OCCURRENCE AND PATHOLOGY OF VARIOUS CONDITIONS OF CARDIOVASCULAR SYSTEM IN BUFFALO (*Bubalus bubalis*)

HkSalo'k (*ccy l ccfy l*) esa âan;okfguh r=a dh fofHkUu voLFkkvksa dk vkikr ,oe 0;kf/kdh

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B.VSc & A.H.

THESIS

MASTER OF VETERINARY SCIENCE
(VETERINARY PATHOLOGY)

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College of Veterinary and Animal Science,
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THESIS

Submitted to the
Rajasthan University of Veterinary and Animal Sciences, Bikaner
In partial fulfillment of the requirements
for the degree of

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BY

KSHITIZ GOSWAMI

2019

RAJASTHAN UNIVERSITY OF VETERINARY AND ANIMAL SCIENCES, BIKANER
COLLEGE OF VETERINARY AND ANIMAL SCIENCE, BIKANER

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This is to certify that this thesis entitled **"OCCURRENCE AND PATHOLOGY OF VARIOUS CONDITIONS OF CARDIOVASCULAR SYSTEM IN BUFFALO (*Bubalus bubalis)*"** submitted for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** embodies bonafide research work carried out by **Mr. KSHITIZ GOSWAMI** under my guidance and supervision and that no part of this thesis has been submitted for any other degree. The assistance and help received during the course of investigation have been successfully acknowledged. The draft of the thesis was also approved by the advisory committee on_____

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Date :

Place: ***Kshitiz Goswami***

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INTRODUCTION

India is a fast growing country and more than 70 percent people of India earn from agricultural and animal husbandry. A large number of farmers in India depend on animals for their livelihood. Animal husbandry and livestock sectors are important for rural economy. Animal husbandry is an integral component of Indian agriculture supporting livelihood of more than two-thirds of the rural population. Ruminants such as buffaloes, cow, sheep and goat are reared for milk, meat, wool and draught power. Animals also provide dung as organic manure and domestic fuel, hides & skin, and are a regular source of cash income for rural households. They serve as an insurance against crop failure and natural calamities. Livestock sector contributes 4.11% GDP and 25.6% of total Agriculture GDP.

India holds ranks first in terms of buffalo population followed by Pakistan and china in the world. According to livestock census 2012, India comprises approximately 56.7 percent of the total world buffalo population. The domestic or water buffalo (*Bubalus bubalis*) is widely dispersed throughout the southern Asia, Africa, Europe and South America. Buffaloes were in the service of man as early as 2500 to 2100 B.C. (Banerjee, 2005). Buffaloes play a pivotal role in overall social development through contributions to milk, meat, hides and draft power for agricultural operations in many regions they are raised for triple purpose (milk, meat and draft). In fact, all body parts are used including horns and hair. The buffalo holds an important place and role in Indian rural economy. Buffalo is the premier animal in Indian dairy industry. Buffalo milk is used to produce various dairy products such as butter, khoya, curd, yoghurt, Mozzarella cheese, milk-powder, shrikhand etc. Buffaloes are preferred over cattle in India because of their distinctive qualities such as better feed conversion efficiency, more resistance to diseases and higher milk fat percentage than in cows (Bandyopadhyay *et al 2003).*

Buffalo milk is healthy as it is richer in saturated fatty acids. Its much higher total solids (18–23% vs 13–16%) is useful for making cheese, butter fat, several kinds of traditional sweets and ice creams. Buffalo meat is lean,

tasty and often indistinguishable from beef. Buffalo meat is tough, dry and dark red in color with specific taste because it is mostly used as processed meat. It contains lower saturated fat than beef and pork, which is a good dietary value. Buffalo meat contains 40% less cholesterol, 55% less calories, 11% more protein and 10% more minerals in comparison to bovine meat so is, therefore, healthier *(*Nanda and Nakoo 2003).

In our country total livestock population is 512.05 million, of which buffalo contributes 21.23 per cent of total livestock population according to livestock census 2012, total buffalo population is 108.7 million from which number of male buffalo is 16.1 million and 92.5 million female buffalo in 2012.Number of milch buffalo is 51.05 million(19th livestock census 2012).

Buffalo rearing in Rajasthan plays a significant role in the animal husbandry practice. According to livestock census 2012 in Rajasthan total buffalo population is 12.97 million.

Cardiovascular system comprises heart, arteries, veins, blood cells and plasma. Heart and blood vessels form two functional units of cardiovascular system. The cardiovascular system allows circulation of blood and transport of nutrients like amino acids, electrolytes, sugar. It also helps in distribution of respiratory gases such as oxygen and carbon dioxide apart from that hormones, excretory products are also circulated by cardiovascular system. Blood cells specially white blood cells fights against the infections and diseases.Cardiovascular system also maintain body temperature and pH and homeostasis.

Buffalo calves suffer from higher mortality than cow calves *(* Tomar and Tripathi*, 1991)* mainly due to managemental problems and diseases. Heart disease in buffalo remains medically challenging both to diagnose and to treat. This is because of its low incidence in the bovine species but also because the prognosis is poor. In buffaloes, cardiovascular disease conditions like atherosclerosis, arteriosclerosis, serous atrophy of suberpicardial fat, hydropericardium, haemopericardium, epicardial calcification, suppurative pericarditis, endocarditis, epicardial haemorrhages, Traumatic pericarditis etc. are common which causes economic loss to the farmers.

Cardiac diseases in bovines are one of the easily overlooked veterinary cases *(*Maillard *et al., 2007)* and the diagnosis is normally made almost at the terminal stage with poor prognosis *(*Reef *et al., 2008).* The proposed work has been planned to study pathological changes in cardiovascular system of buffaloes in the areas of Rajasthan.

No efforts have been made to study the occurrence and pathology of cardiovascular lesions in buffaloes in Rajasthan state. Therefore, the present investigation has been undertaken with the following objectives:

1. To study the occurrence of various pathological conditions of Cardiovascular system in buffalo.
2. To Study the gross and histopathology of various pathological conditions of Cardiovascular system of buffalo with reference to the type, pattern and morphology.

A. Pathological conditions of Heart:

1. Inflammatory conditions:-

1. (a) Pericarditis:-

Alwis (1984) observed incidence of Haemorrhagic septicaemia in cattle and buffaloes through histopathological studies, He showed that the animals which died within 24-36 hours of infection had only widespread petechial haemorrhages, most pronounced at the base of the heart. When the course of the disease extended to 48 hours, the haemorrhages were more severe and ecchymotic, with fibrinous pericarditis. There was serofibrinous pericarditis and pleurisy with adhesions between the pericardium and pleura. Buffaloes die quicker and consequently show fewer lesions than cattle.

Sojka *et al* (1990) studied septic pericarditis as sequel to traumatic reticulitis in cattle. An antemortem diagnosis of pericarditis was made in cattle based on the clinical, clinicopathologic, radiographic, and ultrasonographic findings, which were consistent with those classically described with traumatic pericarditis. Postmortem findings suggested that the cause of the traumatic pericarditis was penetration of the wire through the skin, with subsequent migration into the sternebrae and pericardial sac.

Abo-Shehada *et al.* (1991) studied Traumatic pericarditis in awassi lamb.During necropsy finding a 6 cm long metallic wire was found penetrating the pericardium and the left ventricle through to the other side of the ventricle wall. Fibrinous pericarditis, thickening of the pericardium, fusion of the pericardium and epicardium, and epicarditis were also observed. The heart was enlarged with oedematous myocarditis and focal necrotic endocarditis involving the two ventricles.

Vegad and Katiyar (2001) observed colonization of E. coli on the surface of entrocytes in enterotoxic colibacillosis from E. coli infection. In septicemic colibacillosis, they observed polyserositis (pericarditis, pleuritis and peritonitis), pyelonephritis with bacterial emboli and necrotizing, purulent and fibrinious exudates.

Jesty *et al.* (2005) studied Idiopathic pericarditis with cardiac tamponade. Two cows were examined for clinical signs of right-sided congestive heart failure. Cytologic analysis of the pericardial fluid revealed hemorrhagic inflammation but no evidence of a septic or neoplastic condition such as traumatic reticulopericarditis or lymphoma, respectively.

Anna *et al.* (2006) studied idiopathic hemorrhagic pericardial effusion in cows. They observed idiopathic pericarditis and traumatic pericarditis in cattle. Postmortem examination revealed that the pericardial sac was filled with dark red fluid and the epicardium was covered with fibrin. Myocardial necrosis with intracellular bacilli, fibrinosuppurative epicarditis, and necrohemorrhagic pericarditis were observed histopathologically

Bexiga and Philbey (2008) studied on Clinicopathological presentation of cardiac disease in cattle and its impact on decision making. 116 cattle suffering from cardiac disease were examined. On the basis of the results of postmortem examinations pericarditis were observed in 39 cases.

Abdelaal *et al* (2009) studied clinical and ultrasonographic differences between cattle and buffaloes with various sequelae of traumatic reticuloperitonitis (TRP).Twenty nine cows and 33 buffaloes with TRP were investigated. Various sequelae of TRP in both species were clarified. Acute local peritonitis, chronic local peritonitis, acute diffuse peritonitis, reticular abscesses, thoracic abscesses and pericarditis have been detected as sequelae of TRP in both cattle and buffaloes. Brisket oedema and distended jugular veins have been shown in both cattle and buffaloes with pericarditis and also in four buffaloes with thoracic abscesses.

Braun (2009) studied traumatic pericarditis in cattle: clinical, radiographic and ultrasonographic findings were observed these includes distension of the jugular veins and submandibular , brisket and ventral abdominal oedema. Fibrinopurulent adhesions were also found in necropsy.

Raji *et al.* (2010) observed Pathological conditions and lesions in slaughtered cattle. In the study total 7812 cattle were examined. The examination involved visual examination of carcasses and organs including

hearts. Through palpation and incision of suspected organs, gross pathological lesion of each diseased organ was determined. The results of the study revealed that from total cases, pericarditis 6.3% and fat atrophy of the heart 30.3%.

Ghanem (2010) carried out study on 15 Holstein cows divided into 3 groups of 5. Group 1 was the control, group 2 had traumatic reticuloperitonitis (TRP), and group 3 had traumatic pericarditis (TP).TRP cows had a significant increase in PCV, leukocytes, and neutrophils, and a significant decrease in RBC, hemoglobin, and lymphocytic counts, as compared to the control group. TP cows had significant erythrocytopenia, leukocytosis, neutrophilia, monocytosis eosinopenia, and basopenia, and a significant decrease in the hemoglobin, lymphocytes, eosinophils, and basophils as compared to the controls.

Cevik *et al.* (2010) studied Traumatic reticulopericarditis in a Goat and observed fibrinopurulent exudate and fibrinous adhesions between pericardium and left apical lobe of the lung in necropsy finding. In the pericardial sac, cardiac tamponade resulting from excessive clotting was observed. Histopathologically, fibrinopurulent inflammatory reaction was detected in the wall of the reticulum, pericardium and epicardium.

Buczinski *et al.* (2010) conducted a study on heart diseases. In the study 47 cattle suffering from heart disease without clinical signs of heart failure were considered. Based on clinical and echocardiographic examinations and necropsy findings, the final diagnoses included 19 cases of bacterial endocarditis, 8 of pericarditis.

Buczinski *et al. (*2010) found principal heart diseases of cattle as pericarditis, bacterial endocarditis, congenital heart defects and heart neoplasms.

Tharwat and Buczinski (2011) studied Clinicopathological findings and echocardiographic prediction of bovine endocarditis. Thirty-six cows with confirmed vegetative endocarditis at postmortem examination were examined in this study. Results of the study revealed postmortem findings in 36 cows with tricuspid, mitral and pulmonic valve vegetations. The tricuspid, mitral and

pulmonic valves were affected as a single infection in nine, four and 20 cases, respectively. Two cows had vegetations at both tricuspid and mitral valves and one had mitral valve vegetations together with fibrinous pericarditis.

Braun *et al.* (2011) confirmed Traumatic pericarditis in postmortem of 28 cattle on the bases of clinical signs.

Mohamed *et al.* (2012) studied on Atypical Presentation of Constrictive Pericarditis in a Holstein Heifer. They observed a 9 year old heifer in e gross pathology revealed approximately 8 liters of clear yellow fluid in the pleural cavity mixed with yellow gelatinous material. The pericardium was severely distended, displacing the lung dorsally. The pericardial cavity contained approximately 7 liters of clear yellow malodorous fluid admixed with moderate amounts of tan to white friable material. The parietal pericardium was severely thickened measuring approximately 2.5 cm. The pericardial sac and was totally lined by a large mat of thick yellow fibrinous material.

Athar *et al.* (2012) studied pericarditis in bovines and observed that it is an inflammation of the pericardium and accumulation of pericardial fluid in pericardial sac.

Faisal *et al.* (2013) worked on advanced techniques in traumatic reticuloperitonitis diagnosis in cattle. In post mortem examinations sometimes there were extensive fibrinous adhesions between the cranioventral aspects of the reticulum, the ventral abdominal wall, and the Adhesions and multiple abscesses were observed on either side of the reticulum. Large quantities of turbid, foul-smelling peritoneal fluid that contained fibrinous clots were present. Histopathology showed thickening of the pericardium due to accumulation of fibrinous inflammatory exudate, which was apparent between the pericardium and myocardium. With high magnification there was a fibrinous network observed to trap inflammatory cells: mostly neutrophils and mononuclear cells. The myocardiums in the TP cows have severe inflammatory cell infiltration replacing the cardiac muscle that has atrophied.

Hussein *et al*. (2014) reported congestive heart failure in buffaloes. Fifty-three water buffaloes (*Bubalus bubalis*) were examined for the study. Postmortem examination was performed thoroughly on all animals which

revealed that in the cases with traumatic pericarditis, the pericardium was distended, displacing the lung dorsally. Turbid yellow malodorous fluid, indicating hydropericardium was present in the pericardial sac, along with the thick wall of the pericardium. The heart was covered by yellow fibrinous deposits. Constrictive adhesions between the pericardium and the apex of the heart were seen in 7 cases. In buffaloes with vegetative endocarditis, the tricuspid valve was mostly affected (8 cases), followed by the mitral (5 cases) and pulmonary (3 cases) valves. These lesions appeared primarily as cauliflower-like, verrucose, or wart-like with either cardiac dilatation or cardiac hypertrophy.

Prasad *et al.* (2017) carried out a pathological study to observe traumatic reticulitis, reticulo-peritonitis and pericarditis in bovines. On post mortem examination of animals severe fibrinous pericarditis with thick fibrinous nodules in heart were observed. The contour of the heart altered from its normal structure while the interior of the heart was normal and free from any pathological lesions.

2. (b) Myocarditis:-

Hoffmann *et al*. (1984) studied on clinico-pathology of a malignant catarrhal fever syndrome in the Indonesian swamp buffalo (*Bubalus bubalis*).Postmortem and histopathological lesions were observed in most of the tissues and organs of the body. The occurrence of serofibrinous epicarditis and myocarditis, which was seen in buffaloes in all cases.

Van and Ferrans (1986) studied on myocardial diseases of animals and observed idiopathic or primary cardiomyopathies and myocarditis of viral, bacterial, and protozoal causation. These include various degenerative changes, myocyte necrosis, and inflammatory lesions.

Damayanti *et al*. (1994) in their study they inoculated intravenously *Trypanosoma evansi*, in six Indonesian buffaloes (*Bubalus bubalis*) and examined clinically, haematologically and serologically, and then killed 1, 2, 3, 4, 8 or 12 weeks of infection for detailed pathological study. In histopathology the most consistent lesions were interstitial myocarditis in the heart.

Teankum K. *et al*. (2000) conducted pathological study on Malignant catarrhal fever in swamp buffaloes in Thailand. In their experiment total 26 animals were included from herd and necropsy was performed on 11 buffaloes. Various degrees of haemorrhage in the epicardium, myocardium, and endocardium were observed in all animals. Striking lesions of non-suppurative myocarditis were frequently noted.

V. Gunes *et al.* (2005) carried out Assay of cardiac troponins in the diagnosis of myocardial degeneration due to foot-and-mouth disease in a calf. Postmortem examination was carried out, and heart samples were taken for histopathological examination, routinely processed and stained with haematoxylin and eosin, Von Kossa and Van Gieson stains. Microscopically, intense mononuclear cell infiltration, were seen on the myocardium. There was also hyperaemia, epicardial haemorrhage and edema.

Erica A.Costa *et al.* (2009) studied an outbreak of malignant catarrhal fever in Murrah buffaloes in Minas Gerais, Brazil.Histopathological lesions included multifocal histiolymphocytic epicarditis, myocarditis.

O'Toole D. *et al.* (2009) worked on diagnostic exercise myocarditis due to *Histophilus somni* in feedlot cattle. They found focal red discoloration in papillary muscle of the left ventricular myocardium. Histologically, acute necrotizing myocarditis lesion were observed .

Abdullah kaya *et al*. (2013) studied serum homocystein levels in calves with FMD.They studied on 15 FMD affected calves with 9 healthy calves. They performed necropsy of three calves died due to FMD. In macroscopic findings they observed necrotic myocarditis with pale foci having hyperemia on periphery was found on the ventricular and papillar muscles of heart. In histopathology lymphohistiocytic infiltration were observed in heart muscles.

Yasmin *et al.* (2014) carried out an clinical, biochemical and pathological investigation buffaloes with foot-and-mouth disease. Specimens from tongue, heart, liver, lung and kidneys were taken for histopathological examination. Examination of the dead heifers revealed presence of different lesions in the heart, lungs, liver and kidneys. The heart showed necrosis in the myocardium of the left atrium. Histopathological examination of heart from

the dead heifers revealed severe interstitial myocarditits in which the myocardial fibers were necrosed with heavy infiltration of lymphocytes.

Mustafa S. Aktas *et al* (2015) examined total, 53 calves were to be suspected of having foot-and-mouth disease infection. During this period 6 calves died within 2 days and histopathology confirmed myocarditis.

Adeeb A. Dawood and Kamal M. Alsaad (2018) studied clinical and diagnostic studies of myocarditis result from FMD In lambs. The study was conducted on (125) local suckling lamb breeds (5-30) days old and from both sexes. Macroscopic examinations of the autopsied dead lambs, exhibit necrotic myocarditis with enlargement of the heart and observing of different sizes of pale foci with a zone of hyperemia detected in different parts of heart tissue specially the papillar and ventricular cardiac muscles. In histopathological examinations there were severe inflammatory cell infiltration in the interstitium of myocardial fibers were found.

3. (c) Endocarditis:-

Power and Rebhun (1983) studied bacterial endocarditis in adult dairy cattle and found that the end stage was congestive heart failure. They also observed low-grade anemia and high total serum globulin content developed with chronicity.

Yamaga and Too (1987) worked on Diagnostic ultrasound imagine of vegetative valvular endocarditis in cattle. Bovine vegetative valvular endocarditis in 5 cases was examined using echocardiography to determine its diagnostic capacities, and in addition, observations of heart failure were performed by ultrasonography. In 4 out of 5 cases, the vegetations revealed echogenic or "shaggy" masses of various sizes, and one of them also contained the cystic pattern. Vegetative endocarditis was found on the pulmonary valve and there was a large amount of reddish pericardial effusion. The cauliflower-like vegetation obstructed almost the whole cavity of the pulmonary trunk. The right ventricle was also dilated. Several nodular vegetations (1 to 3 cm) were found on the mitral valve and the left atrium and ventricle were moderately dilated.

Healy (1996) studied endocarditis in 22 cattle. Endocarditis was confirmed by ultrasound and/or at necropsy. The most frequent lesions were on the tricuspid valve which was the only valve involved in eight animals. Multiple valve involvement was present in three cases. Four animals had a mitral endocarditis only.

Maillard *et al.* (2007) conducted a study to determine the role of Bartonella as an endocarditis agent in cattle. For the study twenty-two cases of bovine endocarditis were diagnosed in adult cows endocarditis was diagnosed at physical examination. Lesions of the cardiac valves were confirmed at necropsy for all animals. Most of the damaged valves of these animals had large, cauliflower like lesions.

Bexiga *et al.* (2008) studied on Clinicopathological presentation of cardiac disease in cattle and its impact on decision making. 116 cattle suffering from cardiac disease were examined. On the basis of the results of postmortem examinations 52 cases of endocarditis were observed.

Edwards *et al.* (2009) studied endocarditis in a British heifer due to *Erysipelothrix rhusiopathiae* infection. Examination of haematoxylin and eosin-stained sections of formalin fixed tissues revealed severe subacute to chronic active endocarditis. The pathology observed was consistent with a diagnosis of severe valvular and mural endocarditis orientated on the right side of the heart.

Buczinski *et al.* (2010) studied heart disease in cattle. In their study they observed 106 anmals from which 59 had clinical signs compatible with heart failure, and 47 had a clinical diagnosis of heart disease without clinical signs of heart failure. In their study they also observed 7 cases of bacterial endocarditis along with 25 cases of pericarditis, 5 cases of neoplasm and 9 cases of dilated cardiomyopathy.

Diane J. Waschburger *et al.* (2012) studied streptococcal endocarditis and valvular arteritis in a sheep. In gross examination there were irregular, vegetative structures adhered to the endocardium of the right ventricle and pulmonary semilunar valve. Fibrinous pericarditis and concentric hypertrophy

of the right ventricular myocardium were also observed. In histology they observed mild lymphohistioplasmacytic endocarditis and myocarditis of the right ventricle, with subendocardial fibrosis. They found that main cause of pericarditis was streptococcal infection.

Selvam *et al*. (2014) studied occurrence of vegetative valvular endocarditis in an Indian buffalo. On autopsy of the affected buffalo pin-point to ecchymotic hemorrhages on subcutaneous tissues and visceral surfaces were observed. The heart was enlarged and the endocardium of the cardiac ventricles had suffusive hemorrhage. The cusps of right atrio-ventricular valves had irregular, yellow, loosely adhering, friable masses causing stenosis of the valvular space. Histopathology of the affected valve showed, severe suppurative inflammation and peri-vavular cuffing of thrombotic mass. The thrombotic mass was seeded with multifocal bacterial colonies surrounded by multiple concentric layers of eosinophilic fibrin material.

Adamu *et al* (2014) reported Vegetative valvular endocarditis in a calf. In the study a one month old calf with the history of naval ill was considered. Grossly, the carcass was emaciated and pale. Post mortem examination of the carcass revealed cauliflower like lesions on both valves of the heart. Histopathologically, necrosis and haemorrhage were observed in the cardiac valves with infiltrations of bacterial colonies. Variable sizes of whitish and reddish growth were also found on the valve leaflet. All findings confirmed the diagnosis of septicemic and vegetative valvular endocarditis squeal to navel ill caused by E. coli infection.

