# PATHOLOGY OF CARDIAC DISORDERS IN PIGS REARED ON SWILL

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

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## DECLARATION

I hereby declare that this thesis entitled **"PATHOLOGY OF CARDIAC DISORDERS IN PIGS REARED ON SWILL"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other university or society.

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## CERTIFICATE

Certified that the thesis entitled **"PATHOLOGY OF CARDIAC DISORDERS IN PIGS REARED ON SWILL"** is a record of research work done independently by **Dr. Sivanesan, P** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

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# *Introduction*

# 1. INTRODUCTION

Pig rearing in rural production system based on swill feed is becoming more and more popular in Kerala. The swill feeding has reduced the maintenance cost of the farm. But there are reports of sudden death in apparently healthy pigs and the post mortem examination has revealed damage to the cardiovascular system. The sudden mortality in swill fed pigs with the cardiomyopathic changes, is suspected to be the result of nutritional deficiencies especially the micronutrients. The indigenous breeds have been replaced by cross breds of Duroc, Yorkshire and Landrace. These animals are more prone for multiple micro nutrient deficiencies producing cardiomyopathies. The myocardial diseases of nutritional deficiencies are studied in animals mostly under laboratory conditions by feeding deficient diets. But diseases due to deficiency of selenium- vitamin E resulting in great economic loss in animal production have been reported in many areas of the world.

Cardiomyopathies are heart diseases of multiple etiology, associated with pathological processes within the myocardium or endocardium or both including certain lesions of the conduction system (McKinney, 1974). Cardiomyopathies are common in animals as in man but only few such cases have been reported. This is probably because the cardiovascular lesions are overlooked in the routine post mortem examinations of pigs and the histological lesions are not studied in detail. Previously the only etiology commonly regarded as causing damage to the myocardium was coronary artery obstruction, causing myocardial infarction. The recent studies contributed to the evidence that many cases of heart failure are due to a involvement of myocardium.

The normal heart has a three to fivefold functional reserve capacity that eventually will be lost with cardiac disease and subsequently result in impaired function. A variety of compensatory mechanisms operate in normal and diseased hearts in an attempt to meet both the short- term and long – term demands for adequate cardiac output and these mechanisms include cardiac dilatation, myocardial hypertrophy, increase in heart rate ( Van Vleet and Ferrans, 2001).

The cardiomyopathies in hamsters, mice, rats, cattle may have progressive clinical courses and some morphologic alterations similar to those in certain cardiomyopathies in human patients and may provide useful models for the human cases. But in many of the experimental studies in human cardiology, the swine heart is used as a paradigm.

Considering the significance of micronutrients in the development of cardiac disease the study was undertaken to investigate the influence of feeding practice on the development of cardiomyopathic changes. The objectives of the study are

1. Comparative pathology of gross and histopathological abnormalities in heart of pigs maintained on swill in farm conditions in comparison with the pigs maintained on standard diet.

2. To measure the level of Lactic dehydrogenase (LDH), Aspartate amino transferase (AST), Creatine kinase (CK) and correlate with the pathological observation in swill fed and concentrated fed pigs.

The findings of the study will enable to make a better understanding of the correlation between the cardiomyopathic changes and the swill feeding and will help to suggest remedial measures on the management of pigs under swill feeding.

# *Review of Literature*

# 2. REVIEW OF LITERATURE

## 2.1 ANATOMY OF PIG HEART

The pig's heart is small in proportion to the body weight. It is nearly 0.3% of the body weight and in a large adult is usually between 240 to 500 g. The ventral surface is moderately convex, overlies the sternum from the second sternebra to the cranial part of seventh sternebra. The dorsal surface is more convex. The apex is blunt and almost median, overlies the cranial part of seventh sternebra (Ghoshal, 1975).

A study conducted by Crick *et al.* (1998) on qualitative analysis of porcine and human cardiac anatomy by gross examination and dissection of hearts with macrophotography revealed that the porcine heart had a classic valentine heart shape reflecting its location within the thorax and to the orientation of the pigs body and its right atrium was characterized by the tubular shape of its appendage. They further stated that the apical components of porcine ventricles possessed very coarse trabeculations, much broader than those observed in the human ventricles.

## 2.2 SWILL FEEDING

Osuna *et al.* (1981) reported cadmium toxicosis in pigs when animals fed with recycled sewage sludge in which cadmium is concentrated.

Pritchard *et al.* (1985) observed zinc toxicity among weaned swill fed pigs.

Corso (1997) studied on the probability of exposure for exotic diseases in domestic pigs fed with uncooked household waste. Of the four diseases studied, the

probability of exposure was highest for the classical swine fever (hog cholera) virus compared to lower exposure for foot and mouth disease virus, swine vesicular disease virus and African swine fever virus.

Horst *et al.* (1997) reported that swine fever virus is transmitted by feeding swill to pigs.

Fritzemeir *et al.* (2000) reported that swill feeding is an important factor for swine fever virus transmission and in most cases the outbreaks were due to the feeding of untreated swill or kitchen waste. They also reported that an important factor for virus transmission in the pig's holdings is feeding of wild boar carcasses as swill feed. Due to swill feeding, the virus caused a primary outbreak which was followed by eleven secondary outbreaks.

Animals that have access to potentially infected swill will always constitute a potential public threat with regard to trichinellosis (van Knapen., 2000).

## 2.3 CARDIOVASCULAR DISORDERS IN VARIOUS FEEDING PRACTICES

Moir and Masters (1979) reported that the extensive use of low quality protein source replacing much of the meat meal in pig rations increases the incidences of nutritional myopathy.

Kennedy and Rice (1988) reported that feeding polyunsaturated fatty acids to deficient calves intensified the cardiac cell damage.

Multifocal or diffuse cardiocyte degeneration and necrosis in calves fed on diets containing poly unsaturated fatty acids were observed by Kennedy and Rice (1992).

Kelly (1993) stated that feeding pigs largely of grain and containing protein supplements lacking either quality or quantity could result in death without showing any clinical signs. The lesions described were degeneration of cardiac and skeletal muscle, serous effusions, fibrinoid necrosis of arterioles and ulceration of stomach.

In an experimental study pigs fed with diet lacking antioxidants and enriched with oxidated cod liver oil, developed hyaline degeneration of heart muscle and all pigs displayed an overall pale yellowish colour and translucence of the skeletal muscles (de Gritz *et al.*, 1994).

Nolan *et al.* (1995) investigated the effect of feeding corn oil to vitamin E deficient pigs on lipid peroxidation and induction of dietetic microangiopathy or mulberry heart disease. They concluded feeding of corn oil did not induce dietetic microangiopathy although lipid peroxidation was evident.

Menon *et al.* (2003) observed cardiomyopathic lesions in apparently healthy pigs reared on locally available waste.

Oldfield (2003) reported mulberry heart disease and hepatosis dietetica in pigs raised solely on cereal diet and attributed due to deficiency of both selenium and vitamin E.

## 2.4 CARDIOVASCULAR DISORDERS IN NUTRITIONAL DEFICIENCIES

Selenium- vitamin E, potassium, copper, thiamine and magnesium deficiencies are the nutritional causes of myocardial necrosis in animals (Van Vleet and Ferrans, 2001).

### 2.4.1 Vitamin E and Selenium

Research on swine nutrition contributed to the evidence that selenium was an essential nutrient, when it was shown that even a high level of vitamin E did not eliminate the need for selenium (Ewan *et al.*, 1969).

Hoekstra (1975) proposed synergism between selenium and vitamin E in the process of antioxidation, wherein tocopherols tended to prevent oxidative damage to polyunsaturated fats in cell membranes. Selenium, as part of selenoenzyme glutathione peroxidase, catalyzes the destruction of lipid hydroperoxides. This explains these two nutrients play separate but interrelated roles in the cellular defense system against oxidative damage

Selenium and vitamin E deficiency is associated with various myopathies, such as cardiac, skeletal, intestinal myopathy, hepatosis dietetica, mulberry heart disease in animals (Van Vleet, 1980).

Van Vleet *et al.* (1981) protected ducklings from cardiomyopathy induced by selenium - vitamin E antagonists by feeding appropriate supplements.

Van Vleet and Ferrans (1982) observed cardiomyopathy by feeding selenium - vitamin E antagonist.

Maas *et al.* (1984) reported that white muscle disease can occur in animals of adequate selenium status but deficient in vitamin E.

Korpela (1990 a) concluded that pigs with low selenium status are at risk of microangiopathy and the low selenium status together with vitamin E deficiency increases oxidative stress and development of oxidative damage.

Korpela (1990 b) suggested that increased myocardial and hepatic iron concentration could promote oxidative stress in selenium - vitamin E deficient pigs which contribute to the development of oxidative damage.

Mahan (1991) stated that pigs are born with low tissue reserves of α tocopherol and they further stated that though colostrum contributes α -tocopherol to the neonate, its concentration is dependent on the dietary vitamin E level fed to the sow.

Kelly (1993) explained that pathogenesis of hepatosis dietetica; a polymorphous syndrome in pigs is partly related to the generation of free radicals whose noxious effects are normally limited by a system of free radical scavengers of which both vitamin E and selenium are important components.

Tapiero *et al.* (2003) stated that selenium deficiency can contribute to the development of atherosclerotic cardiovascular disease by increased thromboxane B2 and decreased prostacyclin. They further reported that cardiomyopathy in children

and young women ( Keshan disease ) and coronary artery disease are possibly associated with selenium deficiency in human beings.

Oldfield (2003 ) stated that stress factors may increase pig's requirements for both selenium and vitamin E. Further he explained that vitamin E and selenium, would act synergistically in the process of antioxidation.

### *2.4.1.1 Incidence*

Muth (1955) reported cardiac and skeletal myopathy in varying degrees in young calves and lambs of 3 to 4 weeks of age. He also stated that white muscle disease has been recognised in lambs and calves at birth as well as in older animals.

The study conducted on piglets born to vitamin E and selenium deficient sows showed lesions in cardiac tissue of the neonates, and progressive vascular damage from 3 to 12 weeks of age (Sweeny and Brown, 1972).

Ruth and Van Vleet (1974) observed mortality among rapidly growing swine without showing any clinical signs in selenium and vitamin E deficiency.

Nielsen *et al.* (1989) reported Mulberry Heart Disease (MHD) in young pigs without any nutritional deficiency.

Robinson and Maxie ( 1993) reported that MHD has been observed in animals from 3 weeks to 4 years of age. He further stated that among old pigs the incidence is sporadic where as it is short and snappy outbreaks in young pigs.

Van Vleet and Ferrans (2001) reported that the death of 5 week old pigs from the cardiac form of mulberry heart disease is due to selenium and vitamin E deficiency.

Belchev *et al.* (2002) reported death of young piglets due to vitamin E and Selenium deficiency.

Moreira and Mahan (2002) in an experimental study on vitamin E concluded that serum α -tocopherol concentration declines in the post weaned pig and contribute to the onset of the vitamin E deficiency.

### *2.4.1.2 Gross Pathology*

In the lambs with white muscle disease, heart lesions appeared as white sub-endocardial plaques which coalesced and underlie almost entire endocardium with an appearance of white enamel lining to the organ along with extensive peritoneal and ascitic fluid accompanied by marked congestion and edema of the lungs (Muth 1955).

Van Vleet *et al.* (1970) reported the gross lesions in the heart, varying from extensive subepicardial, myocardial and subendocardial ecchymotic and suffusive hemorrhage to a subtle mottling of the diffusely congested subepicardial myocardium with scattered pale streaks or patches of necrosis. Marked hydropericardium, hydrothorax, scant thick fibrin strands in pericardial, peritoneal and pleural effusions were the lesions accompanied in MHD.

Mulberry heart disease and fluid accumulation in the pericardial sac were reported under field conditions of the vitamin E and Se deficiency in pigs (Mahan *et al.*, 1973).

Van Vleet and Ferrans (1977) reported prominent hydropericardium and extensive serosal and myocardial hemorrhage in pigs with spontaneous mulberry heart disease.

Van Vleet *et al.* (1977) reported severe hemorrhage in the epicardial and visceral pleural surface, skeletal muscle degeneration in selenium- vitamin E deficient pregnant heifers.

Bengtsson *et al.* (1978 b) stated that pigs fed on vitamin E deficient diet revealed pericardial, pleural and abdominal cavity transudation, intermuscular transudation, red mottling of ventricular myocardium.

Generalised skeletal, cardiac muscle degeneration were the features reported to be found at necropsy in selenium- vitamin E deficiency (Van Vleet., 1980).

Ferrans and Van Vleet (1985) reported scattered pale streaks in the ventricular myocardium of pigs, necrosis and calcification of the left ventricular free wall, ventricular septum in calves and of right ventricular subendocaridum in lambs affected with selenium and vitamin E deficiency. They also stated epicardial and myocardial hemorrhages as the most striking features.

Necrosis of myocardium and skeletal muscles is a consistent finding in the numerous animal species in spontaneous or experimental selenium- vitamin E deficiency (Van Vleet and Ferrans., 1986).

Nielsen *et al.* (1989) reported the lesions of acute heart failure and circulatory disturbances in MHD. They described the lesion as mulberry heart based on the hemorrhage in the myocardium. They also reported that these pigs showed hemorrhage in subendocardium, serofibrinous fluid in pericardial sac, transudation in the thorax, serohemorrhagic fluid in the abdomen.

Kennedy and Rice (1992) observed lesions in atrial and ventricular myocardium of calves fed poly unsaturated fatty acids. They stated that there was preferential involvement of left ventricular free wall and ventricular septum but subendocardial, intramural and subepicardial myocardium were equally affected. The observed lesions were multiple white striations or diffuse paleness in the myocardium.

Belchev *et al.* (2002) observed liver dystrophy, hemorrhage, white striation in epicardium and endocardium in piglets died of vitamin E and Selenium deficiency.

### *2.4.1.3 Histopathology*

Sweeny and Brown (1972) stated that microscopic alterations in fibroblasts and the extra cellular compartment of cardiac tissue and progressive vascular damage were evident before any marked changes become evident in muscular compartment. They also observed that the predominant capillary changes involved varying degrees of endothelial degeneration, ranging from images showing slight loss of cytoplasmic density to excessive ballooning.

In areas showing minor signs of degeneration in muscles, the arterioles showed marked alterations and capillary and small vessel changes were manifested

by endothelial swelling and loss of ultrastructural characteristics (Sweeny *et al.*, 1972).

Ruth and Van Vleet (1974) reported that fibrinoid degeneration of the arterioles and capillaries in the heart along with mineralization, fibrosis of atrial and ventricular myocardium. The most widespread lesions were found in those pigs with prolonged deficiency.

Van Vleet *et al.* (1977) reported increased numbers of perinuclear lipofuscin granules in many subendocardial purkinje fibers. They further reported vascular changes which included multiple thrombi within necrotic masses of muscle, interstitial edema and focal hemorrhage.

Bengtsson *et al.* (1978 a) reported that myocardial swelling and a granular cytoplasm together with areas of complete necrosis, hyaline vasculosis of the arterioles and capillaries in the myocardium were reported in deficiency of selenium.

Ferrans and Van Vleet (1985) reported areas of myocardial damage had hyaline necrosis with or without calcification, subsequent macrophagic invasion and eventual fibrosis in calves with vitamin E and selenium.

Van Vleet and Ferrans (1986) reported vascular and myocytic lesions in vitamin E and selenium deficiency. Vascular changes included fibrinoid necrosis in intramyocardial arteries and arterioles and numerous microthrombi in myocardial capillaries. Myocardial edema accompanied the vascular lesions. Multifocal hyaline necrosis and calcification was followed by macrophagic invasion and myocardial fibrosis in pigs with prolonged survival.

