INVESTIGATION ON SUDDEN DEATH IN PIGS

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DECLARATION

I hereby declare that the thesis entitled **"INVESTIGATION ON SUDDEN DEATH IN PIGS"** is a record of research work done by me during the course of research and this thesis has not previously formed the basis for the award of any degree, diploma, fellowship or associateship or other similar title, of any other University or Society.

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Certified that the thesis entitled **"INVESTIGATION ON SUDDEN DEATH IN PIGS"** is a record of research work done independently by **Manjusha A.** 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 her.

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MANJUSHA A.

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LIST OF ABBREVIATIONS

@	- At the rate of
°F	- Fahrenheit
AST	- aspartate amino transferase
B.W	- Body weight
BHI	- brain heart infusion
CK	- creatine kinase
EDTA	- Ethylene Diamine Tetra Acetate
g	- Gram
H&E	- Haematoxylin and Eosin
Kg	- Kilogram
LDH	- lactate dehydrogenase
mg	- Milligram
min.	- Minute
ml	- Milliliter
mm^3	- Cubic Millimeter
rpm	- rotation per minute
Se	- Selenium
SE	- Standard Error
spp.	-species
Sec	- second
TLC	- Total leukocyte count
U/L	- units per liter

Introduction

1. INTRODUCTION

Pig farming is becoming popular among the farmers in Kerala, which is discerned as profitable compared to rearing other livestock. Pigs are considered the supreme among the meat producing livestock. The high prolificacy, fast growth rate, short gestation period and ability to thrive well on unconventional feedstuff are the merits of pigs. They are marketed at a younger age fetching the farmer good returns. Majority of the pig farmers in Kerala were feeding their animals on organic wastes of animal and plant origin .The swill feed thus fed include poultry slaughter waste, hotel or restaurant or vegetable waste. Though pork industry is having a bright future in Kerala, on these days many pig farms have experienced problems with pigs dying without any clinical symptoms. This unexpected death causes great economic loss to the farmers.

A number of causes like bacteria, virus, poisoning by various toxic materials causes sudden death. According to Casteel *et al.* (1987) many infectious, toxicological and miscellaneous causes are attributed to the sudden death in pigs.

The sudden deaths are most commonly observed in growing pigs, which are in apparently good body condition. In most of the pig farms in Kerala, swill is the sole source of feed, which increases the chances for exposure to many infectious diseases. These pigs are reared in confinement with no access to pasture, which increases the chances for nutritional deficiencies. In addition to this many of the defective managemental practices and adverse environmental conditions aggravate the health problems of the pigs. There will not be any premonitory clinical signs in these cases. The precise diagnosis and management of these conditions are often found to be difficult. The history, clinical signs, clinical examination of the contact animals, various laboratory methods like examination

of various blood parameters, necropsy findings and histopathological studies were helpful in reaching a presumptive diagnosis of the condition.

So far no investigations have been undertaken into the various factors involved in the sudden death of pigs in Kerala. Hence the present study "Investigation on sudden death in pigs" has been taken up with the following objectives.

1. To identify the various etiological factors of sudden death in pigs.
2. To formulate suitable control measures to prevent sudden death in pigs.

Review of Literature

2. REVIEW OF LITERATURE

2.1 MORTALITY IN PIGS

Koller and Exon (1986) reported sudden death in young pigs due to myocardial dystrophy.

Casteel *et al.* (1987) attributed various causes to sudden death in pigs including infectious, toxicological and some other miscellaneous causes.

Dyck and Swierstra (1987) reported that the important causes of piglet death from birth to weaning were starvation, diseases, inadequate nutrition and crushing by the sows.

Nielsen *et al.* (1989) reported sudden death without previous clinical signs in rapidly growing young pigs at Danish piggeries.

Chagnon *et al.* (1991) reported that mortality was significantly high during the months of July, August and October and the major causes of death were heart failure, torsions and accidents of abdominal organs.

According to Lay *et al.* (2002) the preweaning mortality was influenced by disease, nutritional status, thermal environment, birth weight, litter size of sow and piglet behavior, sex, genetics etc.

Pathak *et al.* (2004) observed highest piglet mortality due to digestive disorders. Hot and humid environment in the monsoon season resulted in great stress on both the mother and piglets making them susceptible to respiratory infection.

2.2 ETIOLOGY

2.2.1 Bacterial diseases

According to Baldwin (1959) enterotoxemia caused by *Clostridium perfringens* type C was a problem in piglets, characterized by sudden death.

Thomlinson (1963) reported sudden death in pigs during the early stages of an outbreak of *E. coli* infection

Clostridium perfringens type C (per acute enteritis) causes hemorrhagic diarrhoea and spontaneous death in pigs (Barnes and Bergeland, 1970).

According to Chia and Taylor (1978) many outbreaks of swine dysentery by *Treponema hyodysenteriae* resulted from the mixing of infected and susceptible pigs. *Treponema hyodysenteriae* was found to survive for periods of up to 48 days in dysenteric pig faeces stored at temperature between 0° C to10° C and survival rate of the organism reduced on high temperature.