2. Degenerative Changes:-

2. (a) Myocardial necrosis:-

Bradley *et al.* (1981) reported Sudden death and myocardial necrosis in cattle. They studied 26 animals which died suddenly. The only pathological abnormalities detected were definite or equivocal myocardial necrosis. Three types of necrosis were identified: myodegeneration, contraction band necrosis and coagulation necrosis. Vacuolation of myocytes occurred in control hearts only in the sub-endocardial myocardium, but was found more extensively in diseased hearts. Paraffin sections of myocardium stained by von Kossa's

method or by haematoxylin-basic fuchsin-picric acid improved the detection of myocardial necrosis.

Van Vleet *et al.* (1986) studied Clinical, clinicopathologic, and pathologic alterations in acute monensin toxicosis in cattle. Twenty beef calves weighing approximately 180 kg were allotted to 3 groups. Calves were euthanatized and their necropsy examinations were done, lesions of monensin toxicosis were present in the heart, skeletal muscles, and rumen. In observation disseminated pale yellowish-brown areas of necrosis were present in the ventricular myocardium. In Microscopic study the myocardial and skeletal muscular lesions were characterized by sarcoplasmic vacuolation from mitochondrial swelling and lipid accumulation.

Van and Ferrans (1986) studied on myocardial diseases of animals and observed various degenerative changes, myocytic necrosis and inflammatory lesions.

Seamus Kennedy and Desmond A. Rice (1988) observed selective morphological alterations of the cardiac conduction system in calves with deficiency of vitamin E and selenium and reported that vitamin E and selenium deficiency causes necrosis of the contractile myocardium and preferential degeneration and necrosis of Purkinje cardiocytes.

Bradley *et al*. (1991) studied cardiomyopathy in adult holstein friesian cattle. In their study the cardiac lesions were, extensive myocyte vacuolation, endomysial and perimysial fibrosis and focal cardiac myocyte degeneration, atrophy and hypertrophy resulting in an extended range of myocyte size.

Orr and Blakley (1997) Investigated selenium status of aborted calves with cardiac failure and myocardial necrosis. The fetuses were necropsied, tissues were taken for histopathology. Histopathologic examinations of myocardium of fetuses with cardiac failure revealed myocardial necrosis and mineralization in 7 fetuses, lymphocytic myocarditis in 5 fetuses, myocardial fibrosis in 5 fetuses, or no microscopic lesions in 5 fetuses.

Sharma and Shrivastava (2002) studied the pathomorphological changes due to induced colibacillosis in neonatal kids. In histopathological

examination of heart showed marked congestion and haemorrhages with separation and degeneration of myofibers and purkinge fibers.

Gunes *et al.* (2005) carried out Assay of cardiac troponins in the diagnosis of myocardial degeneration due to foot-and-mouth disease in a calf. Postmortem examination was carried out, and heart samples were taken for histopathological examination, routinely processed and stained with haematoxylin and eosin, Von Kossa and Van Gieson stains. Macroscopically, greyish foci of irregular size in the ventricles of the heart were identified. Microscopically, lesions characterised by hyaline degeneration and necrosis, accompanied by an intense mononuclear cell infiltration, were seen on the myocardium. There was also hyperaemia, epicardial haemorrhage and oedema.

Murray *et al.* (2007) studied on field investigation of perinatal mortality in friesian cattle associated with myocardial degeneration and necrosis.Total 504 cows were calved and 215 were heifers. Tissues from eight fetuses were submitted to the University of Liverpool for histopathological examination; all presented lesions consistent with myocardial degeneration and necrosis of the left ventricle.

Hahn *et al.* (2012) studied on acute death in 36 out of 233 predominantly Simmental breed calves. According to their observation the cause of death was myocardial necrosis revealed at post mortem examination. The calves were orally administered with doxcycline at high dosage in their country of origin. Based on previous reported cases and the lack of other pathological evidence, high dose of doxycycline was presumed to be the cause of the myocardial changes.

Asopa (2012) carried out a study on Occurrence and pathology of colibacillosis in buffalo (*Bubalus bubalis*). In the study 610 buffaloes were examined. For histopathological study, samples of liver, lung, spleen, intestine, kidneys, heart and mesenteric lymph nodes were collected. Microscopically, some sections of heart showed degeneration and separation of myofibrils.

Nabi *et al.* (2013) studied Physio-pathology of induced endotoxaemia in bovine. In the study endotoxic shock was induced in five apparently healthy male buffalo calves by i.v infusion of Escherichia coli endotoxin at 5 microgram/kilogram (µg/Kg) body weight/hour (BW/hr) for 3 hours. In necropsy examination they found degenerative changes of cardiac myocytes along with mononuclear cell infiltration in perivascular region of the myocardium were also noticed, suggesting cardiomyopathy.

Mehra (2013) conducted a study to determine occurrence and pathology of colibacillosis in cattle (*Bos indicus*). The isolation and identification of bacteria Escherichia coli were carried out from various tissue samples including heart. Out of total isolates, 11 isolated from hearts. Microscopically, some section revealed degeneration and separation of myofibrils.

Abdullah kaya *et al.* (2013) stidied serum homocystein levels in calves with FMD. They studied on 15 FMD affected calves with 9 healthy calves.They performed necropsy of three calves died due to FMD. They observed hyaline degeneration and necrosis of myocardial cells.

Adamu *et al.* (2014) reported Vegetative valvular endocarditis in the calf. In the study a one month old calf with the history of naval ill was considered. Histopathologically, necrosis and haemorrhage were observed in the cardiac valves.

Mohammad *et al.* (2017) carried out clinicopathological investigation of Foot and Mouth Disease in cattle. In the study tissues from the buccal cavity, lungs, liver, kidney, spleen, pancreas, and heart were taken and processed for histopathology. Myocardial lesion was common in all the dead young and adult cattle investigated; the irregular grey necrotic foci onto the heart gave rise to a stripped appearance commonly known as ‘tiger heart’ disease. Characteristic “currant jelly clot” was seen in the heart of adult cattle. Most specific striking lesions seen were in the heart muscles of both the young and adult cattle, where the muscle fibers were necrosed and hyalinised. Multifocal accumulation of lymphocytes were seen in necrotic heart muscle.

Carlos *et al.* (2018) studied white muscle disease in three selenium deficient beef and dairy calves in Argentina and Uruguay. At postmortem examination, multiple pale areas were observed in skeletal muscles and myocardium. In all necropsied calves, ventricles were dilated and hydropericardium were present. Microscopically, degeneration, necrosis and mineralization of cardiac and skeletal myocytes were observed.They also observed myocardial fibrosis in the affected sections.

2. (b) Coagulative necrosis :-

R. Bradley *et al.* (1981) reported Sudden death and myocardial necrosis in cattle. They studied 26 animals which died suddenly. Three types of necrosis were identified: myodegeneration, contraction band necrosis and coagulation necrosis. Vacuolation of myocytes occurred in control hearts only in the sub-endocardial myocardium, but was found more extensively in diseased hearts.

Narth *et al.* (2016) studied Clinical and pathological features of dilated cardiomyopathy in Holstein-Friesian cattle. In their experiment nine cases of the condition were compared in terms of their clinical and pathological characteristics with nine unaffected animals. In postmortem, the affected hearts were enlarged with all the chambers dilated and walls of variable thickness. In histopathology extensive loss of cardiomyocytes by coagulative necrosis, increased variation in the cross-sectional area of the myocardial fibres were observed.

Adeeb A. Dawood and Kamal M. Alsaad (2018) made clinical and diagnostic studies of myocarditis result from FMD In lambs. The study was conducted on (125) local suckling lamb breeds (5-30) days old and from both sexes. In histopathology myocardial sections showed obvious areas of coagulation of myocardial fibers (coagulative necrosis of myocardial fibers) and marked area of hyalinization, Furthermore, high number of large areas of vacuolated degenerative myocardial muscle cells,several foci and several vacuolated-degenerated myocardial muscle cells with possibility of interstitial cell edema were also detected.

2. (c) Myocardial fibrosis

Leifsson and Agerholm (2004) studied familial occurrence of bovine dilated cardiomyopathy in Denmark. The occurrence of this disorder in the red danish dairy breed, Holsteins, and red holsteins in Denmark was reported. Fourteen cases were diagnosed during a 13-year period. All suffered from congestive heart failure because of progressive myocardial fibrosis.

Carlos (2018) studied white muscle disease in three selenium deficient beef and dairy calves in Argentina and Uruguay. At postmortem examination, multiple pale areas were observed in skeletal muscles and myocardium. In all necropsied calves Ventricles were dilated and hydropericardium, were present. Microscopically,degeneration, necrosis and mineralization of cardiac and skeletal myocytes were observed. They also observed myocardial fibrosis in the affected sections.

2. (d) Endocardial fibrosis:-

Cushing (2013) studied endocardial fibroelastosis in a quarter horse mare. Necropsy examination revealed a markedly enlarged heart. Microscopically, the endocardial thickening was due to deposition of fibrous connective tissue and elastin fibres. These findings are consistent with a diagnosis of endocardial fibroelastosis.

Hananeh and Ismail (2018) studied occurrence of acute bovine pulmonary edema and emphysema and endocardial fibroelastosis in cattle. Necropsy and histopathological examination were performed and endocardial fibroelastosis revealed seven cattle. The left endocardium was markedly and uniformly thickened up to 10× normal thickness caused by abundant connective tissues primarily, elastic fibers.They also observed fibers were loosely arranged in multiple layers with moderate edema that separated the fibers. In multiple areas, the fibers breached the endocardium into the underlying myocardium causing widening the edematous interstitium.

3. Circulatory disturbances:-

3. (a) Congestion :-

Azmi and Jha (1994) conducted an experimental study on pathology of induced *E.coli* infection in kids. In their study they observed sections of heart showed congestion and edema.

Rao *et al.* (1985) conducted postmortem examination of lambs and found diffused congestion of all internal organs including spleen and heart. Spleen and heart were found edematous and swollen.

Sharma and Shrivastava (2002) studied the pathomorphological changes due to induced colibacillosis in neonatal kids. Postmortem examination of infected kid revealed general congestion of the visceral organ with or without petechial over the epicardium, lung, kidney and liver. In histopathological examination heart showed marked congestion and haemorrhages with separation and degeneration of myofibers and purkinje fibers.

Seema *et al* (2007) examined 30 buffaloes/ buffalo calves at Department of Veterinary Pathology, CCS Haryana Agricultural University, Hisar. In her examination, congestion of lungs, kidneys and heart were also observed.

Gupta (2012) carried out a study on Occurrence and pathology of coloibacillosis in Goats (*Capra hircus*).Grossly the hearts showed, congestion and haemorrhages over epicardium. Microscopically, heart sections revealed congestion, haemorrhages, oedema, and infiltration of few mononuclear cells with separation of muscle fibres.

Asopa (2012) carried out a study on occurrence and pathology of colibacillosis in buffalo (*Bubalus bubalis*). In the study, 610 buffaloes were examined. For histopathological study, samples of liver, lung, spleen, intestine, kidneys, heart and mesenteric lymph nodes were collected. Affected hearts showed congestion with mild haemorrhages over epicardium in gross examination. Microscopically, heart section showed marked congestion of myocardial blood vessels.

Mehra (2013) conducted a study to determine occurrence and pathology of colibacillosis in cattle (*Bos indicus*). The isolation and identification of bacteria *Escherichia coli* were carried out from various tissue samples including heart. Out of total isolates, 11 isolated from hearts. Gross examination of affected hearts showed congestion with mild haemorrhages over epicardium.

3. (b) Haemorrhage:-

Alwis (1984) observed incidence of Haemorrhagic septicaemia in cattle and buffaloes through histopathological studies, He showed that the animals which died within 24-36 hours of infection had only widespread petechial haemorrhages, most pronounced at the base of the heart. When the course of the disease extended to 48 hours, the haemorrhages were more severe and ecchymotic, with fibrinous pericarditis.

Damayanti *et al.* (1994) intravenously inoculated *Trypanosoma evansi* in their study, in six Indonesian buffaloes (*Bubalus bubalis*) and examined clinically, haematologically and serologically, and then killed 1, 2, 3, 4, 8 or 12 weeks alter infection for detailed pathological study. In postmortem findings they observed petechial to larger haemorrhages in the pericardium associated with heart lesions. In histopathology the most consistent lesions were interstitial pneumonia, interstitial myocarditis, splenic multifocal necrosis, interstitial myositis and hyperplastic bone marrow.

Singh *et al.* (1996) infused *E.coli* endotoxin in 8 male murrah buffalo calves aged 8-10 months. Four calves died within 24 - 36 hours after injection while one calf died 5 minute after endocardial administration of endotoxin. Post mortem examination revealed sub-epicardial and sub-endocardial haemorrhage. Hyaline degeneration of myocardium coupled with sub-endocardial and myocardial haemorrhages were predominant lesions.

Teankam *et al.* (2000) studied Malignant catarrhal fever in swamp buffaloes. In their study various degrees of haemorrhage in the epicardium, myocardium, and endocardium were observed in all animals. They observed epicardial haemorrhages (23.07 %), endocardial haemorrhages (53.84%), myocardial haemorrhages (30.76%) and non-suppurative myocarditis in (72.72 % cases.

Sastry (2001) recorded enteric form of colibacillosis and observed haemorrhages on the epicardium and endocardium in septicemic form of colibacillosis.

Gunes *et al.* (2005) carried out assay of cardiac troponins in the diagnosis of myocardial degeneration due to foot-and-mouth disease in a calf.

Postmortem examination was carried out, and heart samples were taken for histopathological examination, routinely processed and stained with haematoxylin and eosin, Von Kossa and Van Gieson stains. Microscopically, there was hyperaemia, epicardial haemorrhage and oedema.

Jubb *et al*. (2007) reported serosal haemorrhage with serosanguinous pericardial fluid in the heart in their study on *E. coli* infection.

Buczinski *et al.* (2010) conducted a study on heart diseases. In the study 47 cattle suffering from heart disease without clinical signs of heart failure were considered. One case of pericarditis was associated with pneumonia while another was compatible with idiopathic hemorrhagic pericarditis.

Bratanich *et al.* (2012) confirmed diagnosis of Sheep-associated Malignant Catarrhal Fever in Bison in Argentina. Necropsies were performed and tissue samples taken from all animals for histopathological studies. In necropsy findings epicardium and pericardium exhibited petecchiae and ecchimoses on them with subendocardical hemorrhages.

Gupta (2012) carried out a study on Occurrence and pathology of coloibacillosis in Goats (*Capra hircus*). The hearts were grossly examined and congestion and haemorrhages over epicardium were seen. Microscopically, heart sections revealed congestion, haemorrhages, edema.

Asopa (2012) carried out a study on Occurrence and pathology of colibacillosis in buffalo (*Bubalus bubalis*). In the study 610 buffaloes were examined. For histopathological study, samples of liver, lung, spleen, intestine, kidney, heart and mesenteric lymph nodes were collected. Affected hearts showed congestion with mild haemorrhages over epicardium in gross examination. Microscopically some sections revealed haemorrhages in myocardial region.

Mehra (2013) conducted a study to determine occurrence and pathology of colibacillosis in cattle (*Bos indicus*). The isolation and identification of bacteria Escherichia coli were carried out from various tissue samples including heart. Out of total isolates, 11 isolated from hearts. Gross examination of affected hearts showed congestion with mild haemorrhages

over epicardium. Microscopically, some section revealed haemorrhages in myocardial region.

Ahmed *et al* (2014) investigated an outbreak of hemorrhagic septicemia in a vaccinated herd of domestic water buffalo. All animals in the herd were affected with HS and 11 died out of the total 40 buffaloes. Postmortem examination was performed on 3 out of the total 11dead animal. Petechial hemorrhages were also noted on the epicardial surface of the heart.

Adamu *et al.* (2014) reported Vegetative valvular endocarditis in a calf. In the study a one month old calf with the history of naval ill was considered. Histopathologically, necrosis and haemorrhage were observed in the cardiac valves with infiltrations of bacterial colonies.

Selvam *et al.* (2014) studied occurrence of vegetative valvular endocarditis in an Indian buffalo. On autopsy of the affected buffalo pin-point to ecchymotic hemorrhages on subcutaneous tissues and visceral surfaces were observed. The heart was enlarged and the endocardium of the cardiac ventricles had suffusive hemorrhage.

Camara *et al.* (2014) studied epidemiology, clinical signs, laboratorial and pathological findings in eight outbreaks of botulism in cattle. Post mortem examination of ten cattle were done and several tissue samples were collected (liver, kidney, heart small intestine and central nervous system) for histopathological examination. In heart they observed petechial and ecchymotic haemorrhages in the pericardium.

Hristov *et al.* (2016) investigated Malignant catarrhal fever in wild ruminants. Tissue samples from liver, lungs, kidneys, heart, spleen and small intestines were collected for histological examinations.In their study numerous petechiae and ecchymoses and extensive hemorrhages around adipose tissue of pericardium were found. Coronary and interventricular vessels of epicardium were with extensive hemorrhages and numerous pale zones in cardiac muscle were observed. In histopathology examination, in hearts subendocardiac and subepicardiac haemorrhages were observed.

3. (c) Edema:-

Azmi and Jha (1994) conducted an experimental study on pathology of induced *E. coli* infection in kids. The sections of heart showed edema and congestion in their study.

Rao *et al.* (1985) conducted postmortem examination of lambs and in their study spleen and heart were found edematous and swollen.

H. Furuoka *et al*. (2001) studied Hereditary Dilated Cardiomyopathy in Holstein-Friesian Cattle in Japan. The study was based on pathology and genetic basis of dilated cardiomyopathy in 10 Holstein-Friesian cows aged 3–6 years. At necropsy the animals showed cardiomegaly and cardiac oedema.

Gunes *et al*. (2005) carried out Assay of cardiac troponins in the diagnosis of myocardial degeneration due to foot-and-mouth disease in a calf. Postmortem examination was carried out, and heart samples were taken for histopathological examination. Microscopically, they observed edema along with hyperaemia and epicardial haemorrhage.

Gupta A. (2012) carried out a study on Occurrence and pathology of coloibacillosis in Goats (*Capra hircus*). Microscopically, heart sections revealed congestion, haemorrhages and oedema.

3. (c) Hydropericardium:-

Damayanti *et al*. (1994) intravenously inoculated *Trypanosoma evansi* in their study, in six Indonesian buffaloes (*Bubalus bubalis*) and examined clinically, haematologically and serologically, and then killed 1, 2, 3, 4, 8 or 12 weeks alter infection for detailed pathological study. In postmortem findings they observed emaciation, serous atrophy of fat, hydropericardium, petechial to larger haemorrhages in the pericardium associated with heart lesions.

J.M. Neary *et al*. (2016) studied right sided congestive heart failure in north american feedlot Cattle. In post-mortem findings they observed brisket and ventral edema, hydroperitoneum, hydrothorax and secondary atelectasis, hepatomegaly and chronic passive congestion, intestinal and mesenteric edema, hydropericardium, right-ventricular hypertrophy and dilatation.

4. Parasitic Involvement:-

4 (a). Sarcosporidiosis:-

Foggin (1980) histopathologically examined sections of heart muscles of buffalo carcasses and observed thin walled *S. cruzi* in myocardium.

Bratberg and Landsverk (1980) examined apparently healthy cattle carcasses. They observed focal interstitial myocarditis in 31.6% of samples. They also reported that Interstitial mononuclear cell infiltrations were positively associated with Sarcocystis infection.

Collery and Weavers (1981) did histological examination in calves. They observed large number of immature sarcocysts were present in the skeletal muscles and myocardium along with lymphocytic infiltration.

Szarek (1982) examined 58 cows microscopically and macroscopically for sarcosporidiosis and observed that sarcosporidiosis was present in myocardium of 8 cattle.

Jain *et al*. (1987) examined 200 oesophagus and 25 heart specimens of buffaloes from slaughter houses of Delhi. They found 72 percent heart samples infected from sarcocysts.

Shah (1983) studied presence of sarcocyst in various organs and found highest rate of infection in cardiac muscles (54.66%).

Jensen *et al* (1986) evaluated Eosinophilic myositis and muscular sarcocystosis in the carcasses of slaughtered cattle and lambs. Fifty-three bovine and 7 ovine carcasses were examined and they found 7 (100%) of the ovine carcasses had multiple, small, disseminated lesions in tongue, esophagus, heart, diaphragm, or skeletal muscles.

Jitender Reddy (1989) studied gross and histopathological changes in different organs caused by Sarcocystis in 980 buffaloes of different abattoirs of Hyderabad. In his examination the histopathology showed mononuclear cell infiltration and necrosis of cardiac muscle due to sarcocystis infection.

Adam Bundza *et al.* (1989) studied on Eosinophilic myositis and lymphadenitis in slaughter cattle.They collected Samples of bovine hearts, skeletal muscles and lymph nodes in histopathology they observed

eosinophilic epicarditis, myocardial granulomas surrounded by eosinophils, and the presence of sarcocysts with and without inflammation were common.

Alvin A. *et al.* (1992) studied Ultrastructural and transmission evidence of *Sarcocystis cruzi* associated with myositis in cattle. All samples collected from 19 bovine carcasses that were condemned for EM contained small disseminated lesions of variable intensity from one organ to another. The intensity and prevalence of cysts were highest in myocardial and esophageal muscles.

Silva *et al.* (2002) conducted an experiment for the detection of bovine *sarcocystis cruzi* cysts in cardiac muscles.The results of the microscopic examination indicated that the 500 slides made from 50 hearts were all positive for the presence of sarcocystis.

Latif *et al.* (2013) investigated the prevalence of muscular sarcocystosis in cattle andwater buffaloes. Various muscle samples were collected including heart of 102 cattle and 18 water buffaloes. Out of 120 animals examined, 49 (40.8%) harboured the microscopic type of Sarcocystis spp. In cattle, the organs highly infected were the skeletal muscles and diaphragm (27% each), followed by tongue and esophagus (24.3% each), and the heart (8%). In water buffaloes, the heart was most often infected (66.7%), followed by the oesophagus (50%) and skeletal muscle (33.3%); no sarcocysts were detected in the tongue and diaphragm.

Fazly *et al.* (2015) studied Sarcocystis infection in Kedah-Kelantan crossbred cattle and Murrah Buffalo slaughtered in abattoir in Perak, Malaysia.In their study out of the 86 animals, 19 (22.0%) animals were infected with Sarcocystis spp. 22.5% (16 of 71) of cattle and 20.0% (3 of 15) of buffalo were diagnosed with sarcocystis infection. Microscopically, 10.5% (9 of 86) of heart muscle samples were positive by histological examination.