In an experimental study Kennedy and Rice (1988) reported that vitamin E and selenium deficiency in cattle showed preferential degeneration and necrosis of purkinje cardiocytes. Calves fed deficient diets had sublethal damage characterized histologically by sarcoplasmic accumulation of lipofuscin granules. Necrotic cells were stained deeply with eosin and shrunken with prominent separation between the necrotic cells and apposed normal conduction cells or the surrounding peri- Purkinje connective tissue sheath.

Nielsen *et al.* (1989) described mulberry heart as histologically characterised by myocardial hemorrhage, degeneration of muscle fibers to various degrees, calcification of degenerated fibers and deposition of PAS positive material in arterioles and capillaries.

Myofibrillar lysis, sarcoplasmic vacuolation, granule formation, nuclear enlargement and proliferation in sublethal cardiocytic injury were induced by vitamin E and selenium deficiency. Cells with severe loss of myofibrils had sarcoplasmic vacuoles and granules. PAS positive, acid- fast and sudanophilic granules characteristic of lipofuscin could be demonstrated in degenerated cells. Thickening of tunica media in large arterioles, hyaline material and pyknotic nuclei were reported to be occasionally seen in media of such arterioles. Capillaries had swollen endothelial cells that frequently resulted in apparent luminal occlusion (Kennedy and Rice, 1992).

In pigs fed with diet lacking antioxidants, changes in the heart muscle was reported to be hyaline degeneration of a small proportion of fibers in the left ventricular wall, demonstration of yellowish, acid- fast pigment resembling

lipofuscin and ceroid in the left ventricular wall, subacute plasmacytic- lymphocytic myocarditis and a severe chronic epicarditis ( de Gritz *et al.*, 1994).

Belchev *et al.* (2002) observed hyaline degeneration in heart, skeletal muscles and muscles of diaphragm in experimentally induced vitamin E and selenium deficiency in piglets.

### 2.4.2 Copper

Kopp *et al.* (1983) suggested that a generalized alteration in tissue metabolism occurred as a consequence of dietary copper restriction in rats, resulting in putative cardiomyopathy. They also concluded that the resultant response was similar to the myocardial response to chronic ischemia.

The growing pigs when given a diet consisting entirely of swill with no mineral supplement had developed copper deficiency and anaemia (Pritchard *et al.*, 1985)

Schoenemann *et al.* (1990) reported that the relative cardiac mass of all Cu-deficient pigs was greater and hematocrit was lower than that of animals fed Cu-adequate diets.

In an experimental study conducted by Scholfield *et al.* (1990), copper deficient swine exhibited decreased plasma ceruloplasmin, erythrocyte superoxide dismutase and plasma lysyl oxidase activities and lowered serum copper. The relative heart weight in the copper deficient group was greater than the animals supplemented with copper.

In an experimental study, Vadlamudi *et al.* (1993) reported that copper deficient diet caused reduction in ventricular collagen cross linking which provided the stimulus for the development of cardiac hypertrophy.

Wildman *et al.* (1996) stated that Cu-restricted diet-fed pigs exhibited significantly greater heart: body weight ratio, greater diastolic measures of ventricular wall and internal dimension relative to body weight.

Nath (1997) reported that copper was an essential trace element and has profound influence on cardiomyopathy and heart metabolism.

Concurrent deficiency of copper or manganese in primary selenium deficiency could potentiate the effect of lipid peroxidation in tissues (Tapiero *et al.*, 2003).

### *2.4.2.1 Gross Pathology*

Shields *et al.* (1962) found cardiovascular lesions among 26 swine deficient in copper. The most important cardiac lesions were myocardial infarction and ruptured papillary muscles with intramural hemorrhage in carotid, coronary and other thoracic arteries. They also found that copper deficiency produced greater cardiac enlargement.

Pale skeletal musculature, excessive pericardial fluid and epicardial petechiation adjacent to the coronary groove were the lesions observed in copper deficiency in pigs (Pritchard *et al.*, 1985).

Danks (1989) stated that humans with genetic copper deficiency had elongated and dilated major arteries, rupture and hemorrhage in the vessels.

Rupture of coronary artery, aorta, pulmonary artery can occur in copper deficient young growing swine followed by haemopericardium (Robinson and Maxie, 1993)

Nath (1997) reported that dietary copper restriction produces various cardiac lesions characterized by hemothorax or aneurysms, vascular rupture and muscle weakness in humans.

Klevay (2000) stated that anatomical studies of several species of copper deficient animals revealed cardiac enlargement and rupture, coronary artery thrombosis, myocardial infarction, aortic fissures and rupture.

### *2.4.2.2 Histopathology*

Shields *et al.* (1962) reported medial thickening of the aorta and intramural hemorrhages in carotid, coronary and other thoracic arteries along with aortic fissures and rupture.

The study conducted by Coulson and Carnes (1963) in pigs fed with copper deficient diet, the lesions in the cardiovascular system varied from interruption of the internal elastic lamina in the coronary arteries to myocardial infarction. They observed radially oriented fissures which penetrated the muscular media eventually the adventitia, massive dissections and compression of media eccentrically with in the intact adventitia leading to occlusion of lumen. Small dissecting hemorrhages

with in the media or between the media and the adventitia appeared to be sequelae of such fissures.

Danks (1989) stated subintimal thickening of major arteries with partial occlusion in humans with genetic copper deficiency.

Medeiros *et al.* (1991) reported that glycogen granules, lipid droplets and enlargement were more frequently observed in myofibrils of copper deficient rats. They also explained that valves from Cu-deficient rats had less connective tissue and seemed fragmented in areas.

Nath (1997) reported that dietary copper restriction affects the integrity of the basal lamina of cardiac myocytes and capillaries.

### 2.4.3 Magnesium

#### *2.4.3.1 Gross Pathology*

Heggtveit *et al.* (1964) reported small, pale patches flecked with yellow discolouration in the heart muscle to large areas of necrosis and calcification extending through the entire ventricular wall, congested subepicardial vessels, dilated cardiac chambers in experimental magnesium deficiency.

Myocardial necrosis were usually present as scattered foci of necrosis with calcification and lesion occasionally involved full thickness of the ventricular wall or selective involvement of the inner myocardium in magnesium deficiency (Van Vleet and Ferrans.,1986).

Miller *et al.* (2000) reported necrotizing myocarditis in pigs fed diet low in magnesium and high manganese which resulted in the death of pigs. They further stated that the pigs showed symptoms of paddling their feet, convulsion and hemorrhage from the nose and mouth.

### *2.4.3.2 Histopathology*

Focal areas of myocardial necrosis and inflammation were the microscopic lesions scattered throughout the myocardium predominantly subendocardial regions. The muscle fibers were fragmented, vacuolated; the sarcoplasmic vacuoles contained clusters of minute reddish brown granules and reported to be positive for PAS. These muscle fibers were often accompanied by varying degrees of calcification and proliferation of fibroblasts. Vascular dilatation and hyperemia were common and the distribution of the lesions was not consistently perivascular in magnesium deficiency (Heggtveit *et al.*, 1964).

Necrotic myocytes in magnesium deficiency had extensive mineralization and areas were infiltrated by mononuclear leukocytes and healing of the lesions resulted in residual areas of fibrosis (Van Vleet and Ferrans., 1986).

### 2.4.5 Other Nutritional Deficiencies

Sykes and Moore (1942) reported degeneration of Purkinje fibres characterized by vacuolation as well as the presence of yellowish- brown granules in the cytoplasm due to deficiency of potassium in the diet.

The gross lesions in the hearts of thiamine deficient pigs were dilatation and scattered pale streaks of necrosis in the myocardium. Histologically multifocal myocardial necrosis has been reported in the atria and ventricles (Follis *et al.*, 1943).

The dogs with protein malnutrition had cardiac atrophy with decreased heart weight and decreased myocardial glycogen content. Histologic and ultra structural study of the heart revealed atrophy of myocytes and prominent interstitial edema (Abel *et al.*, 1979).

Neonatal pigs with chronic iron deficiency developed cardiac dilatation and hypertrophy (Lee *et al.*, 1983).

Tanne *et al.* (1994) reported cardiac hypertrophy, marked edema in myocytes, degeneration and discontinuities in myofilaments were common in iron deficiency.

## 2.5 CARDIOMYOPATHY IN STRESS

Van Vleet *et al.* (1977) reported that stress susceptible pigs that were not pre-treated with selenium and vitamin E exhibited severe myocardial damage.

Johansson and Jonsson (1977) investigated pigs died with clinical signs indicative of cardiac failure. They concluded that the myocardial cell damage was caused by different stress factors with liberation of cardio toxic catecholamines.

Turnbull and Cowan (1998) suggested that the physiological and psychological stress were among the probable cause for myocytolysis in the heart.

### 2.5.1 Gross Pathology

Stress induced cardiomyopathy showed mottled appearance in myocardium, myocardial congestion, especially in the papillary muscles of the left ventricle (Van Vleet *et al.*, 1977).

Subepicardial, subendocardial, intramural hemorrhage, hydropericardium, hydrothorax, poorly defined brown areas in left ventricular myocardium were the gross lesions recorded in porcine stress syndrome (Johansson and Jonsson., 1977).

### 2.5.2 Histopathology

Myocardial fibers had prominent hypercontraction bands, pyknotic nuclei and basophilic granular sarcoplasm that contained small vacuoles, edematous interstitium, congestion of blood vessels and endothelial proliferation in stress susceptible pigs (Van Vleet *et al.*, 1977).

Johansson and Jonsson (1977) demonstrated disruption of the regular pattern of cross striations in myocardium, presence of homogenous eosinophilic transverse bands with intervening areas of fine granulation and myolysis in stress induced cardiomyopathy in pigs. They also stated that the cytoplasmic segmentation and granulation were the most common features of the lesion.

Acute degenerative changes, loss of cross striations, vacuolization, interstitial edema, cytoplasmic eosinophilia, vascular congestion in myocardium, nuclear pallor, cytoplasmic fragmentation, wavy fibres and hyalinized fibres were observed in stress induced cardiomyopathy ( Turnbull and Cowan 1998 ).

## 2.6 LABORATORY INVESTIGATIONS ON CARDIOMYOPATHIES

The mean hepatic selenium concentrations in pigs affected with nutritional myopathy were reported to be low compared with that of normal pigs (Moir and Masters, 1979).

Korpela (1990 a) reported lower hepatic selenium concentration in pigs that died suddenly of microangiopathy compared to the healthy pigs.

Nolan *et al.* (1995) reported that lipid peroxidation in dietetic microangiopathy was judged by reduced glutathione concentration in tissues especially in skeletal muscle.

Wildman *et al.* (1996) stated that the pigs fed on Cu-restricted feed exhibited, lower liver and serum Cu concentrations compared to the Cu-adequate diet-fed pigs.

Laboratory evaluation of myocardial necrosis was based on measuring the blood levels of intracellular molecules that leaked out of fatally injured myocardial cells through damaged cell membrane especially cardiac troponin T and troponin I (Schoen, 2004).

### 2.6.1 Enzyme Studies in Cardiomyopathy

Kuttler and Marble (1958) observed elevation of SGOT (Serum Glutamic Oxaloacetate Transferase) level in both cardiac and skeletal type of white muscle diseases.

Increase in the activity of GOT (Glutamic Oxaloacetate Transferase), CPK (Creatine Phospho Kinase) and LDH (Lactic dehydrogenase) were consistently the first to increase in pigs deficient in selenium and vitamin E (Ruth and Van Vleet, 1974).

Plasma AST and CPK activities were markedly increased in the pigs that were not pre-treated with selenium and vitamin E during experimentally induced cardiomyopathy (Van Vleet *et al.*, 1977).

Determination of blood glutathione peroxidase activity was considered as an effective test to screen animals for selenium deficiency (Van Vleet., 1980).

Smith *et al.* (1983) reported that estimates of myocardial infarct (MI) size based on plasma creatine kinase (CK) can be used for prognosis and in the assessment of therapy designed to salvage ischemic myocardium.

Serum creatine kinase (CK) and lactic dehydrogenase (LD) isoenzyme activities were measured in blood serum of pigs having myocardial damage and skeletal muscular lesions induced by restraint stress. But there was no significant increase reported in the serum CK-BB (CK-1) or CK-MB (CK-2) activity whereas a pronounced elevation of the CK-MM (CK-3) activity was found, particularly in the stress-sensitive animals (Tolling and Jonsson, 1983).

Coles (1986) reported that white muscle disease is associated with elevation of AST in sheep and calves that were depleted of selenium and vitamin E and the values of AST varied from 400 to 4000 IU/L.

D'Allaire and DeRoth (1986) reported that stress susceptible pigs had higher levels of serum total creatine kinase and MM isoenzyme compared to resistant pigs.

Increase in the activities of serum GOT (Glutamic Oxaloacetate Transferase), CPK (Creatine Phospho Kinase) and LDH (Lactic Dehydrogenase) were noticed in cardio pathological alterations of calves (Osame *et al.*, 1989).

Stevenson and Jones (1989) evaluated the relationship between plasma alpha tocopherol concentrations, creatine kinase activity. They stated that vitamin E deficient animals were clinically healthy but had elevated CK activities and low alpha tocopherol concentration. The level of creatine kinase activities decreased in response to treatment with vitamin E.

Awaji *et al.* (1990) utilized myocardial isoenzyme activity of creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) as an indicator of myocardial necrosis in animals with diabetic cardiomyopathy.

CK, AST, LDH were three enzymes used in diagnosis of myocardial infarction and LDH activity could increase 5 to 10 times more in myocardial infarction. (Vasudevan and Sreekumari, 1995).

Elevations in total CK activities have been reported in selenium- vitamin E deficiencies of swine, cattle and sheep. A highest concentration of AST was found in cardiac and skeletal muscle. Tissues with essentially aerobic metabolism, such as heart muscle contained mostly heart specific isoenzyme and tissues with flexible metabolic properties especially skeletal muscle contained predominantly the muscle specific isoenzyme (Cardinet, 1997).

Kramer and Hoffmann (1997) reported that serum creatine kinase isoenzymes serve as sensitive and specific indicators of cardiac infarction. He further reviewed

that CK isoenzymes were commonly used to quantify cardiac and muscular damage. AST was a sensitive marker of soft tissue damage and estimation of its level complement CK changes.

Blood levels of CK and LDH could be assessed to evaluate myocardial necrosis. Though the preferred biomarkers for myocardial damage were cardiac specific proteins, particularly troponin, CK- MB remained the best alternative to troponin measurement (Schoen, 2004).

# *Materials and Methods*

# 3. MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology (CEP), College of Veterinary and Animal sciences, Mannuthy to investigate the pathology of cardiac disorders in pigs reared on swill.

## 3.1 MATERIALS

### 3.1.1 Prevalence Study

The autopsy records maintained during the past 3 years (2001 -2003) at the Centre of Excellence in Pathology, College of Veterinary and Animal sciences, Mannuthy were screened to assess the prevalence of cardiac disorders in pigs. The lesions were classified and the prevalence was assessed for the pigs maintained on swill to concentrate fed.

### 3.1.2 Data Collection

The history with regard to the management practices, clinical symptoms of pigs which were brought for autopsy to the CEP during the period of study (January 2004-June 2005) were collected using a questionnaire by interaction with farmers.

### 3.1.3 Sample Collection

One hundred pigs maintained on swill and twenty pigs maintained on standard concentrate feed brought for slaughter at Meat Technology Unit, College of Veterinary and Animal Sciences were utilized for the study. Blood samples were collected at the time of slaughter and the serum was separated and stored at 0 degree. Representative samples of tissues from all the chambers of the heart were collected in 10% Neutral Buffered Formalin

## 3.2 METHODS

### 3.2.1 Relative Weight of the Heart

The body weight of the carcass and the weight of the heart were recorded. The relative weight of the heart was determined by the method used by Robinson and Maxie (1993).