Haemophilus pleuropneumoniae was a serious pathogen in pigs, which could be isolated from any purulent conditions of pigs especially in cases of pneumonia (Morgan and Phillips, 1978).

The acute septicaemic form of swine pastereurellosis due to *Pastereulla multocida* type B had a sudden onset and death may occur within hours (Farrington, 1981).

Septicemic anthrax caused sudden death in pigs without any period of illness being noticed by the owner (Ferguson, 1981).

Nielsen (1981) reported that the first indication of an outbreak of edema disease was often sudden death of one or more pigs in the susceptible group.

Septecaemic samonellosis mainly occurred in weaned pigs and occasionally in adults led to sudden death in single animal or as small epizootics (Wilcock, 1981).

Enteric colibacillosis was the most prevalent cause of diarrhoea, dehydration and death in neonatal pigs. Faeces were watery and yellowish in colour. Piglets became weak and dehydrated and death occurring within 24 hours of the first appearance of clinical signs (Mc Carthy *et al.*1984).

Casteel *et al.* (1987) reported that bacterial diseases like Actinobacillosis, Anthrax, Black leg, Clostridium infections, *Escherichia coli* causing oedema disease, Erysepelas, Glassers disease, *Haemophilus pleuropneumoniae* infection, Pasteurellosis, Salmonellosis, Streptococcal infection, *Treponema hyodysenteriae* infection etc. had caused sudden death in pigs.

According to Tubbs (1987) a presumptive diagnosis of swine dysentery by *Treponema hyodysenteriae* was based on the presence of mucohaemorrhagic diarrhoea, gross lesions limited to large intestine and significant number of spirochetes in stained impression smears of colonic mucosa but isolation and identification of the organism were necessary to confirm the diagnosis.

Gannon *et al.* (1988) reported that verotoxigenic *E. coli* were associated with post weaning diarrhoea, stools with blood and sudden death in pigs.

In peracute cases of *Haemophilus pleuropneumoniae*, sudden deaths without clinical signs were common especially in pigs in good condition. Clinical signs of acute form were dyspnoea, coughing, depression, fever and anorexia. (Tubbs, 1988)

Falade *et al.* (1989) isolated salmonella strains from the pigs and studied the antibiotic sensitivity patterns of the organism. They found out that most of the strains were sensitive to gentamicin and kanamycin.

According to Schwartz (1991) salmonellosis in swine caused by *Salmonella choleraesuis*, *Salmonella typhimurium* and *Salmonella typhisuis*. The clinical signs were variable and include fever, cyanosis of extrimities, pneumonia, diarrhoea, icterus, emaciation, abortion and sudden death. Complete diagnosis of the disease must include necropsy and bacterial isolation.

In an investigation to find out the common causes of diarrhoea in nursing and weanling piglets, Johnson *et al.*(1992) observed *Clostridium perfringens* as one of the cause.

Salmonella cholaraesuis and *Salmonella typhimurium* were the most common salmonella species causing great economic loss to the swine industry (Fedorka-cray *et al.*, 1997).

Haemophilus parasuis infection resulted in a wide variety of clinical entities including pleuritis, polyserositis, pericarditis, arthritis, meningitis and acute death (Rapp-Gabrielson *et al.*,1997).

Streptococcus suis was identified in pigs in U.K which caused acute very painful polyarthritis in suckling and very recently weaned piglets. (Alexander, 1998)

There are thirty five known serotypes of *Streptococcus suis* which cause outbreaks of septicaemia or meningitis in pigs (Amass *et al.* 1999).

Dziva and Mohan (2000) reported that *Pastereulla multocida* infection was prevalent in pig industry and caused pneumonia and meningitis in pigs.

Slavi *et al.* (2000) reported sudden death of growing / finishing pigs in Ontario during an outbreak of *Actinobacillus suis*.

Ken and Bilkei (2003) identified enterotoxigenic *Escherichia coli* and verotoxigenic *Escherichia coli* repeatedly by PCR testing in piglets which died following weaning.

Sujatha *et al.* (2003) reported an outbreak of pasteurellosis in a pig farm causing sudden death in few animals without showing any symptom.

Tarradas *et al.* (2004) reported that serotypes of *Streptococcus suis* isolated from diseased pigs of Spain was the main cause of nervous disorder, septicemia and endocarditis in pigs.

Silvapru and Bilkei (2005) reported *Clostridium difficile* infection in a farm causing a sudden increase in post parturient sow mortality.

2.2.2 Viral diseases

Acland and Littlejohns (1975) reported an outbreak with encephalo myocarditis infection in pigs of New South Wales where they found few cases of brief depression, inappetance, trembling, paralysis, vomiting, dyspnoea, but in most cases pigs died suddenly.

Stewart (1981) reported that in case of hog cholerae rise in temperature, severe leucopenia, and convulsions were the salient features and the pig die with in hours of the symptoms in per acute cases.

According to Casteel *et al.* (1987) pseudorabies infection caused by Suid herpes virus resulted in sudden death of pigs and usually the pigs die within 24 hours of onset of the fever.