JyothiSree *et al.* (2016) reported Prevalence and microscopic studies of *Sarcocystis* infection in naturally infected water buffaloes (*Bubalus bubalis*) of Andhra Pradesh. total of 137 buffalo carcasses were screened grossly and microscopically organ wise viz., esophagus, tongue, heart, diaphragm and

intercostal muscles Out of 137 animals, 91 were infected. The percentage of sarcocyst infection was 29.92% in heart.

Mounika *et al.* (2017) studied prevalence and histomorphology of Sarcocystis species infecting cattle in Andhra Pradesh, and they observed thin walled sarcocysts in the cardiac muscles along with oesophagus and diaphragm muscles.

Tong-Il Choi *et al.* (2018) Detected and Identified *Sarcocystis cruzi* by Molecular and Ultrastructural Studies Infected Korean Cattle (*Bos taurus coreanae*). 210 heart samples were collected from Korean native cattle at an abattoir and Sarcocysts were detected form 31 specimens (14.8%). The cysts were all in the muscle fibers. The sarcoplasms of the myocardia presented with numerous bradyzoites enclosed in the thin and smooth host cell wall.

(5). MISCELLANEOUS

5. (a). Hypertrophy of ventricular wall

James *et al.* (1986) studied Locoweed (Oxytropis sericea) poisoning and congestive heart failure in cattle. They observed Locoweed (*Oxytropis sericea*), when fed to calves at high elevations, increased the prevalence and severity of congestive heart failure. At necropsy of affected calves, there were right ventricular hypertrophy and dilation, subcutaneous edema, hydrothorax.

Furuoka *et al.* (2001) studied Hereditary Dilated Cardiomyopathy in Holstein-Friesian Cattle in Japan. Histologically, hypertrophy and vacuolation of the cardiac muscle fibres and severe fibrosis were noted.

Christopher *et al*. (2012) obseved the cases of right ventricular hypertrophy with heart failure in Holstein heifers. At necropsy, 10 cattle had marked hypertrophy of right ventricular myocardium, dilated right atria, right ventricles, and pulmonary trunks.

Neary *et al.* (2015) studied Right-sided congestive heart failure in North American feedlot cattle. In post-mortem findings they observed brisket and ventral edema, hydroperitoneum, hydrothorax, hepatomegaly and chronic passive congestion, intestinal and mesenteric edema, hydropericardium, right-ventricular hypertrophy.

5. (b).Atrophy:-

Harold *et al. (*1988) studied pathology of the cardiac conduction system in myotonic dystrophy. They observed that histopathological lesions of the cardiac conduction system were fibrosis, fatty infiltration and atrophy. Atrophy was prominent in the AV bundle and bundle branches.

Ho-Seong *et al.* (2006) studied balantidiasis in the gastric lymph nodes of Barbary sheep. During necropsy finding they observed severe serous atrophy of the fat tissues of the coronary and left ventricular grooves, resulting in the transformation of the fat to a gelatinous material.

Raji *et al* (2010) observed Pathological conditions and lesions in slaughtered cattle. In the study total 7812 cattle were examined. The examination involved visual examination of carcasses and organs including hearts. From the total heart lesions they observed fat atrophy of the heart 30.3%.

Christopher *et al.* (2012) studied Right ventricular hypertrophy with heart failure in Holstein heifers at elevation of 1,600 meters. Ten cattle with typical clinical signs were examined postmortem. At necropsy, the 10 cattle had marked serous atrophy of fat of epicardial fat.

B. Pathological condition of blood vessels:-

1. Onchocercosis:-

Patnaik B. (1962) studied incidence of aortic onchocerciasis in adult cattle.He examined 167 aorta and found 165 aorta positive with onchocerca. In his study affected aortas are invariably sclerosed, and the aortic nodules containing worms and microfilariae. He also observed degeneration and calcification of aorta.

Cheema and Ivoghli (1978) studied bovine Onchocerciasis caused by *Onchocerca armillata* and *O.gutturosa.* They observed 28 % cases of cattle aorta affected with *Onchocerca armillata.* Gross lesions included parasitic tunnels, nodules, roughening and calcification in the aortic walls. They observed aortic walls were thicker and intimal surfaces uneven because of numerous tortuous tunnels and nodules. Under light microscopes parasites were found to be covered with thin layers of medial and intimal tissues.

Javed Rashid and Khan (1991) studied incidence and pathology of Onchocerciasis in cattle and buffalo in Pakistan. They studied on 1200 cattle and 1050 buffaloes slaughtered at Lahore abattoirs. They observed rate of infection in cattle and buffalo was 92 % and 2% respectively. The lesions were only observed in aorta.

Joseph Neary *et al.* (2010) studied *Onchocerca armillata* contains the endosymbiotic bacterium *Wolbachia* and elicits a limited inflammatory response.During their study the aortic arch was examined for evidence of *O. armillata* adult worms and 49 positive specimens were collected. In mild infection aortic tunica appeared smooth, in heavier infection thick and less elastic aortic wall were observed. Microscopically lesions were found in tunica intima, media and in heavy infection tunica adventitia was also affected. Cellular responses with no to few inflammatory cells were observed.

Zafer *et al.* (2012) studied parasitic aortitis due to *Onchocerca armillata* in slaughtered cattle. In their study total of 400 aorta suspected with parasitic aortitis was investigated. Macroscopically 32 of 400 (8%), both macroscopically and microscopically 43 (10.75%) cattle were diagnosed as aortic onchocercosis.

2. Calcification and mineralization of Aorta:

Charles C. Capen *et al* (1966) studied pathology of hypervitaminosis D in cattle. In their study total 26 animals were included from which 15 cows received 30 million unit of vitamin D in dividing dose for 7, 10, 21 and 30 days along with regular diet. In necropsy examination they observed cardiovascular mineralization, large arteries were firm, inelastic and asymmetrical in circumference due to mineralization. In arteries transverse ridges projected

into the lumen with hard and irregular surface. Microscopically deposits of irregular plates of mineral crystals were present in tunica media and intima.

Kenneth Dale Wiggers (1971) studied atherosclerosis in domestic and wild ruminants. In the study Aortas from six 6-month-old calves, 11 aged ewes, 10 goats (five males and five females), 35 wild native White-tailed deer (24 males and 11 females) and nine wild Alaskan caribou (six males and three females) were examined grossly and histologically Calcium deposition was evidenced by Von Kossa calcium stain was observed in aortas of one milk-fed calf, eight ewes, one goat, three of six deer examined and two caribou. Large deposit of calcium was observed in tunica media.

3. Atherosclerosis:

Likar *et al* (1966) observed moderate gross lesions of bovine aorta. Aortas with severe gross lesions showed an increase of total cholesterol. They observed spontaneous gross lesions in 53.57% of aortas from 200 Massachusetts cows.

Thomas *et al* (1968) observed ultrastructural evidence of aortic lesions in miniature swine after cholesterol supplemented diet; the lesions were, focal accumulation of smooth muscle cells.These ultrastructural lesions were described as a preproliferative phase of atherosclerosis.

Ratcliffe *et al* (1970),described histological features of atherosclerosis during examination of cerebral arteries of three age-groups in swine,as (1) focal reorientation and proliferation of smooth muscle cells (SMCs) accompanied by apparent medial degeneration, (2) fragmentation and replication of the internal elastic lamina, (3) apparent movement of smooth muscle cells through fragmented internal elastic lamina and proliferation to form stenosing lesions and (4) elastic and collagen fiber formation to enclose the smooth muscle cells.

Kenneth Dale Wiggers (1971) studied atherosclerosis in domestic and wild ruminants. In the study Aortas from six 6-month-old calves, 11 aged ewes, 10 goats (five males and five females), 35 wild native White-tailed deer (24 males and 11 females) and nine wild Alaskan caribou (six males and three females) were examined grossly and histologically. Sudanophilic

streaking was noted grossly in the aortas of three of four milk calves.They observed intimal thickening in most of the young animals of their experiment. Calcium deposition was evidenced by Von Kossa calcium stain. Many histological similarities were seen between domestic and wild ruminants. Plaque formation, fatty streaks, lipid and calcium deposition, internal elastic membrane duplication and disruption and smooth muscle cells in the intima are quite similar to those observed in man and other animals.

MATERIAL AND METHODS

COLLECTION OF SAMPLES

For the proposed investigation, samples of the heart of buffaloes (*Bubalus bubalis*) irrespective of age ,sex and breed were collected from slaughter houses of Alwar,Bikaner,Jaipur and Siker and adjoining areas of these districts.

The tissue specimens were also collected from the carcasses of buffaloes submitted to the department of Veterinary Pathology ,College of Veterinary and Animal Sciences, Bikaner for post mortem examination.The samples received from the field veterinarians at the department of Veterinary Pathology were also included in this study.

During post-mortem examination, the samples were thoroughly examined grossly for alteration in morphology in terms of shape, size, color, consistency, location and presence of cysts,tumours and abcesses etc. lesions in individual parts of cardiovascular system.

In the study,total 473 specimens of Cardiovascular system of buffaloes were examined 163 specimens (148 samples of heart and 15 samples of blood vessels in cluding major arteries,aorta and coronary artery) showing gross lesions were used for further histopathological examination.

Histopathology

Following collection, all the samples were properly preserved in 10 percent formalin after cutting the affected parts. The part of affected tissues measured 2-5 mm thickness and presenting the lesions with normal tissue, were used for fixation and further histopathological examination

For histopathological examination, processing of tissues was done by paraffin embedding using acetone and benzene technique (Lille,1965). The section of 4-6 micron thickness were cut and stained with routine staining methods by hematoxylin and eosin. As far as possible, results were recorded by gross observations and microphotographs.

Processing of tissues

1. All the tissue samples sized at 1cm x 0.5 cm were trimmed properly and fixed for 24 hours or more. To remove the fixative, the tissues were kept in running tap water for overnight.
2. The tissues were dehydrated in ascending grades of alcohol using 50%, 70%, 80%, 95% and in absolute alcohol. The tissues were changed at every 1 hour interval.
3. The tissues were cleared by two changes in chloroform, one and half an hour for each.
4. The tissues were embedded with molten paraffin wax at 56^0 C : 2 changes one and half an hour for each.

Preparation of stains

1.) Preparation of Harris hematoxylin solution

Hematoxylin crystals 5.0 g

Alcohol, 100% 50.0ml

Ammonium or potassium alum 100.0g

Distilled water 1000.0ml

Mercuric oxide (red) 2.5 g

The hematoxylin dissolved in the alcohol and alum in the water by the aid of heat.The two solutions were removed from heat and thoroughly mixed and boiled as rapidly as possible.After removal from heat mercuric oxide was added slowly.It was reheated to simmer until it became dark purple,removed from heat immediately and plunged the vessel in to a basin of cold water until cooled.Just before using ,2-4ml of glacial acetic acid was added per 100ml of solution to increase the precision of the nuclear stain. The prepated solution was filtered before using.

Preparation of stock solution

a) 1% stock alcohol eosin

Eosin Y, water solution 1.0g

Distilled water 20.0ml

Dissolved and adding

Alcohol, 95% 80.0 ml

b) Working eosin solution

Eosin stock solution 1 part

Alcohol, 80.0% 1 part

Just before use 0.5 ml of glacial acetic acid was added to each 100ml of stain and stirred.

Routine hematoxylin and eosin staining procedure

1. The sectioned cardiovascular tissues were deparaffinized in 3 changes of xylene (3 minutes in each).
2. Then the sectioned tissues were dehydrated through descending grades of alcohol. (3 minutes in absolute alcohol,3 minutes in each; 95% alcohol, for 2 minutes; 80% alcohol for 2 minutes; 70 % alcohol for 2 minutes) followed by tap water washing for 5 minutes).
3. The sectioned tissues were stained with Harris hematoxylin for 15 minutes.
4. The were washed in running tap water for 10-15 minutes.
5. After washing the sections were differentiated in acid alcohol: 2 to 4 dips (1 part HCL and 99 part 70% alcohol).
6. Then they were washed in tap water for 5 minutes followed by two to four dips in ammonium water until sections were bright blue.
7. They were stained with eosin for 1 minute.
8. They were differentiated and dehydrated in alcohol: 95% alcohol: 3 changes, 2-4 dips each; absolute alcohol: 3 changes 2-3 minutes for each.
9. They were cleaned in xylene: 3 changes (5 minutes for each).
10. Finally the sections were mounted with cover slips using DPX.

RESULT

In the present investigation total number of 473 specimens of cardiovascular system of buffalo were examined irrespective of age groups, sex and breeds. Out of these 473 specimens, 163 specimens (148 specimens of heart and 15 specimens of Blood vessels including aorta, coronary arteries and major arteries) which were suspected for abnormalities were further processed for histopathological examination.The overall incidence of pathological conditions of cardiovascular system was 34.46 per cent. These conditions were recorded as follows:

Table 1: Pathological conditions involving heart.

S.N.	Name of lesion	No. of Cases	Percentage
1.1	**Inflammatory Conditions of Heart**	**17**	**11.48**
1.1(a)	Pericarditis	9	6.08
1.1(b)	Myocarditis	4	2.70
1.1(c)	Vegetative Endocarditis	4	2.70
1.2	**Degenerative Changes**	**16**	**10.81**
1.2(a)	Myocardial Necrosis	4	2.70
1.2(b)	Degeneration and Separation of Muscle Fibers	5	3.37
1.2(c)	Coagulative Necrosis	2	1.35
1.2(d)	Myocardial Fibrosis	3	2.02
1.2(e)	Endocardial Fibrosis	2	1.35
1.3	**Circulatory Disturbances**	**44**	**29.72**
1.3(a)	Hydropericardium	2	1.35
1.3(b)	Pericardial Congestion	10	6.75
1.3(c)	Epicardial Haemorrhage	8	5.40
1.3(d)	Epicardial Congestion	5	3.37
1.3(e)	Myocardial Haemorrhage	12	8.10
1.3(f)	Myocardial Congestion	6	4.05
1.3(g)	Myocardial Edema	1	0.67
1.4	**Parasitic Involvement**	**56**	37.83
1.4(a)	Sarcosporidiosis	56	37.83
1.5	**Miscellaneous**	**15**	**10.13**
1.5(a)	Hypertrophy of Ventricular wall	2	1.35
1.5(b)	Serous Atrophy of Epicardial Fat Cells	13	8.78
	Total	**148**	

Table 2: Pathological conditions involving Blood vessels

S. No.	Name of lesion	No. of Cases	Percentage
1.	Parasitic involvment (Onchocercosis)	4	26.66
2.	Calcification and mineralization	5	33.33
3.	Atherosclerosis	6	40
	Total	**15**	

1.1. Inflammatory Conditions of Heart:-

1.1 (a).Pericarditis:-

This condition was observed in 6.08 per cent cases.

Grossly there were necrotizing haemorrhagic pericarditis observed (fig. 1).

Microscopically, there were marked infiltration of mononuclear cells such as lymphocytes and monocytes along with diffused haemorrhage (fig.5,7) . Some of the cases of traumatic pericarditis were also reported. Grossly there were large quantity of turbid, foul smelling pericardial fluid observed. There were thickening of pericardial sac, the pericardium showed typical “bread and butter” appearance (fig. 2,3).

Microscopically, thickening of pericardium due to accumulation of fibrinous inflammatory exudates, and there were marked infilteration of leukocytes observed (fig.6).

1.1(b) Myocarditis:-

This condition was observed in 2.70 per cent cases.

Microscopically, it was seen aggregates of lymphocytes in between degenerative myofibrils. (fig.9).Some of the sections showed perivascular infiltration around the coronary artery.(fig.8).

1.1(c) Vegetative Endocarditis:-

This condition was observed in 2.70 per cent cases.

Grossly, the heart showed cauliflower like irregular, yellow vegetative growth in left atrio-ventricular valves (fig. 11).

Microscopically the sections showed atrio-ventricular valve with vegetation containing inflammatory cells, and bacterial clumps (fig. 12) .

1.2. Degenerative Changes:-

1.2 (a) Myocardial Necrosis:-

This condition was observed in 2.70 per cent cases.

Grossly, some of the heart showed pale, irregular necrotic areas over the surface. (fig.19).

Microscopically, there was degeneration and necrosis of the myofibers with loss of striations. In some of these cells the nuclei were disappeared (fig.13).

1.2. (b) Degeneration and Separation of Muscle Fibers :-

This condition was observed in 3.37 per cent cases.

Microscopically, muscles fibers of myocardium showed degeneration and separation. Some of the sections also showed marked haemorrhages along with separated myofibrils (fig. 14,15).

1.2. (c) Coagulative Necrosis:-

This condition was observed in 1.35 per cent cases.

Grossly, diffused pale areas of necrosis were observed in the ventricular wall (fig. 16).

Microscopically, myocardial sections showed coagulation of myocardial fibers with damaged architectural details. Sections showed lighter stained tissue containing no nuclei (fig.16).

1.2 (d) Myocardial Fibrosis:-

This condition was observed in 2.02 per cent cases.

Microscopically the sections revealed severe fibrosis of the myocardial cells.

Abundance of fibroblasts were observed in the myocardial sections with mild infiltration of mononuclear cells along with necrosis.(fig.20).

1.2 (e) Endocardial Fibrosis:-

This condition was observed in 1.35 per cent cases.

Microscopically, sections of endocardium showed marked fibrosis and deposition of fibroblasts along with mild degree of infiltration. Endocardial thickening was due to deposition of fibrous connective tissue (fig. 21).

1.3. Circulatory Disturbances:-

1.3 (a) Hydropericardium:-

This condition was observed in 1.35 per cent cases.

Only gross conditions were obsereved. Pericardial sac was filled with clear transudate (fig. 22).

1.3 (b) Pericardial Congestion :-

This condition was observed in 6.75 per cent cases. Microscopically sections of pericardium showed diffused and marked congestion of blood vessels along with severe infiltration of lymphocytes and monocytes (fig.23).

1.3 (c) Epicardial Haemorrhage:-

This condition was observed in 5.40 per cent cases.

Grossly the epicardial surface of hearts showed varying degree of diffused haemorrhages (fig.25), some sections showed pin point haemorrhages (fig.24).

Microscopic study of epicardial surface revealed diffused haemorrhages. RBCs were observed between the fat cells of epicardium (fig. 26).

1.3 (d) Epicardial Congestion:-

This condition was observed in 3.37% cases.

Grossly there were blood vessels engorged with blood (fig.28).

Microscopically, epicardial blood vessels showed marked congestion (fig.29).

1.3 (e) Myocardial Haemorrhage:-

This condition was observed in 8.10 per cent cases.

Microscopically, haemorrhages were observed in between muscle fibers RBCs were found scattered in the micocardial sections. Some of the muscle cells also showed necrosis and degeneration.(fig.30).

1.3 (f) Myocardial Congestion:-

This condition was observed in 4.05 per cent cases.

Microscopically, sections of myocardium showed diffused and marked congestion of blood vessels, (fig.31).

1.3 (g) Myocardial Edema :-

This condition was observed in only 01 (0.67per cent) case.

Microscopically, pink colored edematous fluid was observed in between the muscle fibers the space between muscle fibers increased due to presence of edematous fluid.(fig.32).

1.4. Parasitic Involvement:-

1.4. (a) Sarcosporidiosis :-

This condition was recorded in 37.83 per cent cases. Grossly, no evidence of sarcocysts in heart muscles.

Microscopically, plenty of sarcocyts were observed throughout muscle bundles. Among the cysts, varied sizes and shapes round, oval and elliptical cysts were noticed (fig.33). Some of the cysts were found ruptured and their bradizoites were found scattered in between the myocardial cells (fig.34). The adjacent muscle parenchyma showed degenerative changes and necrosis in some placs (fig.33).

1.5. Miscellaneous:-

1.5 (a) Hypertrophy of Ventricular wall:-

This condition was observed in 1.35 per cent cases.

Grossly, the wall of ventricles were abnormally thickened with no lesions of necrosis or fibrosis. The lumen of ventricles were extremely narrow due to hypertrophy, (fig.36,37).

Microscopically, the sections showed hypertrophied myocardial cells with infiltration of mononuclear cells and degenerated muscle fibres. (fig. 38).

1.5.(b) Serous Atrophy of Epicardial Fat Cells:-

This condition was observed in 2.02% cases.

Grossly, epicardial fat appeared gelatinous as a result of serous atrophy.(fig.39).

Microscopically, the epicardial fat cells were observed atrophied and serous fluid was present around the fat cells (fig.40).

B.Pathological conditions of blood vessels :

In the present study total 15 blood vessels (aorta and major arteries) were collected for histopathological study, and following conditions were noticed:

Parasitic involvement (Onchocercosis):

This condition was observed in 26.66 per cent Cases.

Grossly the affected vessel walls in general, thickened, crusty in consistency and lost flexibility. The Affected areas of tunica intima revealed tough and an irregular surface. The nodules of varying sizes, especially in areas close to the bifurcation, projecting to the vessel lumen.The nodules were hard in consistency with bright and white surface (fig.46,47).

Microscopically, the parasitic tunnels were present without inflammatory cell.Tunnels had round or ellipsoidal cavities containing one or more intact parasitic sections.Parasitic cyst demarcated by a thin connective tissue without any inflammatory reaction.Some of the areas showed degeneration near to the parasitic tunnels (fig.48,49).

Calcification and mineralization

This condition was observed in 33.33 per cent cases.

Grossly, blood vessel wall showed thickened diffused and localized irregular areas in the wall. Wall of the blood vessels had numerous large, irregularly-shaped plaques projecting into the lumen. The plaques had a rough corrugated surface, which were hard in consistency (fig.41,42).

Microscopically, the tunica intima of blood vessels showed diffused areas of dark calcification.(fig.43).

Atherosclerosis :

This condition was observed in 33.33 per cent cases.

Grossly, the aorta showed mild irregular and diffused atheromatous lipid plaques (fig.50).

Microscopically, the cross sections of aorta showed presence of large atheroma and diffused cholesterol clefts in tunica intima.(fig.51). Atherosclerosis of coronary artery was also observed, which revealed presence of narrow lumen with deposition of large atherosclerotic plaque. (fig.45).

1.1 Inflammatory Conditions of Heart

Occurrence of inflammatory conditions was recorded 11.48 per cent in the present study.

Causes of inflammatory conditions in the present study may include foreign bodies as observed in traumatic pericarditis, infectious conditions such as bacteria, viruses and certain parasites.

1.1(a) Pericarditis

This condition was recorded in 6.08 per cent cases in present study are in close approximation to the incidence recorded by Raji *et al.* (2009) as 6.3 per cent. Gross findings of traumatic pericarditis presence of large quantities of turbid, foul-smelling peritoneal fluid agree well with Faisal *et al.* (2013).