### 3.2.2 Gross Examination

The heart was subjected to detailed gross examination. The pericardium was examined and dissected out. The gross changes in the epicardium were recorded. The heart was opened by an incision through the myocardium, parallel to the descending coronary arteries, from the apex of the heart to the base on either side thus exposing all the chambers including valves. The presences of any abnormalities in the myocardium, endocardium and valves were recorded.

### 3.2.3 Histopathological Examination

The tissues were processed by routine paraffin embedding techniques (Sheehan and Hrapchak, 1980). Sections were cut at 4 micron thickness and stained with routine Haematoxylin and Eosin (Bancroft and Cook, 1995) for histopathological studies. Special staining techniques like Gomori's trichrome, Mallory's PhosphoTungstic Acid-Haematoxylin, Alizarin red, Periodic acid – Schiff were done whenever required as per the method described by Luna (1968). The stained sections were examined in detail under light microscope and the lesions were classified.

### 3.2.4 Enzyme Analysis

The serum samples were analysed for enzymes such as Aspartate amino transferase (Ecoline, Merck Ltd), Creatine Kinase (Labkit, Chemelex) and Lactic dehydrogenase (Ecoline, Merck Ltd) with Microlab 2000 (semi-automatic blood analyser).

# *Results*

# 4. RESULTS

## 4.1 PREVALENCE STUDY

A total number of 787 pig carcasses were examined between 2001 to 2003 at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy. Out of these cases, 41 carcasses of pigs maintained on concentrate and 26 carcasses of pigs fed on swill showed cardiac lesions. The lesions recorded were cardiac dilatation, myocardial degeneration, fibrinous pericarditis, hydropericardium, hemopericardium and cardiac tamponade (Table. 1 and Table. 2).

## 4.2 HISTORY AND CLINICAL SIGNS

The pigs brought for autopsy by the local farmers were fed on swill which included chicken wastes, wastes from the vegetable market and hotels. They had a history of sudden death without showing any clinical symptoms. The animals were reported to be apparently healthy with normal appetite. The pigs were cross bred and between the age group of 3 to 9 months. The carcasses of pigs fed on concentrate were mainly brought from the University pig farm, Mannuthy. The carcasses examined at the slaughter house consisted both swill fed and concentrate pigs.

## 4.3 ENZYME ANALYSIS

The range and mean enzyme levels in swill fed and concentrate fed pigs are shown in table 3. The serum from swill fed pigs revealed a range of 57 to 98 U/L ( mean : 64.6 U/L) for AST, 103 to 316 ( mean : 213.2 U/L) for LDH and 142 to 289 ( mean : 183.3 U/L) for CK. The serum of concentrate fed pigs revealed a

range of 49 to 78 U/L ( mean : 61.6 U/L) for AST, 116 to 216 ( mean : 160.4 U/L) for LDH and 133 to 216 ( mean : 172.2 U/L) for CK. The mean enzyme values are shown in Figure 1.

## 4.4 RELATIVE WEIGHT OF THE HEART

The relative heart weight in pigs of the two groups are given in table 4 and 5. The weight of the animal and the heart were measured and the relative weight of the heart was calculated. Relative organ weight was 0.318 per cent for animals reared on swill and 0.313 per cent for the animals reared on concentrate feed. The relative organ weight for the animals with hypertrophic heart is given in Table 6.

## 4.5 GROSS PATHOLOGY

A total number of 143 hearts from the swill fed and 360 hearts from concentrate fed pigs were examined. Out of these 66 (46.2 per cent) of the swill fed and 22 (6.1 percent) cases of concentrate fed pigs revealed gross lesions. Among the autopsied pigs higher incidence of cardiac disorders were observed in swill fed pigs (65 per cent) compared to 6.2 per cent in concentrate fed pigs. The clinical history and the data on the management practices were collected from the owner of the farm which reported the mortality. The lesions observed were varying degrees of hemorrhage, hydropericardium, hemopericardium, fibrinous pericarditis, myocardial degeneration, cardiac hypertrophy, cardiac dilatation and valvular endocarditis. The lesions and their prevalence are given in Table. 7. Distribution of gross cardiac lesions in swill fed pigs are given in Figure 2.

### 4.5.1 Hemorrhage

#### *4.5.1.1 Petechial hemorrhage*

Varying degrees of hemorrhages were observed in 42 cases out of 143 swill fed animals examined. Petechial hemorrhages were seen mostly in the epicardium, endocardium of the ventricles and atria. Two cases revealed petechial hemorrhages in epicardial fat around the coronary groove. Petechial hemorrhage in papillary muscles was found in 15 cases out of 29 swill fed animals which showed hemorrhagic lesions.

#### *4.5.1.2 Echymosis*

Extensive areas of hemorrhages were observed in eight cases. Echymotic hemorrhage was noticed in three cases each in the epicardium (Fig. 3) and in the myocardium (Fig. 4) and one case in the endocardium (Fig. 5).

### 4.5.2 Congestion

Congestion of coronary vessels was seen in three cases. The coronary vessels were engorged with blood in these cases (Fig. 6).

### 4.5.3 Ventricular Hypertrophy

Ventricular hypertrophy of varying degree was observed in three swill fed autopsied cases. The heart was enlarged due to increased ventricular musculature. These changes were observed mostly in the left ventricle (Fig. 7). The lumen of the

ventricle was reduced considerably. The ventricular walls were thickened and firm. The relative organ weight of the hearts were 0.47 per cent to 0.53 per cent in these cases.

### 4.5.4 Cardiac Dilatation

Cardiac dilatation was observed in six cases. The right ventricular wall was thin and flabby and there was widening of the ventricular lumen. The apex of the heart was rounded, giving a rounded appearance.

### 4.5.5 Degeneration

Mild to moderate degree of degenerative changes were observed in 64.3 per cent of autopsied cases which were swill fed and showed marked difference when compared to slaughtered animals (36.8 per cent). The myocardium of these animals showed alternative areas of pale or yellow discoloration giving mottled appearance (Fig. 8). These changes were predominant in the subepicardial and in the intra- mural region of the left ventricular free wall.

### 4.5.6 Hydropericardium

Hydropericardium was observed in 12 (42.8 per cent) autopsied cases which were swill fed. The pericardial sacs were distended and filled with yellowish straw coloured fluid measuring approximately 100-150 ml (Fig. 9). Only 5 cases among 340 pigs maintained on concentrate feed showed hydropericardium.

### 4.5.7 Hemopericardium

In 8 (28.6 per cent) cases hemopericardium were observed in autopsied swill fed pigs. These cases showed distended pericardial sac and filled with blood measuring approximately 100 to 150 ml. In three cases blood clots were seen encircling the heart giving rise to cardiac tamponade (Fig. 10)

### 4.5.8 Fibrinous Pericarditis

Fibrinous pericarditis was observed in three cases. The pericardium was thick and opaque. These cases revealed loss of transparency and adhesion of parietal pericardium to the visceral pericardium (Fig. 11) in which the attachments were torn away when the pericardial sac was opened. Extensive fibrin deposits were observed in all cases. The examination of blood smear revealed bipolar staining organisms resembling Pasteurella in one case.

### 4.5. 9 Serous Atrophy of Epicardial Fat

Two pigs revealed serous atrophy of the epicardial fat. The adipose tissue around the coronary groove was gelatinous nature with mild edema.

### 4.5.10 Vegetative Endocarditis

Vegetative endocarditis was observed in one case. The gross lesion observed was large, friable, grayish masses adhered to the valvular endocardium (Fig. 12). These masses were found in the right and left atrio- ventricular valves.

## 4.6 HISTOPATHOLOGY

The occurrence of various cardiac lesions at histopathological level is represented in Figure 13.

### 4.6.1 Fibrinous Pericarditis

There was thickening of the pericardium with fibrinous exudates in three cases. Severe degree of infiltration with inflammatory cells could be observed (Fig. 14). The inflammatory cells, predominantly mononuclear cells and scattered polymorphonuclear cells were found in these cases (Fig. 15).

### 4.6.2 Epicardium

#### *4.6.2.1 Hemorrhage*

The extravasated erythrocytes were seen as diffuse collections in the subepicardial adipose tissue or between the connective tissue layers of the epicardium (Fig. 16). There was severe degree of hemorrhage extending from the epicardium into the myocardium in two cases.

#### *4.6.2.2 Serous atrophy of epicardial fat*

Serous atrophy of the epicardial fat was recorded in three cases. There was variation in size of adipocytes and homogenously pink stained serous fluid in between the fat vacuoles (Fig. 17).

### 4.6.3 Myocardium

#### *4.6.3.1 Vascular changes*

4.6.3.1.1 Congestion

There was varying degrees of congestion in the myocardial vessels in 20 cases. Intramural coronary vessels were engorged with erythrocytes (Fig. 18). The congestion was pronounced in the left ventricular myocardium.

4.6.3.1.2 Hemorrhage

Out of the 143 hearts examined 76 cases revealed mild to moderate or severe degree of hemorrhage. The extravasated erythrocytes were seen in between the myocardial fibres and in the interstitial tissue (Fig. 19).

#### *4.6.3.2 Cardiomyopathies*

4.6.3.2.1 Dilatation

Attenuated wavy fibres along with widening of the intermuscular spaces were seen in six cases. The muscle fibres were thin and widely separated giving them a wavy appearance (Fig. 20). A mild degree of interstitial fibrosis was seen in two cases.

4.6.3.2.2 Hypertrophy

*Hypertrophy of the muscle fibres* was recorded in three cases. The muscle fibres were enlarged and there was disarray of the muscle fibres (Fig. 21). The nuclei of the myocytes were enlarged. The adjacent areas showed crowding of muscle fibers with a reduction in the intermuscular spaces.

4.6.3.2.3 Hyaline degeneration

Hyaline degeneration was recorded in five cases. Cardiac muscle fibres showed homogenously pink stained glassy material as focal, multifocal or diffuse degenerative change (Fig. 22). There was severe degree of hemorrhage in the myocardium around the hyalinised area in three cases.

4.6.3.2.4 Fatty infiltration

Fatty infiltration was recorded in three cases. Of these, two cases showed extensive changes in the myocardium of the left ventricle where adipocytes were deposited in between the myocardial fibers (Fig. 23). There was diffuse infiltration of fat vacuoles replacing the myofibers and formation of fat cyst. The fat vacuoles were spherical to oval in shape of varying size. These fat vacuoles appeared as empty spaces in the haematoxylin and eosin stain.

4.6.3.2.5 Vacuolar degeneration

Vacuolar degeneration was recorded in 20 cases. Affected fibres had swelling along with varying degrees of sarcoplasmic vacuolization. Extensive vacuolization occupying the entire sarcoplasm was evident in few cases (Fig. 24).

Loss of myofibrils leading to sarcoplasmic vacuolations was conspicuous when stained with Phosphotungstic acid – Haematoxylin stain. In this staining, myofibrils were stained deep blue and sarcoplasmic areas with myofibril loss failed to stain and appeared as empty spaces (Fig. 25).

4.6.3.2.6 Myocardial necrosis

Myocardial necrosis of varying degrees was found in 72 cases out of 100 slaughtered swill fed pigs. Myocardial fibres were swollen, hypereosinophilic and fragmented at places with indistinct striations (Fig. 26). The fragmentation of myocardial fibers was evident on staining with Phosphotungstic acid – Haematoxylin stain (PTAH), where fragmented areas stained feebly and normal myocardial fibers stained deep blue (Fig. 27). The nuclei of the myocardial cells showed varying degrees of pyknosis and karyolysis along with perinuclear vacuolations. Healing of myocardial necrosis by fibrous tissue proliferation was evident in two swill fed pigs. These cases revealed proliferation of connective tissues and capillaries in between the muscle bundles (Fig. 28). These connective tissues were stained reddish brown with PTAH stain (Fig. 29 and 30) and green with Gomori's trichrome stain (Fig. 31).

*4.6.3.4 Myocarditis*

Focal myocarditis was observed in two cases. There was infiltration of the myocardium with mononuclear cells along with degenerative changes (Fig. 32). Infiltration with eosinophils along with severe degree of myocytolysis, fragmentation of myocardial fibres and perinuclear vacuolations were seen in one case (Fig. 33).

**4.6.4 Purkinje Fibres**

#### *4.6.4.1 Degeneration*

Degenerated Purkinje fibres were seen in 21 cases. The Purkinje fibres appeared swollen and cytoplasm appeared vacuolated (Fig. 34).

### 4.6.5 Coronary Vessels

#### *4.6.5.1 Hyaline degeneration*

Hyaline degeneration of the intramural coronary vessels was recorded in three cases. There was severe degeneration in the medial layer of the intramural artery in the ventricle wall. The vessel was swollen and had a homogenous hyalinized appearance in the focal areas (Fig. 35).

#### *4.6.5.2 Hyperplasia of the vessel wall*

Hyperplasia of the vessel wall was recorded in four cases. There was marked increase in the thickness of the wall in the case of coronary artery due to thickening of the medial layer (Fig. 36). The lumen of the vessel was reduced considerably. There was perivascular fibrosis in two cases.

#### *4.6.5.3 Thrombus*

An intramural coronary artery in the ventricle wall had thrombus in two cases. The thrombus contained pink stained glassy material embedded with fibrin and cellular infiltrates (Fig. 37). The fibrin stained blue with PTAH staining (Fig. 38).

### 4.6.6 Cardiac Valves

#### *4.6.6.1 Vegetative endocarditis*

The vegetative lesion in the cardiac valve was recorded in one case. The valve was enveloped by a mass of coagulated necrotic material mixed with fibrin, small numbers of mixed inflammatory cells and random islands of bacteria (Fig. 39). This proliferated necrotic coagulum was subtended by large numbers of mononuclear and scattered polymorphonuclear inflammatory cells.

