita (1978) recorded four cases of granulosa cell tumor.

Rodriguez *et al.* (1990) studied the prevalence of ovarian tumors in 1489 genital organs, and granulosa cell tumor was found to be the most common ovarian neoplasm.

Granulosa cell tumor accounted for 0.93 % of the cases examined (Wahid *et al.*, 1991).

Ghora (1995) recorded a single case (0.01%) of granulosa cell tumor.

2.1.10.2 Gross pathology

The lesions appeared as small circumscribed areas, with elevated peripheries and depressed centers, hard in texture and yellow in colour (Wahid *et al.*, 1991).

A hard spherical encapsulated mass 7.2 cm. in diameter, weighing 280 gm was located on the left ovary. The cut surface of the growth was white in colour and lobulated (Ghora, 1995).

2.1.10.3 Histopathology

The tumors were composed of basophilic masses of cells, encapsulated and divided by trabeculae into small masses. Some masses were solid and others had tendencies for follicle formation evidenced as rounded, fluid filled cavities with homogenous acidophilic materials. Individual cells had the typical picture of granulosa cells possessing scanty cytoplasm, polyhedral shape, ovoid nucleus and minimal stromal tissue (Wahid *et al.*, 1991).

Microscopically, the growth revealed numerous tubular structures surrounded by fibrous connective tissue. In the large tubules, there were masses of dense fibrous connective tissue within the lumen. The tubular structures had cylindrical to polyhedral type of cells with centrally placed elongated nuclei. In the neoplastic cells, enlarged and hyper chromatic nuclei were observed with vacuolated cytoplasm. At a few places, there was occasional formation of mitotic figures. At other places the lesions simulating pseudo alveolar pattern were also noticed (Ghora, 1995).

2.2 OVIDUCT

The incidence of congenital and acquired tubal abnormalities was 9 %. (Kessy and Noakes, 1985). Tubal disease was fairly consistently found in 6 to 15 % of the adult cows at slaughter houses. Heifers had a lower incidence of around 3 % (Ellington and Schlafer, 1993). Reproductive tract disorders were detected in 9 cases (0.81 %) involving the oviduct (Hatipoglu *et al.*, 2002).

2.2.1 Hydrosalpinx

2.2.1.1 Prevalence

Fallopian tubes of 39 buffaloes (2.21 %) were affected by hydrosalpinx. Bilateral and unilateral hydrosalpinx were present in 24 and 15 buffaloes respectively (Dwivedi and Singh, 1971).

Nair (1974) recorded two cases (0.16%) of hydrosalpinx, both of which were bilateral.

Hydrosalpinx was a relatively common lesion (0.52%) and occurred both unilaterally and bilaterally (Summers, 1974). Kessy and Noakes (1985) recorded 0.35 % incidence of this abnormality of the uterine tubes while Kavani *et al.* (1986) reported an incidence of 12.51 %.

Herenda (1987) recorded 12 cases (0.22%), three of which were bilateral.

The incidence of unilateral hydrosalpinx was 2.56 % in heifers and 2.22 % in aged buffaloes (Ahmad and Khan, 1993). Rao *et al.* (1993) recorded an incidence 3.8% of hydrosalpinx.

Ghora (1995) recorded 52 cases (1.01%) of hydrosalpinx. It was unilateral in 37 cases and bilateral in 15. In the unilateral cases of hydrosalpinx, the right

fallopian tube was involved in 21 cases and the left in 16. Assey *et al.* (1998) recorded five cases of hydrosalpinx.

Lawton *et al.* (2000) recorded an incidence of 0.88 % of hydrosalpinx.

Hatipoglu *et al.* (2002) recorded four cases (0.36%).

2.2.1.2 Gross pathology

Macroscopically, hydrosalpinx was characterized by the distention of the uterine tube from the uterotubal junction to the infundibulum. The infundibulum was firmly adherent to the ovarian surface. The distended tube was either coiled or straight and tended to fluctuate upon palpation. The uterine tube contained clear and colorless watery fluid (Kessy and Noakes, 1985).

The oviducts were filled with a clear amber colour fluid causing mild dilation. In one case, extreme dilation was observed, with oviduct having a maximum diameter of 2 cm (Herenda, 1987).

The fallopian tubes were distended and translucent. The distension was more towards ampulla. The wall of the tube was thin, semitransparent and grayish white in colour. The lumen of the tube was unilocular in appearance (Nair, 1974).

Grossly, in the simple form of hydrosalpinx the fallopian tube was considerably distended, elongated and tortuous forming many coils in the mesosalpinx. The wall was thin, translucent and distended with 10 to 250 ml of clear fluid. Unlike in the simple form, the fallopian tube in the follicular form was

distended with less amount of fluid but was tortuous, hard, irregularly beaded and the lumen revealed a multilocular appearance (Ghora, 1995).

2.2.1.3 Histopathology

Microscopically, the mucous folds in simple form were considerably atrophied and lined by low cuboidal to columnar epithelium without cilia. The muscular coat and lamina proria were completely thin. In the case of the follicular form, the fibrous septa were thin but in some places, there was marked thickening. On both the sides the trabeculae were lined by low cuboidal or flat epithelium. Infiltration of mononuclear cells and few plasma cells was noticed in the lamina proria. The compartments were filled with homogenous eosinophilic material. The muscular coat was thin and atrophic along with mild lymphocytic infiltration in the serosa (Dwivedi and Singh, 1971; Nair., 1974;Ghora, 1995).

Histologically, there was complete absence of the extensive mucosal folds which normally fill the tubal lumen. The lamina propria which forms the frame work of the folds was absent. However, the oviductal epithelium was preserved with its cilia and rested on a thin muscle layer (Kessy and Noakes, 1985).

Microscopically, fibrosis and adhesions between plicae were detectable in early lesions. Later, the uterine tube appeared broadly dilated with cyst formation and thinning of the mucosal folds. Flattening of the lining epithelium was also observed (Bollo *et al.*, 1990). Microscopic examination showed mucosal atrophy and dilation of the lumen of the oviduct (Hatipoglu *et al.,* 2002).

2.2.2 Salpingitis

2.2.2.1 Prevalence

The incidence of acute salpingitis was reported by different workers. Nair (1974) recorded an incidence of 0.16 % while Kavani *et al.* (1986) reported 16.17 % of cases.

Herenda (1987) recorded 37 cases (0.68%) of salpingitis and Rao *et al.* (1993) recorded an incidence of 3.8 %. Ghora (1995) recorded nine cases (0.17 %) of salpingitis.

2.2.2.2 Gross pathology

In acute salpingitis the salpinx was enlarged nearly two to three times and thickened throughout its length. When exposed, the thickened mucous surface was found to be smeared with tenacious yellowish discharge (Nair1974).

In chronic catarrhal salpingitis there was no apparent change in the size, colour and consistency of the salpinx (Nair1974).

Grossly, the affected fallopian tube showed slight or no enlargement (Ghora, 1995).

2.2.2.3 Histopathology

Microscopically, focal areas of deciliation, degeneration, necrosis and desquamation of the epithelial lining of the villi were observed. The stroma was densely packed with a large number of neutrophils. There was moderate thickening

of the tubal wall. The lumen was packed with necrotic material and neutrophils (Nair, 1974).

In chronic catarrhal salpingitis there were deciliation, necrosis and desquamation of the epithelium lining the villi. The lumen of the tube contained necrotic debris. The hypertrophied epithelial cells become large round and vesicular in appearance. The nuclei of the cells were stained pale (Nair, 1974).

Salpingitis was characterized by widening of plicae and mild to moderate lymphocytic and plasma cell infiltration. Microcysts with in the fibrous wall were observed (Rao *et al.*, 1975).

In the case of acute salpingitis, loss of cilia and focal desquamation of epithelium on the tips of some folds along with leukocytic infiltration could be observed. However, degeneration and desquamation were more prominent in suppurative cases. In the chronic cases of salpingitis depending on the severity of the lesions the salpinx, mesosalpinx and ovary commonly revealed adhesions to one another of variable extents (Kumar and Singh, 1985).

Salpingitis was microscopically characterized by leukocytic exudation into the lumen and thickening of the mucosal folds. The epithelium was still evident, and ciliary changes were difficult to appreciate (Bollo *et al.*, 1990).

Microscopically, there was desquamation of the epithelium and cellular debris was found in the lumen. Scanty to diffuse infiltration of mononuclear cells and fibrocellular reaction were observed in the lamina propria and lymphofollicular aggregates were noticed in the musculature (Ghora, 1995).

2.2.3 Ovaro bursal adhesion

2.2.3.1 Prevalence

Adhesion at the ovarobursal region was observed in 146 cases (8.5 %) in buffaloes. It was unilateral in 71 cases and bilateral in 75 cases (Dwivedi and Singh, 1971).

Al-Dahash and David (1977) in a study conducted at a local abattoir in south western England observed that out of 8071 genital tracts examined, 148 (1.83%) had ovarobursal adhesions. Out of these, 119 were unilateral (65 right and 54 left) and 29 were bilateral.

The adhesions of ovaries with bursa and mesosalpinx were encountered in 17 cases (3.99 %) 11 with unilateral and six with bilateral adhesions. Seven showed complete adhesions (1.64%) and the rest partial adhesions with bursa and mesosalpinx (Potekar *et al.,* 1982).

Kessy and Noakes (1985) examined 2000 specimens of genital tracts for gross abnormalities and lesions of the uterine tubes, out of which ovarobursal adhesion was the most common lesion found from abattoir survey and it was identified in 137 specimens (6.85 %).

Ovarian bursal adhesions were found in 292 cases (5.4%). The majority of cases involved both ovaries (171) with unilateral adhesions occurring in 121 cases (Herenda, 1987).

Ovarobursal adhesions were recorded in 5.12 % heifers, 7.69 % adults and 6.66 % aged buffaloes (Ahmad and Khan, 1993).

Ellington and Schlafer (1993) stated that as much as 44 to 80 % of the uterine tubal disease seen in tracts was attributed to the extensive adhesion between the fimbria and ovary.

The lesion was found in 31 % of the non-cycling animals and in 11% of all the animals and accounted for 65 % of abnormalities in the total herd and 60 % in the non-cycling animals (Assey *et al.*, 1998).

According to Lawton *et al.* (2000) the incidence of ovarobursal adhesions was 0.97%.

2.2.3.2 Gross pathology

Lesions ranged from the presence of slight connective tissue strands to extensive lesions in which the ovaries were completely embedded (Herenda, 1987). Ovarobursal adhesions were different from ovulation tags, which are strands of fibrous tissue extending from the ovarian surface to the bursa. The adhesions were classified as mild, consisting of few fibrous strands or moderate with more than a few strands of fibrous tissue or finally extensive with the concealment of the ovaries by fibrous tissue (Fathallal *et al.*, 2000).

The adhesions varied from thread like strands of fibrous tissue attached to the ovary to extensive adhesions that completely covered the ovary and part of the uterine tube (Kessy and Noakes, 1985).

Grossly such adhesions began with a thin fibrous cord connecting the corpus albicans and mesosalpinx (Assey *et al.*, 1998).

2.3 UTERINE DISORDERS

2.3.1 Hydrometra

2.3.1.1 Prevalence

Nair (1974) reordered three cases of hydrometra (0.24%).

Hydrometra was observed in five buffaloes (Ghora, 1995).

2.3.1.2 Gross pathology

Grossly, the affected uterus had distended uterine horns, depending upon the amount of accumulated fluid. The fluid was transparent and watery without any foul smell. The endometrial mucosa was smooth and cotyledons were small and inactive without any developing follicles or corpus luteum (Nair, 1974., Ghora, 1995).

The wall of the uterus was flaccid and very thin and endometrial surface had no caruncles (Tafti and Darahshiri, 2000).

2.3.1.3 Histopathology

Microscopically, the endometrium was atrophied and lined by a single layer of cuboidal or low columnar epithelium. The uterine glands were reduced in number. The stroma consisted of loose fibrous tissue with occasional oedema. The

myometrium was compressed and had engorged blood vessels. A mild lymphocytic infiltration was noticed in the stroma of the endometrium. Severe atrophy of the endometrial glands was associated with edema of the endometrium and hydropic degeneration of the surface muscle cells of the myometrium (Nair, 1974. Tafti and Darahshiri, 2000).

2.3.2 Mucometra

2.3.2.1 Prevalence

Herenda (1987) reported that the most common uterine abnormality found was mucometra (5%).

Mucometra was noticed in 7.69 % heifers and 3.84 % in aged buffaloes (Ahmad and Khan, 1993). Mucometra was observed in four cases (0.07%) by Ghora (1995) while Assey *et al.* (1998) recorded two cases of mucometra.

According to Lawton *et al.* (2000) the recorded incidence of mucometra was 0.26 %.

2.3.2.2 Gross pathology

The affected uterine horn was distended containing opalescent, viscid fluid. The endometrial mucosa was smooth, having small cotyledons (Ghora, 1995). Thick dense mucus was present in the affected uterus (Herenda, 1987).

2.3.2.3 Histopathology

Microscopically, the endometrium was thin and lined by single layer of low columnar epithelial cells. Myometrium was atrophied, containing congested blood vessels. The endometrial stroma was composed of loose fibrous tissue with reduced number of active uterine glands. A few mononuclear cell infiltrations were observed beneath the endometrial epithelial lining only (Ghora, 1995)

2.3.3 Adenomyosis

2.3.3.1 Prevalence

Nair (1974) recorded two cases (0.16%) of adenomyosis.

2.3.3.2 Gross pathology

No visible alteration in the gross appearance of the uterus (Nair, 1974).

2.3.3.3 Histopathology

Epithelial folds of the endometrium were found within the muscular layer. The epithelial folds were low and lined by tall columnar cells (Nair, 1974).

2.3.4 Endometritis

2.3.4.1 Prevalence

The incidence of endometritis recorded by Nair (1974) was 0.86%. Singh and Rajya (1976) recorded 51 cases of endometritis in goat.

Namboothiripad *et al.* (1978) identified 10 cases of endometritis out of the 900 buffaloes examined.

Out of 26 endometrial biopsy materials examined, 18 (69.2%) showed endometritis (Gosh *et al.*, 1983).

Uterine tissues obtained by biopsy revealed mild endometritis in five (45.45 %) and moderate form in six cows (54.55 %) (Javed and Khan, 1991).

Incidence of endometritis was 2.26 % in heifers, 7.69 % in adults and 13.33 % in aged buffaloes (Ahmad and Khan, 1993).According to Rao *et al.* (1993) the recorded incidence of endometritis was 32.8 % out of the 611 cows cases examined.

Saiyari *et al.* (1994) examined 196 uterine specimens out of which 39(19.90%), 37(18.87%), 23(11.73%) and 18cases (9.18%) were category1, category2, category3 and category4 respectively. Ghora (1995) recorded 59 cases (1.20 %) of endometritis in buffaloes.

Lawton *et al.* (2000) recorded an incidence of 0.35 % of endometritis.

Tafti and Darahshiri (2000) examined the uterine specimens of 470 cows from a slaughter house in the Fares Province of Iran, out of which they recorded endometritis in 39 % of the cases. According to Blanc *et al.*. (2002) the recorded prevalence of endometritis was16.9 %.

The prevalence of cytologically diagnosed endometritis was 53 %; within the herds. The incidence varied from 73 to 74 %(Gilbert *et al.*, 2005).

2.3.4.2 Gross pathology

Lesions of endometritis were classified into 4 categories depending on the severity of the lesions. Category 1: Grossly, endometrium was congested with the presence of varying quantities of mucous in the lumen. Category 2: Grossly, endometrium was congested and edematous.Category3: Grossly, the uterine mucosa showed grayish discoloration and the mucus was scanty. Category 4: Grossly, the uterine mucosa was dry, wrinkled at places and showed discoloration (Saiyari *et al.*, 1994).

In the case of acute non suppurative endometritis, the uterine wall was moderately thickened and edematous. On sectioning, the horns revealed brownish to reddish exudate covering the endometrium. The cotyledons appeared enlarged and mucosa was congested. But in the case of acute suppurative endometritis, the uterine horns were enlarged and soft containing whitish thick pus in variable amounts. The mucosal surface appeared dark red. In sub acute non suppurative endometritis, the affected uterine horns were enlarged and flabby. The lumen of the uterus contained viscous, yellowish exudate mixed with flakes. The mucosa was dark brown to black in colour. Caruncles were sloughed from the epithelial surface. In chronic non suppurative endometritis, the affected uterine horns were thickened, firm and

indurated. Mucosal surface was corrugated and chocolate brown coloured. In chronic suppurative endometritis, the uterine horns were asymmetrically enlarged and flabby. Uterine walls were thickened and the cotyledons were small in size bearing rough surfaces. The uterine lumen contained yellowish to green sticky pus (Singh and Rajya, 1976, Ghora, 1995., Tafti and Darahshiri 2000)).

2.3.4.3 Histopathology

Cupps (1973) examined a group of repeat breeder cows having abnormal uterine glands. Morphologically, these glands were characterized by enlargement of the lumen, various degrees of degeneration of the glandular epithelium and localized modification of the stroma accompanied by infiltration of either eosinophils or lymphocytes or both.

Capillaries and larger vessels were greatly dilated. In the mucous membrane there was considerable collection of blood out side the blood vessels. Intense infiltration of the mucosa with neutrophils and lymphocytes in the superficial layers of the endometrium was noticed. Focal areas of necrosis with moderate stromal edema were also noticed (Nair, 1994).

Rao *et al.* (1975) described endometritis as being characterized by variable degrees of periglandular fibrosis comprising closely packed, concentrically arranged, spindle shaped cells with a slender nucleus. The cystic glands had wide branched lumina lined by low cuboidal epithelium. The lymphoid aggregates were spherical or oval and randomly distributed in the endometrial stroma.

There was focal or moderately diffuse infiltration of the mucosa with lymphocytes, plasma cells, macrophages and polymorphonuclear leucocytes.

Vascular changes associated with inflammation and focal desquamation of epithelium were also noticed. There were concentrically arranged layers of fibrous connective tissue around the endometrial glands, with a large number of actively dividing spindle shaped fibroblast. (Gosh *et al.,* 1983).