The microscopic findings of pericarditis such as severe infiltration of mononuclear cells in the pericardium correspond well with the findings of Faisal *et al.* (2013). Microscopic findings of traumatic pericarditis such as marked thickening of pericardium along with infiltration of polymorphonuclear cells and mononuclear cells are in accordance with Faisal *et al.* (2013).

On the basis of the present study, it is reasonable to conclude that pericarditis might be caused by many etiological factors. It may be because of bacterial infection such as Haemorrhagic septicemia (Alwis, 1984) and *E. coli* infection (Vegad and Katiyar, 2001). It may be because of penetration of foreign body in case of traumatic pericarditis (Cevik *et al,* 2010).

1.1 (b) Myocarditis

This condition was recorded in 2.70 per cent cases in present study. The microscopic findings such as aggregates of mononuclear cells in between degenerative cardiac muscle fibers are in accordance with Corbeil *et al.* (2009) and Amir *et al* (2014).

Myocarditis in the present study may be caused by some infectious agents such as foot and mouth disease in calves (Amir *et al,* 2014), malignant

catarrhal fever (Costa *et al,* 2009) and trypanosomiasis (Damayanti *et al,*1994).

1.1 (c) Vegetative Endocarditis:-

This condition was recorded in 2.70 per cent cases in present study are in close approximation to the incidence recorded by Buczinski *et al* (2010) as 6.60 per cent. The gross findings of vegetative endocarditis were cauliflower like irregular, yellow vegetative growth in left atrio-ventricular valves are in close approximation to the observation reported by Adamu *et al* (2014).

The microscopic findings showing vegetation of atrio-ventricular valve with inflammatory cells, and bacterial clumps are in close conformity with the earlier report of Adamu *et al* (2014).

This condition may occur due to etiological factors such as bacteria Bartonella (Maillard *et al,* 2007) and *Staphylococcus aureus* (Waschburger *et al.* 2012).

1.2 Degenerative Changes:-

Occurrence of degenerative changes was recorded 10.81 per cent in the present study. The causes of degenerative changes in the present study may include bacteria, viruses some other conditions such as cardiomyopathy, toxicity of substances and due to deficiency of nutrients such as selenium deficiency.

1.2 (a) Myocardial Necrosis

In the present study, this condition was recorded in 2.70 per cent cases. The gross findings such as pale, irregular necrotic areas over the heart surface in accordance with Gunes *et al* (2005)

The microscopic findings such as degeneration and necrosis of the myofibers with loss of striations. In some of these cells the nuclei were disappeared are in accordance with Kennedy and Rice (1988) and Sharma and Shrivastava (2002).

In the present study, this condition may occur due to various etiological factor such as *E.coli* infection (Sharma and Shrivastava, 2002), (Asopa, 2012). Myocardial necrosis may also occur due to endotoxaemia (Nabi *et al*,

2013), toxicosis (Van Vleet *et al,*1986) and due to foot and mouth disease in calves (Islam *et al,*2017). Other causes may include selenium deficiency (Carlos, 2018).

1.2 (b) Degeneration and Separation of Muscle Fibers:-

In the present study, this condition was recorded in 3.37 per cent cases. In the microscopic findings, degenerated and separated muscle fibers. In some cases degeneration and separation along with marked haemorrhages are in close conformity with the Asopa (2012), Mehra (2013) and Adamu *et al* (2014).

In the present study this condition may occur due to various etiological factor such as infectious disease including foot and mouth disease (Gunes *et al,* 2005), colibacillosis (Asopa, 2012),(Mehra,2013).

1.2(c) Coagulative Necrosis

In the present study, this condition was recorded in 1.35 per cent cases. Gross findings such as diffused pale areas of necrosis of ventricular wall were observed in the study.

Microscopic findings, coagulation of myocardial fibers with damaged architectural details along with lighter stained tissue containing no nuclei are in close conformity with the earlier report of Alsaad (2018).

This condition may occur due to etiological factor like dilated cardiomyopathy (Narth *et al*, 2016) and foot and mouth disease (Alsaad, 2018).

1.2(d) Myocardial Fibrosis

In the present study, this condition was recorded in 2.02 per cent cases. Microscopic findings such as presence of fibrosis and necrosed areas of myocardium are in close conformity with the earlier report of Carlos *et al* (2018).

In the present study this condition my occur due to various etiological factors such as deficiency of selenium (Carlos *et al*, 2018), this may also occur due to dilated cardiomyopathy (Leifsson and Agerholm, 2004) and (Marta *et al*, 2010).

Endocardial Fibrosis:-

In the present study this condition was recorded in 1.35 per cent cases. In the present study, microscopic findings such as marked fibrosis with deposition of fibroblasts with mild degree of infiltration in the endocardial sections, the endocardial section was enlarged due to deposition of fibroblast cells are in accordance with Cushing (2013).

1.3 Circulatory Disturbances

Occurrence of circulatory changes was recorded 29.72 per cent in the present study. The causes of circulatory disturbances may include Parasites, bacteria, viruses and it may also occur due to impairment in protein metabolism.

Hydropericardium

In the present study this condition was recorded in 1.35 per cent cases.

Gross findings such as presence of moderate quantity of transparent fluid in the pericardial sac are in accordance with Damayanti *et al* (1994) because of occurrence of *Trypanosoma evansi* and Neary *et al* (2016) because of right side heart failure.

Pericardial congestion

In the present study this condition was recorded in 6.75 per cent cases. Microscopic findings of pericardium showing diffused and marked congestion of blood vessels along with severe infiltration of lymphocytes and monocytes are in close conformity with the earlier report of Gupta (2012).

This condition may occur due to etiological factor like coloibacillosis (Gupta, 2012), (Asopa, 2012) and (Mehra, 2013).

Epicardial Haemorrhage

In the present study this condition was recorded in 5.40 per cent cases. Higher incidence was recorded by Teankam *et al* (2000) as 23.07 per cent because of occurrence of malignant catarrhal fever.

The gross findings such as severe haemorrhages over the pericardial surface, agreed well with the earlier report of Bratanich *et al* (2011) and Mehra (2013) and Oliveira *et al.* (2014).

Microscopic findings such as haemorrhages between epicardial cells correspond well with the findings of Hristov *et al* (2017).

Epicardial haemorrhages in the present study may be caused by some etiological factors. It may be due to various infectious diseases such as coloibacillosis (Asopa 2012) botulism (Camara *et al.* 2014), haemorrhagic septicemia (Ahmed *et al* 2014).

Epicardial Congestion

In the present study, this condition was recorded in 3.37 per cent cases. The gross finding such as engorged blood vessels of epicardium agreed well with the earlier report of Gupta (2012) and Mehra (2013).

The microscopic findings, congested blood vessels and severe infiltration of mononuclear cells were in accordance with Gupta (2012).

In the present study epicardial congestion may be due to infectious agent like coloibacillosis (Sharma and Shrivastava, 2002), (Gupta, 2012) and (Asopa, 2012).

Myocardial Haemorrhage

In the present study this condition was recorded in 8.10 per cent cases. Higher incidences were recorded by Teankam *et al* (2000) as 30.76 per cent.

The microscopic findings such as haemorrhages along with degenerated muscle fibers are in accordance with Singh *et al.* (1996), Asopa (2012) and Mehra (2013).

In the present study, myocardial haemorrhage may be due to various infectious agents such as Malignant catarrhal fever (Teankam *et al*, 2000), foot-and-mouth disease (Gunes *et al,* 2005), myocardial haemorrhages may also occur due to coloibacillosis (Gupta,2012) and (Asopa,2012).

Myocardial Congestion

In the present study, this condition was recorded in 4.05 per cent cases. The microscopic findings such as congested blood vessels and some of the myocardial muscle fibers were separated are in close conformity with the earlier report of Gupta (2012).

In the present study, myocardial congestion may be due to infectious agent like coloibacillosis (Sharma and Shrivastava,2002),(Gupta,2012) and (Asopa, 2012).

Myocardial edema

In the present study this condition was recorded in only 01 (0.67 per cent) case.

The microscopic findings such as presence of pink colored edematous fluid in between the muscle fibers are in accordance with Gunes *et al* (2005) and Gupta (2012).

In the present study myocardial edema may be due to some infectious agents such as *E. coli* (Azmi and Jha, 1994) and in case of foot and mouth disease (Gunes *et al*, 2005).

Parasitic Involvement

In present study only sarcosporidiosis was observed in the cardiac muscles as *sarcocystis* are most commonly found in domestic ruminants and it mostly affect the musculature of the animals including cardiac muscles.

In the present study this condition was recorded in 37.83 per cent cases. Higher incidence was recorded by Saha *et al* (1983) as 54.66 per cent and lower incidence was recorded by Jyotishree *et al* (2016) as 29.92 per cent.

The microscopic findings such as presence of multiple cysts were observed in the myocardium with infiltration of mononuclear cells are in accordance with Bratberg and Landsverk (1980), Collery and Weavers (1981).

Miscellaneous

In the present study miscellaneous conditions were observed in 10.13 per cent cases. These conditions were hypertrophy of ventricular wall and serous atrophy of epicardial fat cells.

Hypertrophy of Ventricular wall

In the present study this condition was recorded in 1.35 per cent cases. The gross findings such as abnormal thickening of ventricular wall are in accordance with Christopher *et al*. (2007) and Neary *et al* (2015).

The microscopic findings such as hypertrophy of ventricular wall and infiltration of mononuclear cells were in accordance with Furuoka *et al* (2001) and Christopher *et al*. (2007).

In the present study, ventricular hypertrophy may occur due to some etiological factors such as poisoning of Locoweed (*Oxytropis sericea*), (James *et al*, 1986). Hypertrophy may also occur due to heart failure (Christopher *et al,* 2007) and (Neary *et al,* 2015).

Serous Atrophy of Epicardial Fat Cells

In the present study, this condition was recorded in 8.78 per cent cases. Higher incidences were recorded by Raji *et al* (2010) as 30.3 per cent. The gross finding such as epicardial fat appeared as gelatinous mass is in close approximation to the observations reported by Cho *et al* (2006).

In the microscopic findings, epicardial fat cells were observed atrophied and serous fluid was present around the fat cells are in close conformity with the earlier report of Nguyen *et al. (*1988).

We can conclude that in the present study serous atrophy of epicardial fat cells may occur due to myotonic dystrophy (Nguyen *et al.*1988),some infectious diseases like balantidiasis (Cho *et al.*2006) and in case of heart failure heart (Christopher *et a.*2012)

Pathological conditions involving Blood vessels:

Parasitic involment (Onchocercosis):-

In the present study presence of aortic onchocercosis was recorded in 26.66 percents (from total blood vessels collected). Incidence recorded by Cheema and Ivoghli (1978) slightly higher as 28 per cent.

The gross finding such as rough parasitic nodules covered with connective tissue, extending outward the aorta serosa are in agreement with the earlier report of Javed and Khan (1991). Parasitic cysts were located close to each other which were compatible with findings of Zafer *et al* (2013).

The microscopic findings such as presence of one or more round to irregular parasitic tunnels and space between parasites and thin lining are in accordance with the earlier report of Cheema and Ivoghali (1978).

Calcification and mineralization

In the present study this condition was recorded in 33.33 per cent cases(from total blood vessels collected).Gross findings such as thickened diffused and localized irregular areas in the wall of blood vessels along with numerous large, irregularly-shaped plaques projecting into the lumen are in agree with the earlier reports of Charles *et al* (1966).

Microscopic findings such as the tunica of blood vessels showed diffused areas of dark colored calcification in the tunica of blood vessels are in accordance with the earlier report of Kenneth (1971).

We can conclude that in the present study calcification and mineralization of blood vessels may occur due to hypervitaminosis D (Charles *et al*,1966).

Atherosclerosis:

In the persent study this condition was recorded in 40 per cent cases (from total blood vessels collected). A higher incidence was recorded by Likar *et al* (1966) as 53.57 per cent.

Gross findings such as irregular and diffused atheromatous lipid plaques in aorta were noticed. Microscopic findings such as presence of large atheroma and diffused cholesterol clefts in tunica intima are in accordance with the earlier findings of Kenneth (1971). Atherosclerosis of coronary artery was also observed, which revealed presence of narrow lumen with deposition of large atherosclerotic plaque.In the present study the causes of atherosclerosis might be high cholesterol level or feeding of cholesterol rich diet (Likar *et al*,1966) and (Thomas *et al*,1968).

SUMMARY

The present study was undertaken to elucidate occurrence and pathology of Cardiovascular system in buffalo in Rajasthan. During the course of study, 473 specimens of Cardiovascular system were examined. Out of these,163 specimens (148 heart samples and 15 samples of blood vessels) showing gross lesions were further processed for histopathological examination. The gross and histopathological changes in the present investigation were identified as:

In heart, haemorrhages, congestion over pericardium, serous atrophy of epicardial fat cells, hypertrophy, vegetative growth on heart valves, pale necrotic diffused areas with coagulative necrosis were noticed grossly. Some of the cases of traumatic pericarditis were also observed both grossly and microscopically. Microscopically, haemorrhages and congestion of the epicardial and myocardial layers were noticed, various inflammatory conditions were observed in pericardium, epicardium, myocardium and endocardium. Some sections showed degeneration and separation of muscle fibers, coagulative necrosis and study of some sections revealed edematous fluid, serous atrophy and hypertrophy. Microscopic findings confirmed presence of *Sarcocystis* in the cardiac muscle fibers with mild infiltration of eosinophils and degeneration of muscle fibers, some of the cyts were found ruptured.

Grossly, some blood vessels showed numerous large and small, irregularly-shaped plaques of calcification, while some specimens of aorta showed presence of *Onchocerca* nodule projecting toward the outer surface. Microscopically, single or multiple onchocerca parasites were observed inside connective tissue lining, dark colored calcium depots were noticed in the tunica intima of blood vessels. Atherosclerosis was also observed in coronary arteries and major aortas.

Almost all pathological conditions found in cardiovascular system were accordance with findings of other researchers mentioned in various texts. In this study, all the histopathological changes were observed and described in detail and photographs were taken, where ever necessary.

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OCCURRENCE AND PATHOLOGY OF VARIOUS CONDITIONS OF CARDIOVASCULAR SYSTEM IN BUFFALO (*Bubalus bubalis*)

M.V.Sc. Thesis

DEPARTMENT OF VETERINARY PATHOLOGY COLLEGE OF VETERINARY AND ANIMAL SCIENCE, RAJASTHAN UNIVERSITY OF VETERINARY AND ANIMAL SCIENCES, BIKANER-334001

Submitted by: **Kshitiz Goswami**
Major Advisor: **Dr. (Prof.) Hemant Dadhich**

ABSTRACT

The present investigation was carried out to study the occurrence and pathology of various conditions of cardiovascular system in buffalo.at different arid regions of Rajasthan. This study revealed that the overall incidence of cardiovascular system 34.46 per cent in buffalo in Rajasthan state.

Total 473 samples of cardiovascular system were examined from which 148 hearts and 15 blood vessesls irrespective of age, breed and sex, were collected and examined and preserved in 10 percent formaline and processed histopathologically by paraffin embedding using acetone and benzene technique.The pathological conditions of heart were categoriezed as follows: inflammatory conditions (11.48 percent),degenerative changes (10.81 percent) , circulatory disturbances (29.72 percent), sarcosporidiosis (37.83 percent) and miscellaneous conditions (10.13 percent) cases. Pathological conditions of blood vessels were classified as follows: calcification and mineralization (33.33 percent), onchocircosis (26.66 percent) and atherosclerosis (40 percent).In the present study the incidence of sarcosporidiosis in the heart were reported maximum as 37.83 percent, while in blood vessels atherosclerosis was most commonly observed finding as 40 per cent.

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वर्तमान परीक्षण राजस्थान के विभिन्न शुष्क क्षेत्रों,भैंस में हृदय वाहिनी तंत्र की विभिन्न अवस्थाओं के आपात एवं व्याधिकी का अध्ययन करने के लिए किया गया। इस अध्ययन से ज्ञात हुआ कि राजस्थान राज्य में भैंस के हृदय वाहिनी तंत्र के कुल आपात 34.46 प्रतिशत है।

हृदय वाहिनी तंत्र के कुल 473 नमूनों की जाँच की गई जिनमें से 148 हृदय तथा 15 रक्त वाहिनियों के नमूनें थे जो कि विभिन्न नस्लों, आयु व लिंग की भैंसों से एकत्र किए, 10 प्रतिशत फार्मेलिन में संरक्षित किए गए तथा उनका एसीटोन व बेंजीन के प्रयोग से पैराफिन एम्बेडिंग तकनीक द्वारा ऊत्तक व्याधिक परीक्षण किया गया है।

हृदय की व्याधिकी अवस्थाओं को निम्न प्रकार से वर्गीकृत किया गया : सूजन की स्थितियाँ (11.48 प्रतिशत), अपक्षयी परिवर्तन (10.81 प्रतिशत), संचार संबंधी व्यवधान (29.72 प्रतिशत), सार्कोस्कोरेडियोसिस (37.83 प्रतिशत) तथा विविध स्थितियाँ (10.13 प्रतिशत) मामलों। रक्त वाहिकाओं की व्याधिकी स्थितियों को निम्नानुसार वर्गीकृत किया गया, कैल्सीफिकेशन और मिनरलाइजेशन (33.33 प्रतिशत), ओन्कोसर्कोसिस (26.66 प्रतिशत) और धमनियों का सख्त होना (40 प्रतिशत), वर्तमान अध्ययन में हृदय में संचार संबंधी व्याधिकी का आपात अधिकतम 29.72 प्रतिशत था जबकि रक्त वाहिकाओं में धमनियों के सख्त होने की स्थिति सबसे अधिक 40 प्रतिशत के रूप में था।

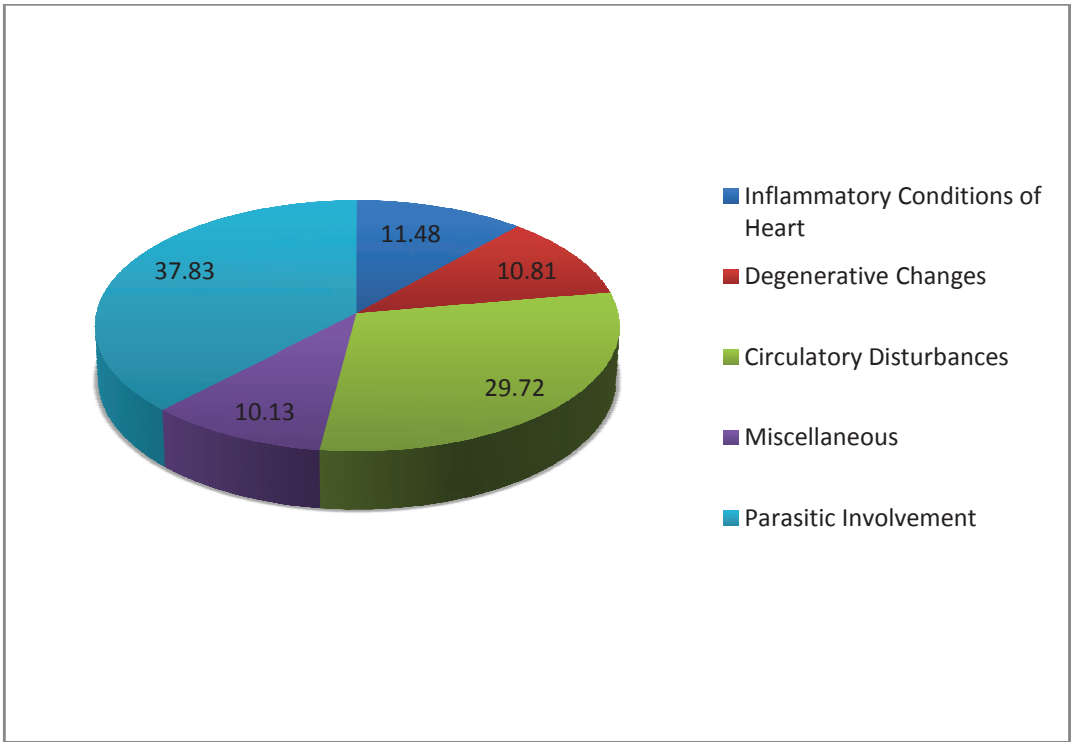

Fig 1 : Pathological conditions involving heart.

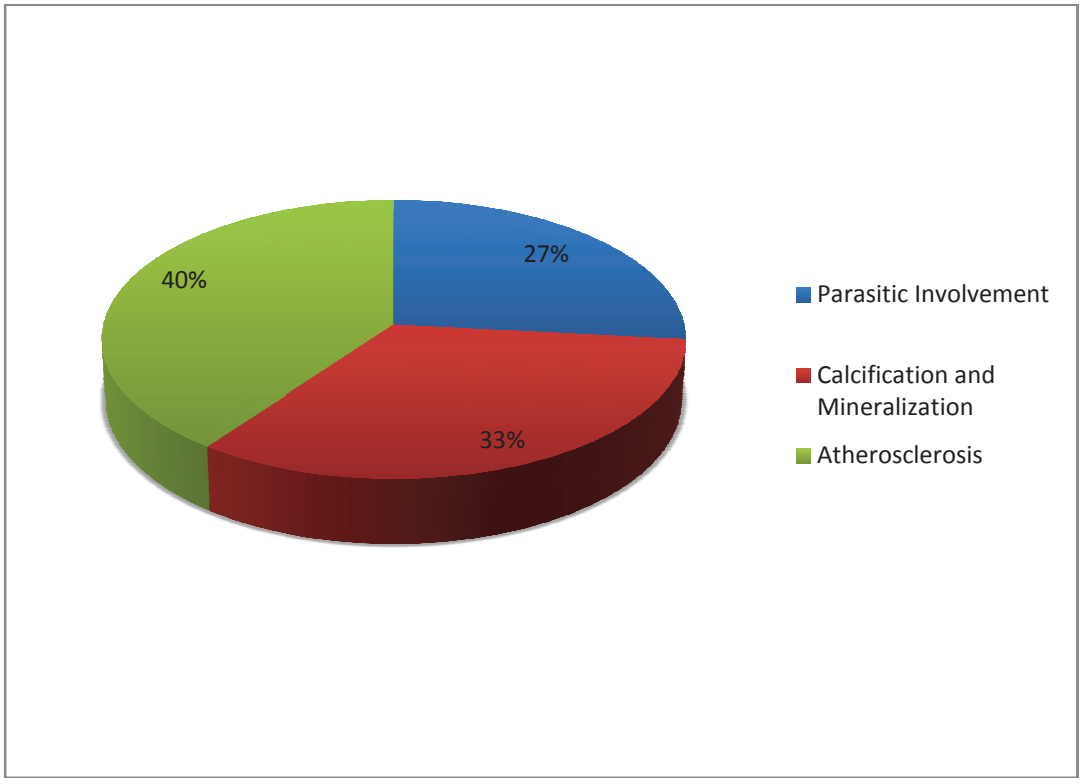

Fig 2 : Pathological conditions involving Blood vessels

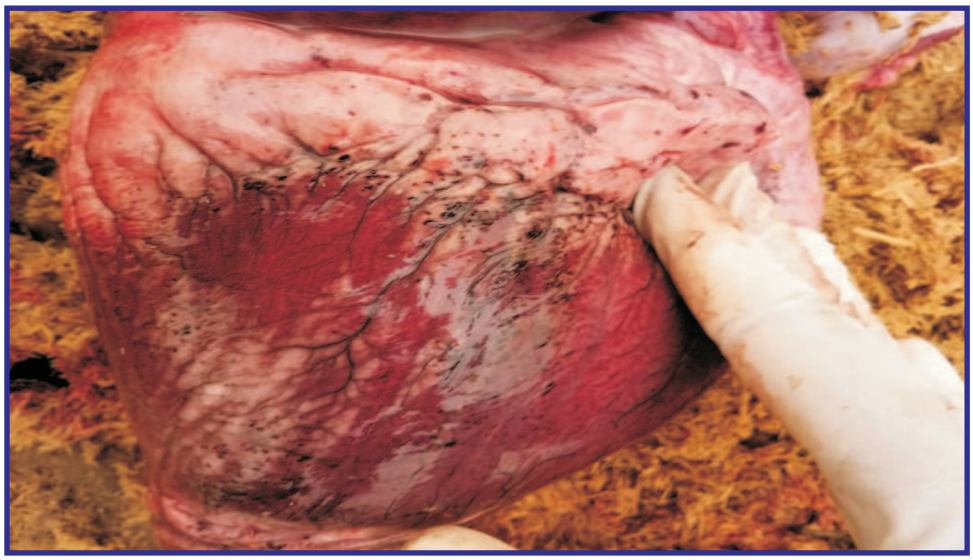

Fig.1 : Gross photograph showing necrotizing haemorrhagic Pericarditis.