Table 1. Prevalence of cardiac lesions in concentrate fed pigs

| Year | Total No of Pigs | No. of cases with cardiac lesions. | Hemorrhage | Degeneration | Valvular changes | Hydroperi-cardium | Hemoperi cardium | Fibrinous Pericarditis | Cardiac hypertrophy | Cardiac dilatation |
|---|---|---|---|---|---|---|---|---|---|---|
| 2001 | 270 | 10(3.7) | 6(60.0) | 2(20.0) | 1(10.0) | 3(30.0) | 2(20.0) | 1(10.0) | 2(20.0) | 2(20.0) |
| 2002 | 245 | 15(6.1) | 5(33.3) | --- | --- | --- | 2(13.3) | 3(20) | 3(20) | 4(26.6) |
| 2003 | 206 | 16(7.8) | 6(37.5) | 4(25.0) | --- | --- | --- | 2(12.5) | --- | 5(31.3) |

* The values in parenthesis indicates percentage

Table 2. Prevalence of cardiac lesions in swill fed pigs

| Year | Total No of Pigs | No. of cases with cardiac lesions. | Hemorrhage | Degeneration | Valvular changes | Hydroperi-cardium | Hemoperi cardium | Fibrinous Pericarditis | Cardiac hypertrophy | Cardiac dilatation |
|---|---|---|---|---|---|---|---|---|---|---|
| 2001 | 30 | 10(3.7) | 3(30.0) | 2(20.0) | --- | 2(20.0) | 1(10.0) | --- | --- | 3(30.0) |
| 2002 | 15 | 6(40.0) | 3(50.0) | 2(33.2) | 1(16.6) | 2(33.2) | 1(16.6) | --- | --- | --- |
| 2003 | 21 | 10(7.8) | 3(30) | --- | --- | 3(30.0) | 4(40.0) | 3(30.0) | 1(10) | 3(30.0) |

* The values in parenthesis indicates percentage

Table 3. Levels of AST, LDH and CK in swill fed and concentrate fed animals.

| S.NO | Enzymes | Swill fed | | Concentrate fed | |
|---|---|---|---|---|---|
| | | Range U/ L | Mean U/ L | Range U/ L | Mean U/ L |
| 1 | Aspartate amino transferase | 37-98 | 64 | 49-78 | 61 |
| 2 | Lactic dehydrogenase | 103-316 | 213 | 116-216 | 160 |
| 3 | Creatine kinase | 142-289 | 188 | 133-216 | 172 |

Table 4 . Relative weight of the heart in pigs maintained on swill

| S.NO | Body weight (kg) | Heart weight ( Kg) | Relative Organ weight(%) |
|---|---|---|---|
| 1 | 77 | 0.216 | 0.28 |
| 2 | 67 | 0.194 | 0.28 |
| 3 | 61 | 0.226 | 0.37 |
| 4 | 65 | 0.216 | 0.33 |
| 5 | 60 | 0.198 | 0.33 |
| 6 | 68 | 0.224 | 0.32 |
| | | Mean | 0.318 |

Table 5. Relative weight of the heart in pigs maintained on concentrate feed

| S.NO | Body weight (kg) | Heart weight ( Kg) | Relative Organ weight(%) |
|---|---|---|---|
| 1 | 62 | 0.218 | 0.35 |
| 2 | 60 | 0.194 | 0.33 |
| 3 | 72 | 0.208 | 0.28 |
| 4 | 64 | 0.196 | 0.31 |
| 5 | 74 | 0.226 | 0.31 |
| 6 | 71 | 0.214 | 0.30 |
| | | Mean | 0.313 |

Table 6. Relative weight of the heart of animals with ventricular hypertrophy

| S.NO | Body weight (kg) | Heart weight ( Kg) | Relative Organ weight(%) |
|---|---|---|---|
| 1 | 80 | 413 | 0.516 |
| 2 | 118 | 626 | 0.530 |
| 3 | 92 | 432 | 0.470 |

TABLE 7. PREVALENCE OF CARDIAC LESIONS IN PIGS.

| Type of pigs | Source of carcass | Total No of cases | No. of cases with cardiac lesions | Haemorrhage | Degeneration | Valvular changes | Hydro pericardium | Hemo pericardium | Fibrinous pericarditis | Cardiac hypertrophy | Cardiac dilatation |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Swill fed | Slaughter house | 100 | 38 (38) | 29 (76.3) | 14 (36.8) | --- | --- | --- | --- | --- | --- |
| | Post mortem ( CEP) | 43 | 28 (65.1) | 13 (46.4) | 18 (64.3) | 1 (3.57) | 12 (42.8) | 8 (28.6) | 3 (10.7) | 3 (10.7) | 6 (21.4) |
| | **Total** | **143** | 66 (**46.2**) | 42 (63.6) | 32 (48.4) | 1 (1.5) | 12 (18.1) | 8 (12.1) | 3 (4.5) | 3 (4.5) | 6 (9.0) |
| Concentrate fed | Slaughter house | 20 | 1 | 1 | --- | --- | --- | --- | --- | --- | --- |
| | Post mortem ( CEP) | 340 | 21 (6.2) | 8 (38.1) | 8 (38.1) | --- | 3 (14.3) | 1 (4.8) | --- | --- | 3 (14.2) |
| | **Total** | **360** | 22 (**6.1**) | 9 (40.9) | 8 (36.3) | --- | 3 (13.6) | 1 (4.5) | --- | --- | 3 (13.6) |

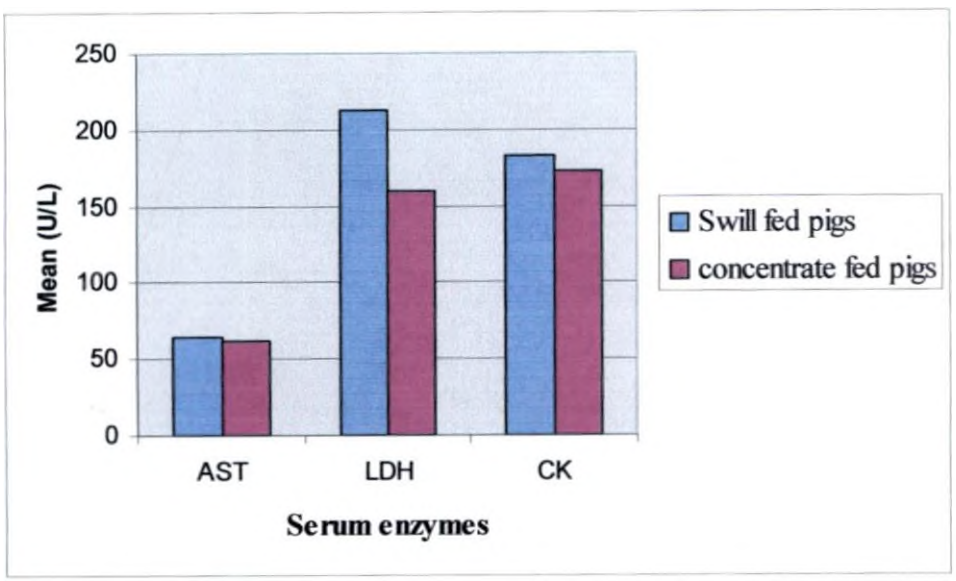


Fig. 1. Mean values of AST, LDH and CK in swill fed and concentrate fed pigs.

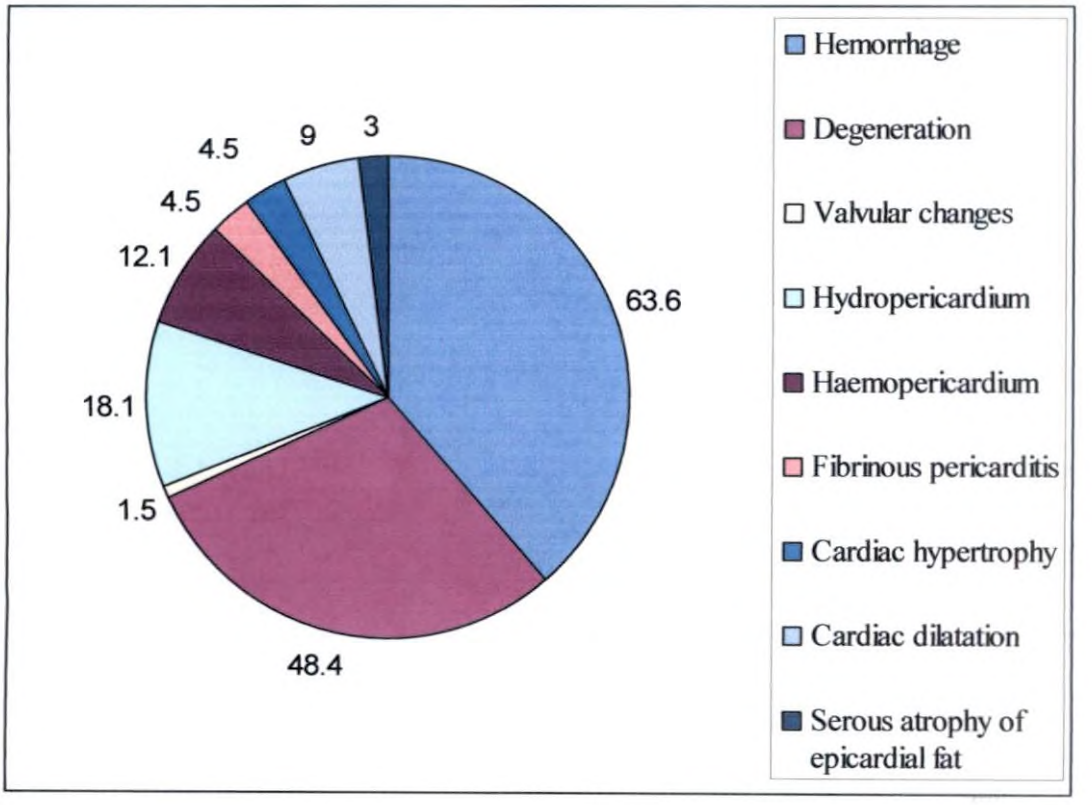


Figure 2. Distribution of different cardiac lesions in swill fed pigs ( In Percentage)

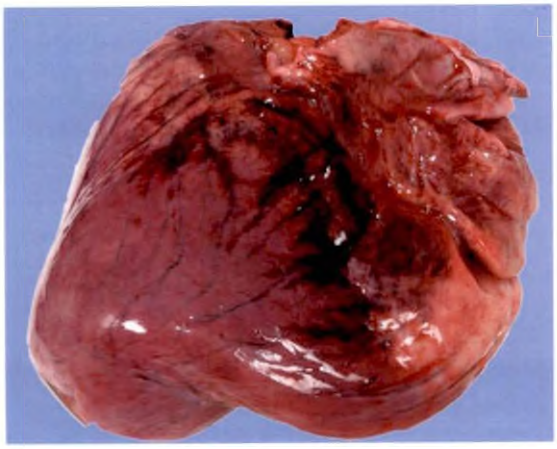

Fig.3

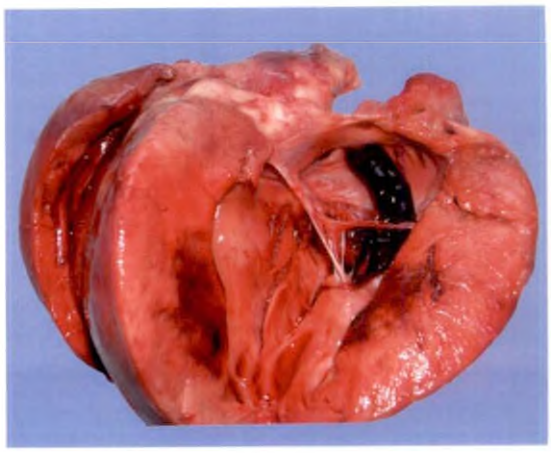

Fig.4

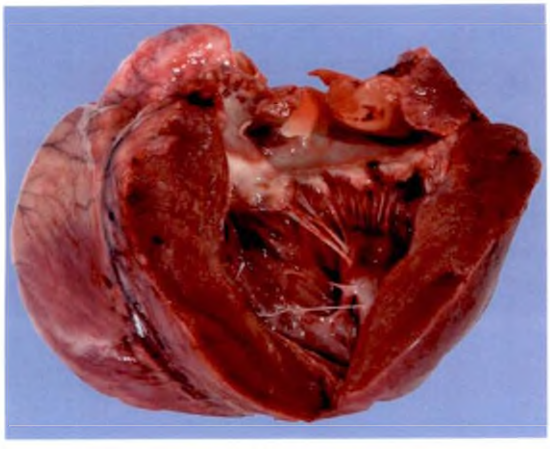

Fig.5

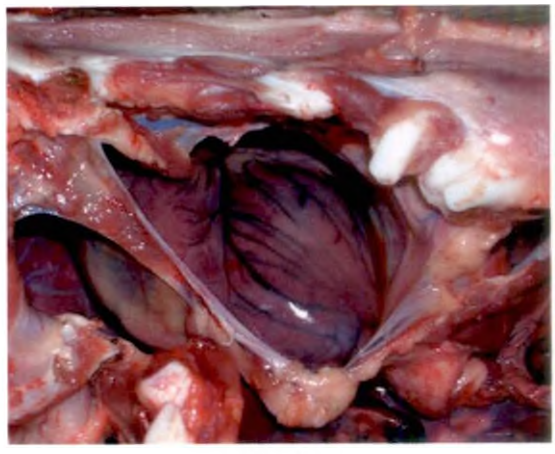

Fig.6

Figure 3. Epicardial hemorrhage in swill fed pig.

Figure 4. Extensive myocardial hemorrhage.

Figure 5. Extensive endocardial hemorrhage in swill fed pig.

Figure 6. Congestion of coronary vessels.

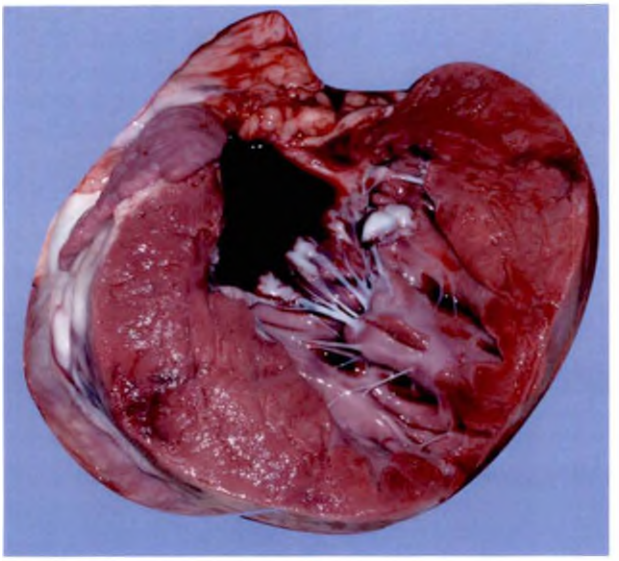

Fig.7

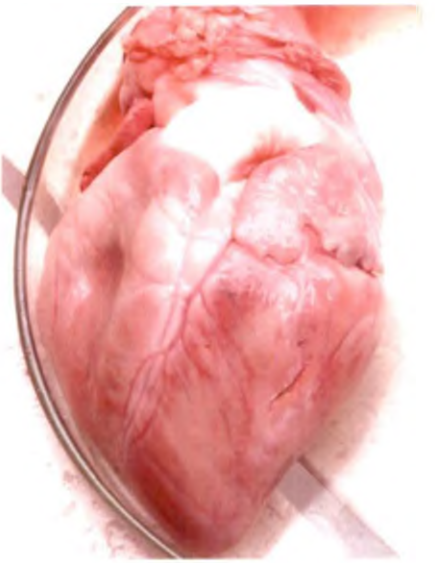

Fig.8

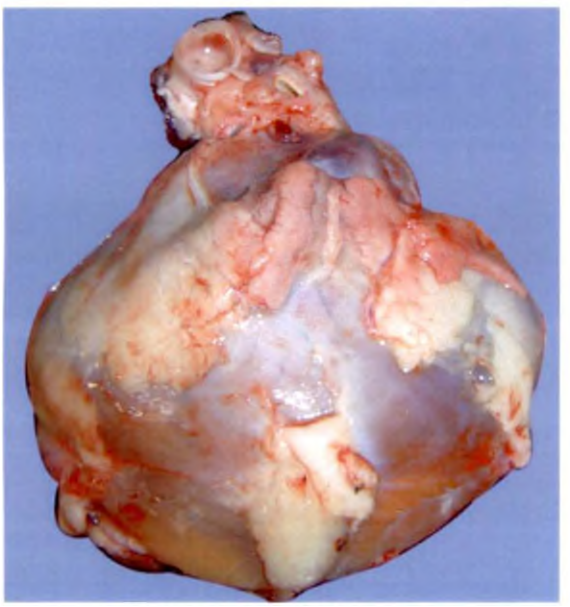

Fig.9

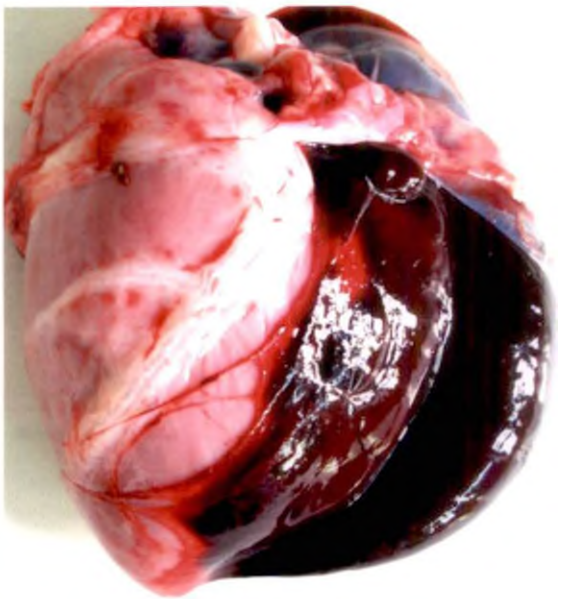

Fig.10

Figure 7. Ventricular hypertrophy in a swill fed pig

Figure 8. Myocardial necrosis in a swill fed pig.