The endometrium was edematous with densely packed with chronic inflammatory cells and young fibroblasts. The entire endometrium was highly vascular with many thin walled and thick walled vessels. Most of the glands were atrophied with periglandular accumulation of inflammatory cells. Desqumation of the lining cells was also observed. (Kanjllal *et al.,* 1984)

Gonzalez *et al.* (1985) evaluated reproductive tracts with grading system to determine the severity of pathologic changes. Category 1 was characterized by simple or columnar epithelium or pseudo stratified columnar epithelium. The epithelium had eosinophilic homogenous or vacuolar cytoplasm. Lymphocytes, plasma cells, neutrophils and mast cells were evenly distributed in the lamina propria. The uterine glands varied from straight tubular to tortuous and had no evidence of glandular degeneration. Category 2 endometrium had one to three layers of fibrocytes around the glands. Lymphocytes, plasma cells, neutrophils and mast cells were evenly distributed in the lamina propria with some tendency to be localized in the stratum compactum. Category 3 endometrium had diffusely distributed or few focal aggregates of lymphocytes and four to five layers of fibrocytes around the glands. Some glands were slightly dilated and contained an eosinophilic granular material in the lumen. Category 4 endometrium had more than six layers of fibrocytes surrounding the acini. Along with this, category 4 endometria had the following characteristics: frequent necrolytic vacuoles in the epithelium, marked exocytosis, multifocal aggregates of inflammatory cells, focal aggregates of mast cells and eosinophils around blood vessels and glands. Distended glands

containing inflammatory exudate, cellular debris, or secretory substance were observed.

According to Kavani *et al.* (1986) endometritis was microscopically characterized by oedema of the endometrium causing compression of the uterine glands, focal lymphocytic infiltration forming aggregates, cystic dilatation and degenerative changes in the uterine glands, periglandular fibrosis and atrophy with inflammatory cells.

In mild endometritis, the changes included denudation of lining epithelial cells and moderate infiltration of neutrophils and lymphocytes in the lamina proria. The endometrial glands showed degeneration and necrosis of their epithelial cells. In the case of moderate endometritis, in addition to the above changes, there was dense infiltration of lymphocytes and plasma cells in the stratum compactum and early fibroblastic proliferation around some endometrial glands. There was also periglandular and perivascular leukocytic infiltration. The endometrial glands at some places were distorted and cystic (Javed and Khan, 1991).

Category 1: Microscopically, mucosa showed edema and congestion of the blood vessels. The lamina propria was infiltrated by almost equal numbers of neutrophils, lymphocytes, plasma cells and mast cells. Uterine glands did not show any abnormality. Category 2: The inflammatory cells comprised of neutrophils, eosinophils, mast cells and lymphocytes; the last one predominated and at places formed aggregates. Fibrosis of one to three layers was found around glands. The glandular epithelium showed vacuolation in their cytoplasm. Category 3: The inflammatory cells increased in number and lymphoid aggregates simulated lymphoid follicles. Fibroblasts were seen around the degenerating uterine glands. Category 4: Multifocal lymphoid aggregates especially around blood vessels and

uterine glands were prominent. Fibrosis around the glands was extensive. The glands at places were atrophied and cystic (Saiyari *et al.*, 1994).

Microscopically, subepithelial, perivascular and periglandular lymphoid aggregates were usually noticed. In chronic cases, fibrosis was the prominent lesion in endometrium along with infiltration of lymphocytes with a tendency to form lymphoid follicles at places. The uterine glands varied greatly in number, size and shape concurrently with the pattern and severity of stromal and periglandular fibrosis (Namboothiripad *et al.*, 1978, Kumar and Singh, 1985).

In acute endometritis, the epithelial lining was intact and desquamated at places. Endometrial stroma was heavily infiltrated with neutrophils and occasional mononuclear cells. Blood vessels in the sub mucosa and muscular layer were congested. Few endometrial glands showed degenerative changes (Nair, 1974).

In the case of acute suppurative endometritis, the endometrial lining epithelium was eroded at most of the places with frank necrosis. Lamina propria showed massive infiltration of polymorphs together with congestion. Uterine glands revealed dilatation at places and few glands showed degenerative changes (Nair, 1974).

In sub acute non suppurative endometritis, the surface epithelium was denuded in most of the cases .Varying degrees of lymphocyte and plasma cell infiltration were evident in the endometrial stroma. Focal areas of degeneration with few neutrophilic infiltrations were noticed involving subepithelial tissue and superficial layers of zona compacta .The uterine glands were slightly dilated and the blood vessels were engorged (Nair, 1974).

In chronic endometritis, the lining epithelium along with subepithelial stroma was desquamated at places. Lamina propria revealed extensive infiltration of lymphocytes, macrophages and occasionally plasma cells. Endometrial glands showed atrophy with mild periglandular fibrosis and periglandular lymphocytic infiltration (Nair, 1974).

2.3.5 Metritis

2.3.5.1 Prevalence

Nair (1974) recorded an incidence of 0.08% for metritis.

Namboothiripad *et al.* (1978) examined reproductive organs from 900 buffaloes out of which they recorded eight cases of metritis. But Ahmad and Khan (1993) recorded an incidence of 3.84 % for metritis.

Saiyari *et al.* (1994) recorded an incidence of 4.60 % of metritis.

2.3.5.2 Histopathology

On microscopic examination varying degrees of mononuclear cell reaction was prominent in different layers of the uterus (Namboothiripad *et al.*, 1978).

Microscopically metritis was characterized by infiltration of neutrophils and a few mononuclear cells in the mucosa, muscular layer and serosa along with moderate fibrosis in muscular layer (Saiyari *et al.*, 1994).

2.3.6 Perimetritis

2.3.6.1 Prevalence

Rao *et al.* (1993) recorded an incidence of 0.4% for perimetritis .But according to Nair (1974) the incidence was 0.16% in cow.

2.3.6.2 Gross Pathology

The serous surface of the uterus appeared coarsely granular and the broad ligament was thickened and sclerotic with partial adherence to the serosa (Nair, 1974).

2.3.6.3 Histopathology

Hyalinization of the serosa and infiltration with neutrophils were the only lesions observed in the histological sections (Nair, 1974).

The thickened serosa was infiltrated by mononuclear cells, large number of haemosiderin laden macrophages and mast cells. At places the serosa showed fingerlike projections of fibrous tissue. Mild fibrosis extended into the muscle layer as well. The blood vessels in the muscle layer showed calcification and hypertrophy in their medial layer (Saiyari *et al.,* 1994).

2.3.7 Pyometra

2.3.7.1 Prevalence

The incidence of pyometra was 0.12% in cow. All cases of pyometra contained several liters of mucopurulent material and were associated with retained corpora lutea (Summers, 1974).

Up to 4.44 % of the aged buffaloes suffered from Pyometra (Ahmad and Khan, 1993). Ghora (1995) recorded 12 cases (0.23 5%) of pyometra. While Assey *et al.* (1998) recorded eight cases of pyometra.

Lawton *et al.* (2000) recorded an incidence of 1.23 % of Pyometra in cow.

2.3.7.2 Gross pathology

Grossly, the affected horns were enlarged and there was thinning of uterine wall in most cases. On opening, the uterine lumen showed whitish pus emitting a fetid smell (Ghora, 1995).

2.3.7.3 Histopathology

Microscopically, the thinning of the uterine wall was evident .The musculature was atrophied. The endometrial stroma also showed atrophy with a decrease in the number of uterine glands. The mucosal epithelial lining was sloughed off. A diffuse infiltration of neutrophils along with mononuclear cells was seen in the endometrial stroma (Ghora, 1995).

2.3.8 Leiomyoma

2.3.8.1Prevalence

This tumor was recorded in 3 cases (0.03 %) (Bhowmik, 1985) in cow.

2.3.8.2 Gross Pathology

It was pink in colour, firm in consistency, spherical in shape, dry in appearance and protruding from the organ surface (Bhowmik, 1985).

2.3.8.3Histopathology

Histopathologically, the muscle fibers were spindle shaped with elliptical nuclei possessing chromatin. The bundles of muscles were separated by connective tissue with abundant blood supply (Bhowmik, 1985).

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 investigate the pathology of utero ovarian disorders in the cow.

3.1 MATERIALS

3.1.1 Sample collection

A total of the 1000 genital organs collected from the Corporation slaughter house, Thrissur were utilized for the study. Out of these, fifty cases with lesions were subjected to detailed gross and histopathological examination.

After evisceration of the carcasses, the genital tract as whole were collected, brought to the laboratory and spread out in position. Then a careful examination was undertaken and pathoanatomical abnormalities whenever present were recorded.

3.2 METHODS

3.2.1 Gross examination

The ovaries were opened by longitudinal incision extending from free border to the attached border. The ovarian bursa was spread out by inserting middle and three fingers to detect the presence of lesions. The tubular genital tracts were opened by incising dorsally commencing from the body of uterus to cornua. The exposed mucous membrane was examined for any change 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. Following gross examination, representative pieces of tissue were immediately fixed in 10% formalin solution.

3.2.2 Histopathology

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 the lesions were classified.

Results

4. RESULTS

A total number of 1000 genital tracts were examined for utero-ovarian disorders. Out of which 115 genitalia showed one or more lesions in the ovary, oviduct and uterus.

4.1 OVARIES

Out of the 1000 genital tracts examined, 84 cases showed lesions of varying frequency in the ovaries. The follicular cyst was the most common condition and was recorded in 29 cases. The other conditions encountered were luteal cyst, cystic corpus luteum, embedded corpus luteum, persistent corpus luteum, ovarian haematoma, ovarian sclerosis, senile atrophy and parovarian cyst.

The pathological features observed in each case are as follows.

4.1.1 Follicular cyst

4.1.1.1 Incidence

Follicular cysts were noticed in 29 cases (2.9 per cent). All were unilateral. Among the unilateral cases, the left ovary was involved in 11 cases and right ovary in 18 cases. Multiple follicular cysts were found in eight cases. Among these the left ovary was involved in three cases and right ovary in five cases.

4.1.1.2 Gross pathology

Follicular cysts were thin walled with the wall being tense and distended with pale yellow clear fluid. The single follicular cysts (Fig. 4) diameter varied from 2.5 to 4.9 centimeter. Multiple follicular cysts (Fig. 5) numbering from 2 to 9 had diameter ranging from 1.2 to 2.7 centimeter. In a single case, the multiple follicular cysts (three cysts) in the right ovary were associated with bursal hydrops and ovarian encapsulation.

4.1.1.3 Histopathology

The cysts varied widely in their histological appearance. The granulosa cells showed considerable variation in different cysts. In some of the cases it was quite wide with as many as 10 rows (Fig. 22), while in others it was observed few rows (one to three) of cells (Fig. 23), but had wider intercellular spaces with darkly stained pyknotic nuclei. Granulosa cells were completely detached in a few cases. In some cases degenerations were seen in the granulosa cells (Fig. 24) and in some, the granulosa layer was completely absent (Fig. 25). In one case, the follicular cyst had a fibrous wall enclosing a few degenerated granulosa cell layers along with cellular debris and acidophilic material in its lumen. In case of the larger follicular cysts, the granulosa cells were completely missing; the walls appeared irregular and were lined by strands of connective tissue stroma (Fig. 26). Degenerated and necrotic cumulus oophorus with remnants of ova could be seen in some of the cysts which were not too large. The basement membranes were absent in all the cysts examined. It was also difficult to distinguish the two layers of theca interna and theca externa. Some blood vessels were seen at the periphery.

4.1.2 Luteal cyst

4.1.2.1 Incidence

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

4.1.2.2 Gross pathology

Luteal cysts had thick walls and the fluid in each of the cyst was more amber or darker yellow or brown in colour and cloudy in consistency. The diameter of the cyst varied form 2.6 to 3.1 centimeter. In one of the cases, the cyst wall was lined by orange coloured tissues (Fig.6).

4.1.2.1 Histopathology

Microscopically, the granulosa cells were completely absent in all cases. The cyst wall comprising of three layers surrounded the central cavity that contained homogenous eosinophilic contents. The inner layer consisted of a thin band of loose connective tissue that separated adjacent luteal tissue from the cystic contents. The middle layer had lutein cells of varying thickness. The luteal cells were large cells with irregular cell boundaries, vacuolated cytoplasm and large spherical lightly stained nuclei. The outer wall contained concentrically arranged dense connective tissue stroma of the ovary (Fig. 27).

4.1.3 Cystic corpus luteum

4.1.3.1 Incidence

Cystic corpus luteum was found in three cases (0.3 per cent). All the cases were unilateral two being in the right ovary and one in left.

4.1.3.2 Gross pathology:

Corpus luteum cysts were soft, with the cavities ranging from 0.5 to 0.8 centimeter in diameter and the cavities contained light straw coloured fluid (Fig.7).

4.1.3.3 Histopathology

The cystic cavities were lined by a layer of connective tissue without any lining epithelium. The thick luteal tissue did not show any normal lobulation and was least vascular but in one case considerable amount of fibrous connective tissue breaking up the luteal tissue into irregular cell masses could be seen. There was extreme vacuolation in the luteal cells (Fig.28) due to regressive changes. The nuclei of luteal cells were lightly stained and the out line of the cells were indistinct. There was an outer investment of connective tissue which separated the corpus luteum from the ovarian stroma.

4.1.4 Parovarian cyst

4.1.4.1 Incidence

Parovarian cysts were recorded in 22 cases (2.2 per cent), two cases being bilateral while the remaining 20 cases were unilateral. Among the unilateral cases, the left side was involved in eleven cases and right side in nine cases. The cysts were seen on the mesovarium, mesosalpinx and the ovary. A single case had multiple parovarian cysts on the left mesovarium.

4.1.4.2 Gross pathology

The cysts were small, soft and thin walled and contained colorless watery fluid (Fig.8). The shape of the cysts varied from circular to oval.

4.1.4.3 Histopathology

The cyst wall was 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 does not contain any material (Fig. 29).

4.1.5 Embedded corpus luteum

4.1.5.1 Incidence

Embedded corpus luteum was recorded in 12 cases (1.2). All the cases were unilateral in which eight cases were in the right ovary and four cases in the left.

4.1.5.2 Gross pathology

Grossly there were no lesions but cut surfaces of the ovaries revealed encapsulated, yellowish brown coloured corpus luteum varying from 0.5 to 1.29 centimeters in diameter within the ovary (Fig.9). 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 traversed into the lutein tissues and divided it into number a of lobes (Fig.30). The luteal cells had extreme vacuolation due to regressive changes. The nuclei of luteal cells were spherical and lightly stained and the cell boundaries were indistinct. There was abundant vascularisation in the parenchyma. This corpus luteum was seen located both in the cortex and medullary regions. The density of the ovarian cortex was reduced. There was no evidence of any developing follicles in the ovarian cortex but some of the existing developed follicles underwent cystic transformation.

4.1.6 Persistent corpus luteum

4.1.6.1 Incidence

Persistent corpus luteum was recorded in a single case (0.1 %). It involved in the left side of the ovary.

4.1.6.2 Gross pathology

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

4.1.6.3 Histopathology

The persistent corpus luteum had a 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 were transformed into cystic follicles.

4.1.7 Ovarian haematoma

4.1.7.1 Incidence

Ovarian haematomas were recorded in two cases.

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 laminated appearance on sectioning (Fig.11). The blood clots were easily removed from the ovary.

4.1.7.3 Histopathology

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

4.1.8 Serous inclusion cyst

4.1.8.1 Incidence

Serous inclusion cyst was recorded in a single case (0.1 %).

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 a single case.

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 (Fig. 33) Large dilated lymphatic ducts were seen in the medulla.

4.1.10 Atretic follicles

4.1.10.1 Incidence

Incidence of atretic follicles was recorded in two cases (0.2 %).All were bilateral.

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 (Fig. 34). 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 cell were seen. Theca enclosed the remnants of the zona pellucida.

4.1.11 Ovarian sclerosis

4.1.11.1 Incidence

Ovarian sclerosis was recorded in 2 cases (0.2 %). All the cases were unilateral, one case in right ovary and one in the left.

4.1.11.2 Gross pathology

Sclerosed ovaries were small and hard. Neither corpus luteum nor follicles were evident on the surface (Fig.12). The cut surface revealed a dense stroma.

4.1.11.3 Histopathology

The surface epithelium was absent in 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 five cases (0.5 %) 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. 13).

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 in a few cases but in two cases it remained intact. 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 a single case.

4.1.13.2 Gross pathology

Grossly the ovarian surface was shaggy in appearance.

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 in a few places. The tunica albugenia was infiltrated with lymphocytes and few a 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 and hyperplasia of the salpingeal epithelium.

The pathological features observed in each case are as follows.

4.2.1 Hydrosalpinx

4.2.1.1 Incidence

Hydrosalpinx was recorded in a single case (0.1 %) out of 1000 genital tracts. It was seen in the right oviduct.

4.2.1.2 Gross pathology

The fallopian tube was distended with a clear amber coloured fluid (Fig.14). The distension was more towards ampulla. The wall of the tube was thin and translucent.

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 could be observed (Fig. 37).

4.2.2 Hyperplasia of the salpingeal epithelium

4.2.2.1 Incidence

Hyperplasia of the salpingeal epithelium was recorded in three cases (0.3%).

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 sieve like obliteration in the tubular lumen was characteristic (Fig. 38).

4.3 UTERUS

Out of the 1000 genital tracts examined 24 cases showed lesions of varying frequency in the uterus. The conditions encountered were endometrial hyperplasia, adenomyosis, mucometra, perimetrial cyst, endometritis and metritis.
The pathological features observed in each case are as follows.

4.3.1 Endometrial hyperplasia

4.3.1.1 Incidence

Endometrial hyperplasia was recorded in two cases (0.2 %).

4.3.1.2 Gross pathology

The affected uterus revealed no gross lesions except moderate thickening.

4.3.1.3 Histopathology

Microscopically, there was an increase in the size and number of glands which appeared irregular in their distribution and course. The normal parallel

alignment was lost. The glandular epithelium in all cases were tall columnar and the lumen contained secretions. Endometrial vessels were engorged (Fig. 39).

4.3.2 Adenomyosis

4.3.2.1 Incidence

Adenomyosis was recorded in three cases (0.3 %).

4.3.2.2 Gross pathology

The affected uterus revealed no gross lesions.