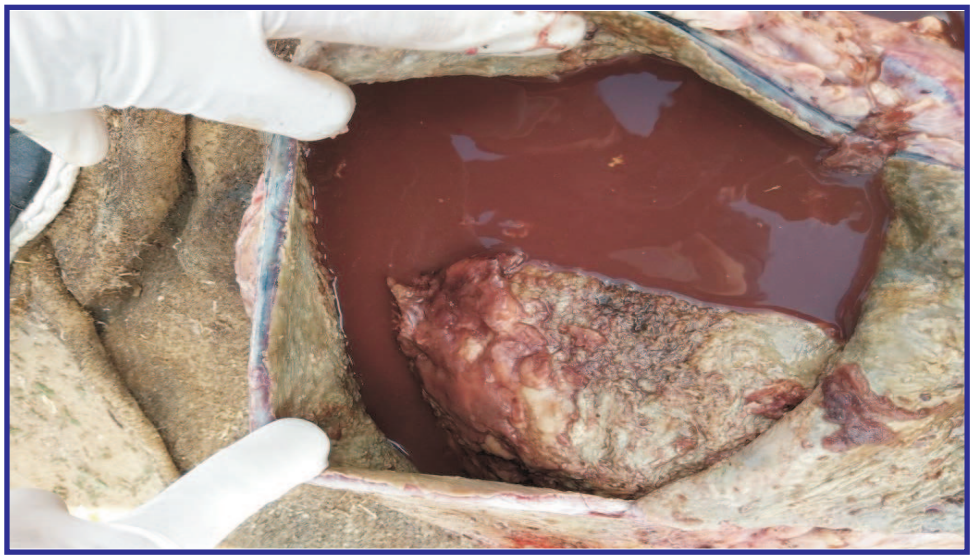

Fig.2 : Gross photograph showing pericardial sac containing large quantity fluid.

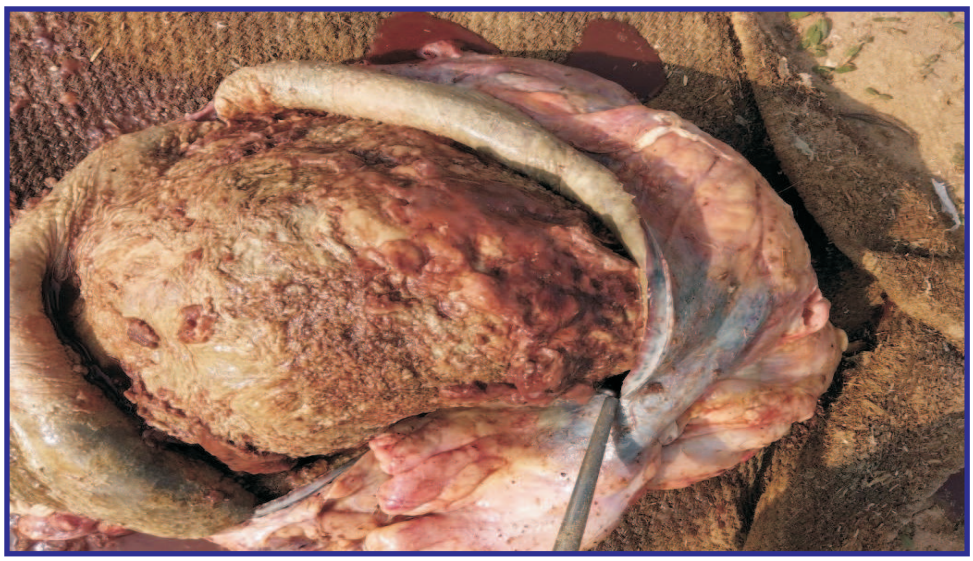

Fig.3 : Gross photograph of heart showing typical bread and butter appearance along with fibrinous pericarditis.

Plate-I

Fig.4 : Gross photograph showing cut surface of myocardium which is hard and thickened due to fibrosis.

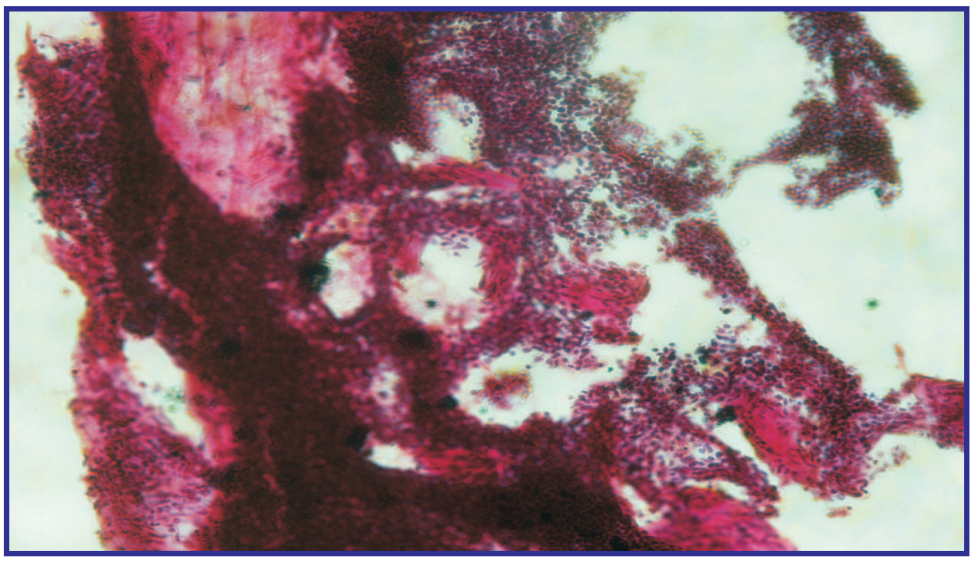

Fig.5 : Microphotograph showing severe infiltration of leucocytes along with diffused haemorrages in pericardium. H & E 200X .

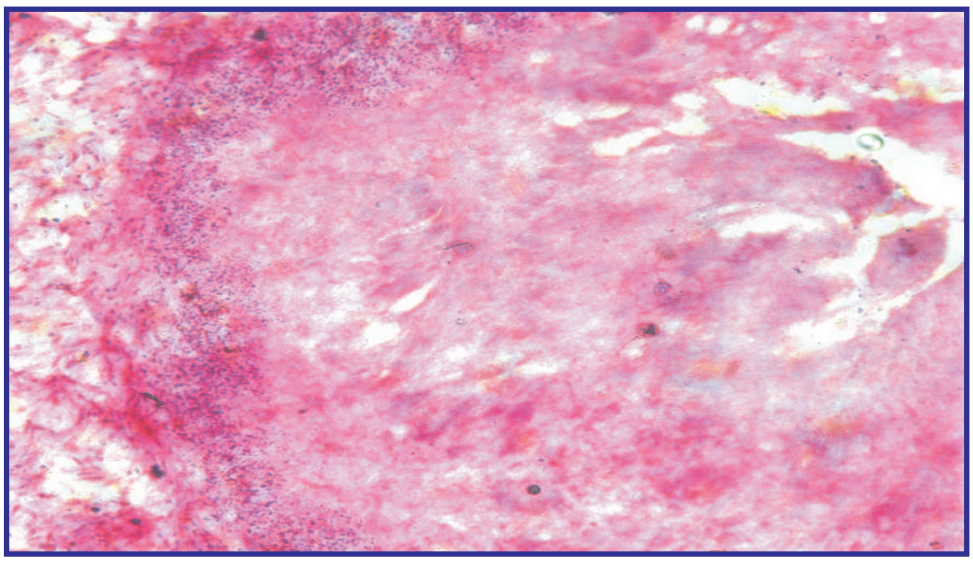

Fig.6 : Microphotograph showing necrotic debris, fibrosis and inflammatory cells in pericardium. H & E 200X.

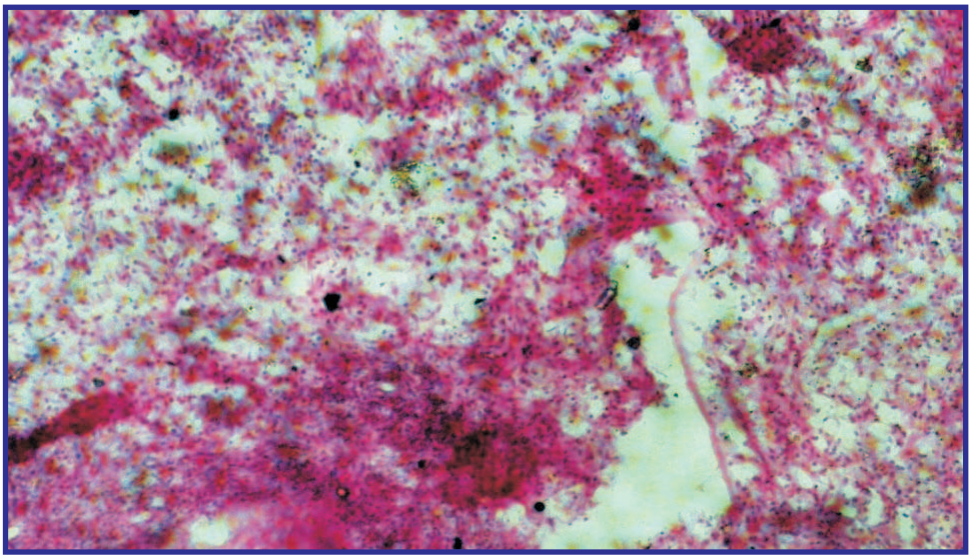

Fig.7 : Microphotograph showing pericarditis along with infiltration of lymphocytes and monocytes. H & E. 100 X.

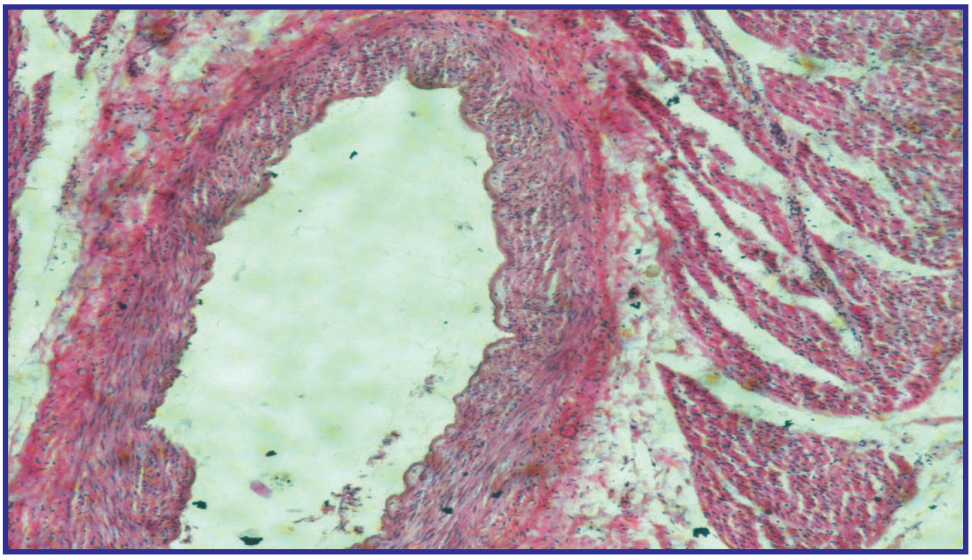

Fig.8 : Microphotograph showing perivascular infiltration around coronary artery along with myocarditis. H & E 100X.

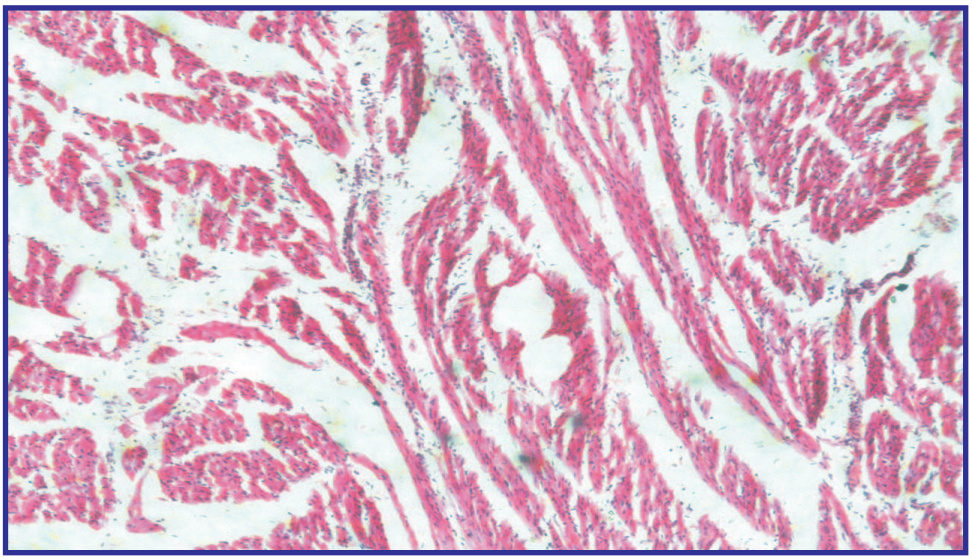

Fig.9 : Microphotograph showing marked infiltration of mono nuclear cells between the degenerated myofibrils . H & E 100X.

Plate-III

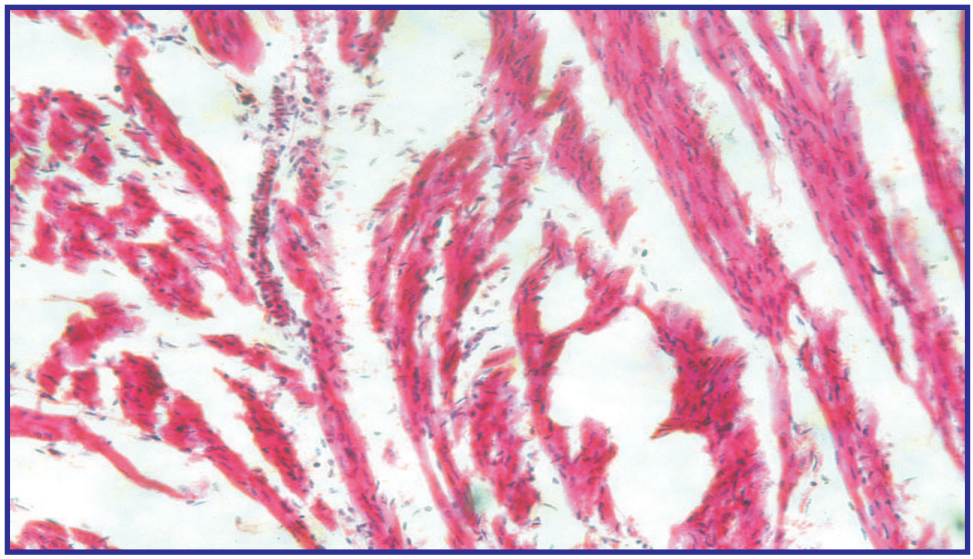

Fig.10 : Higher magnification of Figure 9 showing myocarditis. H&E 200X.

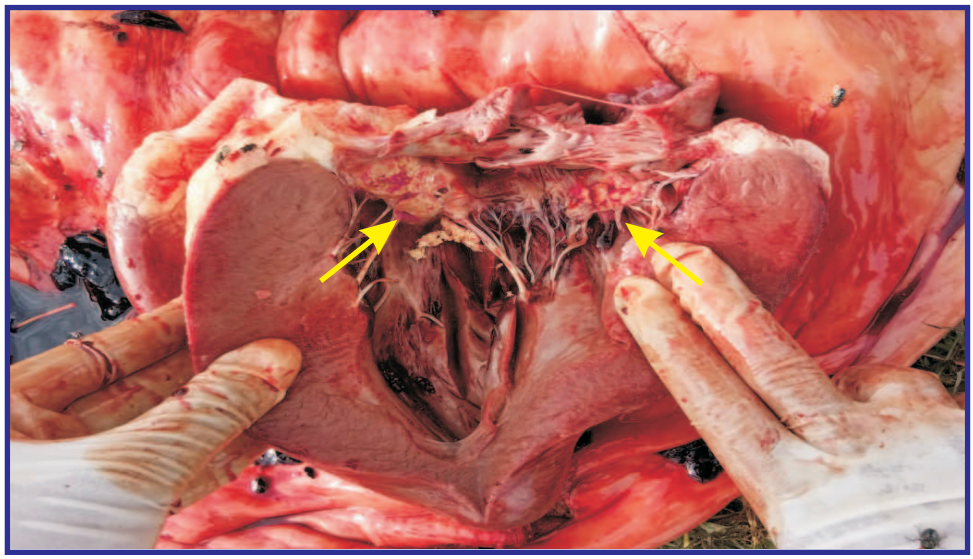

Fig.11 : Gross photograph showing vegetative valvular endocarditis in left-atrio ventricular valve with cauliflower like vegetative growth (Arrows).

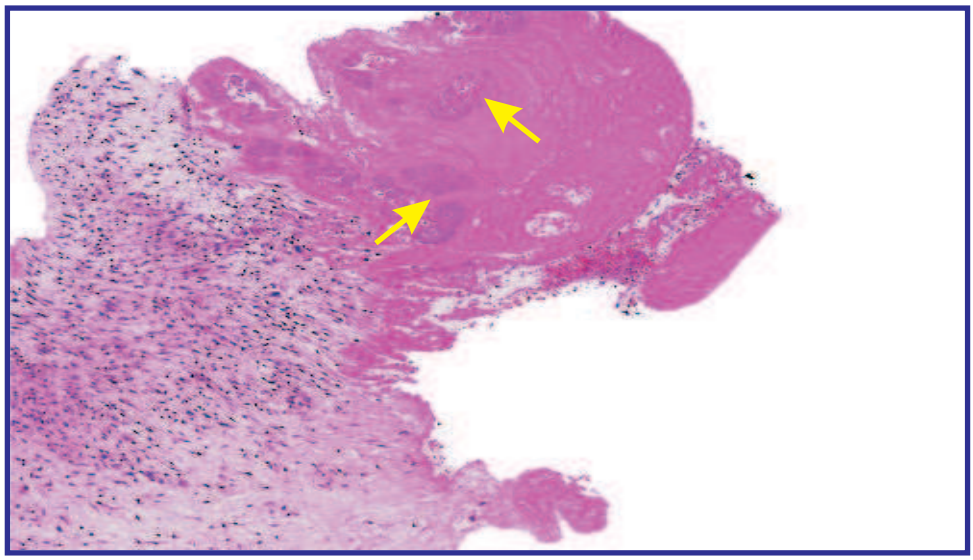

Fig.12 : Microphotograph showing atrio-ventricular valve with vegetative endocarditis containing inflammatory cells and bacterial clumps (Arrow). H & E 100X.

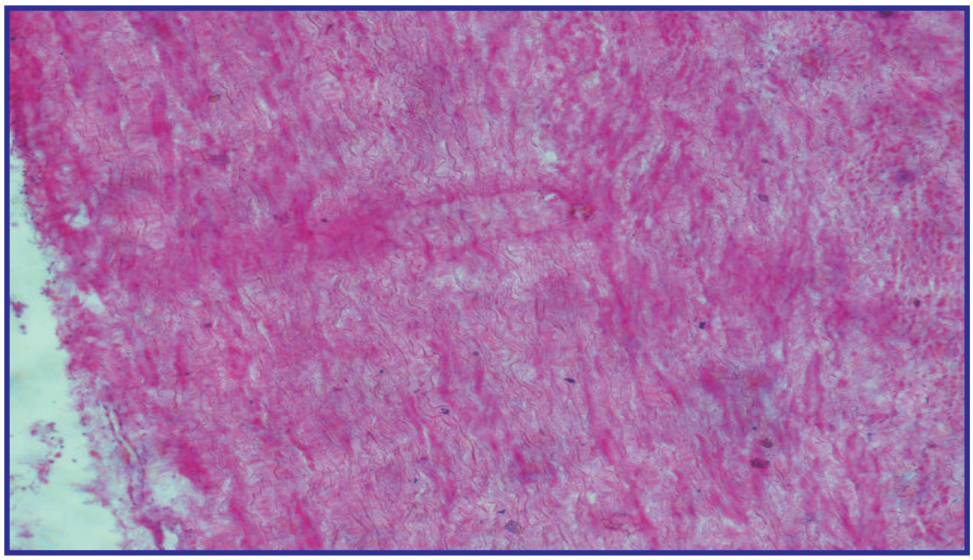

Fig.13 : Microphotograph showing myocardial necrosis along with lighter areas and loss of striations .H & E 100X.