Figure 9. Hydropericardium in a swill fed pig.

Figure 10. Cardiac tamponade in a swill fed pig.

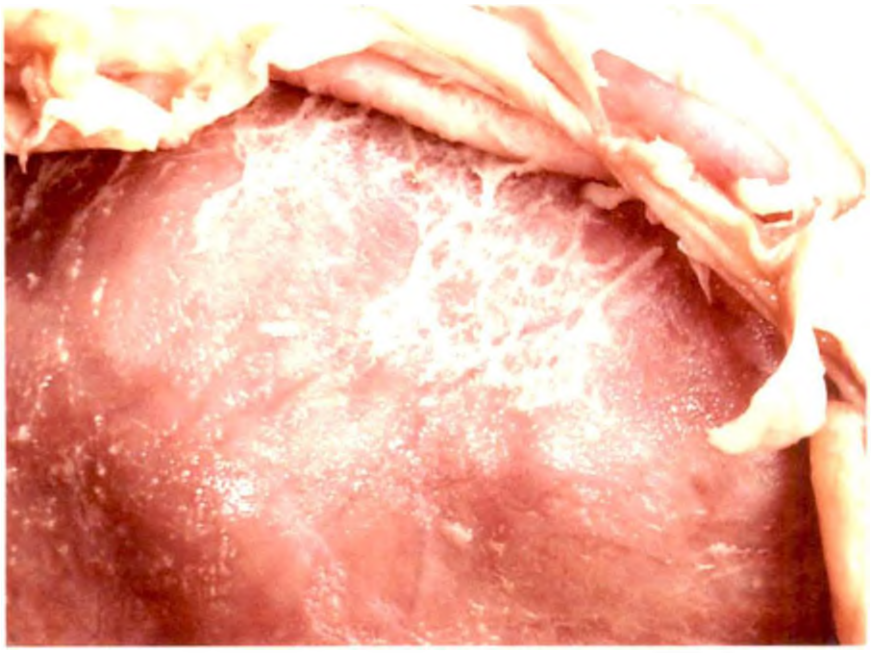

Figure 11. Fibrinous pericarditis. Extensive fibrin deposits in the visceral pericardium adhering the parietal percardium.

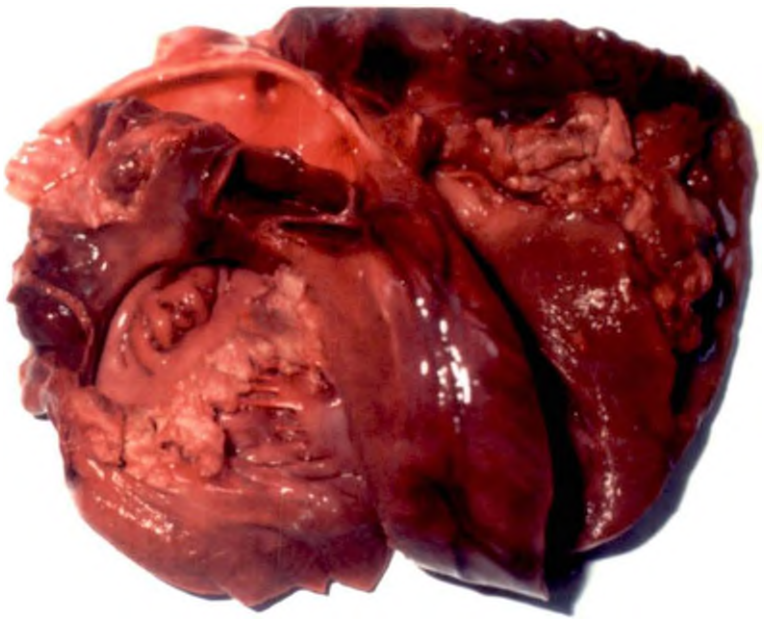

Figure 12. Vegetative endocarditis in a swill fed pig. Large and grayish masses adhered to the atrio ventricular valves.

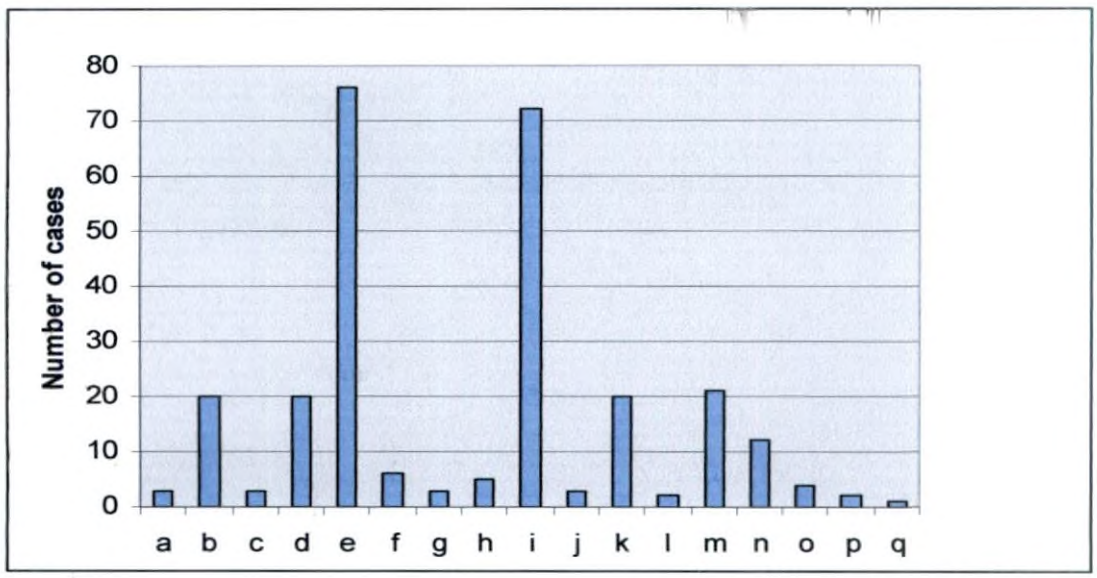


Figure 13. Distribution of various cardiac lesions in swill fed pigs.

a. Fibrinous pericarditis
b. Epicardial hemorrhage
c. serous atrophy of epicardial fat
d. Congestion of myocardial vessels
e. Myocardial hemorrhage
f. Cardiac dilatation
g. Cardiac hypertrophy
h. Hyaline degeneration
i. Myocardial necrosis
j. Fatty change
k. Vacuolar degeneration
l. Myocarditis
m. Purkinje fibre degeneration.
n. Fibrinoid degeneration of vessel.
o. Hyperplasia of the vessel wall.
p. Thrombus in the coronary vessels.
q. Vegetative endocarditis.

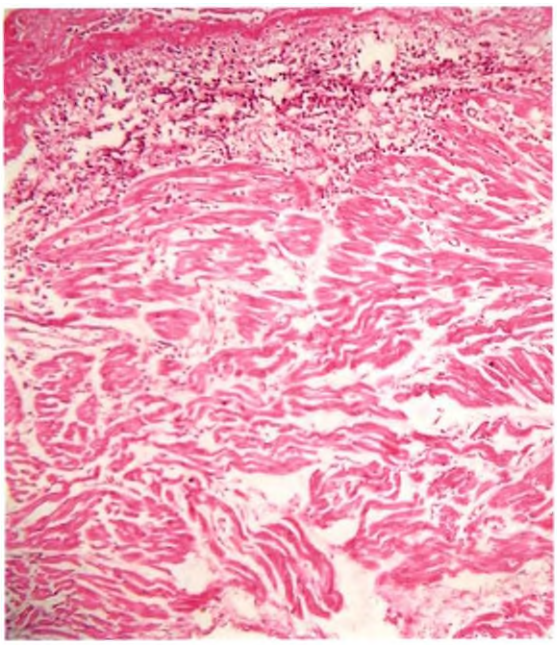

Fig.14

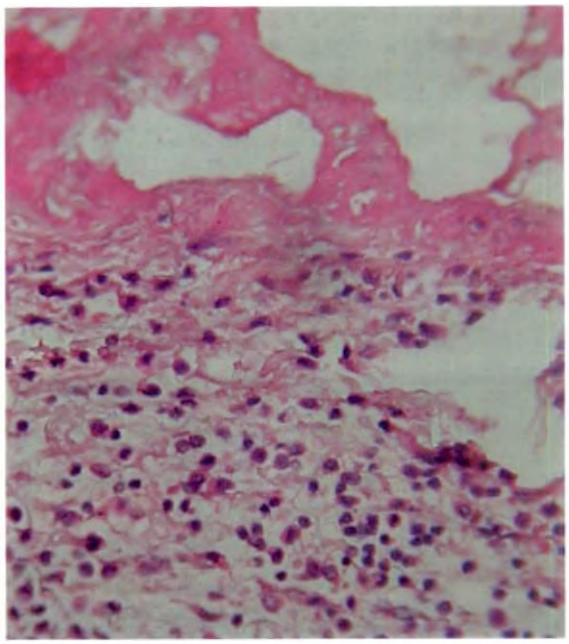

Fig.15

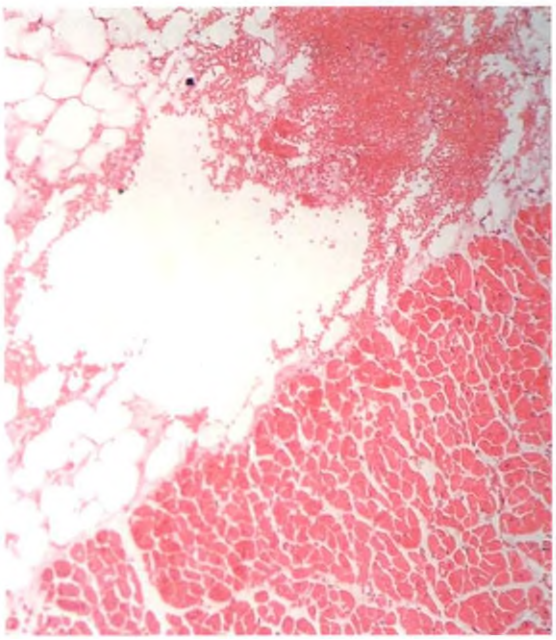

Fig.16

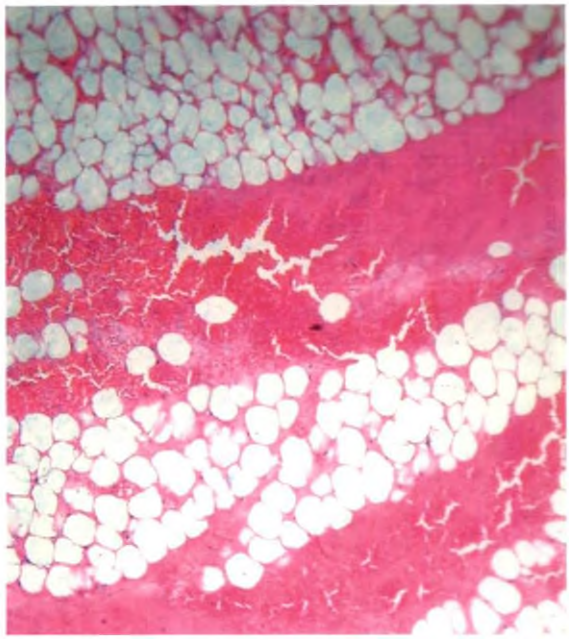

Fig.17

Figure 14. Fibrinous pericarditis. Thickening of the pericardium with fibrinous exudates and severe degree of infiltration with inflammatory cells - H & E x 100.

Figure 15. Fibrinous pericarditis. Infiltration of the pericardium with mononuclear cells - H & E x 400.

Figure 16. Epicardial hemorrhage. Diffuse collections of extravasated erythrocytes in between adipocytes of epicardial fat - H & E x 100.

Figure 17. Serous atrophy of epicardial fat. Variation in size of adipocytes and accumulation of serous fluid in between the adipocytes - H & E x 100.

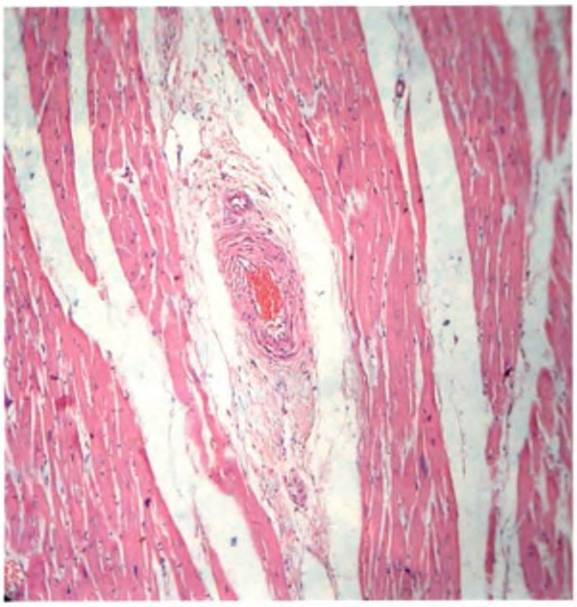

Fig.18

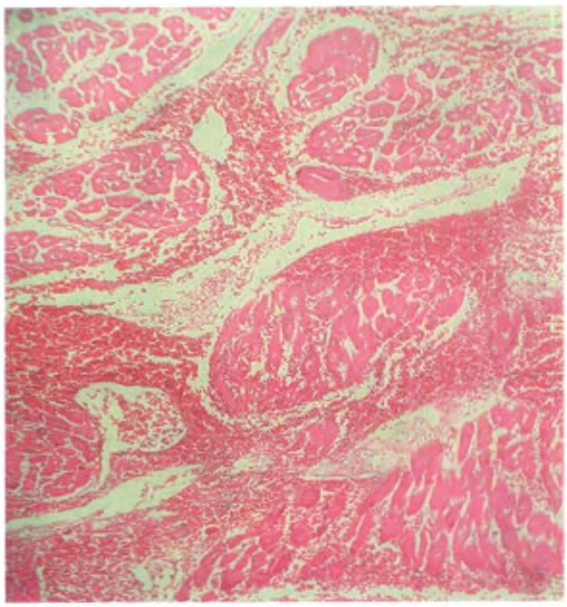

Fig.19

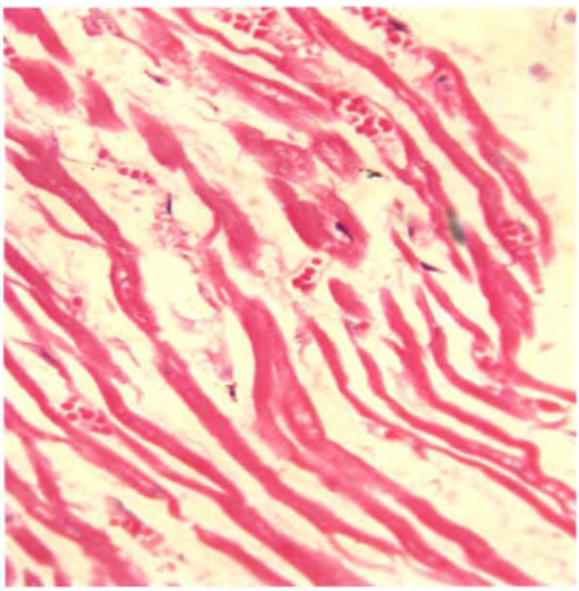

Fig.20

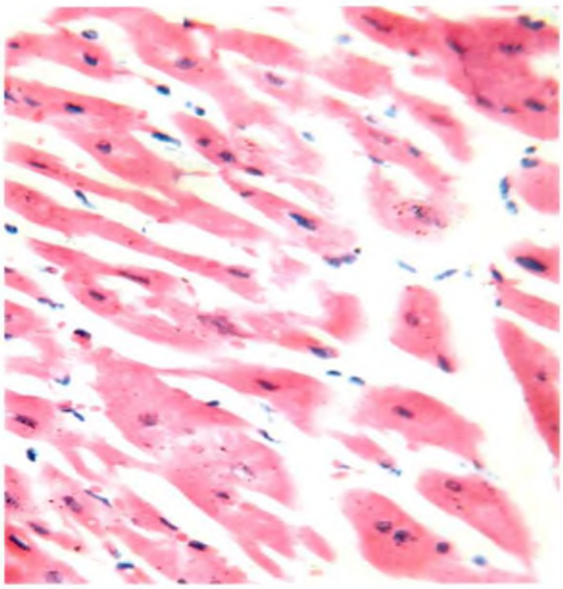

Fig.21

Figure 18. Myocardial blood vessels engorged with erythrocytes. H & E x 100.