4.3.2.3 Histopathology

Microscopically, endometrial glands were seen embedded 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 a single case (0.1 %).

4.3.3.2 Gross pathology

Both uterine horns were distended with 100 to 150 ml of opalescent, viscid fluid (Fig. 15). The endometrial mucosa was smooth and had small cotyledons.

4.3.3.3 Histopathology

The endometrium was thin and lined by single layer of low columnar epithelium with reduced number of active glands in the endometrial stroma (Fig.41). Myometrium was congested.

4.3.4 Serous vascular protrusions

4.3.4.1 Incidence

Serosal vascular protrusions of the uterus were found in five cases (0.5 %).

4.3.4.2 Gross pathology

Grossly soft nodules of 3-6 mm diameter simulating varicose veins were seen at the dorso lateral aspects of the uterine horns (Fig.16).

4.3.4.3 Histopathology

Microscopically, the walls of the vessels were thick (Fig. 42) and all the vessels were distended with blood. Some of the vessels revealed thrombi. Hyperplasia and thickening of the blood vessels were observed.

4.3.5 Perimetrial cyst

4.3.5.1 Incidence

Perimetrial cyst was observed in a single case (0.1 %).

4.3.5.2 Gross pathology

The cyst was oval in shape and contained clear watery fluid. Cyst was noticed on the serosal surface (Fig. 17).

4.3.5.3 Histopathology

The cyst wall was made up of smooth muscle fibers and lined by a layer of flattened epithelium.

4.3.6 Endometritis

4.3.6.1 Incidence

Endometritis was recorded in 11 cases.

Endometritis was graded based on the degree of fibrosis of the uterine glands and inflammatory reaction in the uterine mucosa.

4.3.6.1.1 Category 1

This type of endometritis was recorded in 4 cases.

4.3.6.1.1.1 Gross pathology

Grossly endometrium was congested with the presence of varying quantity of mucus in the lumen.

4.3.6.1.1.2 Histopathology

Microscopically, mucosa showed congestion of the blood vessels. The uterine glands varied in shape from straight tubular to tortuous and were lined by cuboidal epithelium and showed no evidence of degenerative or necrotic changes. Cellular infiltrate or periglandular fibrosis was not seen (Fig. 43).

4.3.6.1.2 Category 2

This type of endometritis was recorded in 2 cases.

4.3.6.1.2 .1 Gross pathology

Grossly endometrium was congested (Fig. 18).

4.3.6.1.2 .2 Histopathology

Microscopically, there was periglandular fibrosis. One to three layers of fibrous tissue was seen around the glands (Fig. 45). The glandular epithelium showed vacuolation in their cytoplasm. Inflammatory cell infiltration was diffuse and comprised of neutrophils, mast cells and lymphocytes (Fig.44). The lymphocytes in some places formed aggregates.

4.3.6.1.3 Category 3

This type of endometritis was recorded in 2 cases.

4.3.6.1.3 .1 Gross pathology

Grossly the uterine mucosa showed grayish discoloration (Fig. 19) and the mucus was scanty.

4.3.6.1.3 .2 Histopathology

Microscopically, periglandular fibrosis was extensive. Many layers of fibrous tissue were observed around the glands (Fig. 46). Glandular atrophy and various cellular changes were seen. Lymphoid aggregates were seen in the stroma.

4.3.6.1.4 Category 4

This type of endometritis was recorded in 3 cases.

4.3.6.1.4.1 Gross pathology

Grossly endometrium was dry, devoid of mucus, wrinkled at places and showed grayish discoloration (Fig. 20) of the uterine mucosa.

4.3.6.1.4 .2 Histopathology

Microscopically, there was extreme periglandular fibrosis. All the glands appeared atrophic and slit like (Fig. 47). The lining cells showed degeneration and desquamation (Fig. 48) and some appeared as clumps within the lumen.

4.3.7 Puerperal metritis

4.3.7.1 Incidence

Puerperal metritis was recorded in a single case.

4.3.7.2 Gross pathology

The uterine lumen contained chocolate colored lochia, which was slightly tenacious and without any foul odour. The endometrium was congested and haemorrhagic (Fig. 21).

4.3.7.3 Histopathology

Microscopically, the mucosal, muscular and serosal layers were infiltrated with neutrophils and a few mononuclear cells (Fig. 49). The vessels in these layers were congested. Moderate perivascular and periductular fibrosis were evident.

Table 1. Prevalence of Utero-ovarian lesions

S.No	Type of organ	No. of cases examined	No. of cases with lesions
1	Ovary	1000	84
2	Oviduct	1000	7
3	Uterus	1000	24

Table 2. Prevalence of Ovarian lesions

S.No.	Type of ovarian abnormality	Left ovary	Right ovary	Bilateral	Total	Per cent
1	Follicular cyst	11	18	-	29	2.9
2	Luteal cyst	1	2	-	3	0.3
3.	Cystic corpus luteum	1	2	-	3	0.3
4	Parovarian cyst	11	9	2	22	2.2
5	Embedded corpus luteum	4	8	-	12	1.2
6	Persistent corpus luteum	1	-	-	1	0.1
7	Ovarian haematoma	1	1	-	2	0.2
8	Serous inclusion cyst	-	1	-	1	0.1
9	Epoophoron	1	-	-	1	0.1
10	Atretic follicles	2	-	-	2	0.2
11	Ovarian Sclerosis	1	1	-	2	0.2
12	Senile atrophy	-	-	5	5	0.5
13	Perioophoritis	-	1	-	1	0.1
	Total				84	

Table 3. Prevalence of Oviduct lesions

S.No	Type of oviductal abnormality	Left	Right	Bilateral	Total	Per cent
1	Hydrosalpinx	-	1	-	1	0.1
2	Salpingeal hyperplasia	-	-	3	3	0.3
3	Mucosal cyst	1	2	-	3	0.3
	Total				7	

Table 4. Prevalence of Uterine lesions

S.No	Type of uterine abnormality	No. of cases	Per cent
1	Endometrial hyperplasia	2	0.2
2	Adenomyosis	3	0.3
3	Mucometra	1	0.1
4	Serous vascular protrusions	5	0.5
4	Perimetrial cyst	1	0.1
5	Endometritis	11	1.1
6	Puerperal metritis	1	0.1
	Total	24	

Fig.1. Distribution of different utero ovarian lesions

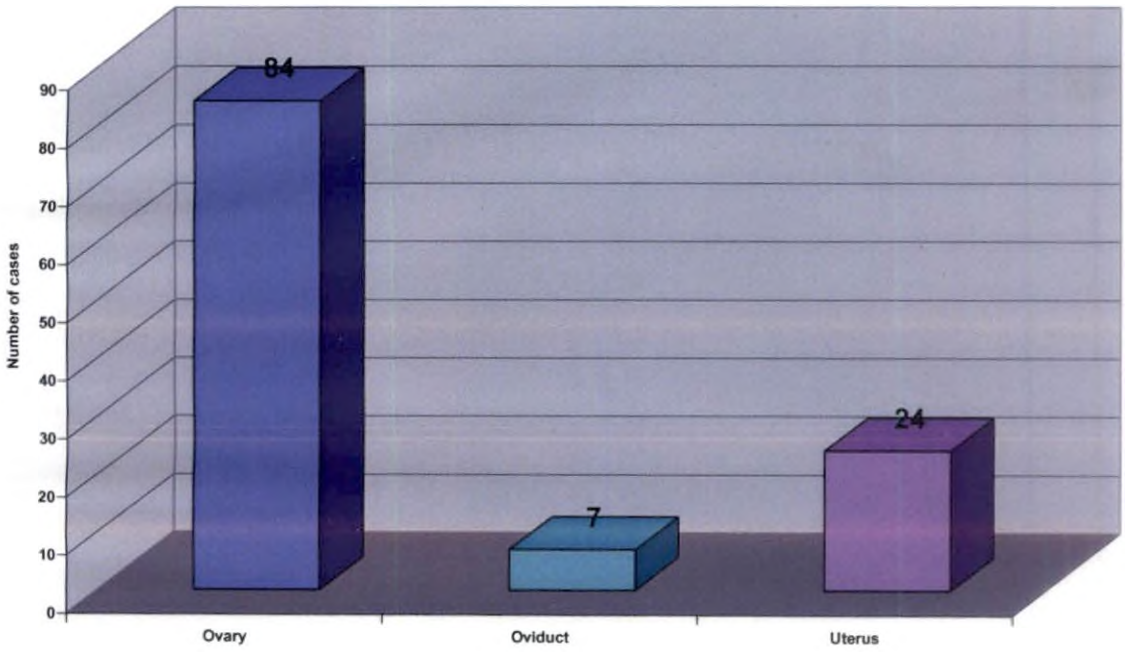


Fig.2. Distribution of different ovarian lesions

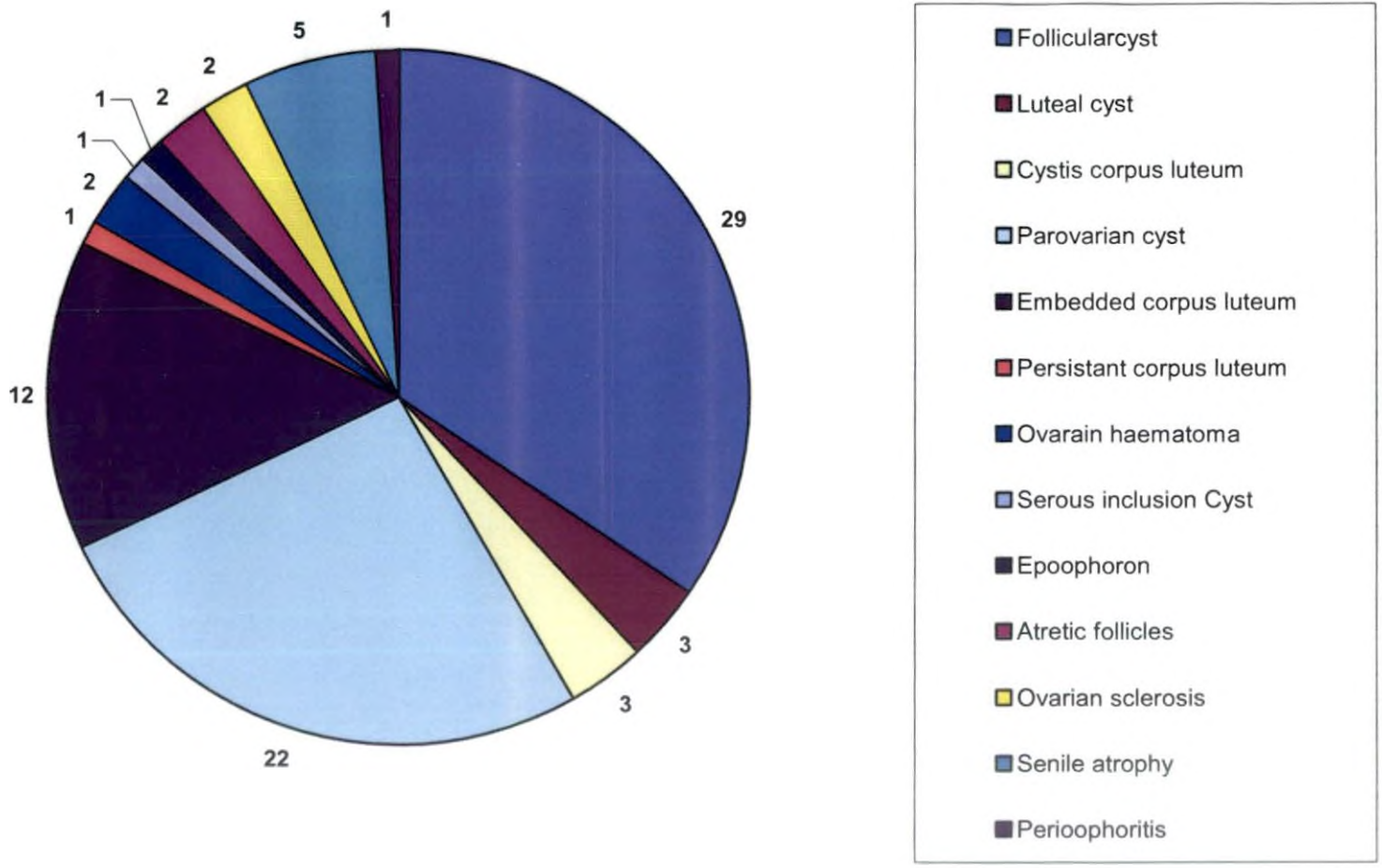
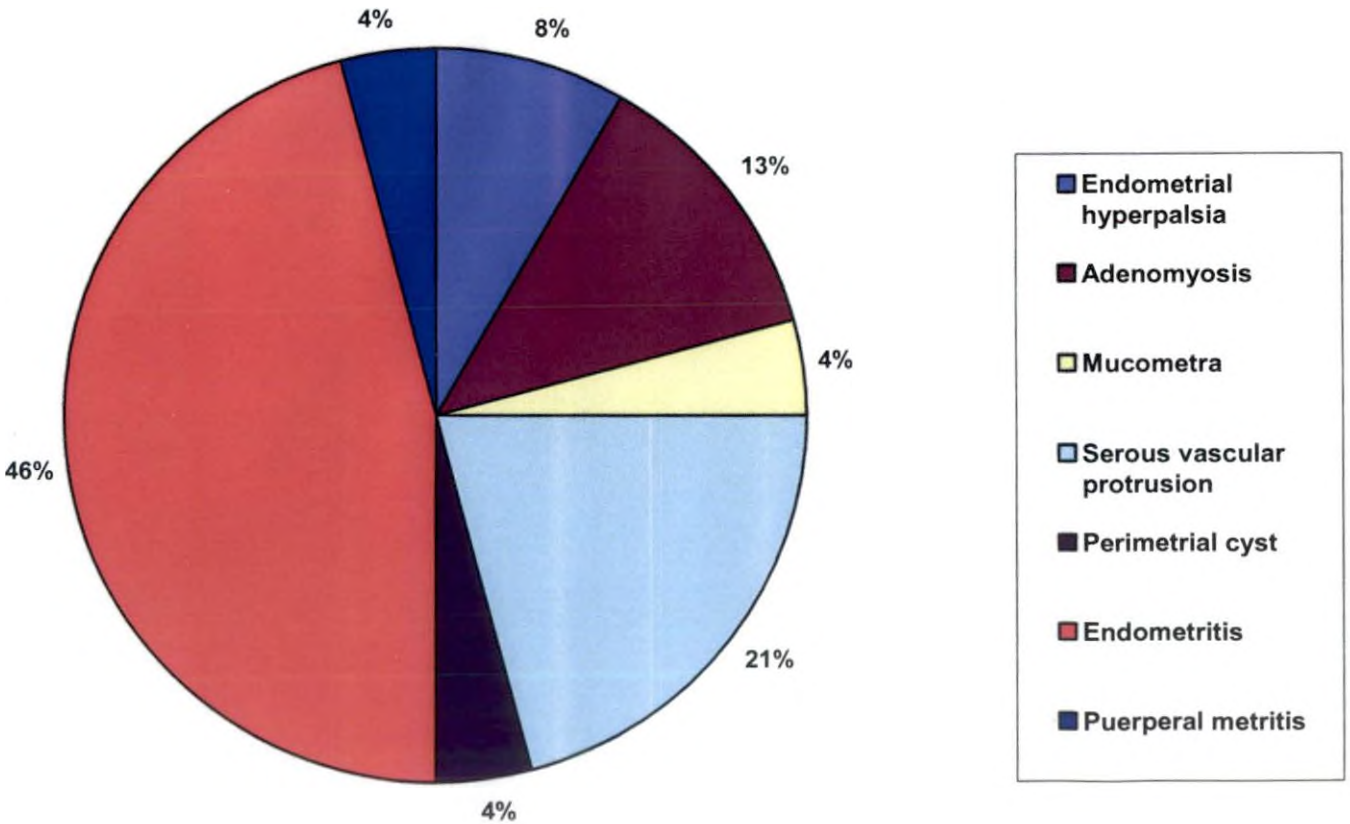