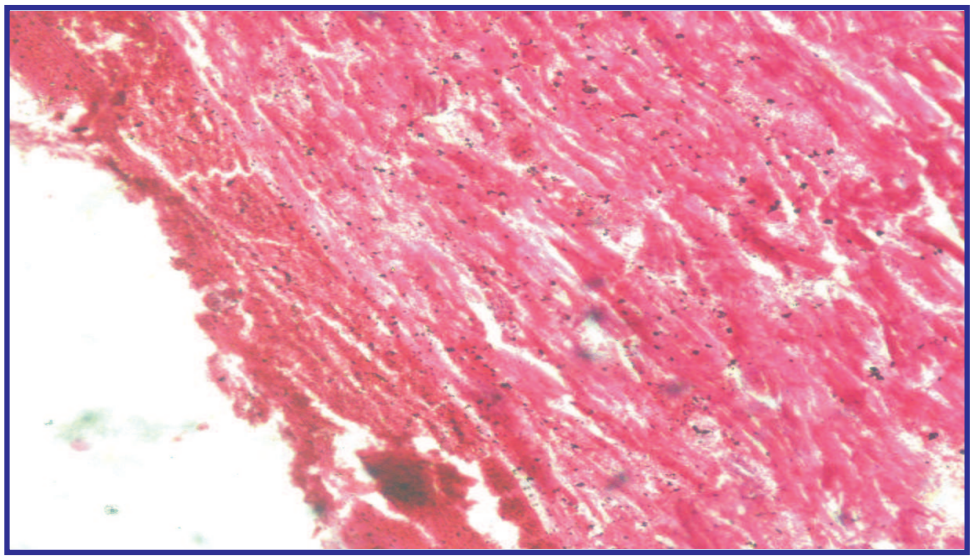

Fig.14 : Microphotograph showing degeneration and separation of myofibers with marked haemorrhages. H & E 100X.

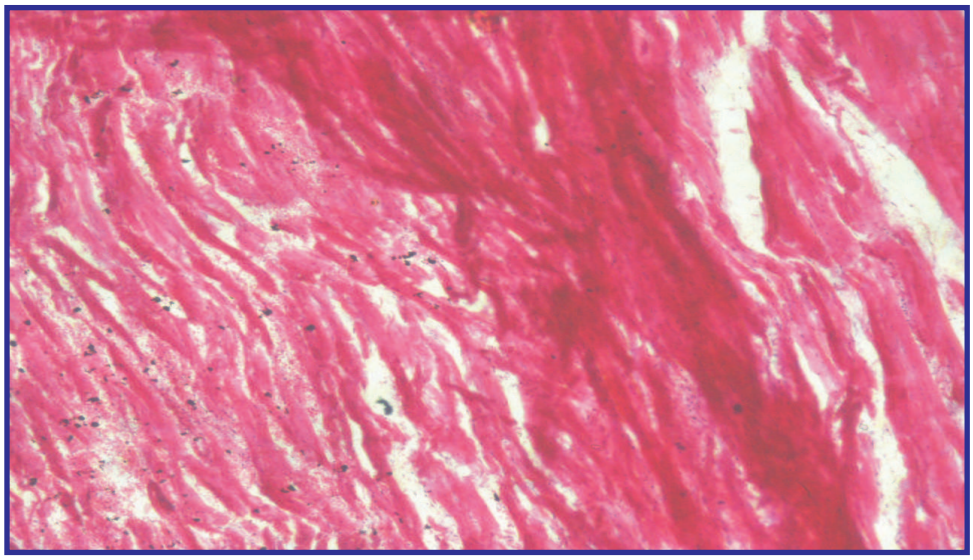

Fig.15 : Microphotograph showing degeneration and separation of myofibers with mild haemorrhages. H & E 100X.

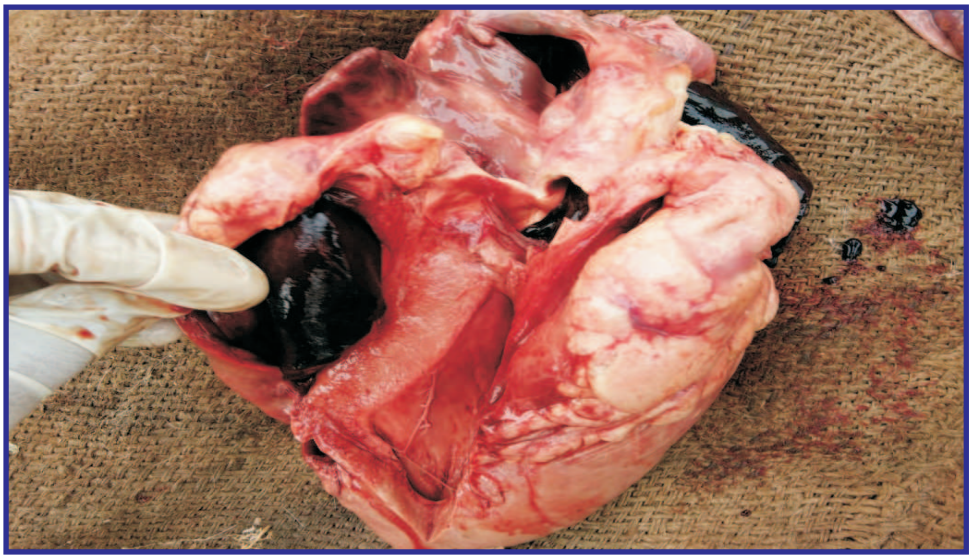

Fig.16 : Gross photograph showing pale areas of coagulative necrosis in heart.

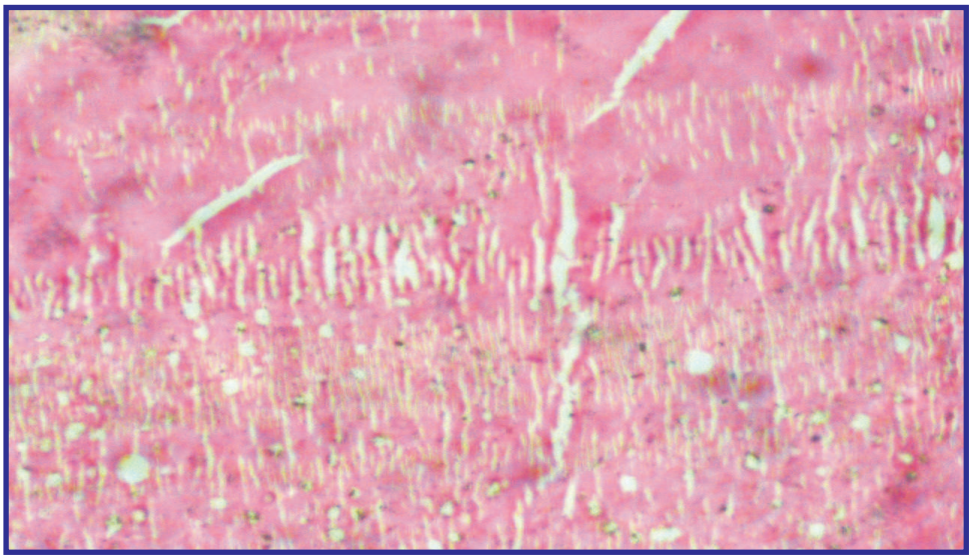

Fig.17 : Microphotograph showing coagulative necrosis of cardiac muscles with no nuclei and very little structural details. H & E 100X.

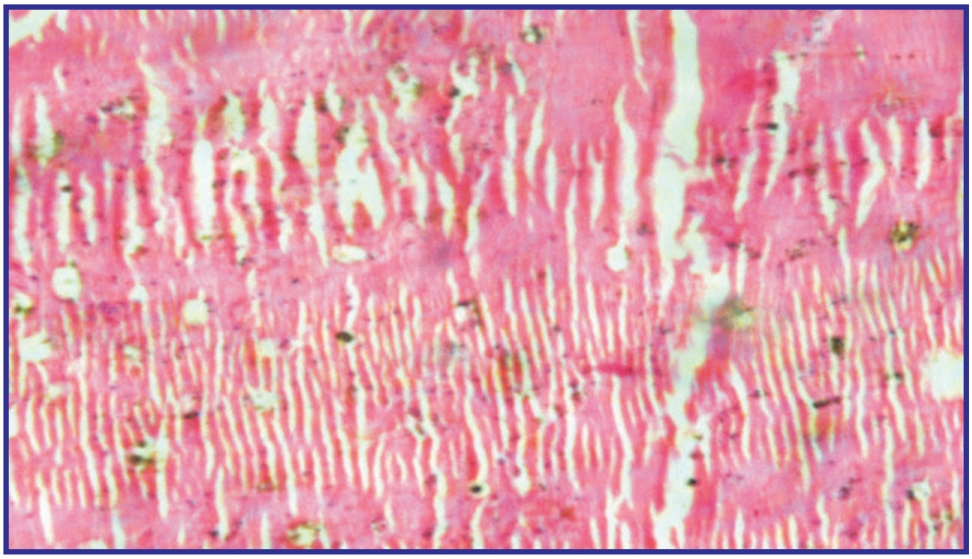

Fig.18 : Higher magnification of figure 17 showing coagulative necrosis of cardiac muscles. H & E 200X.

Plate-VI

Fig.19 : Gross photograph showing lighter necrotic areas over the surface of heart (arrow).

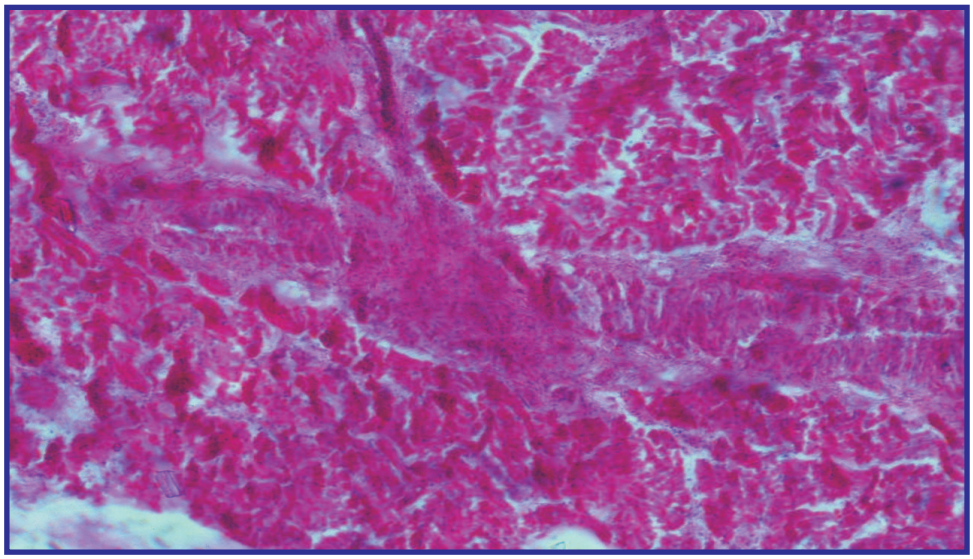

Fig.20 : Microphotograph showing myocardial fibrosis. H& E .100X.

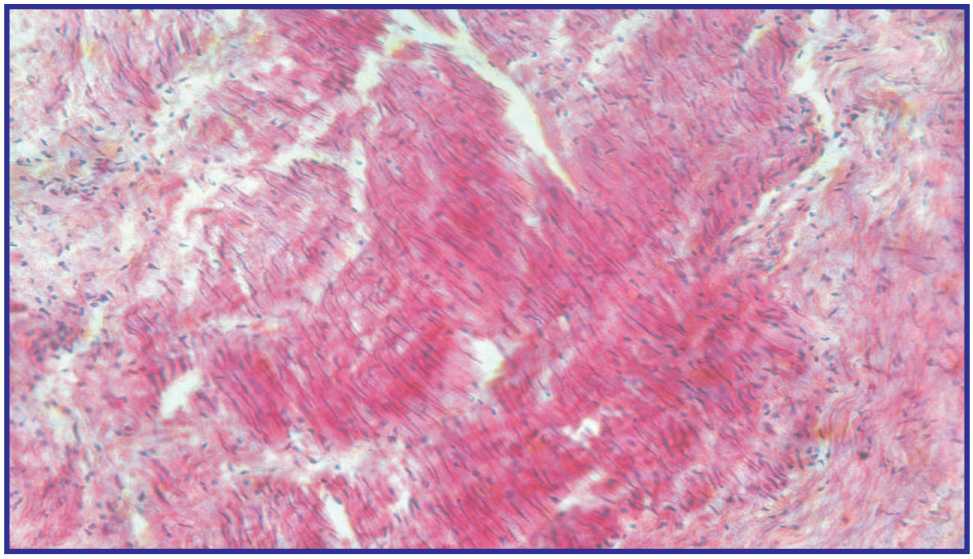

Fig.21 : Microphotograph showing endocardial fibrosis and thickening along with infiltrating cells. H&E 100X.

Plate-VII

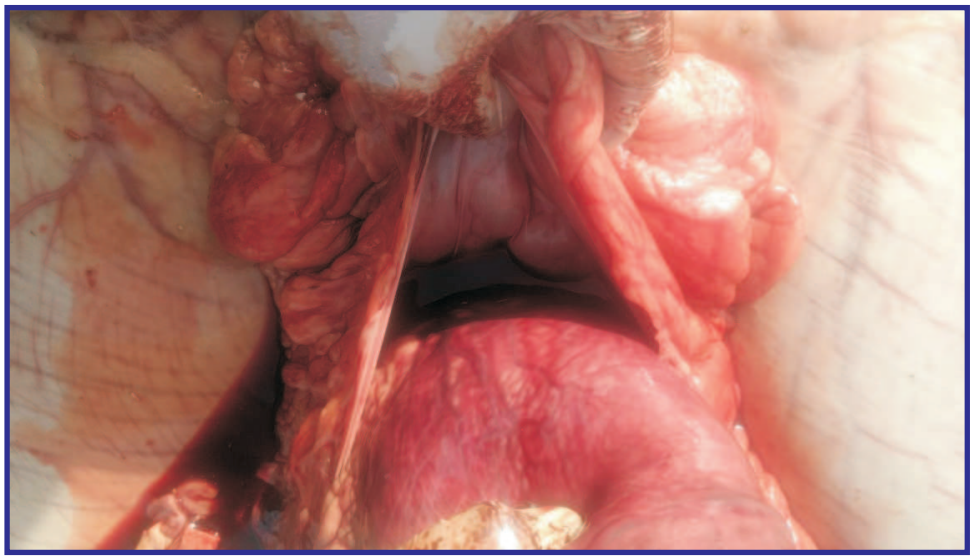

Fig.22 : Gross photograph showing hydropericardium and presence of clear transudate in pericardial sac.

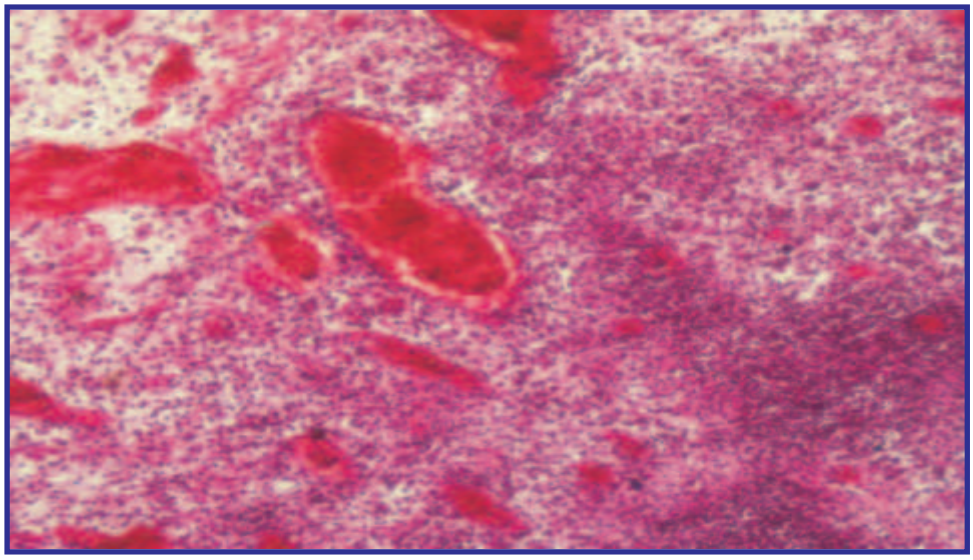

Fig.23 : Microphotograph of pericardium showing congested blood vessels along with infiltrating cells. H & E 100X.

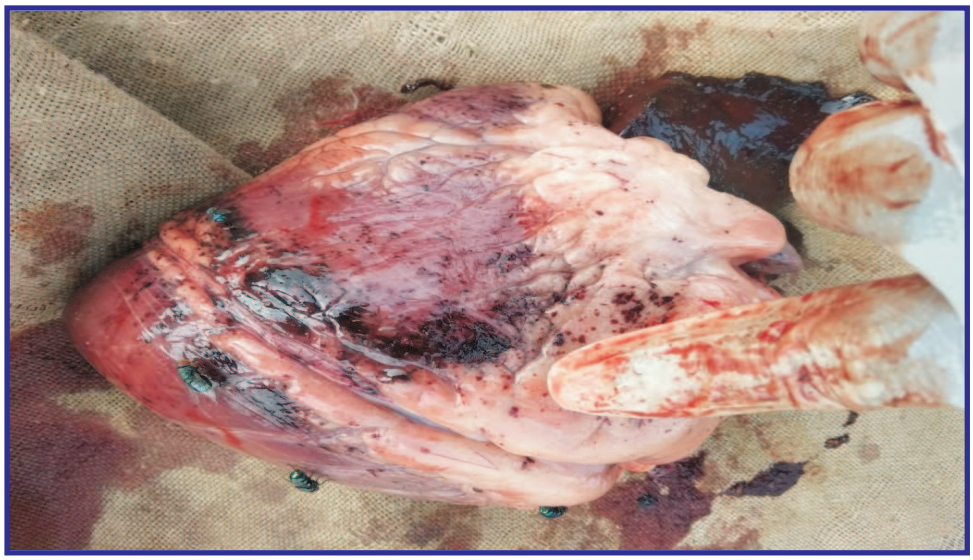

Fig.24 : Gross photograph of heart showing pin point haemorrhages over epicardial surface.

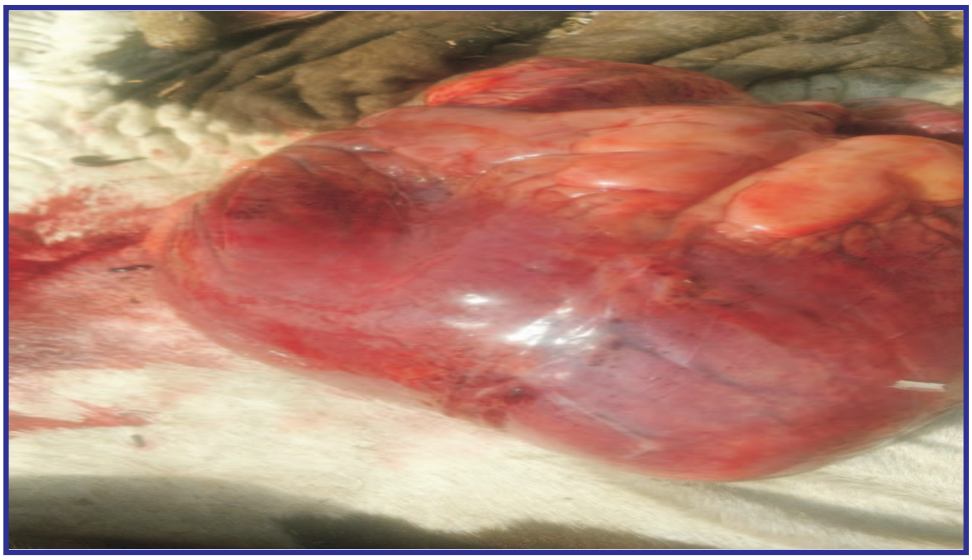

Fig.25 : Gross photograph of heart showing diffused haemorrhages over epicardial surface.

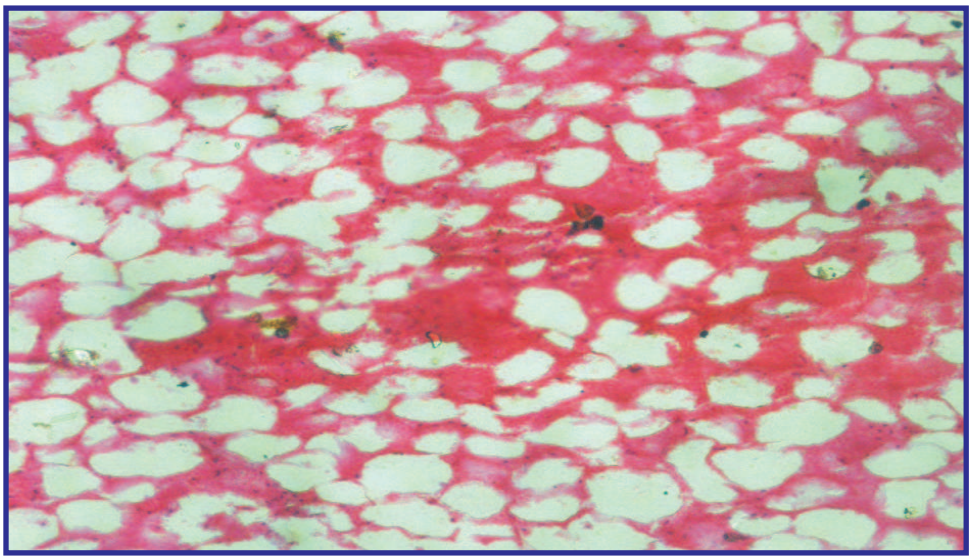

Fig.26 : Microphotograph of epicardium showing diffiused haemorrhages between fat cells of epicardium. H & E 100X.