Figure 19. Myocardial hemorrhage. Extravasated erythrocytes in the interstitial tissue. H & E x 100.

Figure 20. Cardiac dilatation. Presence of thin, attenuated wavy fibers along with widening of interstitial space. H & E x 100.

Figure 21. Cardiac hypertrophy . Enlargement of myocardial fibers with increase in the size of the nuclei. H & E x 400.

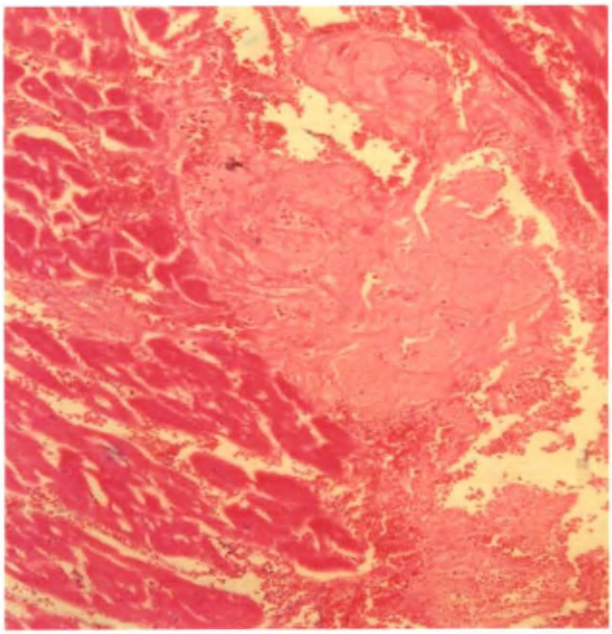

Fig.22

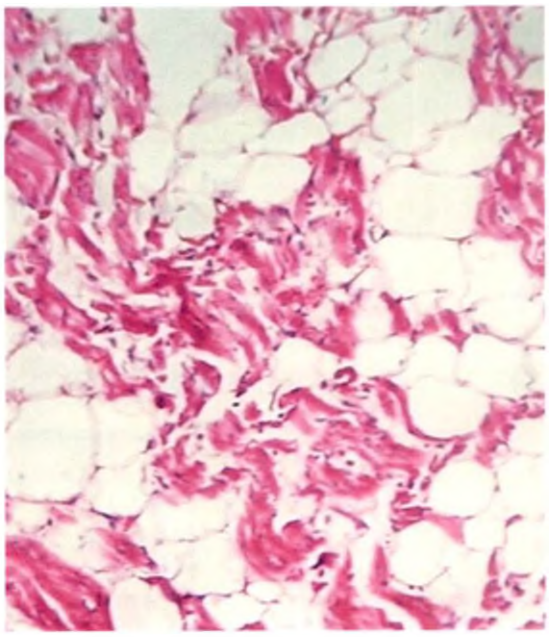

Fig.23

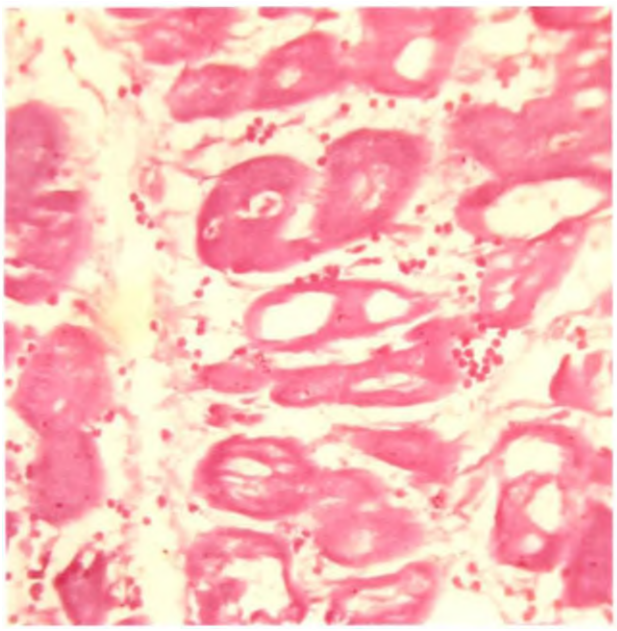

Fig.24

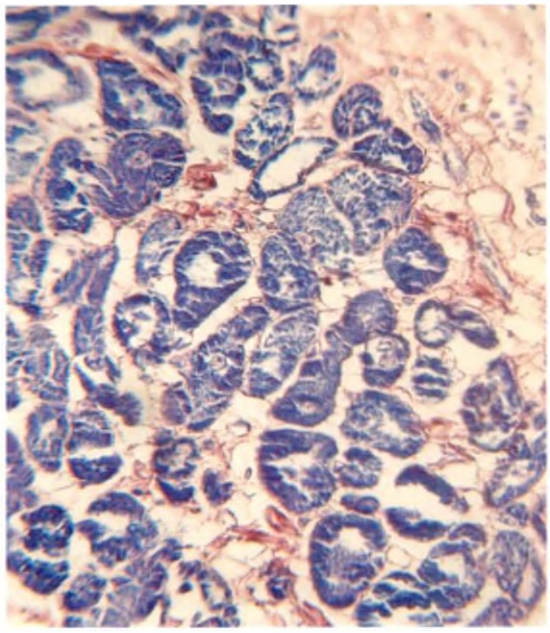

Fig.25

Figure 22. Hyaline degeneration. Diffuse degenerative changes in the myocardium with homogenous and glassy appearance. H & E x 100.

Figure 23. Fatty infiltration. Deposition of lipocytes interposed between the myocardial fibers. H & E x 400.

Figure 24. Vacuolar degeneration. Swelling of myocardial fibers along with varying degrees of sarcoplasmic vacuolations. H & E x 400.

Figure 25. Vacuolar degeneration. Sarcoplasm showing vacuolations and normal one appears deep blue. PTAH x 400.

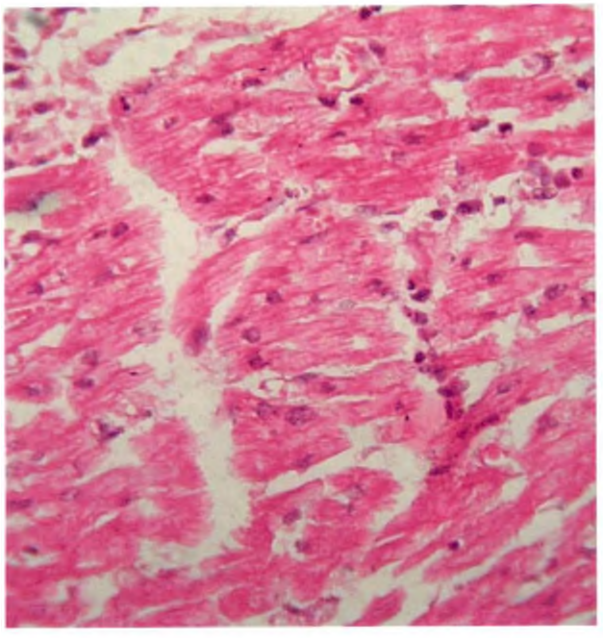

Fig.26

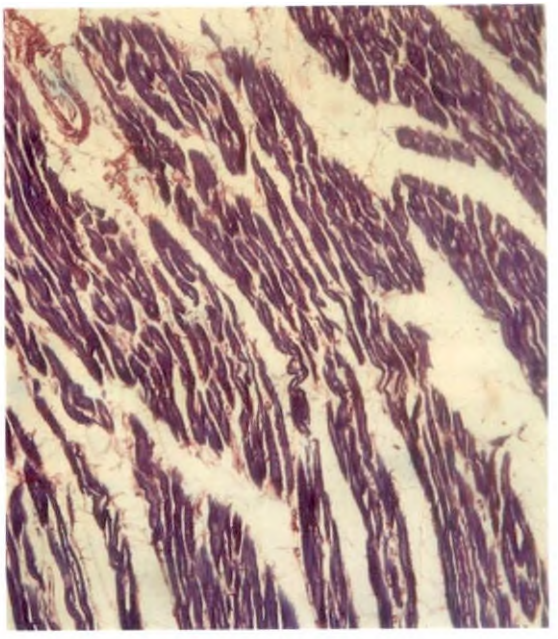

Fig.27

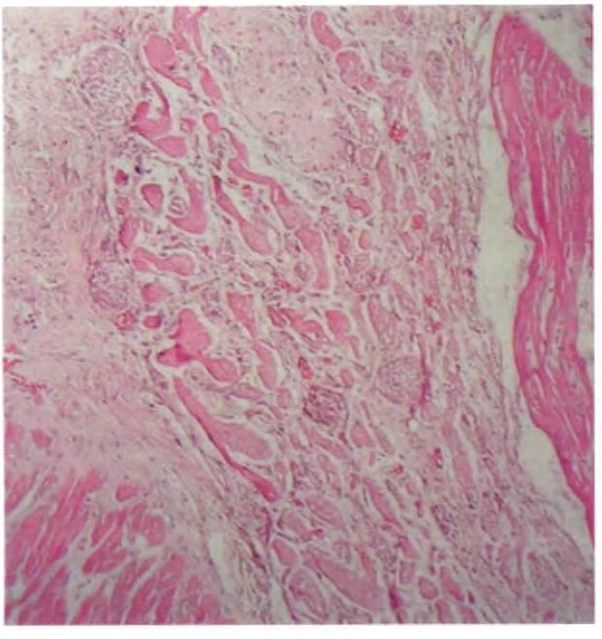

Fig.28

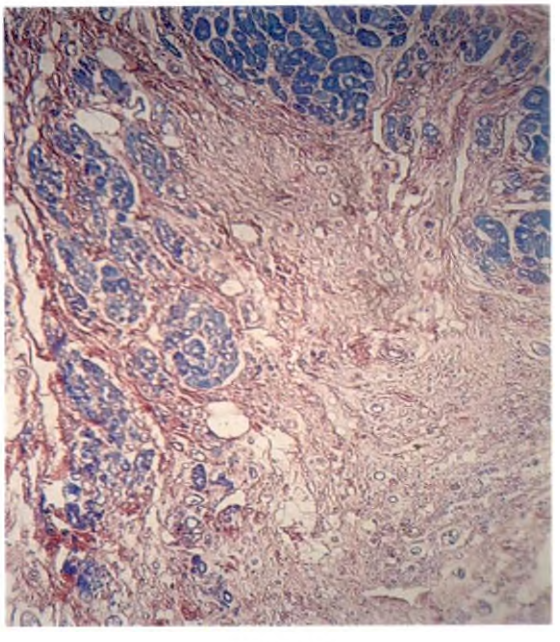

Fig.29

Figure 26. Myocardial necrosis. Swollen, hypereosinophilic myocardial fibers with fragmentations and indistinct striations. H & E x 400.

Figure 27. Myocardial necrosis. Normal myocardial fibers stained deep blue and necrotic areas feebly stained. PTAH x 100.

Figure 28. Healing myocardial necrosis. Proliferation of connective tissues and capillaries in the necrotic myocardium. H & E x 100.

Figure 29. Fibrovascular tissue in myocardium. Connective tissues stained reddish brown. PTAH x 100.

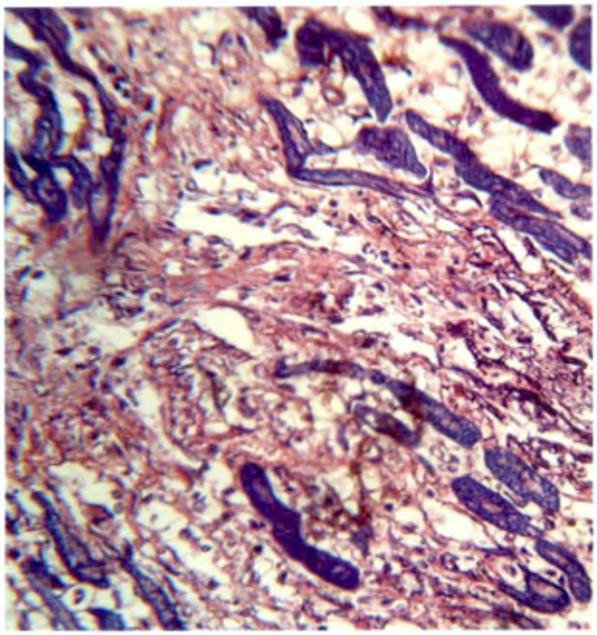

Fig.30

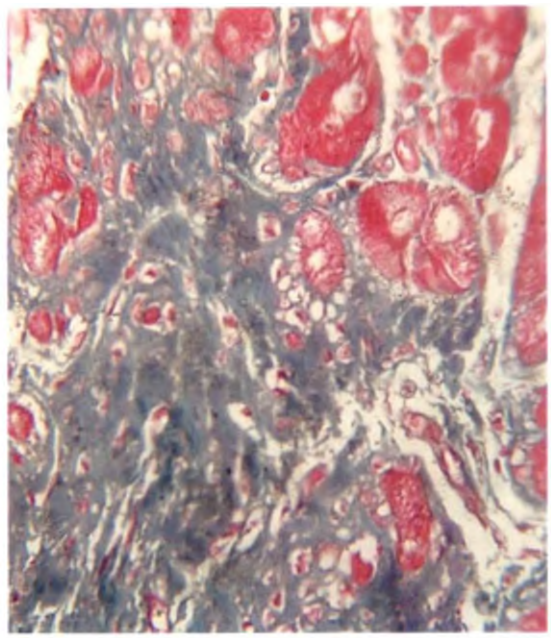

Fig.31

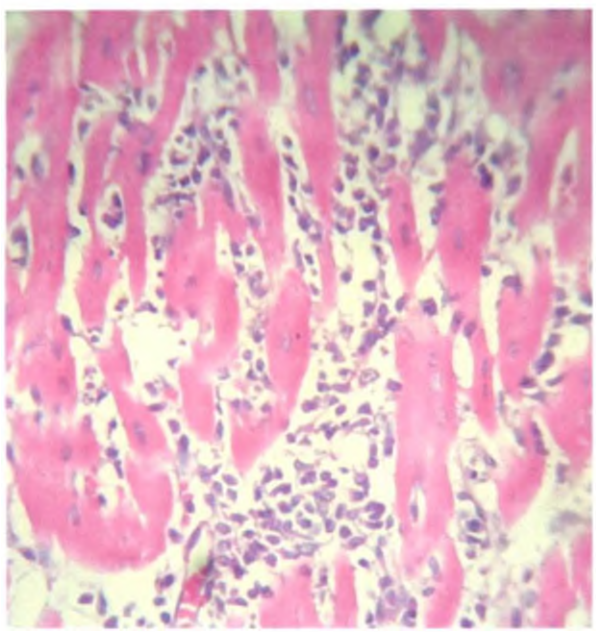

Fig.32

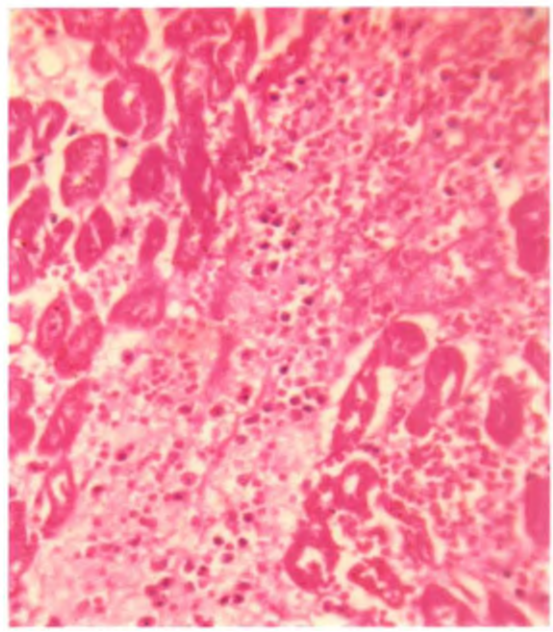

Fig.33

Figure 30. Fibrous tissue proliferation ( reddish brown) in the necrotic myocardium. PTAH x 400.