Fig.3. Distribution of differnt uterine lesions



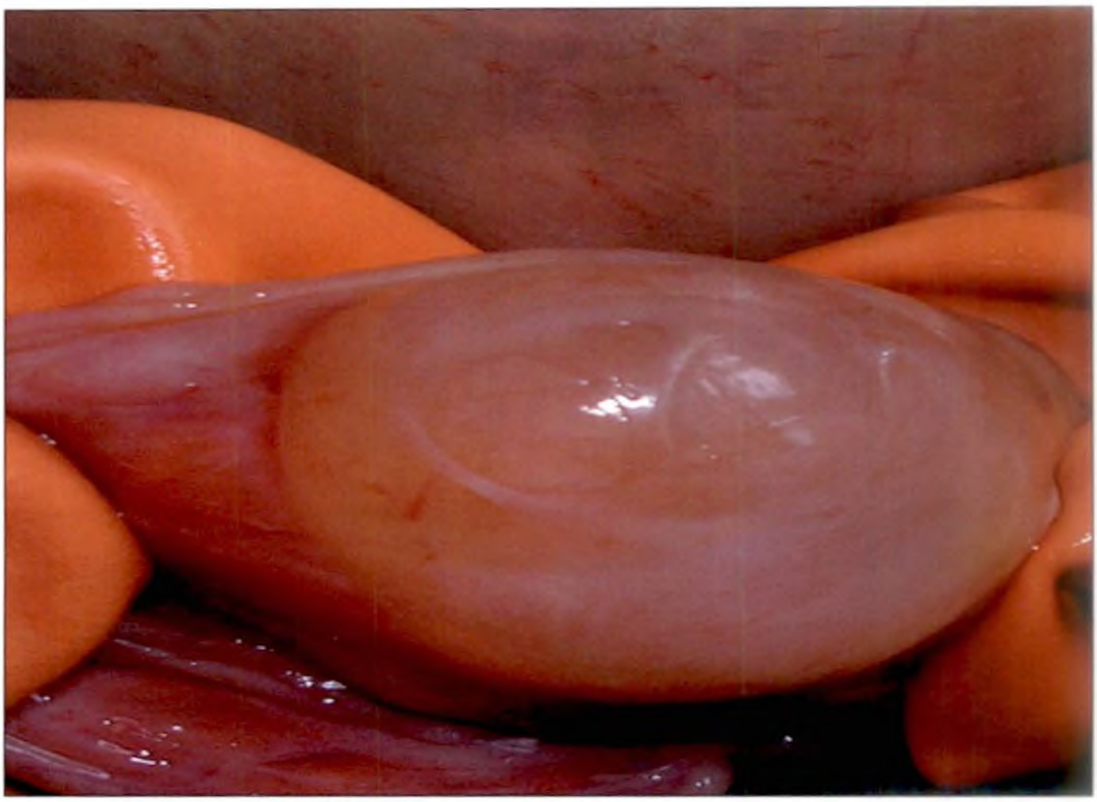


Fig.4.Single follicular cyst in the right ovary

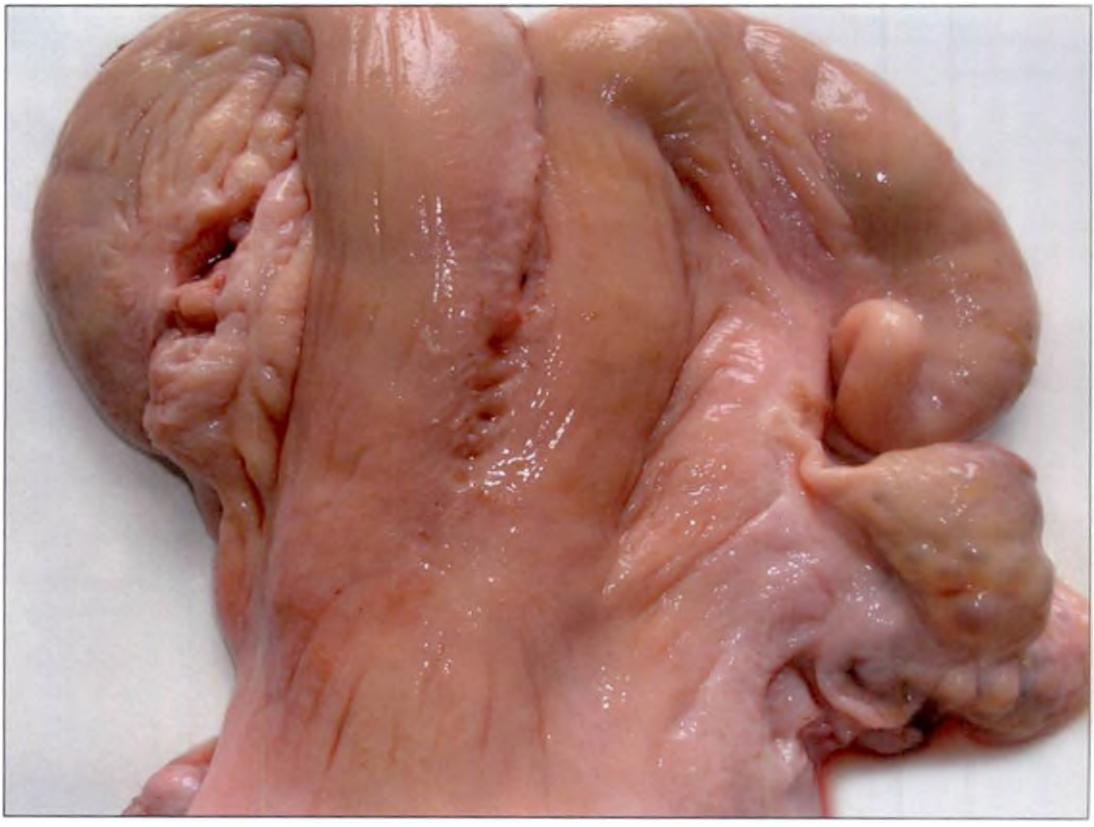


Fig. 5. Multiple Follicular cyst in the right ovary

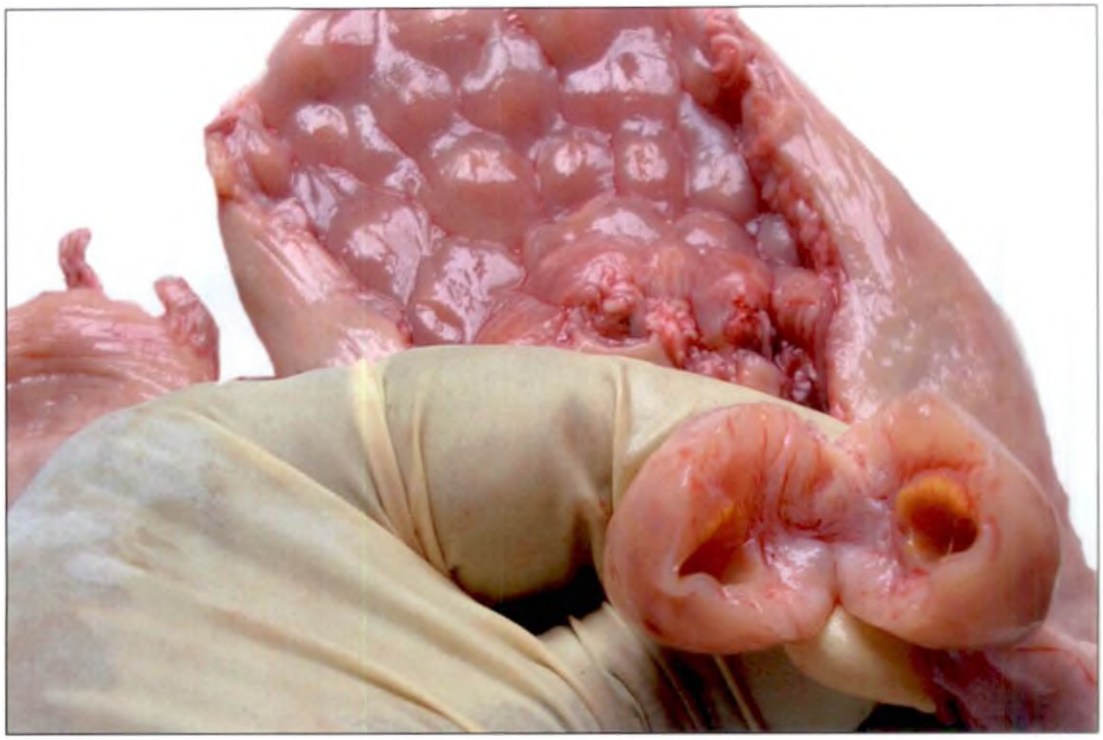


Fig. 6. Luteal cyst in the right ovary, cyst wall having orange colour tissue

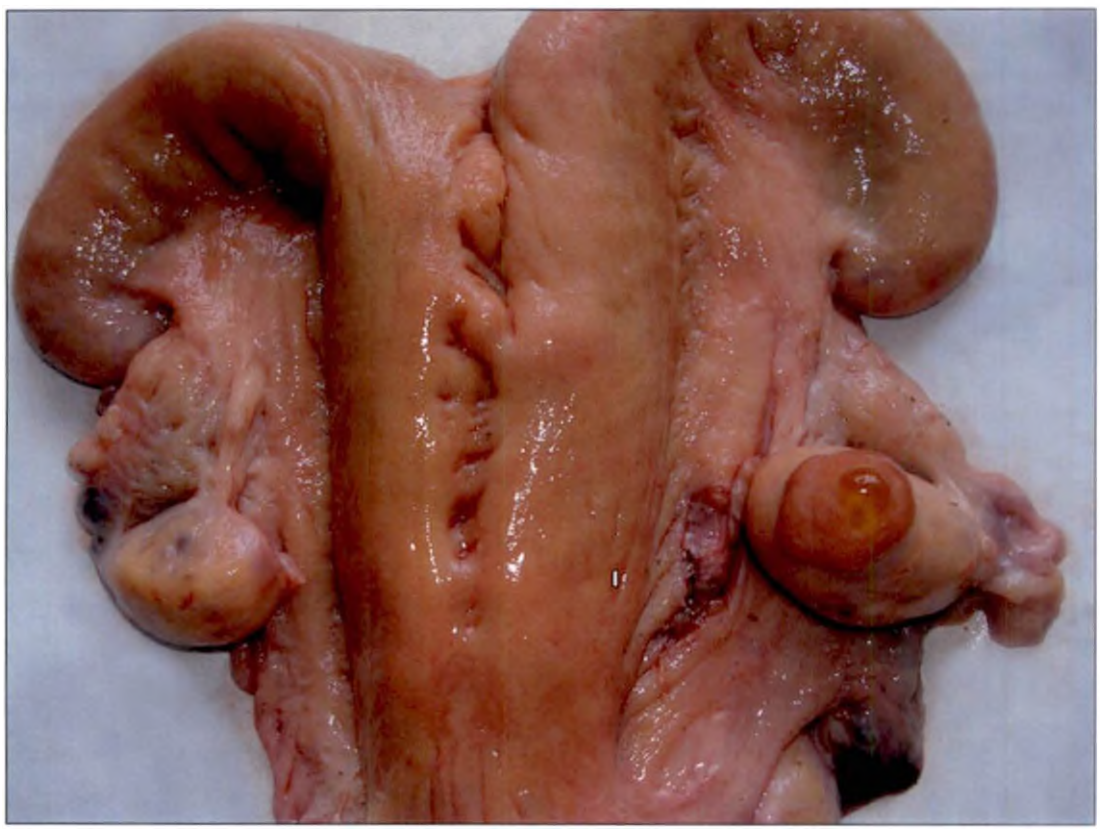


Fig. 7. cystic corpus luteum in the right ovary

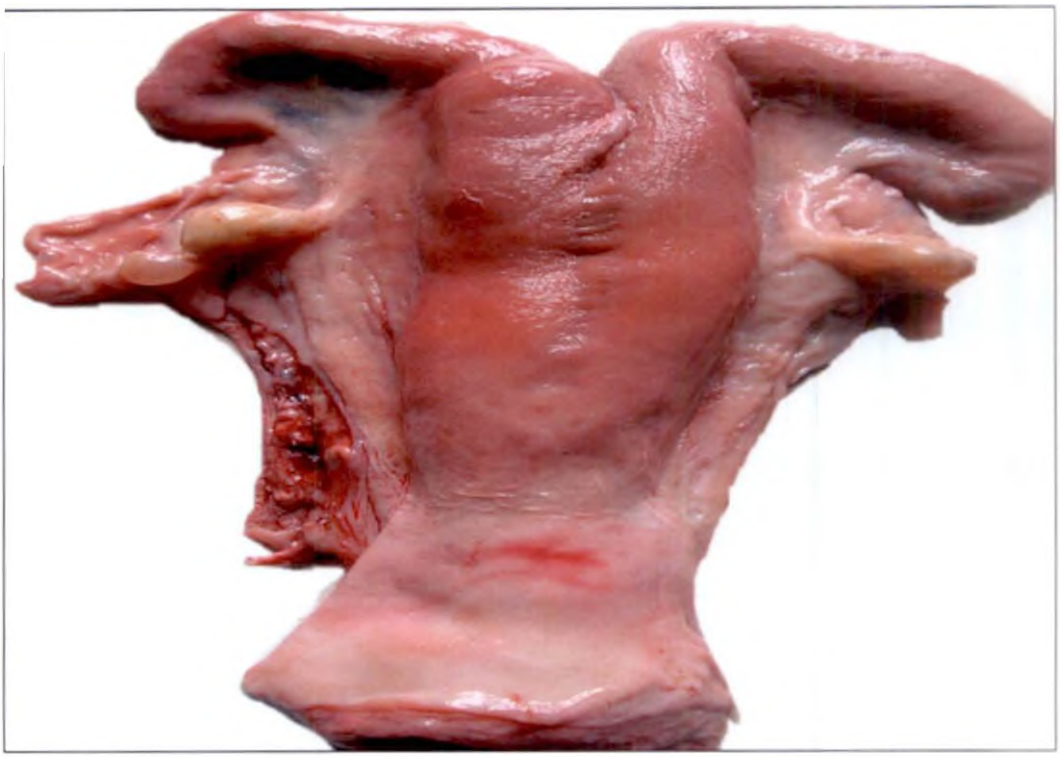


Fig. 8.Parovarian cyst in the left side, near the fimbriated end of the ovary

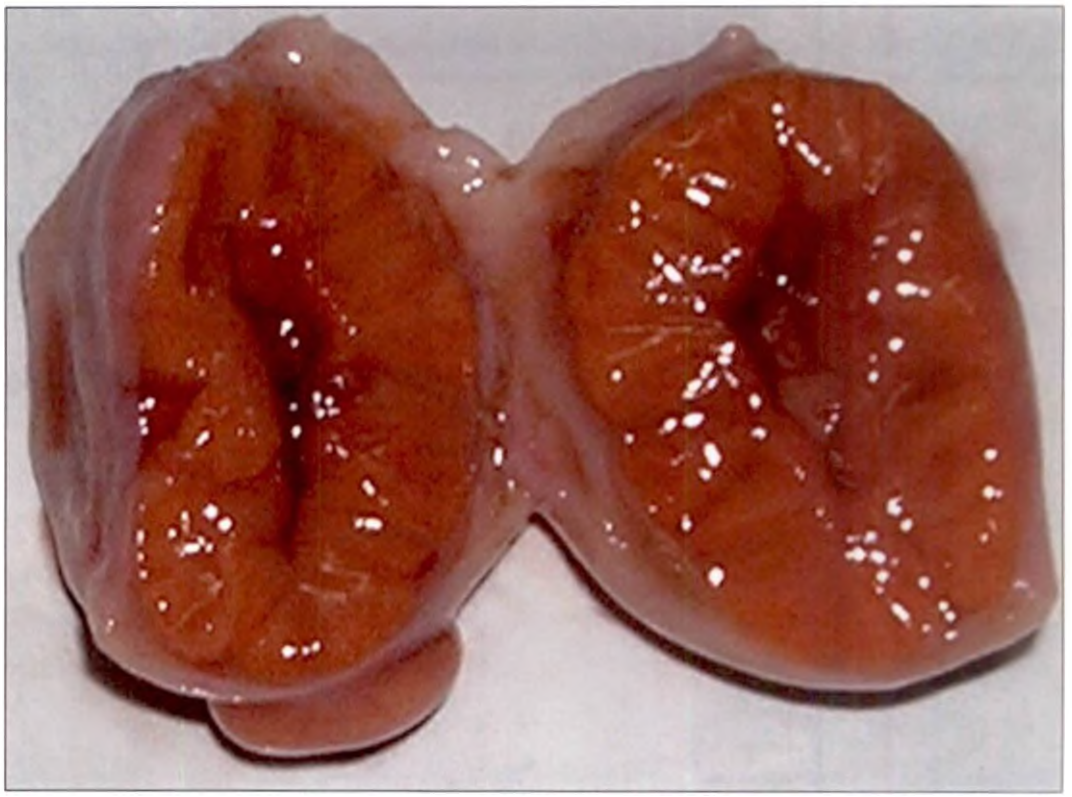


Fig. 9. Encapsulated corpus luteum within the ovary

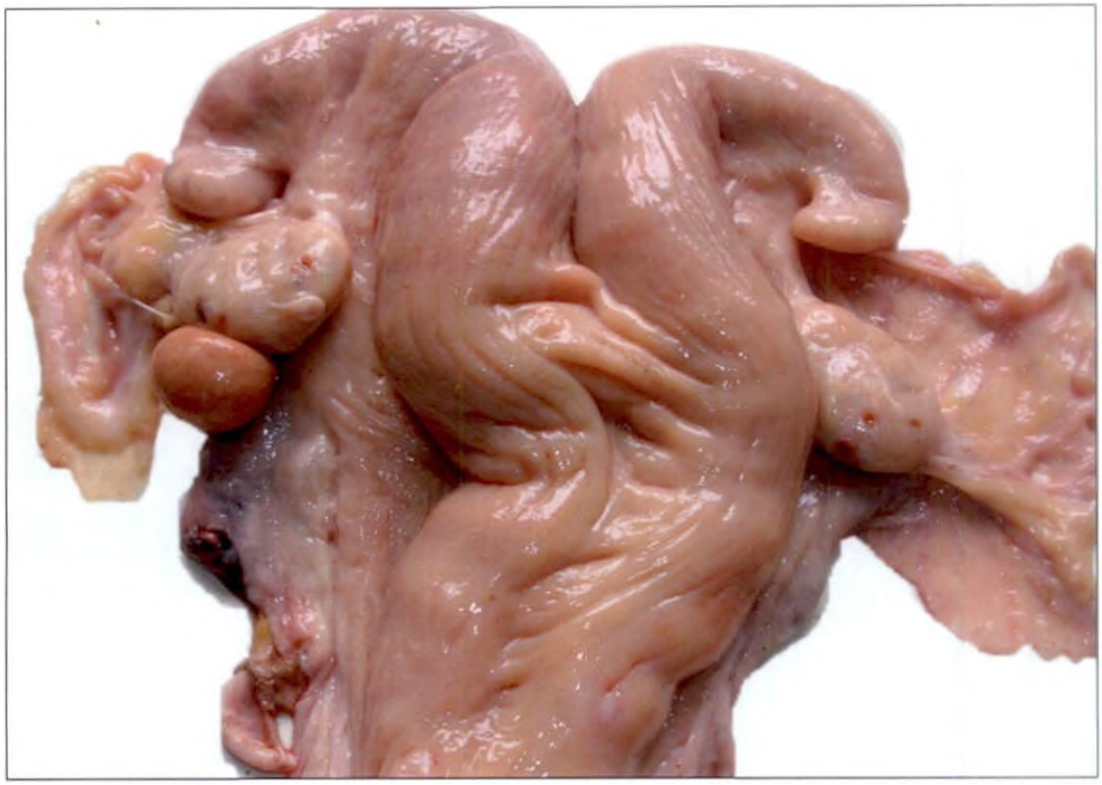


Fig. 10. Persistent corpus luteum in the right ovary. The crown is projecting on the ovarian surface.