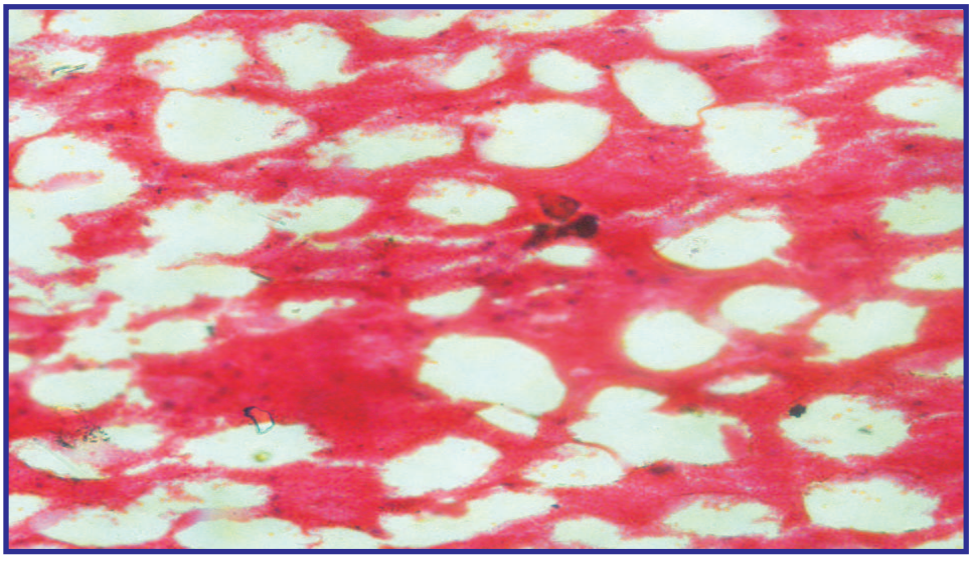

Fig.27 : Microphotograph of epicardium showing diffiused haemorrhages between fat cells of epicardium. H & E 200X.

Plate-IX

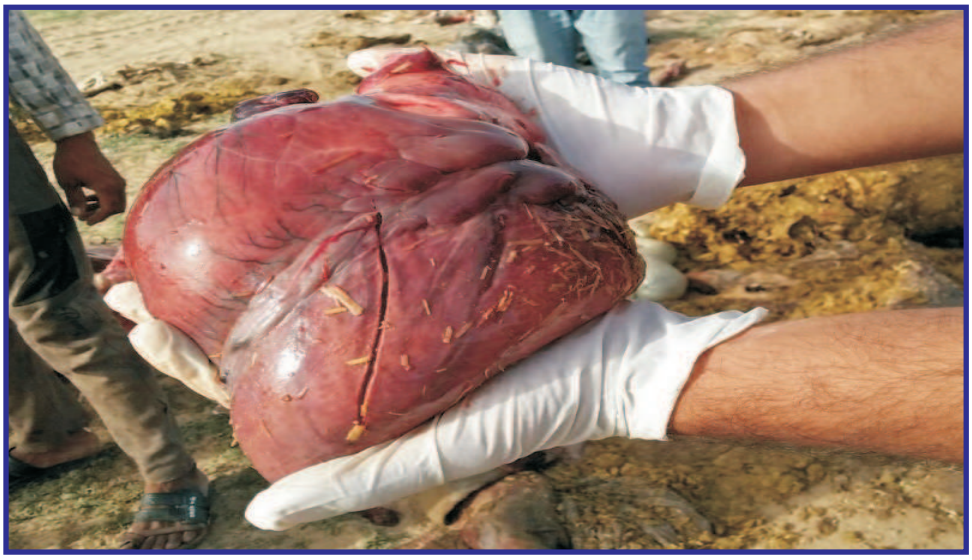

Fig.28 : Gross photograph of heart showing congested blood vessels.

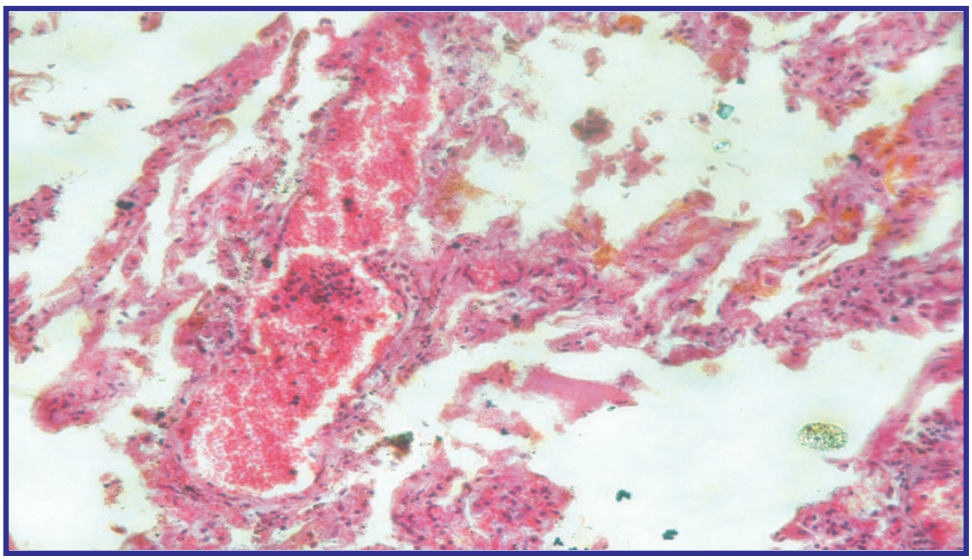

Fig.29 : Microphotograph of epicardium showing congested blood vessels. H & E 200X.

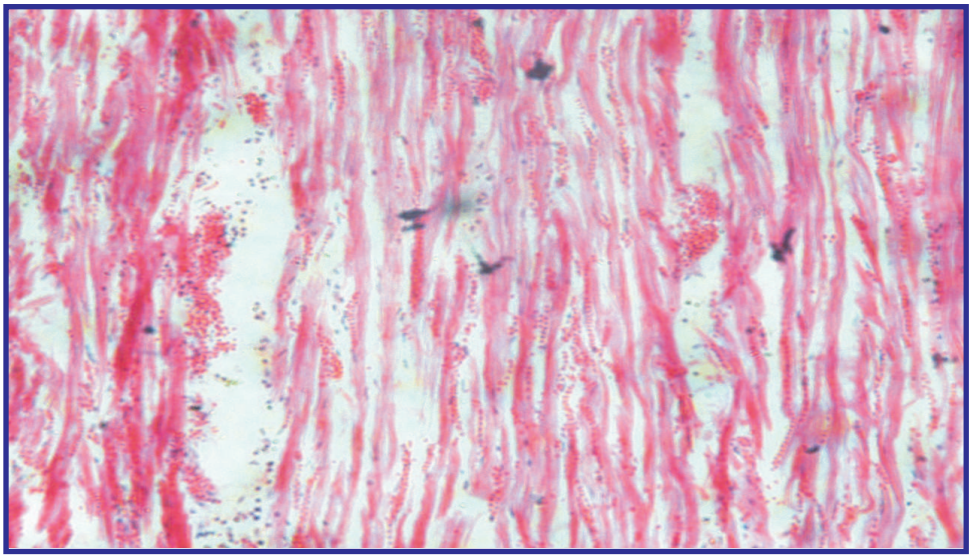

Fig.30 : Microphotograph of myocardium showing haemorrhages and degenerative muscle fibers mild infiltration of leukocytes . H & E 200X

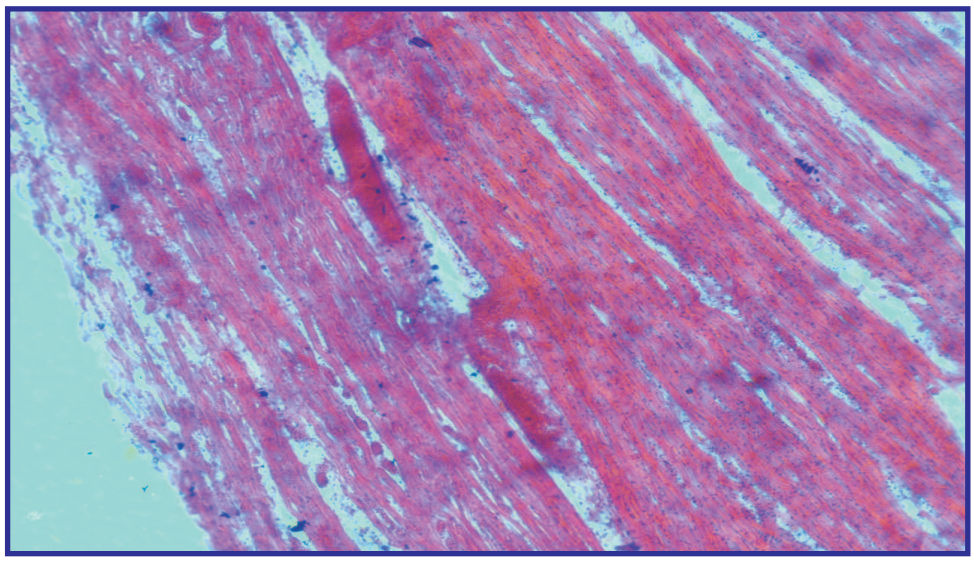

Fig.31 : Microphotograph of myocardium showing congestion of blood vessels. H & E 200X.

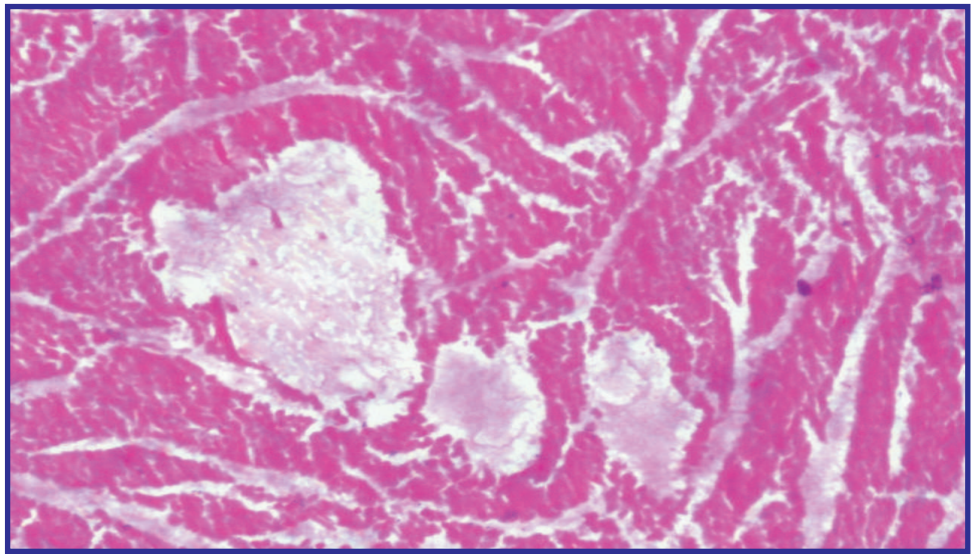

Fig.32 : Microphotograph of myocardium showing pink colored edematous fluid and separation of muscle fibers. H & E 200X.

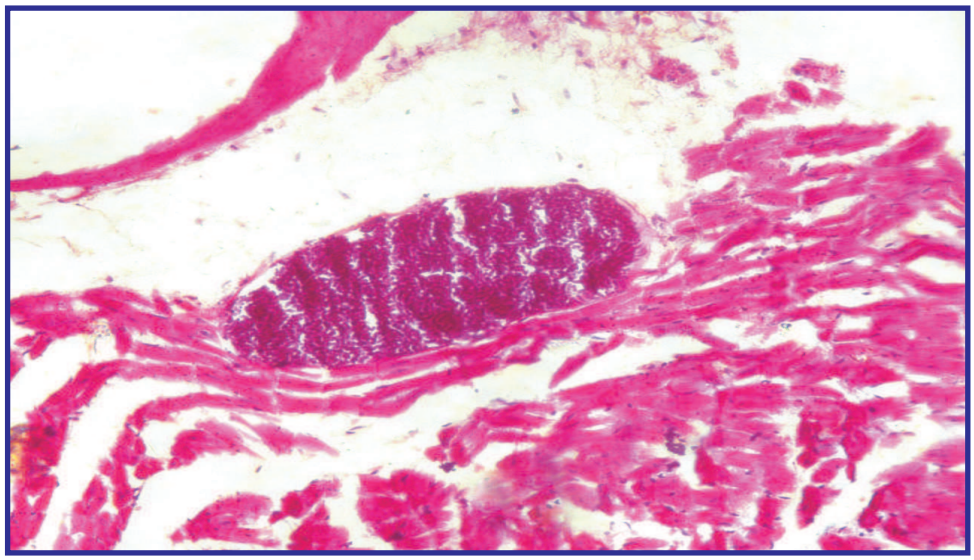

Fig.33 : Microphotograph showing sarcocyst in cardiac muscles along with degenerated muscle fibers and mild infiltration of eosinophils. H & E.200 X.

Plate-XI

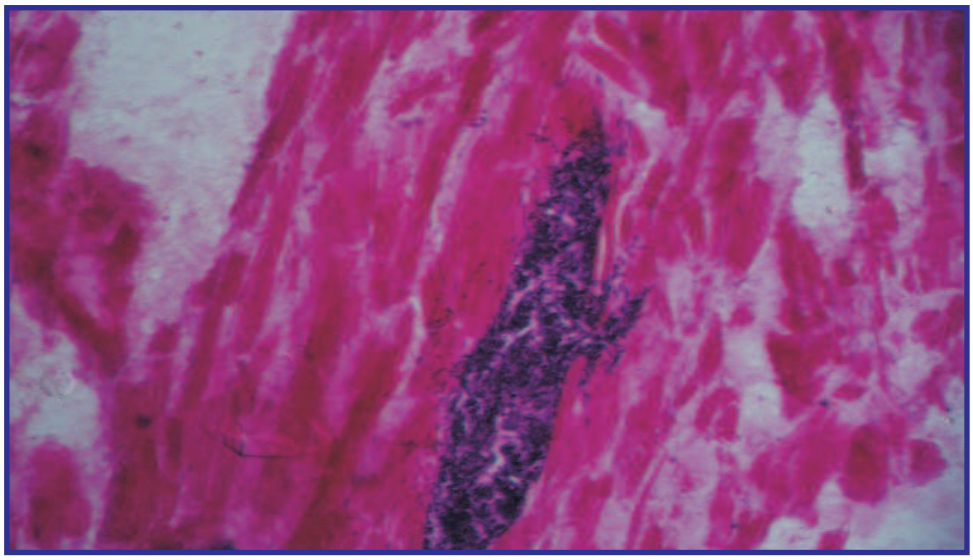

Fig.34 : Microphotograph showing ruptured sarcocyst in cardiac muscles. H & E 200 X.

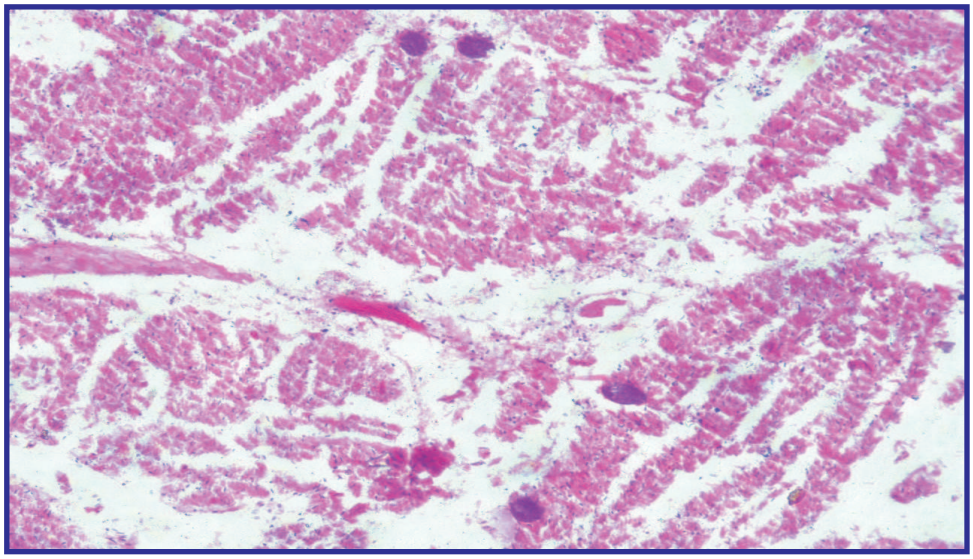

Fig.35 : Microphotograph showing multiple sarcocysts in between cardiac muscle fibers. H & E.100X

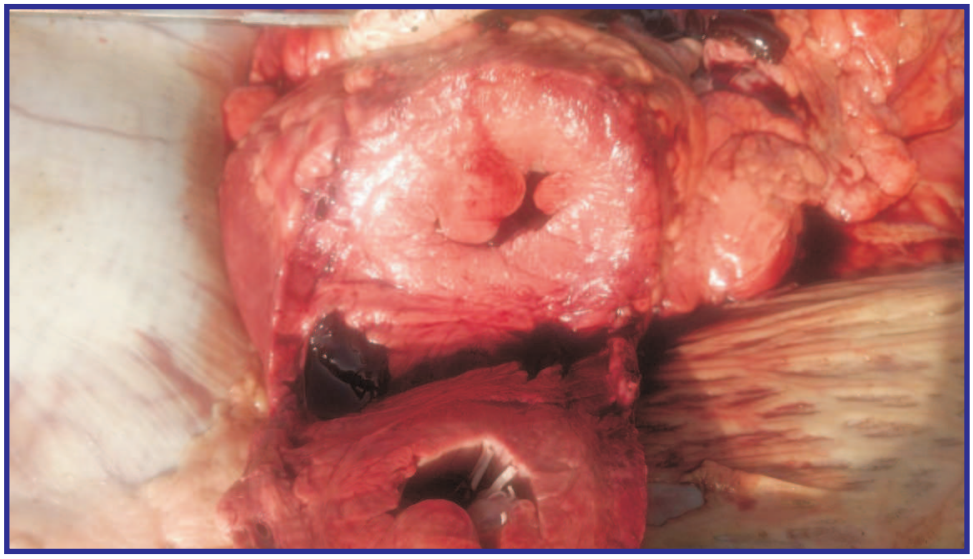

Fig.36 : Gross photograph showing hypertrophy of ventricular wall with narrow lumen.

Plate-XII

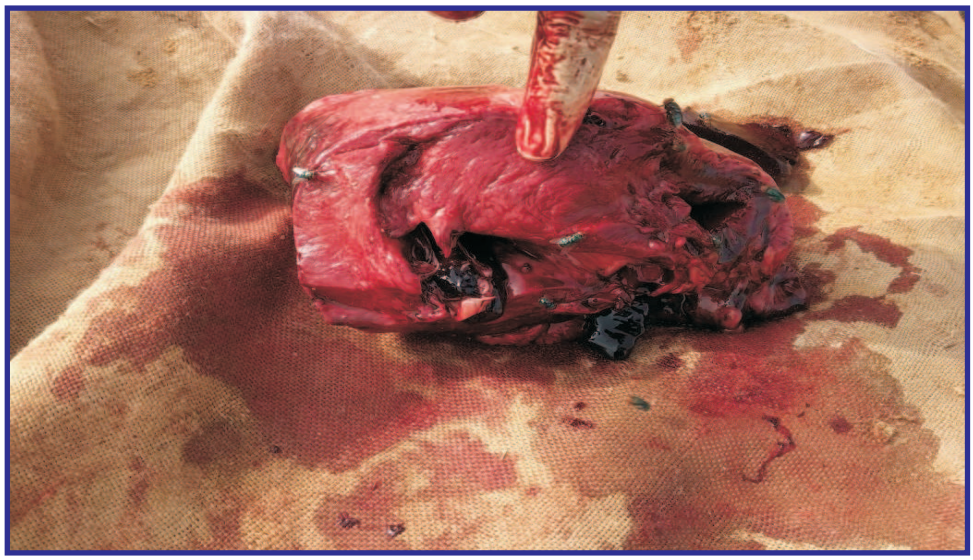

Fig.37 : Gross photograph showing hypertrophy of ventricular wall.

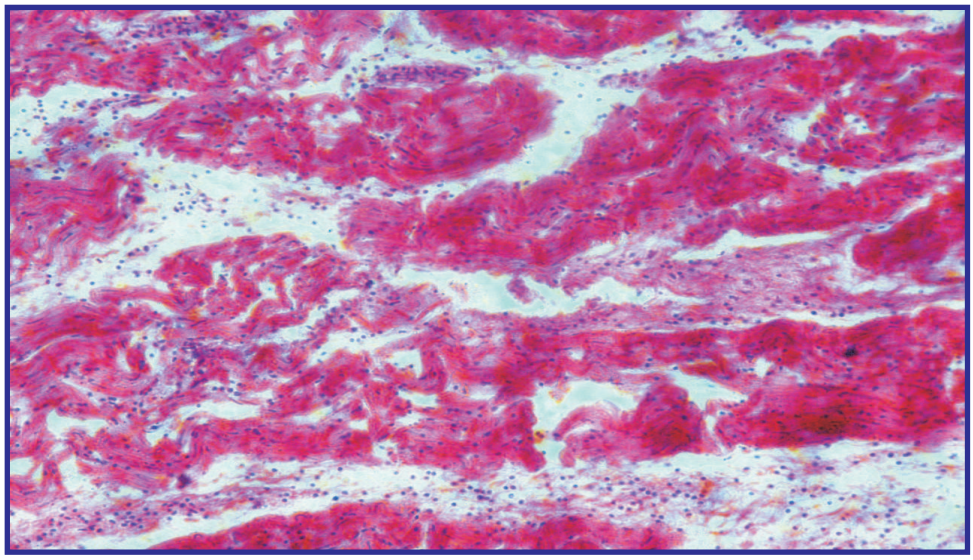

Fig.38 : Microphotograph showing hypertrophy of myocardial cells and infiltration of mononuclear cells. H & E .200X.

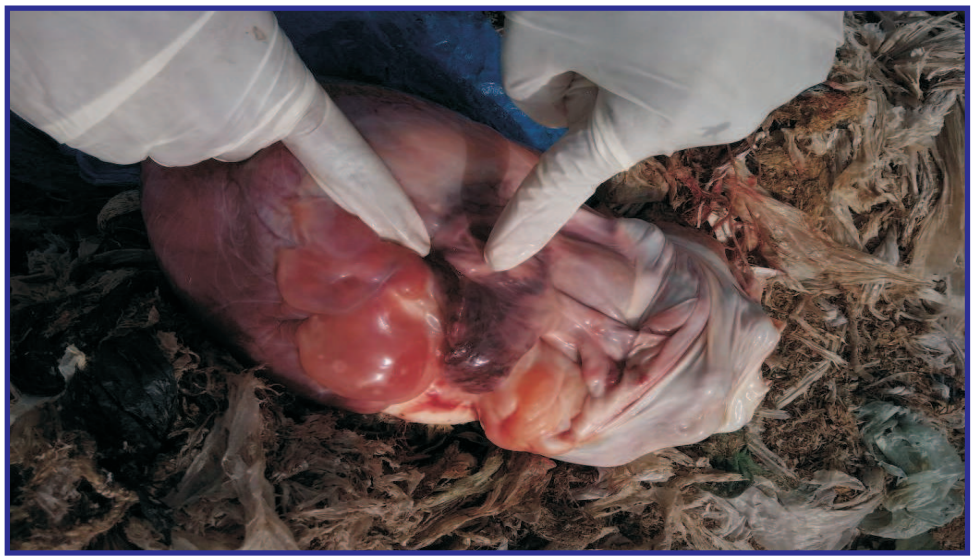

Fig.39 : Gross photograph showing serous atrophy of epicardial fat cells with gelatinous mass.