Figure 31. Healing myocardial necrosis. Connective tissue proliferation in the interstitium stained green. Gomori's Trichrome stain x 400.

Figure 32. Myocarditis. Infiltration of mononuclear inflammatory cells in the interstitium of myocardium. H & E x 400.

Figure 33. Myocarditis. Infiltration of eosinophils in the interstitium of myocardium. H & E x 400.

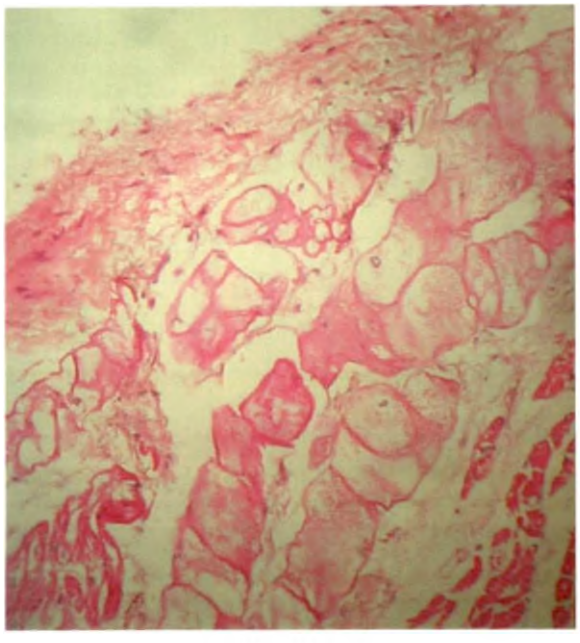

Fig.34

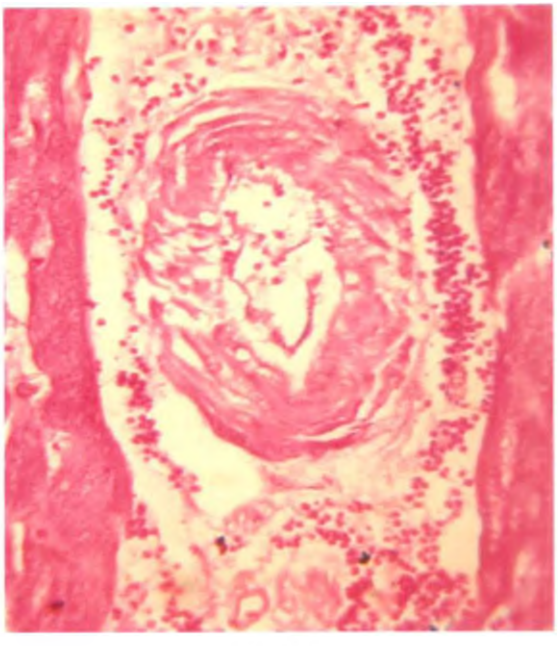

Fig.35

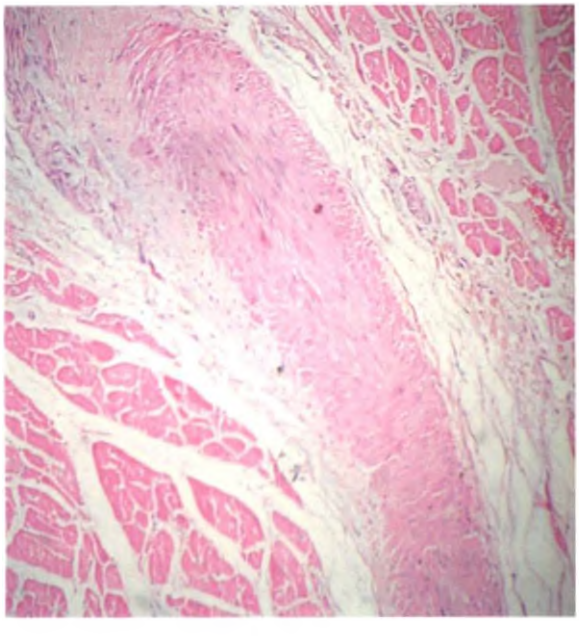

Fig.36

Fig.37

Figure 34. Purkinje fiber degeneration. Swollen Purkinje fibers and the Vacuolated cytoplasm. H & E x 400.

Figure 35. Hyaline degeneration in the vessels. Severe degenerative changes in the tunica media leading to homogenous eosinophilic areas. H & E x 400.

Figure 36. Coronary vessel. Medial hypertrophy. Proliferation of smooth muscle resulted in the thickening of the media and luminal narrowing. H & E x 100.

Figure 37. Coronary vessels. Thrombus. Thrombus containing extensive fibrin deposits and cellular infiltrates. H & E x 63.

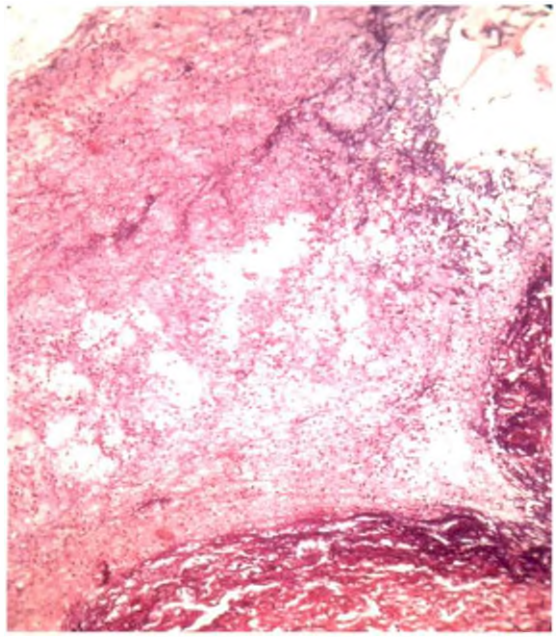

Fig.38

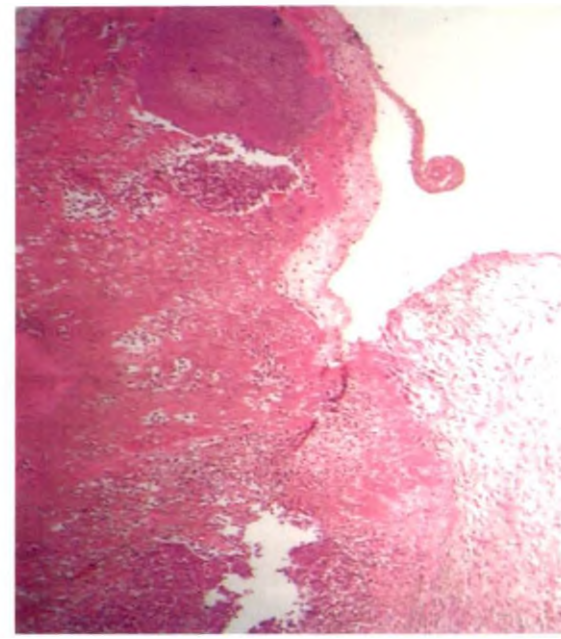

Fig.39

Figure 38. Coronary vessel thrombus. Fibrin stained blue. PTAH x 400.

Figure 39. Vegetative endocarditis. Mass of coagulated necrotic material mixed with fibrin and a zone of inflammatory cells and bacteria. H & E x 100.

# *Discussion*

## 5. DISCUSSION

The present study was undertaken to elucidate the limitations of swill feeding in the development of various cardiac disorders in pigs. The data collected from the autopsy records maintained at the Centre of Excellence in Pathology, clinical history, serum enzyme analysis, gross and histopathological features of pigs maintained on standard ration and swill feed formed the materials for the study. Analysis of data collected from the autopsy records revealed 5.9 per cent and 39.9 per cent occurrence of various cardiac lesions in pigs maintained on standard rations and swill feed respectively for the period from 2001- 2003. In the present investigation 46.2 per cent of swill fed pigs had various types of cardiac disorders compared to 6.1 per cent in concentrate fed pigs. There was a marked difference in the prevalence of cardiac lesions among the animals brought for autopsy and those observed at slaughter. This difference could be attributed to the fact that animals slaughtered could be apparently healthy in condition.

The relative weight of the hearts showed difference between the individual pigs of those reared under standard ration and those reared on swill but the average weight of the organ was within the normal range observed by Robinson and Maxie (1993) and Ghoshal ( 1975 ).

The clinical history collected during autopsy of swill fed pigs revealed that the animals died without showing any clinical symptoms in apparently healthy condition. All those pigs were between the age group of three to eight months. This coincides with the observation made by Menon *et al.* (2003) as she reported the death of young pigs which were reared on locally available market wastes. Belchev *et al.* (2002) reported death of young piglets due to vitamin E and Selenium deficiency. The possible reasons for the death among the rapidly growing pigs

could be due to the development of oxidative damage in pigs born with low tissue reserves of alpha tocopherol and low dietary supplementation of vitamin E to the sow. Van Vleet *et al.* (1973) reported the control of vitamin E deficiency in growing swine by parenteral administration of Vitamin E to pregnant sows and piglets. Increased myocardial and hepatic iron concentration could promote oxidative stress in nutritionally deficient pigs which contribute to the development of oxidative damage (Korpela, 1990 b). Feeding pigs with protein supplements lacking either quality or quantity could result in death due to cardiac muscle degeneration without showing any clinical signs as reported by Kelly (1993). In the present study conducted, the nutritional status of the pigs maintained on swill is doubtful since the feed are not subjected for the proximate analysis especially for the micronutrients.

Hydropericardium with hydrothorax, hydroperitoneum or hemopericardium with hemothorax, hemoperitoneum associated with varying degrees of epicardial and endocardial hemorrhages were the constant gross findings at autopsy. Van Vleet *et al.* (1970) reported similar gross lesions in mulberry heart disease. Excessive pericardial fluid and epicardial petechiation adjacent to the coronary groove were the lesions observed in copper deficiency in swill fed pigs (Pritchard *et al.*, 1985). Similar observation was reported in rats fed with combined copper and iron deficient diet (Van Vleet and Ferrans, 1986). In the light of the above statements, the lesions observed in the present study could be attributed to multiple deficiencies.

Haemopericardium is accumulation of pure blood in the pericardial cavity and death occurs suddenly from cardiac tamponade. In one case rupture of coronary artery caused accumulation of blood in the pericardial cavity. Van Vleet and Ferrans (2001) reported that hemopericardium occurs following rupture of coronary artery, aorta or spontaneous atrial rupture. Robinson and Maxie (1993) reported that

hemopericardium in animals with defective cross linking of collagen and elastin and degeneration of elastica in blood vessels as in copper deficient swine.

Histopathological lesions in the present study varied from mild to severe degrees of degeneration, moderate inter muscular hemorrhage to suffusion in the myocardium and inflammation. The above lesions could be due to nutritional deficiencies, toxic conditions and infectious etiologies. The microbiological examination did not show any specific organism of pathological significance except in one case where bipolar organism could be detected. Hence, most of the cases which showed varying degree of degenerative change could not be attributed to an infectious etiology. More over the extra cardiac lesions did not suggest an infectious nature. Fatty change in the cardiac muscle was associated with congestion and hemorrhage of varying degree. A case of serous atrophy of epicardial fat was encountered in which the piglet was anemic and emaciated which indicated the chronic nature of the condition. Robinson and Maxie (1993) reported that fatty change occurs in a variety of acute systemic intoxications, and it accompanies the dilatation of heart and being best expressed in piglet anemia of iron deficiency. Hypoxic injury to the cell due to anemia could be a reason for fatty change in the myocardium. Malnutrition is also one of the factors causing fatty change. Malnutrition mobilizes the fat and leads to the enhancement in the entry of fat in to the cells. In two cases the pigs with fatty degeneration showed elevations of LDH, AST and CK levels.

Hyaline degeneration of the cardiac muscle fibers was observed in the myocardium along with severe myocytolysis and extensive hemorrhage in five cases. Mc Kinney (1974) observed similar lesions in human beings in various cardiomyopathies and attributed these to injury produced by toxic plants. The possibility of toxic injury to these animals can not be ruled out since these animals

were maintained on various locally available market wastes. Van Vleet and Ferrans (1986) also described similar lesions in selenium- vitamin E deficiency in calves. Hyaline degeneration of heart muscle was reported in pigs fed with diet lacking antioxidants and enriched with oxidated cod liver oil (de Gritz *et al.*, 1994). Thus the underlying etiology of this type of degenerative cardiomyopathies could be either nutritional factors or toxic. Marked elevation of serum enzyme levels suggest that these animals were subjected to severe degree of myolysis.

Degeneration of Purkinje fibers characterized by swelling and vacuolation were observed in few cases. Kennedy and Rice (1988) reported preferential degeneration and necrosis of purkinje cardiocytes in calves fed deficient diets which had sublethal damage characterized histologically by sarcoplasmic accumulation of lipopigment granules. Sykes and Moore (1942) reported degeneration of Purkinje fibers characterized by vacuolation as well as the presence of yellowish- brown granules in the cytoplasm due to deficiency of potassium in the diet. However, in the present study no granular deposit could be observed.

Focal myocarditis observed in the study characterized by infiltration with mononuclear, polymorphonuclear leucocytes in the myocardial fibers could indicate an infection as mentioned by Bolt *et al.* (1997). The pigs in the present study were fed with nutritionally imbalanced feed, thus immune function of those animals could be affected. Myocarditis with severe degree of degenerative changes in one case had elevated levels of LDH, AST and CK levels. Van Vleet and Ferrans (1986) reported that parvo viral myocarditis was characterized by diffuse lymphocytic infiltration with necrotic myocytes and increased number of fibroblasts in the interstitium. Bolt *et al.* (1997) reported non suppurative myocarditis in piglets associated with porcine parvovirus infection characterized by several foci of mild to moderate infiltration of mononuclear cells and hemorrhages between the myocytes and perivascular

accumulation of lymphocytes and macrophages. Corso (1997) suggested that probability of exposure for exotic diseases in domestic pigs fed with uncooked household waste was highest for the classical swine fever (hog cholera) virus compared to lower exposure for foot and mouth disease virus, swine vesicular disease virus and African swine fever virus. In this present study it was observed that most farmers were feeding uncooked swill to pigs. Myocarditis with predominant eosinophil infiltration was noticed in one case. This indicated the parasitic or allergic type of inflammation. However no evidence of any parasites could be seen in these cases.