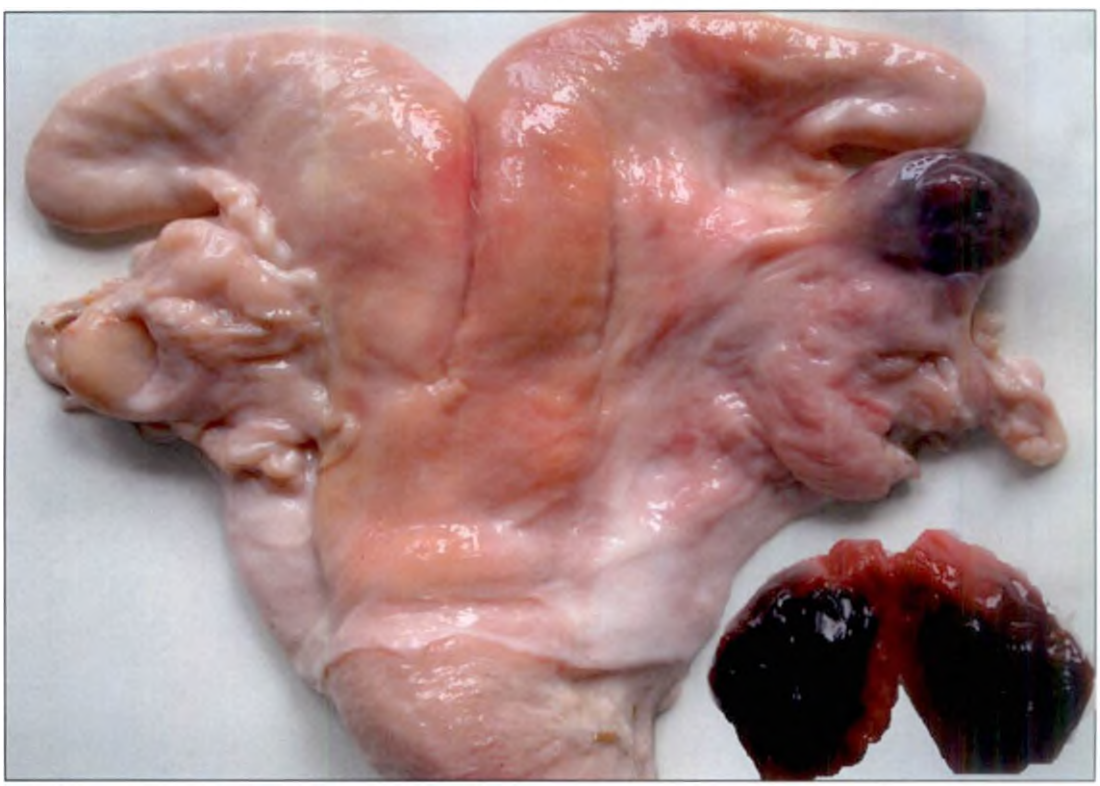


Fig. 11. Ovarian haematoma in the right ovary

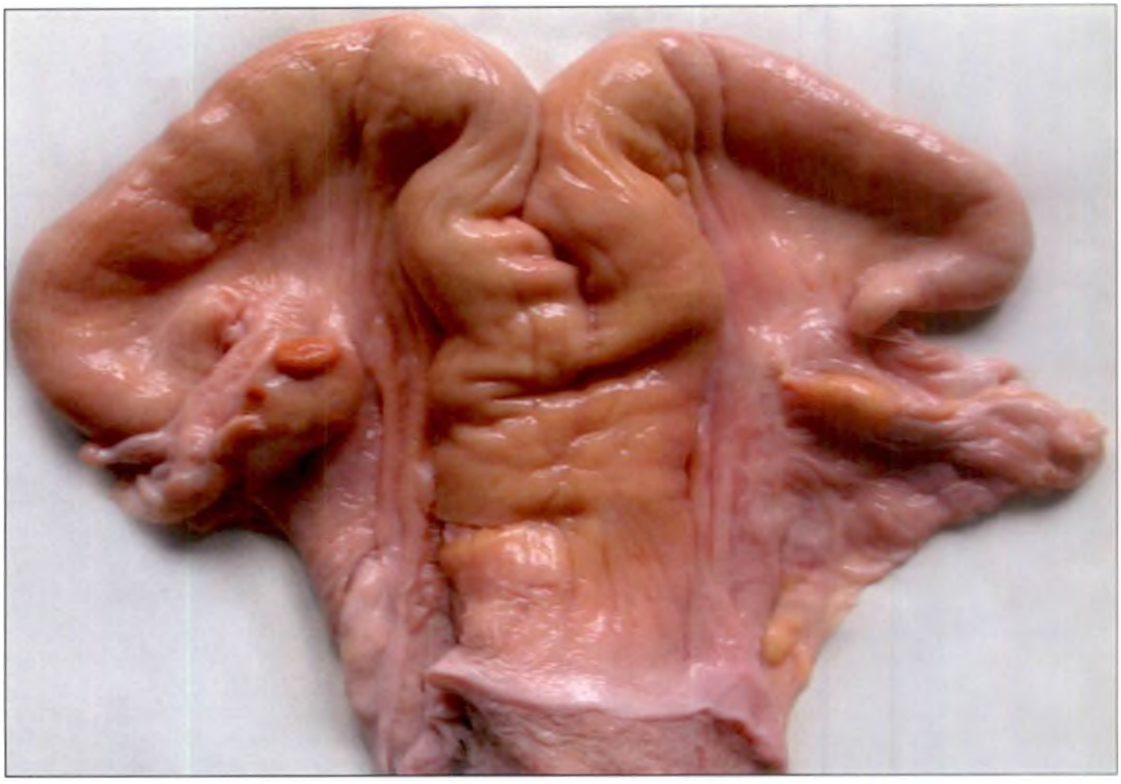


Fig. 12. Small sclerosed ovary in the right side

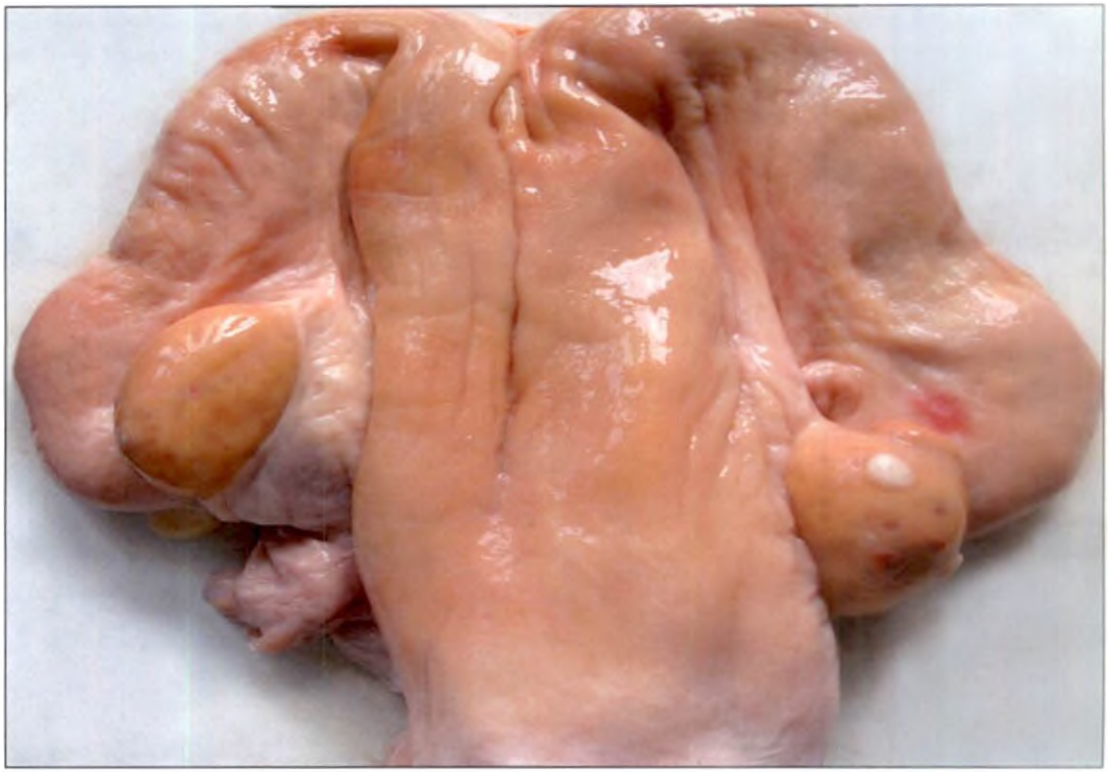


Fig. 13. Senile ovaries

with rough pitted surface with pin head sized follicles,red bodies and corpus albicans

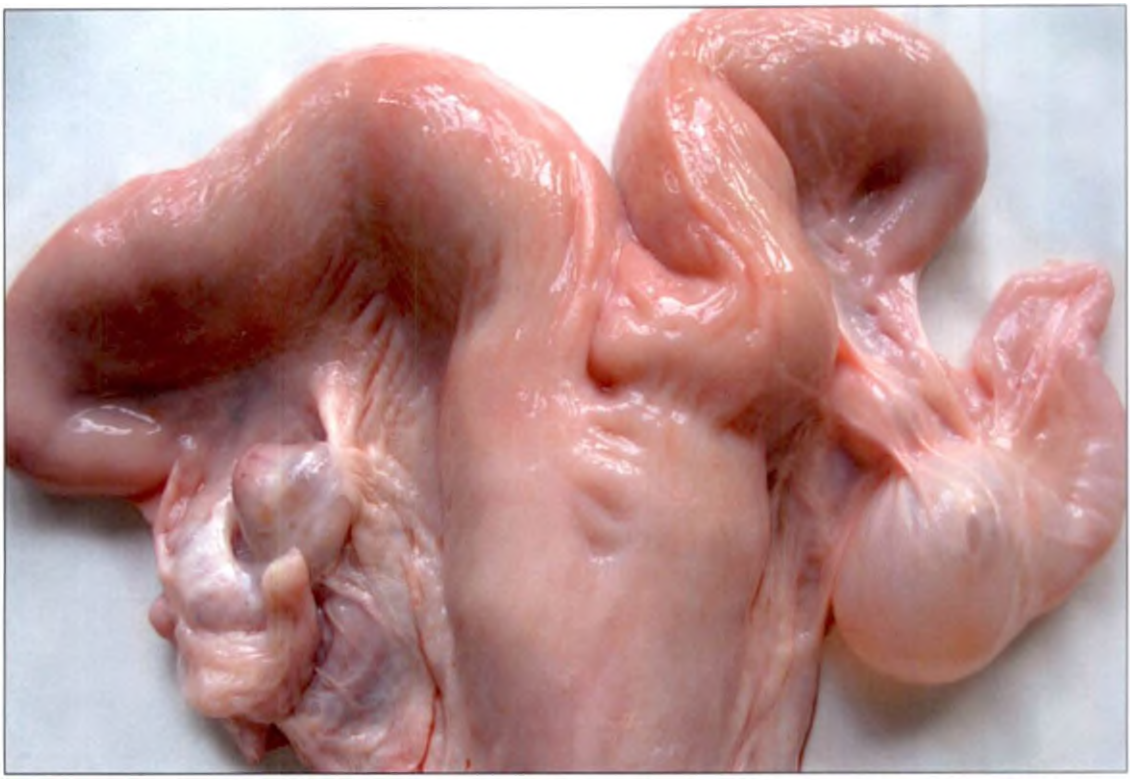


Fig. 14. Hydrosalpinx. Distension of fallopian tube with clear amber coloured fluid in the right oviduct