Plate-XIII

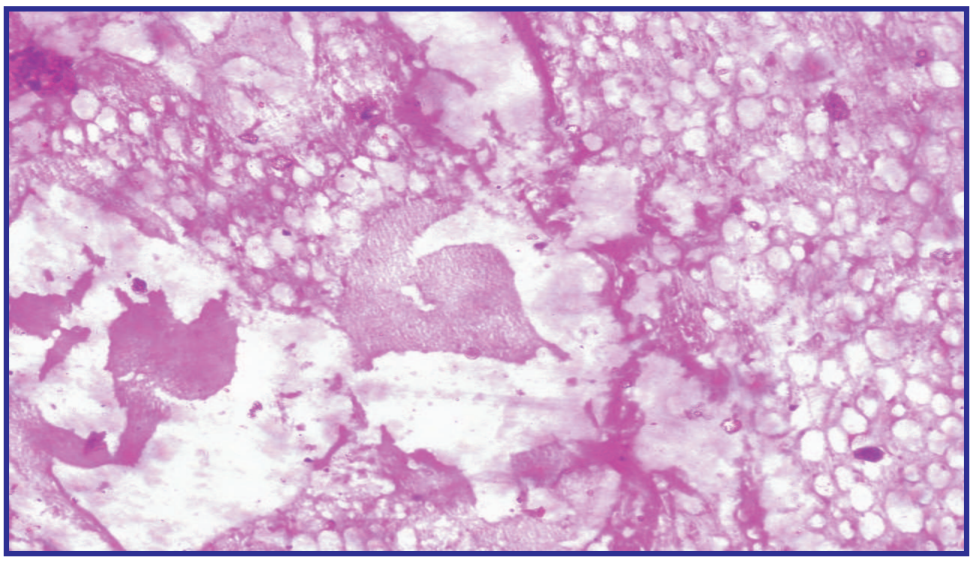

Fig.40 : Microphotograph showing serous atrophy of epicardial fat cells surrounded by serous fluid. H&E 100X.

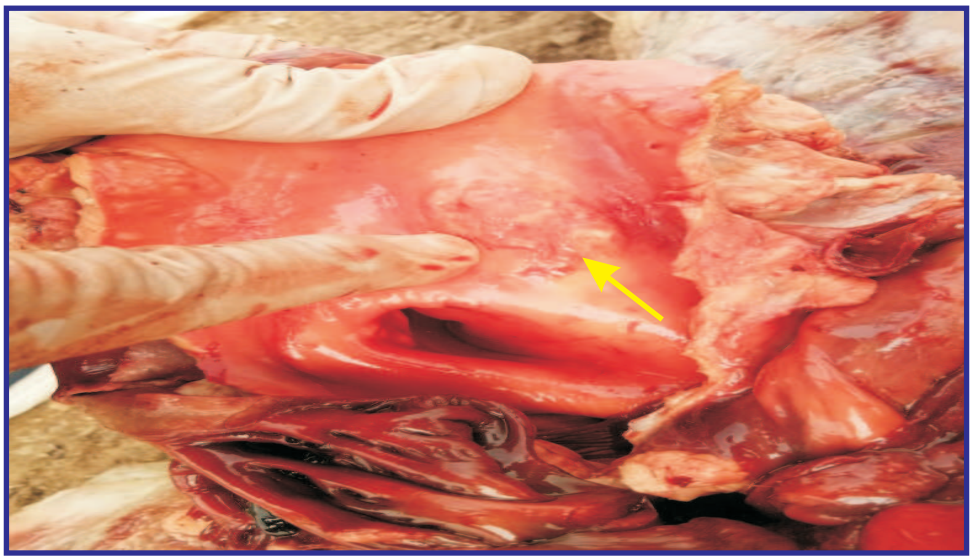

Fig.41 : Gross photograph showing thickened, diffused and localized irregular areas in the inner wall of aorta (arrow).

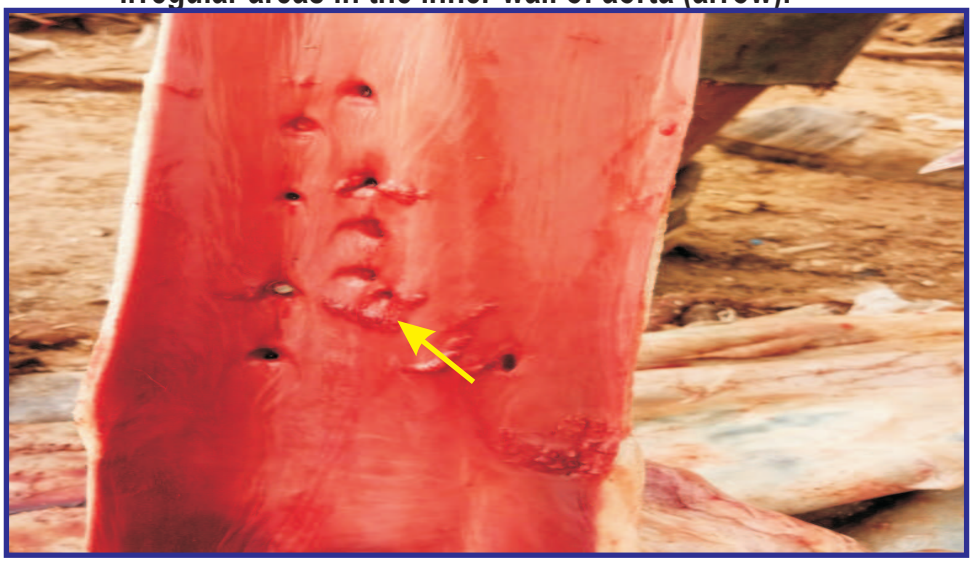

Fig.42 : Gross photograph showing thickened, small and localized irregular areas in the inner wall of aorta (Yellow arrow).

Plate-XIV

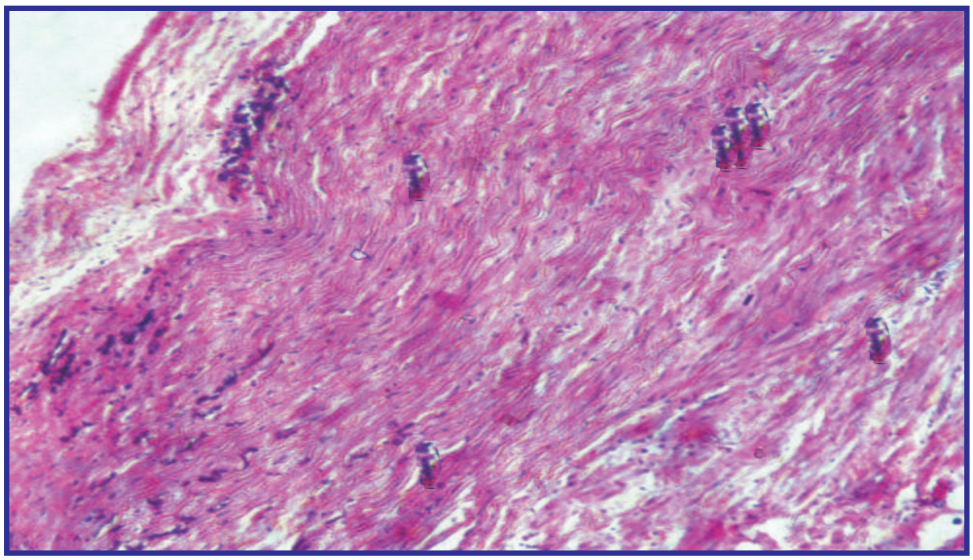

Fig.43 : Microphotograph of aortic wall showing diffused calcification and mineralization in the form of dark depots. H&E 100X.

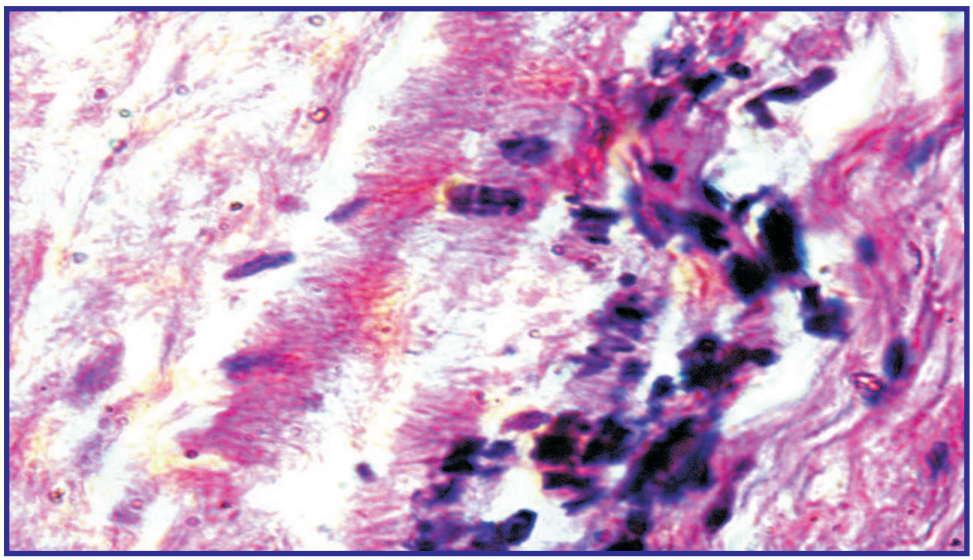

Fig.44 : Higher magnification of fig. 43 showing aortic wall calcification and mineralization. H & E 200 X.

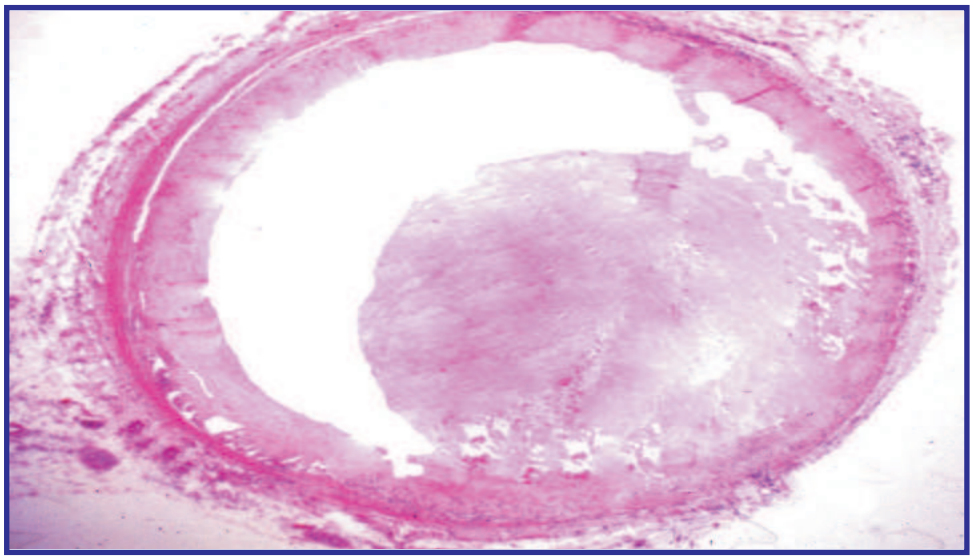

Fig.45 : Microphotograph showing atherosclerosis plaque in the cavity of coronary artery. H & E. 100 X.

Plate-XV

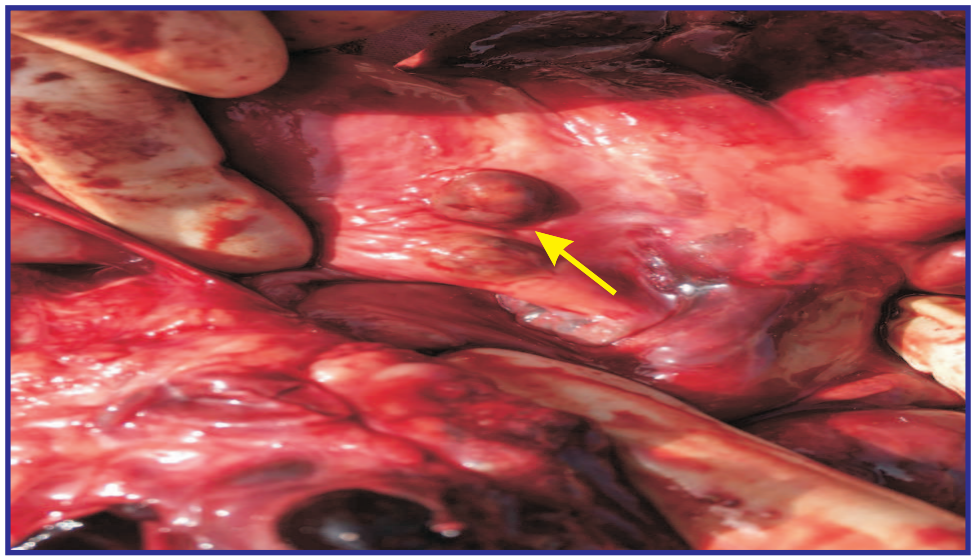

Fig.46 : Gross photograph showing presence of Onchocerca nodule on aortic wall directing outer surface (arrow).

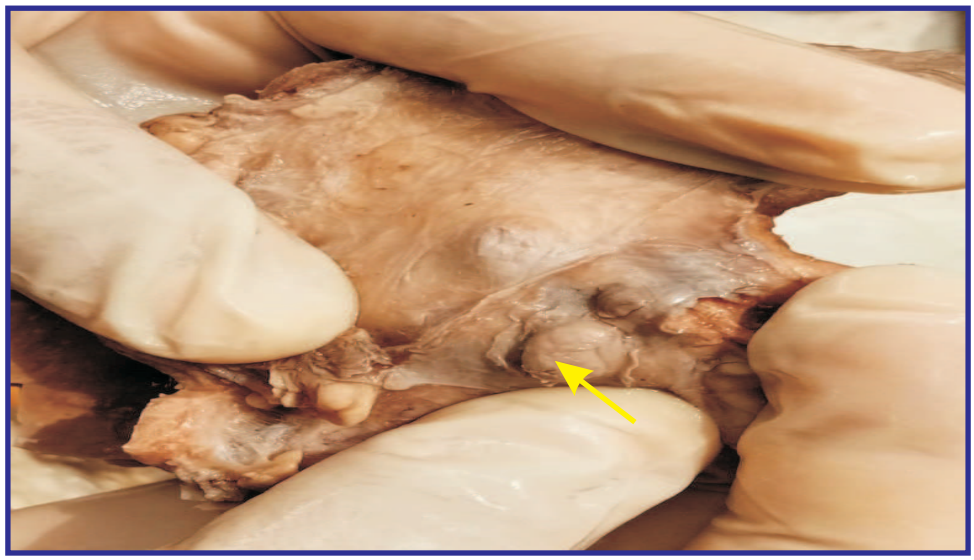

Fig.47 : Gross photograph showing parasitic granulomas, surrounded by tough connective tissue, extending outward the aorta serosa (arrow).

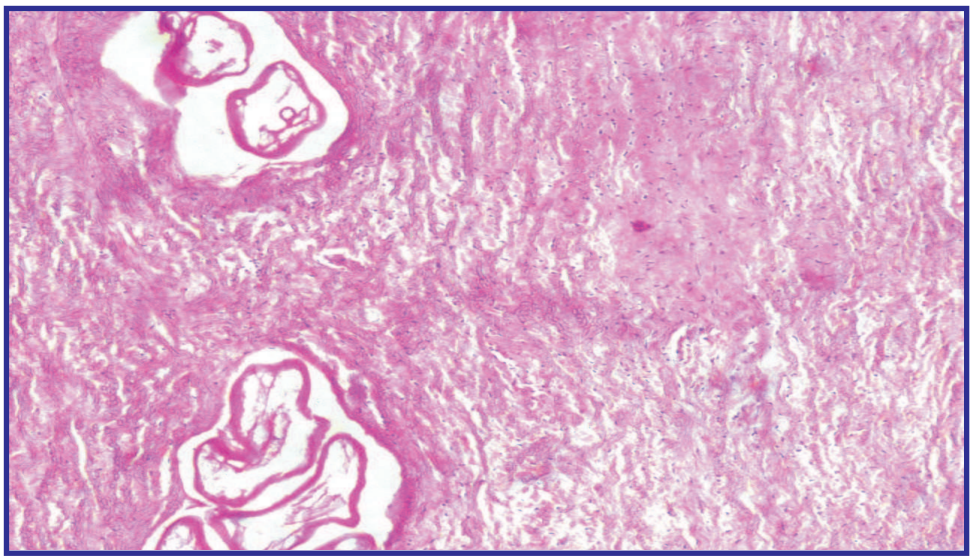

Fig. 48 : Microphotograph showing presence of Onchocerca cyst surrounded by connective tissue lining in the tunica intima of aorta. H &E 100X.

Plate-XVI

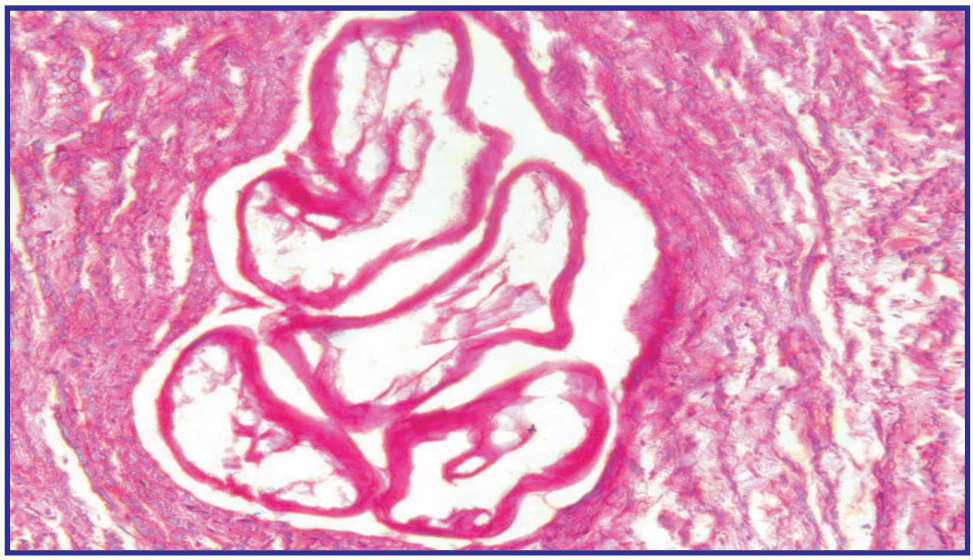

Fig.49 : Higher magnification of figure 48 showing presence of Onchocerca cyst in the tunica intima of aorta. H &E 200X.

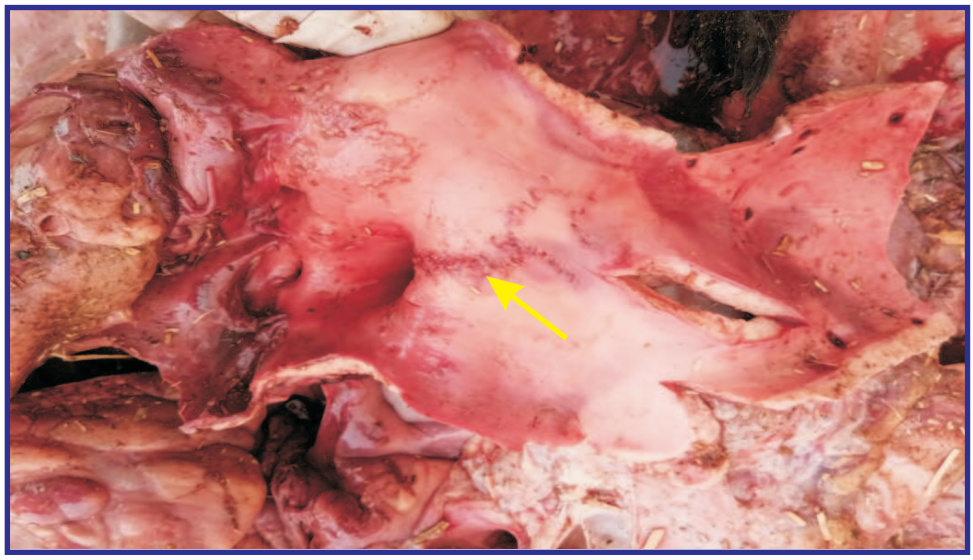

Fig.50 : Gross photograph showing open systemic aorta with fatty streaks in early atherosclerosis. (Arrow)

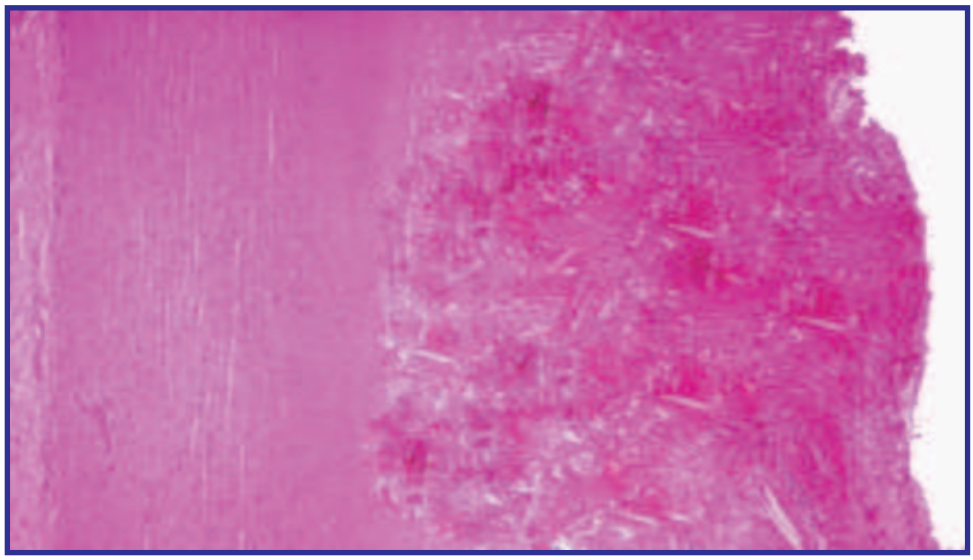

Fig. 51 : Microphotograph showing cross section of aorta with large atheroma and cholesterol cleft. H & E 100 X.

Plate-XVII