Fibrinous pericarditis was recorded in three cases. Fibrinous pericarditis is usually the result of hematogenous infections. All the pigs were between the age group of 4 to 8 months. There was the thickening of the pericardium along with fibrinous exudation and severe degree of infiltration with inflammatory cells. The inflammatory cells predominantly found were mononuclear cells. Robinson and Maxie (1993) reported that in swine it occurs in Glasser's disease and pasteurellosis, is a common complication of porcine enzootic pneumonia and occasionally observed in salmonellosis and streptococcal infection of piglets. Blood smear taken from the autopsied pigs revealed bipolar organisms. Thus fibrinous pericarditis observed in these cases could be due to Pasteurella infection. The sudden and early death in two cases could be due to infection with virulent bacteria and concurrent septicemia. If survival is prolonged in those animals there would be a formation of fibrous adhesions between the pericardial surfaces from fibrous organization of the exudates as described by Van Vleet and Ferrans (2001).

Vegetative endocarditis in atrio ventricular valves was recorded in one case. The valve was enveloped by a mass of coagulative necrotic material mixed with fibrin, inflammatory cells and random islands of bacteria. The similar lesion was

observed by Sanford *et al.* (1982) in *Streptococcus suis* type II infection and associated diseases in swine. Jones (1980) described streptococcus induced vegetative endocarditis where in acute changes were characterized by infiltration with fibrin, inflammatory cells and bacteria. Geissinger *et al.* (1973) isolated *Staphylococcus aureus* in endocarditis in pigs. Johnson *et al.* (1986) isolated Lancefield group C streptococcus from the blood cultures in experimental porcine infective endocarditis. Robinson and Maxie (1993) reported that *Erysipelothrix rhusiopathiae* and streptococcus species were the commonly encountered organisms in valvular endocarditis in pigs. Death in these cases could be due to cardiac failure resulting from the valvular dysfunction along with the effects of septicemia.

Hypertrophy of the intramural coronary artery leading to the occlusion of the vessel and ischemic myocardial necrosis were observed in few cases. Shields *et al.* (1962) reported medial thickening of the aorta and intramural hemorrhages in coronary arteries in copper deficiency. Kennedy and Rice(1992) reported thickening of tunica media in large arterioles, hyaline material and pyknotic nuclei were reported to be occasionally seen in media of arterioles in vitamin E and selenium deficient animals. Capillaries had swollen endothelial cells that frequently resulted in apparent luminal occlusion. Danks (1989) stated that humans with genetic copper deficiency had elongated and dilated major arteries, rupture and hemorrhage in the vessels.

Varying degrees of congestion and hemorrhage were observed in the present study. The changes associated with the tunica media needs to be viewed more seriously in swill fed pigs because vascular changes is reported to be the initial pathological change resulting in cardiomyopathy. These observations need to be confirmed by more controlled experimental study.

Thrombus in the intra mural coronary artery could be observed in two swill fed autopsied pigs associated with ischemic myocardial necrosis in both the cases. Robinson and Maxie (1993) reported that ischemic myocardial necrosis occurs in thrombotic disease such as microangiopathy of vitamin E and selenium deficiency, in inflammatory vascular disease such as periarteritis nodosa. Klevay (2000) stated that copper deficient animals revealed coronary artery thrombosis, myocardial necrosis, aortic fissures and rupture. In both the cases the lesions of myocardial necrosis were observed.

Extensive myocardial hemorrhage along with severe myocardial necrosis and hyaline degeneration was observed in four autopsied cases. The extensive extravasations suggested that the integrity of the basal lamina of the intramural arteries or arterioles might be disrupted leading to rupture of the blood vessels and suffusions. Nath (1997) reported that dietary copper restriction affects the integrity of the basal lamina of cardiac myocytes and capillaries. Coulson and Carnes (1963) reported the lesions varied from interruption of the internal elastic lamina, massive dissections and compression of media eccentrically within the intact adventitia leading to occlusion coronary arteries and myocardial infarction in copper deficient pigs.

Myocardial necrosis of varying degrees was found in majority of autopsied and slaughtered swill fed pigs. Myocardial fibers were swollen and hyper esionophilic and the striations were indistinct. The nuclei of the myocardial cells revealed pyknosis and karyolysis along with perinuclear vacuolations. Myocardial necrosis can result from a number of causes, including nutritional deficiencies, toxic injuries and metabolic disorders. Selenium- vitamin E, potassium, copper, thiamine and magnesium deficiencies are the nutritional causes of myocardial necrosis in animals (Van Vleet and Ferrans, 1986). The areas of necrosis were characterized

histologically by hypereosinophilic and swollen myocardial fibers, indistinct striations with pyknotic nuclei (Van Vleet and Ferrans, 2001). Cellular swelling, nuclear hyperchromasia, early loss of striations and a granular appearance of the myocytes were the features recorded in coagulative type of necrosis. Focal to massive necrosis or residual scars has been reported in selenium- vitamin E deficiency (Robinson and Maxie, 1993). However in the present study extensive fibrous tissue proliferation in the myocardium was evident in two slaughtered swill fed pigs. These could be the cases of non fatal myocardial necrosis.

Myocardial necrosis also occurs in stress susceptible pigs in porcine stress syndrome or in malignant hyperthermia. The distinctive feature of myocardium in such cases would be the presence of homogenous esionophilic transverse bands with intervening areas of fine granulation and myolysis in histopathology as demonstrated by Johansson and Jonsson (1997). In the present study no such contraction bands could be detected in any case. This suggested that the animals were not inherited for PSS trait and myocardial necrosis observed in the swill fed slaughtered pigs were not precipitated by stress conditions encountered in transportation and other physical restraints. D'Allaire and DeRoth (1986) reported that stress susceptible pigs had higher levels of serum total creatine kinase compared to resistant pigs. Elevation of serum AST, LDH and CK level in present investigation might not be under the influence of stress induced cardiomyopathy because histologically hypercontraction bands could not be observed.

In the comparative study on swill fed and concentrate fed pigs, though the lesion could not be detected grossly, the elevation of enzymes indicated a subtle change in the cardiac musculature in swill fed pigs. This elevated serum enzyme levels correlated with the findings of Ruth and Van Vleet (1974) in which increase in the activity of GOT (Glutamic Oxaloacetate Transferase), CPK (Creatine Phospho

Kinase) and LDH (Lactic dehydrogenase) were reported in pigs deficient in selenium and vitamin E. Plasma AST and CPK activities were markedly increased in the pigs that were not pre-treated with selenium and vitamin E during experimentally induced cardiomyopathy (Van Vleet *et al.*, 1977). Coles (1986) reported white muscle disease associated with elevation of AST in sheep and calves that were depleted of selenium and vitamin E. Stevenson and Jones (1989) stated that clinically healthy vitamin E deficient animals had elevated plasma CK activities and low alpha tocopherol concentration and the level of creatine kinase activities decreased in response to treatment with vitamin E.

Hypertrophic cardiomyopathy in pigs is characterized either by asymmetric form with septal : free wall thickness ratio of 1.1 or symmetric form associated with 50 per cent increase in the relative heart weights. Histologically hypertrophy is characterized by disarray of myocytes most frequently associated with asymmetric form and myocyte hypertrophy (Van Vleet and Ferrans, 1986). Only in one case myocardium had prominent disarray or disorganization of myocytes with interweaving rather than parallel arrangement of fibers could be seen as described by Van Vleet and Ferrans (2001). Thus, it seems that hypertrophic cardiomyopathy in pigs is more frequently of the symmetric type and is less frequently associated with myocytes disarray than in the case in humans. Robinson and Maxie (1993) reported that hypertrophic cardiomyopathy in humans is inherited as an autosomal dominant character and asymmetric enlargement of the interventricular septum may lead to obstruction of the left ventricular outflow. Myocardial fiber disarray was cited as most consistent feature of hypertrophic cardiomyopathy in human (Maron, 1985)

In the present study, dilated cardiomyopathy showed thin and wavy fibers with increase in the interstitial tissue space and proliferation of connective tissue.

This is in consonance with the observation made by Van Vleet and Ferrans (1986), that dilated cardiomyopathy or congestive cardiomyopathy occurs in association with nutritional deficiency or in toxic conditions. Tidholm *et al.* (1998) reported that attenuated wavy fibers were a sensitive indicator of dilated cardiomyopathy. Everett *et al.* (1999) suggested that the lesion of the dilated cardiomyopathy was usually characterized by myofiber degeneration and atrophy and replacement of myocardium with dense bundles of collagen and clusters of adipocytes.

The pig farming on swill feed has become an accepted and profitable practice in Kerala. The sudden mortality in pig units and the absence of monitoring of the nutrients in feed has led to a pathological study of this nature. Hence, the systematic investigation was undertaken to study the influence of swill feeding in the development of cardiac disorders in pigs. This has helped to document various cardiac lesions encountered in pigs and to assess the prevalence of such disorders in swill fed animals compared with those fed on the standard ration. The study further helped to assess the usefulness of serum enzyme levels as a diagnostic marker in various cardiomyopathies in pigs. Myocardium, the principle component of the heart may subject to various disease conditions which may be due to multiple etiology. In the present study, the myocardium of swill fed pigs revealed mild to severe histopathological changes although the animals were slaughtered under apparently healthy conditions and had no gross lesions. The serum enzyme levels of AST, LDH and CK were found to be higher in swill fed pigs with cardiomyopathic changes than that of serum of pigs reared on standard ration. The study also showed higher incidences of gross and histopathological changes especially in the swill fed pigs brought for autopsy. The pathological changes associated with various cardiac disorders are suggestive of multiple nutritional deficiencies. The general health status and immune status of swill fed animals also needs to be studied in the light of the pathological changes and the subtle changes reflected by the elevated enzyme

levels. The result of the study also highlights the need to monitor the nutrient content of the swill, especially the availability of micronutrients.

# *Summary*

# 6. SUMMARY

Pig rearing in rural production systems based on the swill feed has become very popular in Kerala. But cases of sudden mortality in apparently healthy animals are also reported. Earlier reports of the post mortem examination of such carcasses revealed severe degree of cardiovascular damages. These reports prompted to undertake a study to elucidate the effects of swill feed on the cardiovascular system of the pigs.

A retrospective prevalence study was conducted based on the data of the autopsy records maintained at the Centre of Excellence in Pathology during 2001-2003. The analysis of the data revealed 5.9 per cent and 39.9 per cent occurrence of various cardiac lesions in pigs maintained on standard rations and swill feed respectively for the period from 2001- 2003.

The present study consisted of examination of carcass brought for autopsy by the local farmers practicing swill feeding and also carcass brought from the University pig farm where standard concentrate feeding is practiced. The detailed examination of the cardiovascular system was also done at the slaughter house, where both swill fed and concentrate fed pigs were brought for slaughter.

A total number of 143 hearts from the swill fed and 360 hearts from concentrate fed pigs were subjected to detailed gross and histopathological examination. Out of which 46.2 per cent of the swill fed and 6.1 per cent of the concentrate fed pigs were found to have lesions which could be detected grossly.

The clinical history collected showed that the mortality in swill fed pigs occurred in apparently healthy condition and died without showing any clinical symptoms. Most of those pigs were between the age group of three to eight months.

Though there were difference in the weight of the heart between individual pigs of those reared on swill and concentrate feed, there was no significant difference in the average weight of the organ.

The gross lesions detected were mainly vascular lesions like epicardial and endocardial hemorrhages, hydropericardium, hemopericardium, fibrinous pericarditis, degenerative and necrotic changes in the myocardium, myocarditis, cardiac hypertrophy, cardiac dilatation and vegetative endocarditis.

Histopathological lesions varied from mild to severe degrees of congestion, hemorrhage, degenerative changes in the myocardium like hyaline degeneration to fatty change, inflammatory conditions and hypertrophic changes. Mild to moderate degree of swelling and vacuolations were characteristic in the degenerative changes affecting the Purkinje fibers.

Stenosis of the vessels following hypertrophic changes in the intramural coronary artery and ischemic myocardial necrosis associated with thrombus were observed in few cases.

The histopathology of the hypertrophic myocardium revealed prominent disarray of myocytes. The hypertrophic cardiomyopathy in pigs was more frequently of the symmetric type. The dilated cardiomyopathy revealed thin, atrophic and wavy fibers with increase in the interstitial tissue space.

Myocardial necrosis of varying degrees was found in majority of autopsied and slaughtered swill fed pigs. Fibrous tissue proliferation seen in few of these cases suggested the regenerative response of the heart leading to scar formation.

The enzymes like AST, LDH and CK were assayed in serum samples collected from the slaughtered pigs of both groups. The enzyme assay showed an elevation of AST, LDH and CK enzymes in pigs reared on swill compared to the concentrate fed pigs.

The high vascular and degenerative changes of the myocardium in the swill fed pigs indicated multiple nutritional deficiencies as the cause of this condition. The changes were further reflected on the increased levels of AST, LDH and CK in swill fed pigs.

*Inflammatory conditions like fibrinous pericarditis, myocarditis and* vegetative endocarditis were also noticed in the study. Bipolar staining Pasteurella was identified in a case of fibrinous pericarditis. It is a fact that animals which are deficient in micronutrients become more susceptible to the flaring up of latent infections. The presence of inflammatory cells predominantly mononuclear cell and scattered polymorphonuclear leucocytes also indicated a multiple infectious nature.

The study has helped to document various cardiac lesions encountered in pigs and to assess the prevalence of such disorders in swill fed animals compared to those on the standard ration. The study further helped to assess the usefulness of serum enzyme levels as a diagnostic marker in various cardiomyopathies in pigs. In the present study, the myocardium of swill fed pigs revealed mild to severe histopathological changes although the animals were slaughtered under apparently healthy conditions. The study also showed higher incidences of gross and

histopathological changes especially in the swill fed pigs brought for autopsy. The serum enzyme levels of AST, LDH and CK were found to be higher in swill fed pigs with cardiomyopathic changes than that of serum of pigs reared on standard ration. The observation pointed to the need for monitoring the general health and immune status of swill fed animals and assessment of the micronutrient availability of the swill.

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* Originals not consulted

# PATHOLOGY OF CARDIAC DISORDERS IN PIGS REARED ON SWILL

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## ABSTRACT

Pig rearing in rural production systems based on the swill feed has become very popular in Kerala. The cases of sudden mortality in apparently healthy animals and earlier reports of the cardiovascular damages in such animals prompted to study the effects of swill feed on the cardiovascular system in pigs. The study confirmed, the cardiomyopathies are more common in the pigs which were maintained on swill than those maintained on concentrate feed. Cardiac lesions to the tune of 46.2% were observed in swill fed pigs examined compared to a mere 6.1% cases in concentrate fed pigs. The cardiac lesions recorded were varying degrees of congestion, hemorrhage, hydropericardium, hemopericardium, fibrinous pericarditis, myocardial degeneration, cardiac hypertrophy, cardiac dilatation and valvular endocarditis. The histopathological changes of all these conditions have been studied in detail and the possible pathogenesis described. The vascular changes associated with myocardial necrosis have to be studied in detail by more controlled experimental study. The pathological changes associated with various cardiac disorders are suggestive of multiple nutritional deficiencies. The serum enzyme levels of AST, LDH and CK were found to be higher in swill fed pigs with cardiomyopathic changes than that of serum of pigs reared on concentrate feed. The high enzyme level in the absence of any gross changes in the heart indicated that these could be used as marker for monitoring the subtle cardiomyopathic changes. The various observation made in the study highlighted that the general and immune status of swill fed animals needs to be studied in the light of pathological changes and the subtle changes reflected by the elevated enzyme levels. The result of the study also highlights the need to monitor the nutrient content of the swill, especially the availability of micronutrients.