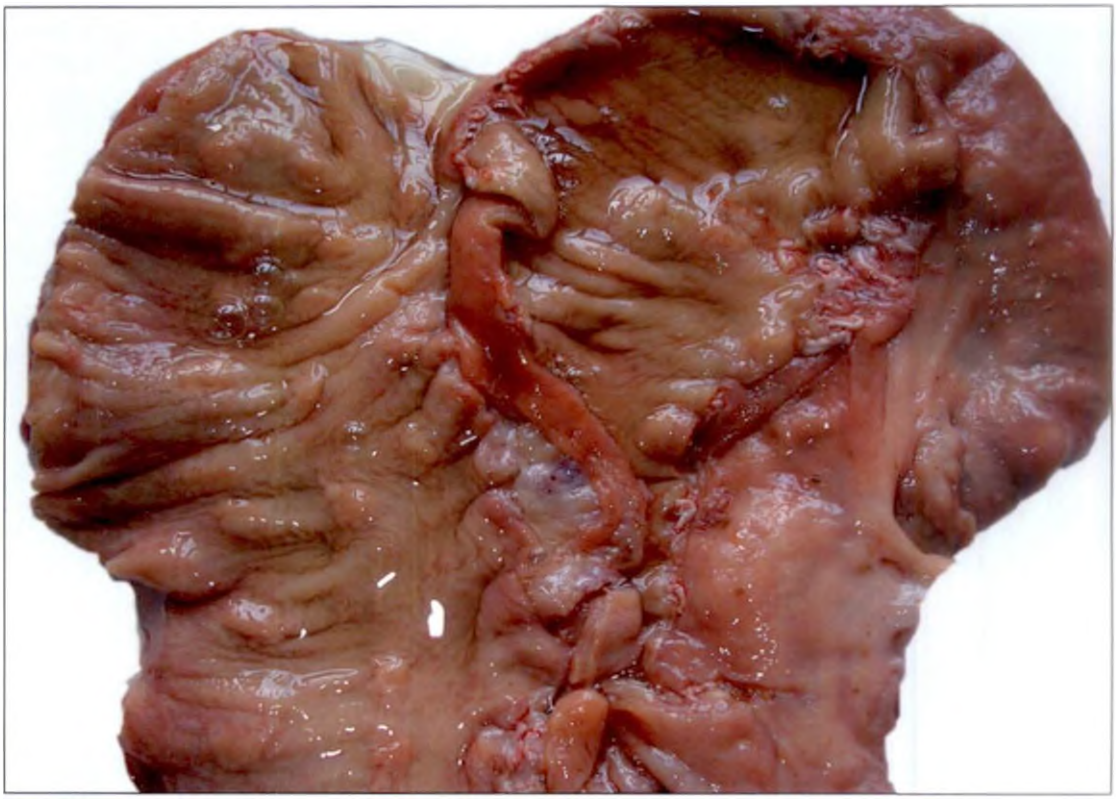


Fig. 15. Mucometra. Uterine horns with opalescent, viscid fluid

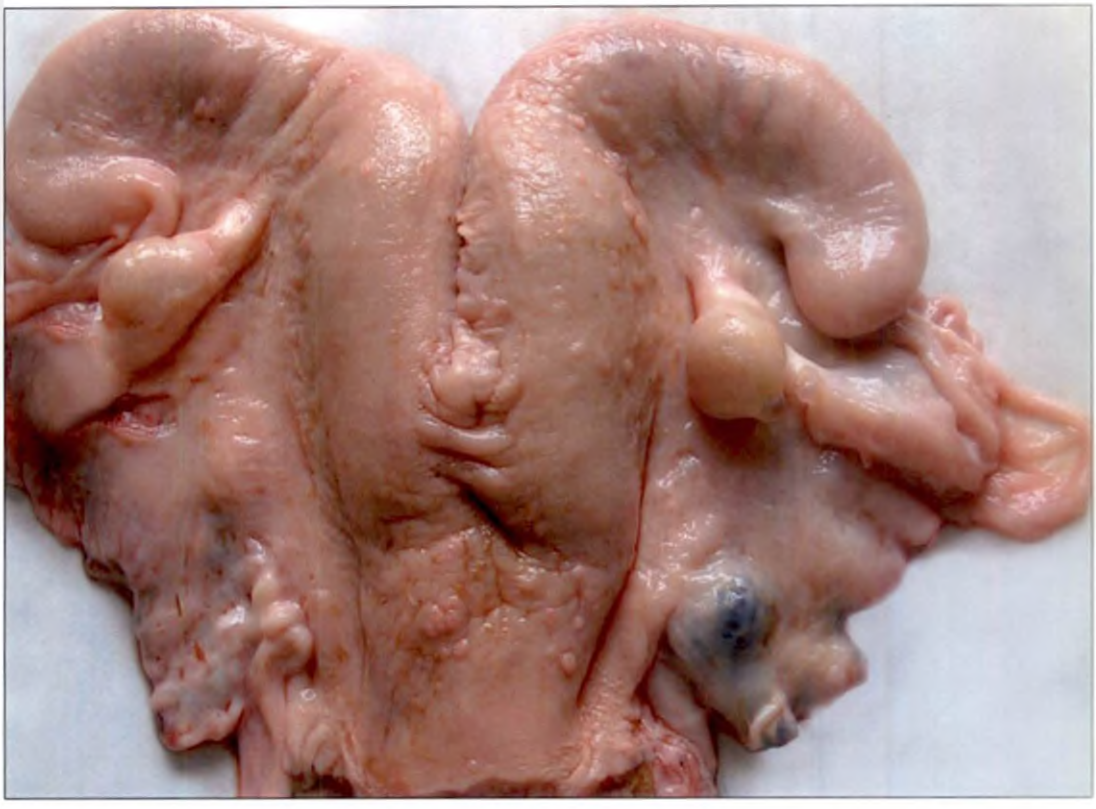


Fig. 16. Serous vascular protrusions on the uterine surface

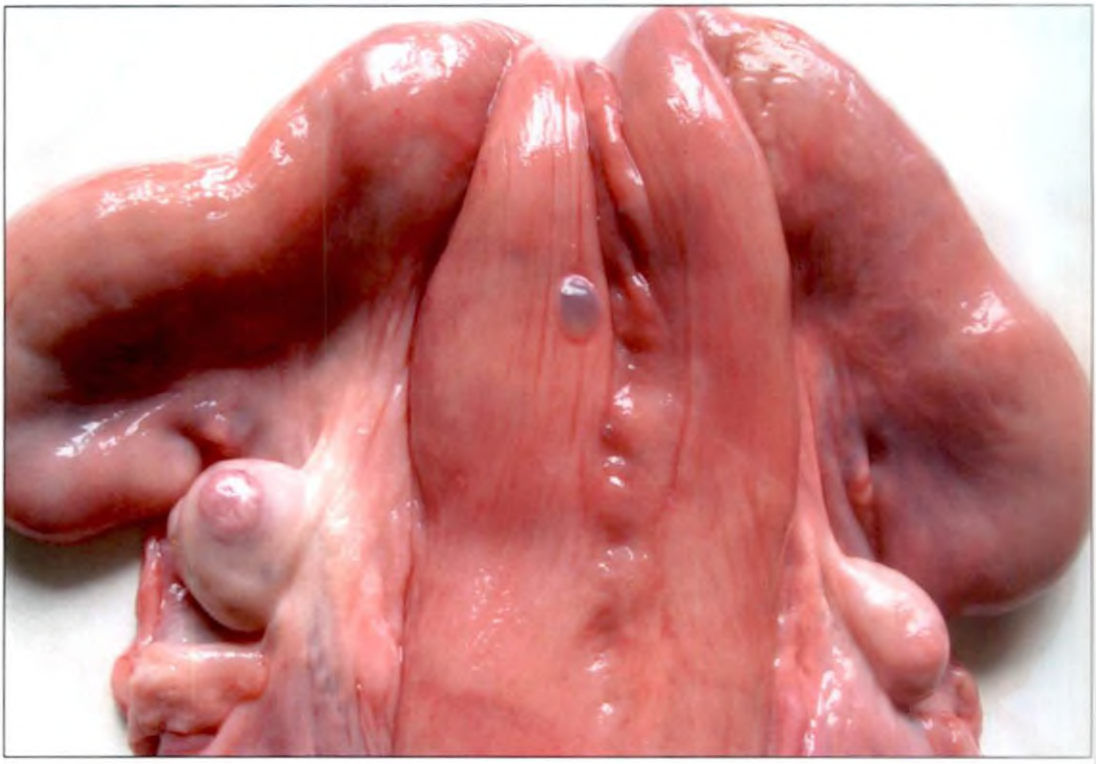


Fig. 17. Perimetrial cyst

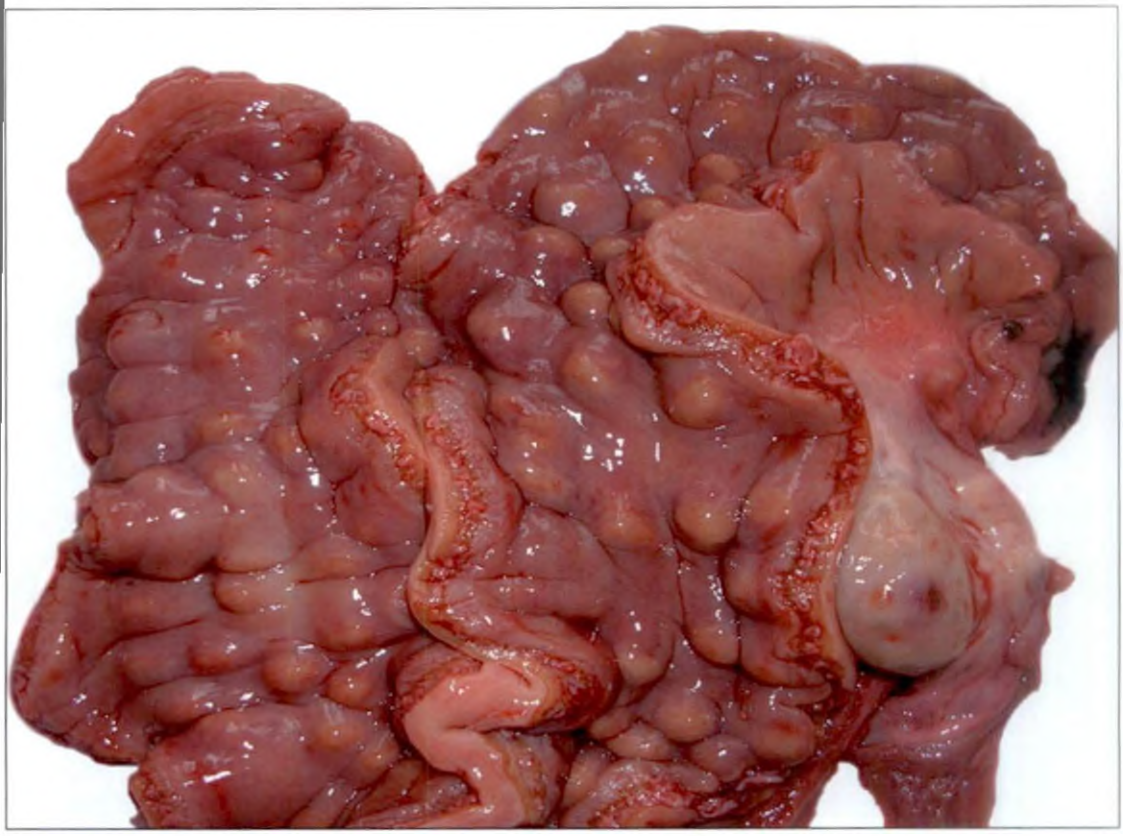


Fig. 18. Category 2 endometritis. Endometrial Mucosal congestion

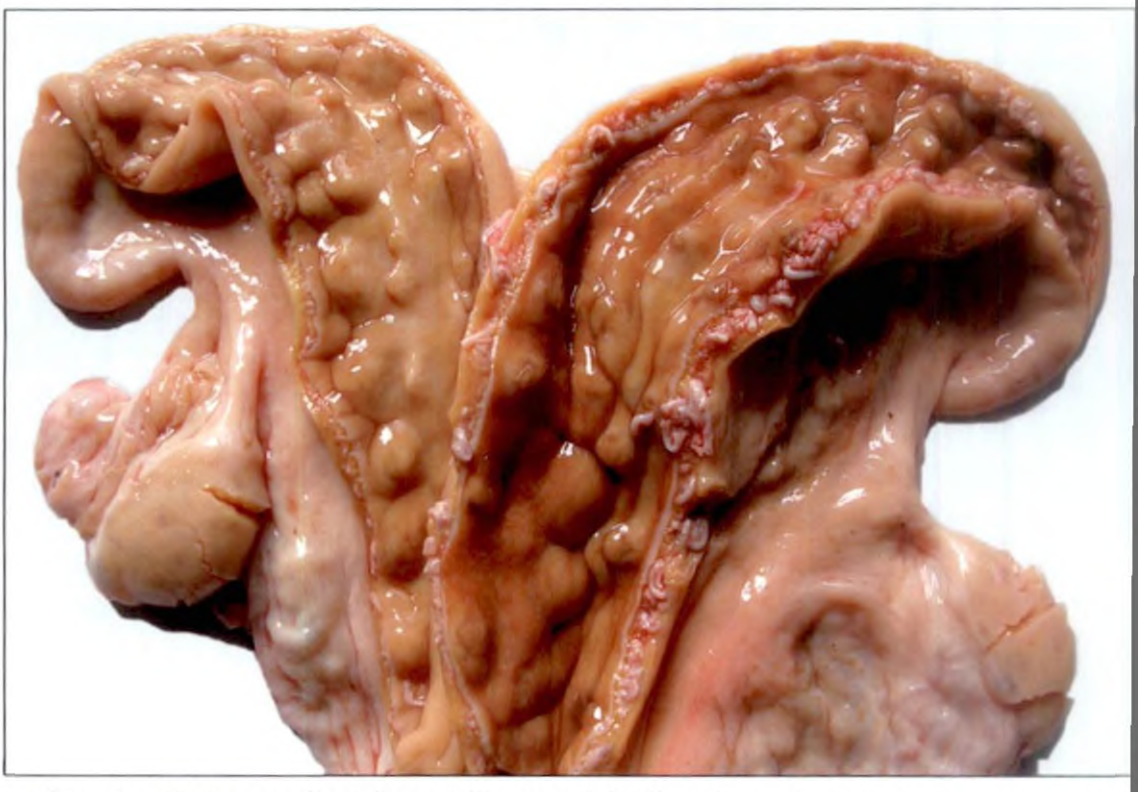


Fig. 19. Category 3 endometritis. Grayish discolouration of uterine mucosa

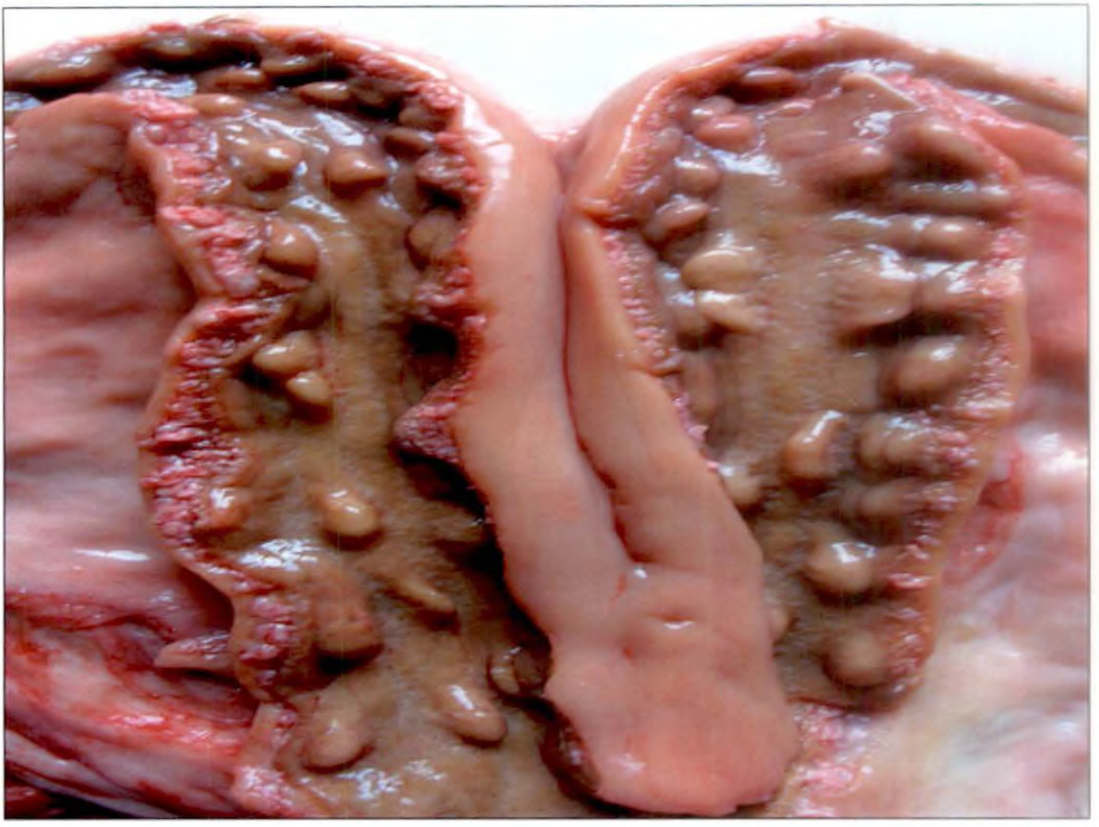


Fig. 20. Category 4 endometritis. Grayish discolouration of uterine mucosa

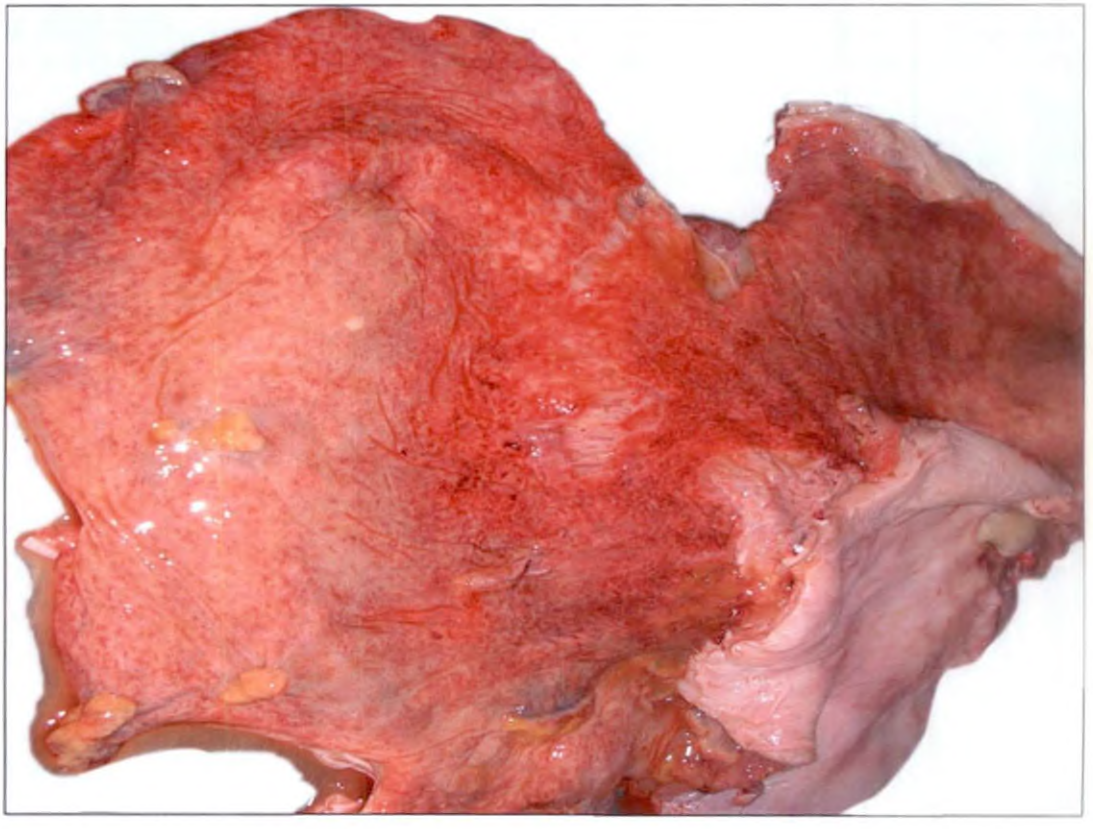


Fig. 21. Puerperal metritis. Endometrium : congested and haemorrhagic

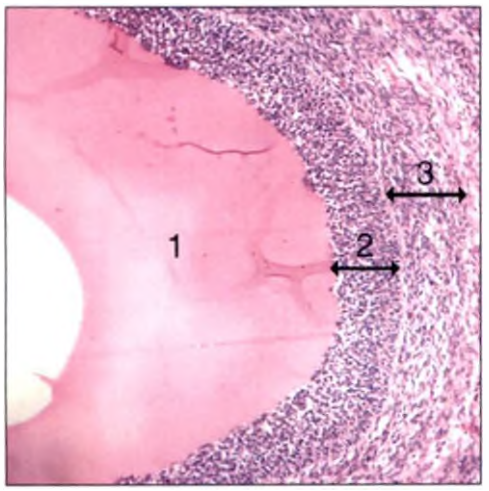


Fig. 22 Follicular cyst. H&E x 100

1. **Acidophilic material in the lumen**
2. **Multiple rows of granulosa cells**
3. **Thecal layers**

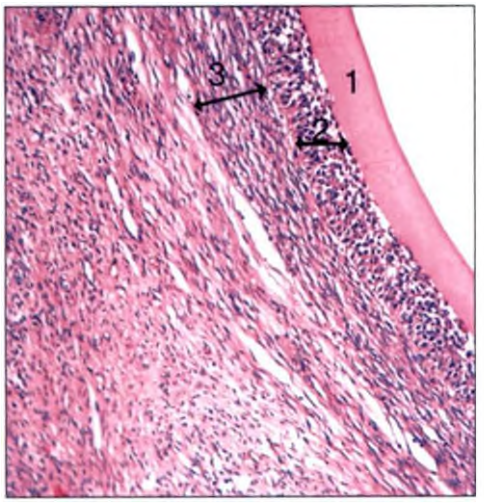


Fig. 23 Follicular cyst. H&E x 100

1. **Acidophilic material in the lumen**
2. **Few rows of granulosa cells**
3. **Thecal layers**

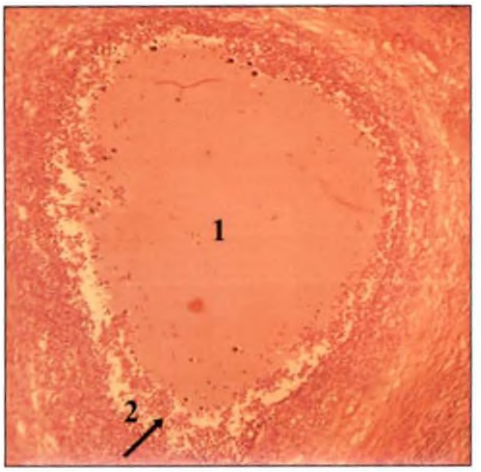


Fig.24 Follicular cyst. H&Ex100

1. **Acidophilic material in the lumen**
2. **Degenerating granulosa cells**

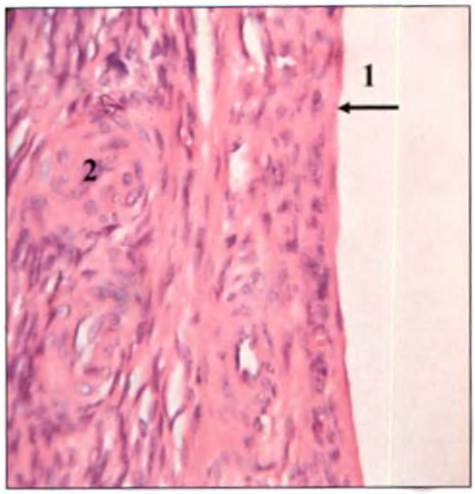


Fig.25. Follicular cyst. H&Ex400

1. Follicular cyst without granullosa cell

2. Peripheral blood vessels

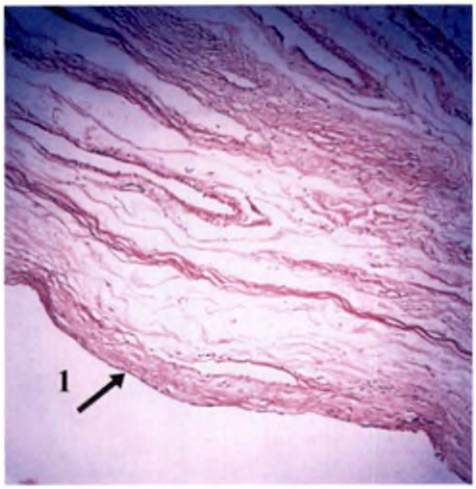


Fig.26. Follicular cyst. H&Ex100

1. Cyst wall with fiberous tissue stroma

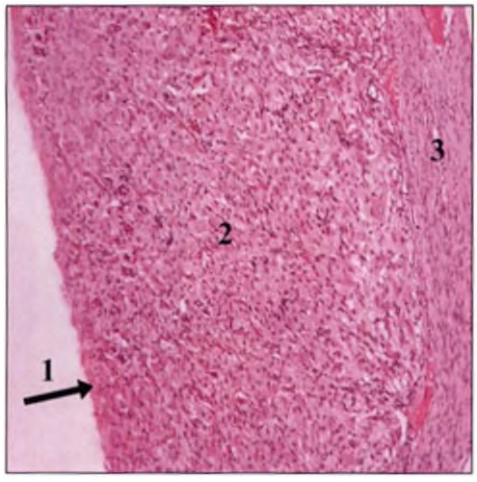


Fig. 27. Luteal cyst. H&Ex100

1. Layer of connective tissue

2. Luteal cells

3. Connective tissue stroma

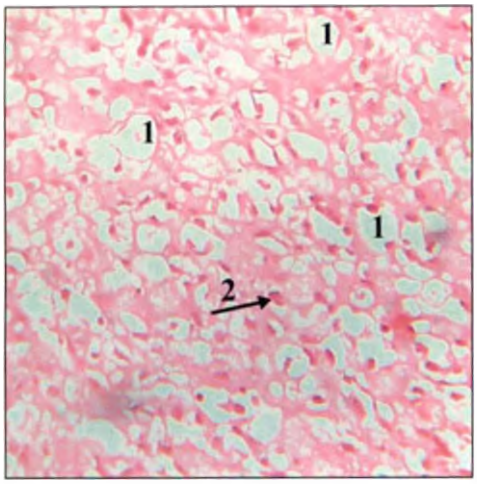


Fig. 28. Cystic corpus luteum.
H&E x 400

1. Vacuolated cytoplasm

2. Lightly stained nuclei

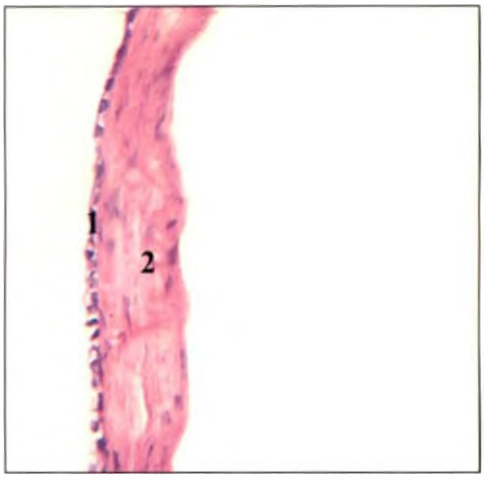


Fig. 29. Parovarian cyst.
H&E x 400

1. Cyst wall lined by layer of cuboidal epithelium

2. Layer of muscle and connective tissue fibre

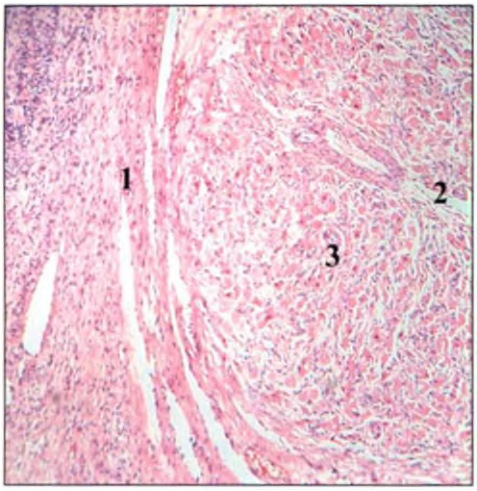
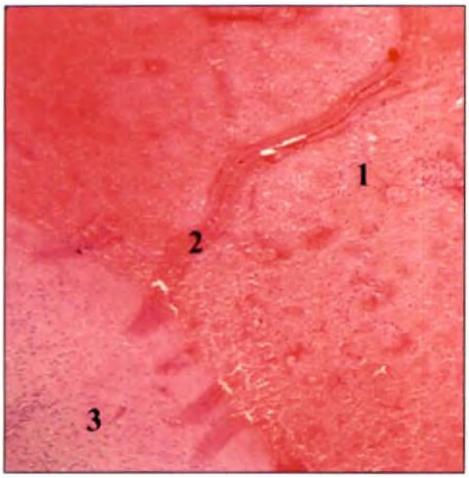


Fig. 30. Embedded corpus luteum.
H&E x 100

1. Outer connective tissue capsule

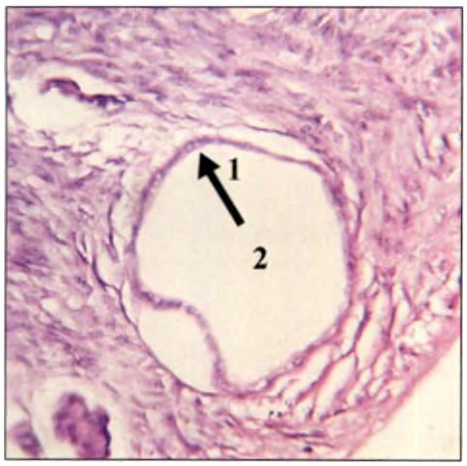
2. Connective tissue septa

3. Lutein cells



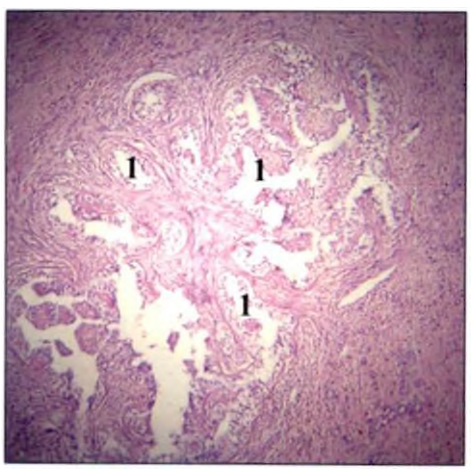
1. Haemorrhage
2. Fibrous tissue septa
3. Ovarian stroma

Fig. 31. Ovarian haematoma.
H&E x 100



1. Cyst wall lined by layer of cuboidal epith
2. Lumen without any contents

Fig. 32. Serous inclusion cyst
H&E x 400



1. Group of acini like structures in the ovarian medula

Fig. 33. Epoophoron
H&E x 100

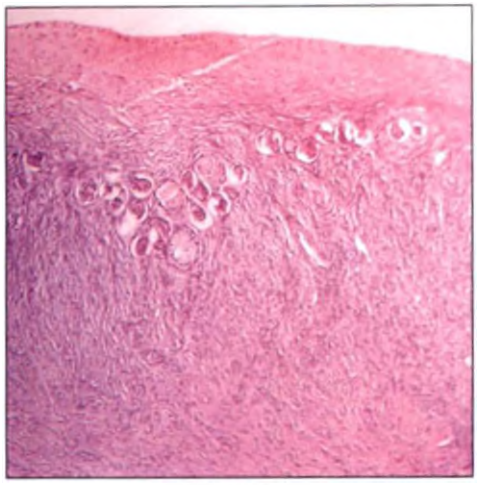


Fig. 34

Figure. 34. Atretic follicles. Group of follicles of various type showing atersia in the ovarian cortex . H&Ex100

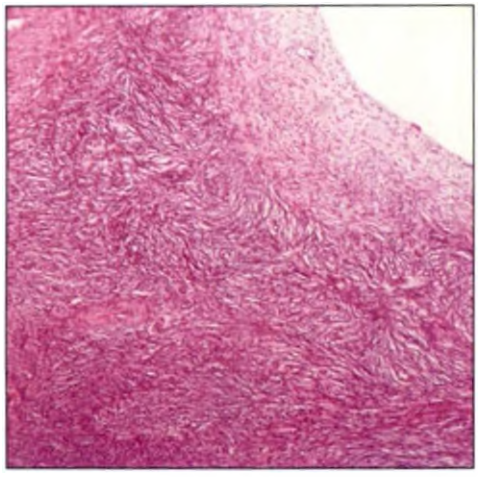


Fig. 35

Figure. 35. Ovarian sclerosis. Dense fibrous connective tissue stroma in the ovarion cortex, devoid of devloping follicles. H&Ex100

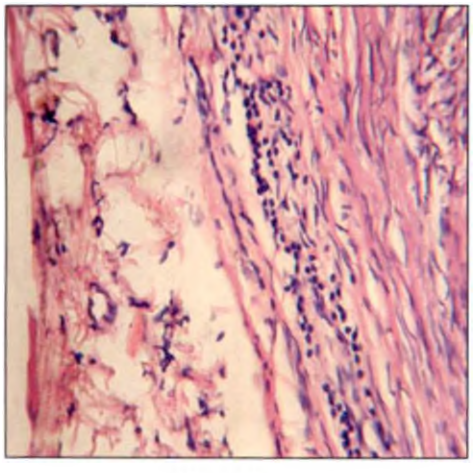


Fig. 36

Figure. 36. Perioophoritis. Periovarion connective tissue and tunica albugenia infiltrated by lymphocytes and few macropages. H&Ex400

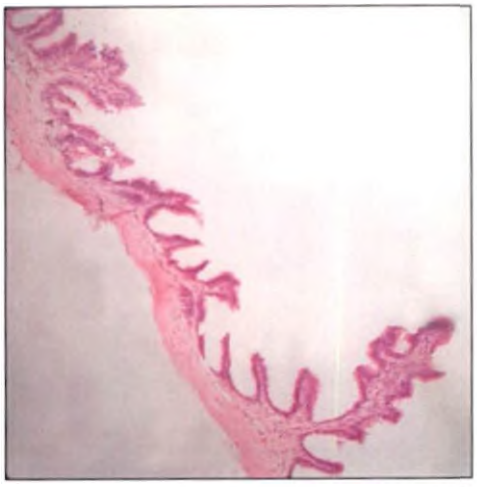


Fig. 37

Figure .37. Hydrosalpinx.
Flatening of mucosal epithelial folds and dilation of the lumen of the oviduct. H&E 100

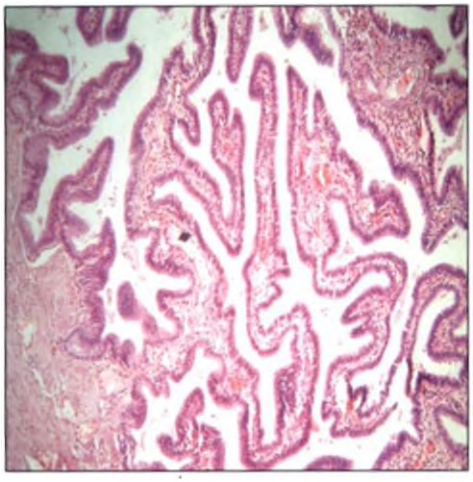


Fig. 38

Figure . 38. Salpingeal hyperplasia.
Papillary hyperplasia of epithelial folds and vascular congestion. H&E x 100

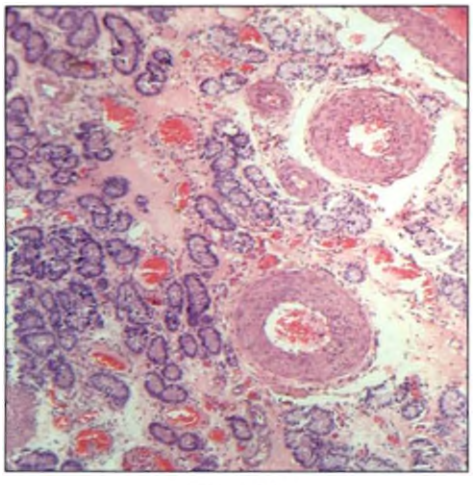


Fig. 39

Figure. 39. Endometrial hyperplasia.
Irregularly distributed multiple number of glands with engorgement of the blood vessels. H&E x 100

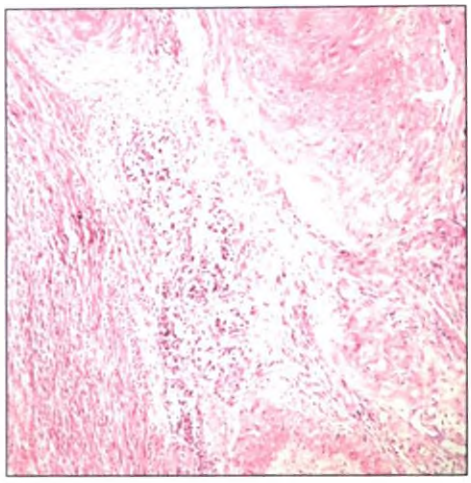


Fig. 40

Figure. 40. Adenomyosis. Group of endometrial glands in the myometrium showing degeneration.H&E x 100

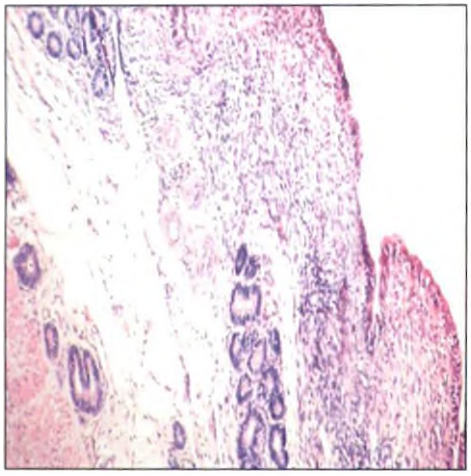


Fig. 41

Figure. 41. Mucometra. Thin endometrium with reduced number of uterine glands.H&E x100

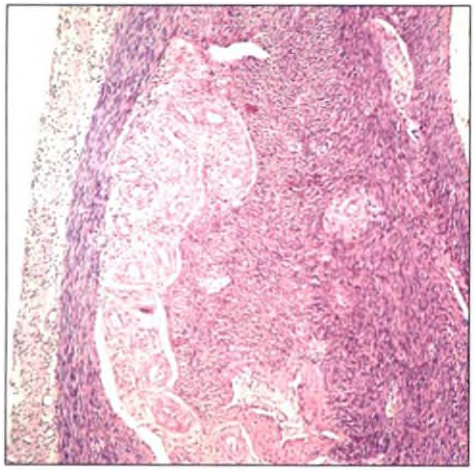


Fig. 42

Figure. 42. Serous vascular protrusion. Thick walled distended blood vessels in the serosa. H&E 100

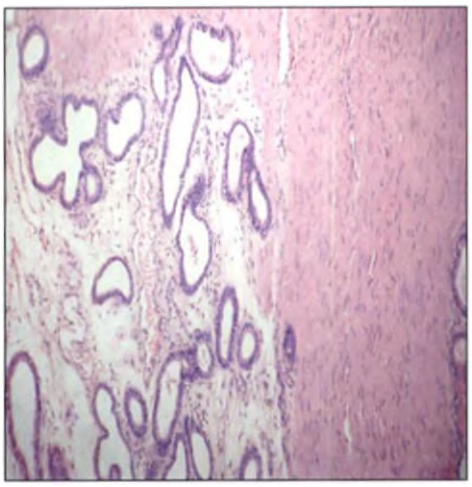


Fig. 43

Figure. 43. Category 1 endometritis. Tubular and coiled endometrial glands lamina propria without cellular infiltrate and periglandular fibrosis H&E x100

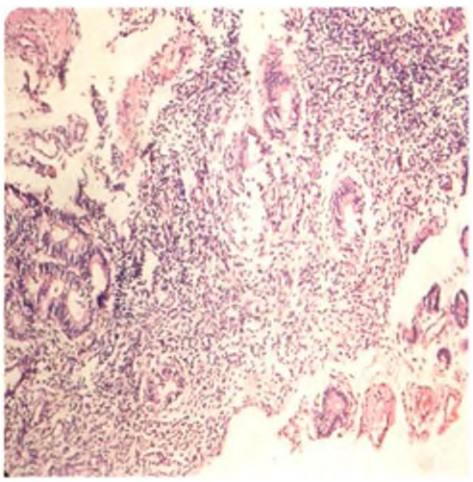


Fig. 44

Figure. 44. Category 2 endometritis. Diffuse infiltration of the endometrial stroma by inflammatory cells. H&E x100

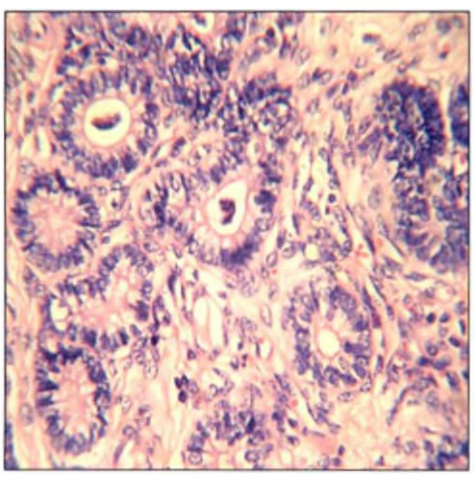


Fig. 45

Figure. 45. Category 2 endometritis. One to three layers of fibrocytes surrounding the glands.H&E x 400

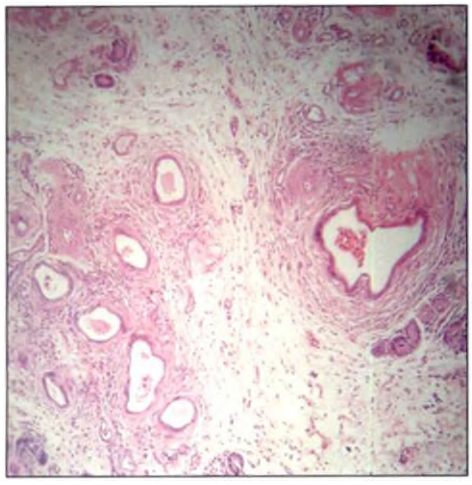


Fig. 46

Figure . 46. Category 3 endometritis. Many layers of fibrocytes surrounding the uterine glands and glandular lumen containing secretions. H&E x 100

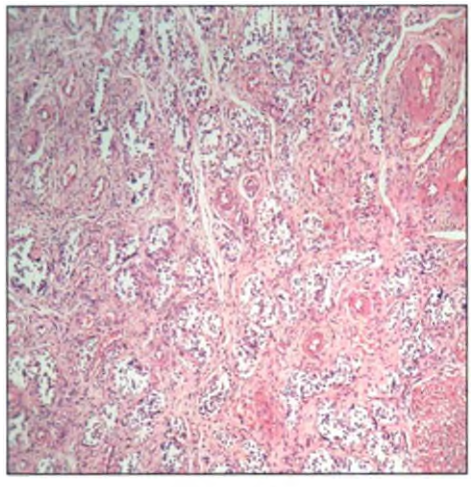


Fig. 47

Figure .47. Category 4 endometritis. Extensive glandular fibrosis. All glands appearing atrophic. H&E 100

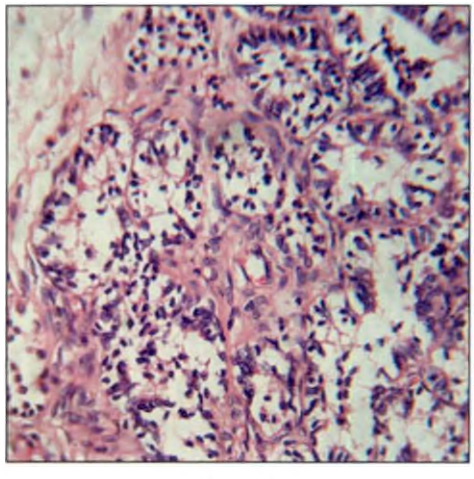


Fig. 48

Figure. 48. Category 4 endometritis. Degeneration and desquamation of endometrial glands. H&E 400

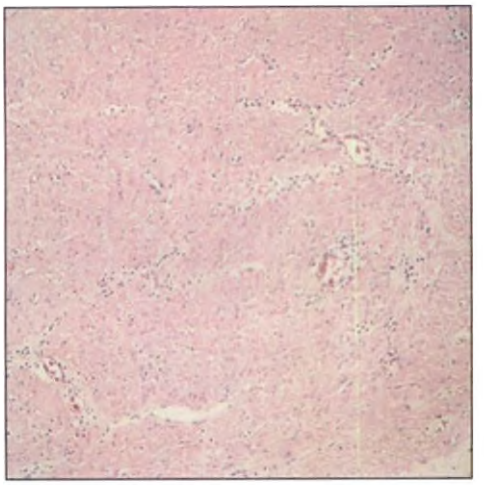


Fig. 49

Figure .49 Puerperal metritis. Muscular layer infilterated by neutrophils and a few mononuclear cells.H&E x100

Discussion

5. DISCUSSION

The present investigation was aimed at studying the occurrence of various conditions affecting the utero-ovarian components of the genital tract of cows and their pathomorphological characteristics and the possible correlation with etiological agents.

A total number of 1000 genital tracts were screened for utero-ovarian disorders. Out of these, 115 genitalia showed lesions in the ovary, oviduct and uterus. Among these 84 cases showed lesions of varying frequency in the ovaries, seven cases showed lesions in the oviduct and 24 cases showed lesions in the uterus. Detailed gross and histopathological examinations were carried out on 50 cases of ovary and uterus which showed grossly visible lesions and the nature of occurrence.

The follicular cyst was recorded in 29 cases (2.9 per cent) in the present study which were in agreement with the reports of previous workers (Derannan and Mac Pherson, 1966., Ahmad and Khan, 1993). A higher incidence of 3.8 to 18.8 per cent was observed by Al-Dahash and David (1977), Wahid *et al.* (1991) and Garverick (1997), while a lower incidence of 0.6 – 1.88 % was recorded by Mylera (1962), Lawton *et al.*(2000) and Hatipoglu *et al.*(2002). The causes of development of follicular cysts are manifold. The cysts develop as a result of a failure of coordination of various endocrine mechanism. Significantly lower luteinizing hormone concentration during proestrus and oestrus in cows with follicular cysts was reported by Parkinson (2001). He also that reported stress, mediated by the adrenocortical hormone as a contributing factor for the development of follicular cysts. Heredity, breed, age, milk production, feeding, management and season are other factors leading to the development of follicular cyst (Herenda, 1987., Garverick, 1997). In the present study, in certain cases of follicular cysts,

conditions like salpingeal hyperplasia, 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 observation made by Derannan and Mac Pherson (1966). Galvan *et al.* (1982) and Wahid *et al.* (1991) who observed a higher incidence of 2.34 to 6 %. 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 reports of Wahid *et al.* (1991) and George (1994). The reason for the development of the cyst could be a failure of hypophysis to release an adequate surge of luteinizing hormone. Ghora (1995) suggested that luteal cyst was a sequel to genital tract infections reaching the ovary through the ovulation crater. The luteal cyst without any genital tract infection observed in this study suggested endocrinal imbalance as a probable cause.

The cystic corpus luteum was recorded in three cases (0.3%). Mylera (1962) Herenda (1987) Wahid *et al.* (1991) and George (1994) recorded a higher incidence of 2.78 to 36.9 %. The low incidence recorded in this study could be attributed to the small sample size utilized in this investigation. Microscopically, the cystic cavities were lined by a layer of connective tissue without any lining epithelium. There was extreme vacuolation in the luteal cells due to regressive changes. There was an outer investment of connective tissue. These findings were in agreement with those of George (1994) Hatipoglu *et al.* (2002) and Sujata *et al.* (2003). Roberts (1971) reported that this could be caused by a mild faulty release of luteinizing hormone. This implies that it is important to assess the hormone status of such animals to find out the exact cause.

The incidence of parovarian cyst was observed in 22 cases (2.2 %). This coincided with those reported by Fathallal *et al.* (2000), Nair (1994), Lawton *et al.* (2000) and Hatipoglu *et al.* (2002) who observed a lower incidence of 0.32 to 0.94 %. The gross and histological findings were in agreement with those of Roberts (1971), Potekar *et al.* (1982), Nair (1994), Fathallal *et al.* (2000) and Hatipoglu *et al.* (2002). 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 (Roberts, 1971., Parkinson, 2001., Hatipoglu *et al.*, 2002).

The incidence of embedded corpus luteum in the present study was 1.2 % which coincided with that reported by Rahman *et al.* (1977). On the other hand the present study does not agree with that of Sujata *et al.* (2003) who observed a higher incidence of 4 %. Gross and microscopical features were in agreement with those of Nair (1974), Ghora (1995) and Sujata *et al.* (2003). This deficiency will interfere with the reproductive performance as continuous production of progesterone from such animals could enhance the anestrous condition.

Persistent corpus luteum was recorded in a single case. Gross and microscopical features were in agreement with those reported by George (1994). Any factor that interfered with the production or release of PGF2 alpha results in the development of persistent corpus luteum. Uterine infections interfere with the production and release of PGF2 alpha (Parkinson, 2001).

The incidence of ovarian haematoma in the present study was 0.2 % which coincided with that reported by Wahid *et al.* (1991). Severe trauma during manual enucleation of the corpus luteum or rupture of the follicular cysts might be the etiological factors. Haemorrhage into the ovary could occur on account of toxic infectious diseases (Roberts, 1971., Nair, 1974., Parkinson, 2001., Ghora, 1995). In the present study none of the animals had any detectable disease on antemortem.

Serous inclusion cysts were recorded in a single case. 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 the ovulation (Kennedy and Miller, 1991).

Incidence of epoophoron was recorded in a single case. Microscopically, acinar 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 Rao *et al.* (1975). Kennedy and Miller (1991) 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 two cases. 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 (1991).

Sclerosed ovaries were recorded in three 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 Nair (1974). Malnutrition and hypothyroidism could have played a significant role in the onset of such conditions and it needs to assess the nutritional, thyroid status of the animals.

Senile atrophic ovaries were recorded in five cases. All the five cases, the animals were weak. The senile ovaries were pale and waxy with a rough pitted surface. The ovarian cortex showed 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, wherein the squamous cell had transformed into a single layer of columnar granulosa cells. Tunica albugenia was thickened. The surface epithelium was almost completely missing in a few cases but in two cases it remained intact. These cells had lost their cuboidal character and became flattened with pyknotic nuclei. These findings were in agreement with those of Nair (1974) and George (1994).

The incidence of hydrosalpinx was recorded in a single case (0.1%) which did not agree with those reported by Dwivedi and Singh (1971), Kavani *et al.* (1986), Ahmad and Khan (1993) and Rao *et al.* (1993) who observed a higher incidence varying from 2.21 % to 12.5 %. Both gross and microscopical features in this present study were in agreement with those of Herenda (1987), Nair (1974) and Ghora (1995). 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 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, 1991). None of these factors could be observed in this case and the etiology could not be ascertained.

Hyperplasia of the salpingeal epithelium was recorded in three 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. Ghora (1995) discussed the incidence of salpingeal hyperplasia as a result of hyperestrogenism.

Endometrial hyperplasia was recorded in two 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 agreement with those of Ghora (1995) Acland (2001) and Kennedy and Miller (1991). In these cases the hyperplasia was associated with follicular cysts. Endometrial hyperplasia in the cow is associated with ovarian follicular cysts or granulosa cell tumors. Both these conditions could bring about prolonged hyperestrogenism leading to endometrial hyperplasia and clinically manifesting as anoestrus or nymphomania in cows (Kennedy and Miller, 1991).

Adenomyosis was recorded in three cases. Microscopically, endometrial glands were located in the myometrium. This could be due to the result of prolonged hyperestrogenism or developmental disturbances (Nair, 1974., Acland, 2001).

Adenomyosis is a rare condition and occasionally reported in buffaloes (Rao *et al.*, 1976).

Mucometra was encountered in a single case. 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 are in agreement with that of Ghora (1995). Factors such as congenital or acquired obstructions to the outflow of the normally produced mucus resulted in the development of mucometra (Acland, 2001). In the present case, the cervical canal was tightly closed giving rise to mucometra.

Serous vascular protrusions were noticed in five specimens of uterus. This anomaly of blood vascular system grossly simulated varicose veins. Ghora (1995) reported that this had no bearing on conception.

A single case of perimetrial cyst was observed on the uterine surface 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 11 cases which was in agreement with that of Namboothiripad *et al.* (1978). On the other hand, it does not agree with those of Gosh *et al.* (1983), Rao *et al.* (1993) and Blanc *et al.* (2002) who observed a higher incidence of 16.9 to 69.2 %. In the present investigation only grossly affected organs and those suspected to be affected were subjected to histopathological studies. Subclinical cases of endometritis might have been left out in the process of selection and this might be the reason for the lower incidence. The gross and histological findings were in agreement with those of Gonzalez *et al.* (1985) and Saiyari *et al.* (1994). Endometritis might follow abnormal parturitions,

traumatic lesions and unhygienic artificial inseminations. These factors were associated with delayed uterine involution and conception rate (Roberts, 1971). Endometritis and fibrosis are thought to be important causes of infertility in the cows. The cellular infiltrates might be cleared whereas fibrosis persists leading to infertility (Gonzalez *et al.*, 1985).

Puerperal metritis was recorded in a single case 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 haemorrhagic. Microscopically, the mucosa, muscular layer and serosa were infiltrated with neutrophils and a few mononuclear cells. These findings were in agreement with those of Parkinson (2001). Etiological factor for this condition might be abnormal parturitions.

The investigation carried out, has helped to document various utero-ovarian disorders encountered in cows. 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 periglandular fibrosis with glandular degenerative changes in the endometrial glands. The pathological changes associated with various utero-ovarian disorders encountered were suggestive of endocrine imbalances. 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 control. 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. Over and above, cervix, vagina and vulva were not included in this investigation. A comprehensive study taking into consideration all these deficiencies are must.

Summary

6. SUMMARY

The present investigation was carried out, to study the pathological features of spontaneously occurring utero-ovarian disorders in cows. One thousand specimens of cow genitalia collected from the municipal slaughter house, Thrissur formed the material for the study. These were subjected to detailed gross and histopathological examinations. Of these, 115 genitalia showed one or more lesions in the ovary, oviduct and uterus.

The conditions encountered in the ovaries were follicular cyst, luteal cyst, cystic corpus luteum, embedded corpus luteum, persistent corpus luteum, ovarian haematoma, ovarian sclerosis, senile atrophy, serous inclusion cyst, epoophoron, atretic follicles, parovarian cyst and perioophoritis.

Seven cases showed lesions in the oviduct. The conditions encountered were hydrosalpinx and hyperplasia of the salpingeal epithelium.

Out of the 1000 genital tracts examined, 24 cases showed lesions in the uterus. The conditions encountered were endometrial hyperplasia, adenomyosis, mucometra, perimetrial cyst, endometritis and metritis.

Among the ovarian lesions, follicular cyst was the most common condition encountered. It was observed in 29 cases (2.9 %). The right ovary (18 cases) was found to be more frequently involved than the left ovary (11 cases). Follicular cyst was thin walled and distended with pale yellow clear fluid. The cyst was found to be associated with salpingeal hyperplasia, endometrial hyperplasia and adenomyosis as well. The role of luteinizing hormone in the development of these cysts has been discussed.

Luteal cysts were recorded in three cases (0.3 per cent) and were found to be due to the inadequate release of luteinizing hormone from the hypophysis or alternatively as a result of infection reaching the ovary through the ovulation crater. The luteal cyst a had thick wall and contained more amber or darker yellow or brown coloured fluid. The diameter of the cysts varied from 2.6 to 3.1 centimeters.

Cystic corpus luteum was found in three cases (0.3 per cent), two being in the right ovary and one in the left. Corpus luteum cysts were soft, with the cavities ranging from 0.5 to 0.8 centimeter in diameter and containing light straw coloured fluid. The cystic cavities were lined by a layer of connective tissue.

Parovarian cysts were recorded in 22 cases (2.2 per cent), two cases being bilateral while the remaining 20 cases were unilateral. Those on the left side (11 cases) were found to be more frequently involved than those on the right side (nine cases).The cysts were found to be vestiges of the wolffian or mesonephric duct system. Larger parovarian cysts causing stenosis of the oviductal lumen by compression, could affect fertility.

Embedded corpus luteum was recorded in 12 cases (1.2 %); eight cases were in the right ovary and four in the left. This corpus luteum was seen located both in the cortex and medulla. The embedded corpus luteum had a fibrous connective tissue capsule from which connective tissue fibers traversed into the lutein tissue dividing it into lobes.

The persistent corpus luteum was observed in a single case. This was solid and larger than normal corpus luteum in size. The crown was projected on the ovarian surface. The persistent corpus luteum had fibrous connective tissue stroma

that divided the luteal tissue into lobes. There was reduced vascularisation in the parenchyma with regressive changes in the luteal cells.

Ovarian haematomas were 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 a single case (0.1 %). Microscopically, these were seen close to the ovarian surface. 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 a single case. 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 two cases (0.2 %). In the cortex, multiple follicles of various types showed follicular atresia.

Ovarian sclerosis was recorded in 2 cases (0.2 %), one in the right ovary and one in the left. Sclerosed ovaries were small and hard. Neither corpus luteum nor follicle was evident the surface. The surface epithelium was absent in a few places. Tunica albugenia was thickened. Stroma of the cortex became more fibrous.

Senile atrophic ovaries were recorded in five cases (0.5 %) and all were bilateral. The senile ovaries were pale and waxy with a rough pitted surface. The

surface epithelium was absent. Tunica albugenia was thickened. There were only a few primary follicles in the cortex.

In perioophoritis the ovarian surface was shaggy. Periovarian connective tissue was seen to be infiltrated by lymphoid cells, plasma cells and a few mononuclear cells.

Hydrosalpinx was recorded in a single case (0.1 %). 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. The mucosa was lined by low cuboidal to columnar epithelium. Muscular coat and lamina propria were atrophied.

Hyperplasia of the salpingeal epithelium was recorded in three cases (0.3%). Thickening of the mucosa was prominent. Microscopically, papillary hyperplasia was observed. This was likely to lead to infertility as the passage of ovum through such a duct is prone to obstruction.

Two recorded cases of endometrial hyperplasia showed microscopic features like increased number and size of the glands with the glandular epithelium being tall and columnar.

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 two cases.

In a single case of mucometra resulting from the congenital or acquired obstructions to the mucous outflow, the uterine horns were distended with opaque and viscid fluid. The microscopic features were reduced number of active endometrial glands and thinning of the endometrial lining.

Serosal vascular protrusions on the dorsolateral aspects of the uterus simulating varicose veins was seen grossly as soft nodules. A single case of this condition was represented microscopically by engorged blood vessels and thick walls.

A case of clear watery fluid containing oval perimetrial cyst was observed which was lined by a layer of flattened epithelium.

Eleven cases of endometritis was observed and graded into four 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 a single case of puerperal 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.

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PATHOLOGY OF UTERO-OVARIAN DISORDERS IN COW

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ABSTRACT

The present study aimed to find out various utero ovarian disorders affecting cows. A total number of 1000 genital tracts were examined for utero-ovarian disorders. Out of which 115 genitalia showed one or more lesions in the ovary, oviduct and uterus. Among these 84 cases showed lesions in the ovaries. The follicular cyst was the most common condition and was recorded in 29 cases (2.9%). The other conditions encountered were luteal cyst (0.3 %), cystic corpus luteum (0.3%), embedded corpus luteum (1.2 %), persistent corpus luteum (0.1 %), ovarian haematoma (0.2 %), ovarian sclerosis(0.2 %), senile atrophy(0.5 %), follicular atresia (0.2 %), epoophoron (0.1 %), serous inclusion cyst (0.1 %), par ovarian cyst (2.2 %) and perioophoritis(0.1 %). Seven cases showed lesions in the oviduct. The conditions encountered were hydrosalpinx (0.1 %) and hyperplasia of the salpingeal epithelium (0.3 %).Uterine disorders recorded in 24 cases. The conditions encountered were endometrial hyperplasia (0.2%), adenomyosis (0.3 %), mucometra (0.1 %), perimetrial cyst (0.1%), serous vascular protrusions (0.5 %), endometritis (1.1 %) and metritis (0.1%). 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 origin, managemental factors and developmental defects. The results of the study also highlight the need to monitor the hormonal levels in the blood, micronutrients and managemental factors.