Pseudo rabies virus was shed at high levels by clinically diseased swine and sub clinically infected swine. Pseudo rabies virus was expelled through nasal and oral secretions. The virus was wide spread in the tissues of carcasses of swine that die of Pseudo rabies. (Beran, 1993)

Van Oirschot (1994) reported that clinical signs of pseudo rabies virus infection depend primarily on the virulence of the virus and the age of the infected pigs and younger pigs showed more severe signs.

2.2.3 Nutritional causes

2.2.3.1. Vitamin E - Selenium deficiency

Ewan *et al.* (1969) observed that most of the pigs deficient in Vitamin E and selenium died suddenly showing no outward signs of ill health.

Trapp *et al.* (1970) reported that Vitamin E - selenium deficiency in a swine herd characterised by sudden death in feeder pigs.

According to Whitehair (1970) the rancidity and peroxidation of the fatty acids aggravated the deficiency of vitamin E in animals.

Sharp *et al.* (1972) in an investigation about the diets which would result in high incidence of sudden death in pigs from mulberry heart disease and hepatosis dietetica found that increased frequency of the condition occurred when fed low level selenium diet containing torula yeast.

In a study of cardiac tissue of piglets deprived of Vitamin E and selenium, Sweeny *et al.* (1972) observed that the effects of the deficiency were most pronounced on those muscle populations undergoing rapid growth with super imposed functional demands.

Mahan *et al.* (1973) opined that mulberry heart, fluid accumulation in pericardial sac and sudden death were salient pathological changes in the field conditions of the Vitamin E and selenium deficiency in pigs.

Nutritional muscular dystrophy caused by vitamin E - Se deficiency lead to sudden death in both weaned pigs and slaughter animals (Johansson and Jonsson, 1977).

Bengtsson *et al.* (1978) observed spontaneous mortality in pigs fed diet deficient in Vitamin E and Selenium.

Mahan and Moxon (1978) reported that post weaning pig deaths and necropsy symptoms attributable to the vitamin E - Se deficiency occurred when animals fed with corn soy bean meal starter diets. Therefore possibility of marginal or chronic vitamin E-Se deficiencies with young swine persists in areas where natural concentrations of the minerals were low in grains.

Kwatra *et al.* (1980) reported mulberry heart disease in pigs for the first time in Assam, India that caused sudden death in pigs.

The diagnosis of vitamin E - Se deficiency was made by observing increased activity of many serum enzymes originating from damaged skeletal muscles, heart or liver, low blood or tissue Se content and characteristic gross and histopathological alteration in skeletal muscles, heart and liver. (Vanvleet, 1980).

In a study about the value of dietary vitamin E and Se for weanling swine Mayer *et al.* (1981) reported a reduction in plasma tocopherol level within one week after weaning.

Ullery (1981) reported mortality in piglets fed diets containing 15 IU of Vitamin E and 0.03mg selenium/kg and that the problem was significantly reduced by the supplementation of 30IU of Vitamin E and 0.03mg Se/kg

Nielson *et al.* (1989) reported sudden death in rapidly growing pigs due to mulberry heart disease.

Mahan (1990) reported that pigs from sows not fed supplemental Se exhibited vitamin E- Se deficiency more rapidly during the post-weaning period than those of pigs from sows fed with the Se-fortified diet.

Lessard *et al.* (1991) suggested that the Vitamin E-selenium deficiency induce the formation of immuno suppressive factors and increase the release of

oxygenated metabolites and hence it is essential to feed the pigs adequate levels of vitamin E and selenium to control oxidative reactions and to maintain normal functions of immuno competent cells consequently to improve resistance to infectious diseases.

The work of Degritz *et al.* (1994) suggested that deficiency of Se and vitamin E could cause hyaline degeneration of the heart muscle.

Dhanya *et al.* (2003) observed sudden mortality of healthy pigs in swill fed farms in Kerala. They suggested that this could be the result of the deficiency of vitamin E and Se.

2.2.4 Miscellaneous causes

Rothen bacher *et al.* (1963) reported sudden death losses in slaughter age or younger feeder age pigs in good to excellent condition due to hemorrhage into the stomach from the ulceration. The visible mucous membrane was always extremely pale in these conditions.

Obrien (1968) reported that gastric ulceration in bacon pigs in Ireland were greatest in those areas where cheese whey was used for fattening pigs.

Carson and Lloyd (1981) stated that sudden death of the pigs occurred in the acute toxicosis of Arsenic along with other clinical manifestation like vomiting, diarrhoea, dehydration, collapse, convulsions and death. Inorganic or aliphatic forms of Arsenic which were used as ant and grass hopper bait, a non specific herbicide cause sudden death in pigs which consumed it accidentally. In a study about the anticoagulant toxicosis they suggested that the diagnosis should include demonstration of the defect in the clotting mechanism which was evidenced by increase in clotting time, one-stage prothombin time or activated partial prothromboplastin time. Chemical detection of the rodenticide samples in blood, liver or suspect baits was also helpful. They also reported that Carbamate

and Organo phosphates used as insecticides, Cockleburs including *Xanthium strumarium* was also the causes of sudden death in pigs.

According to Glock (1981) intestinal haemorrhagic syndrome characterized by sudden death was common in pigs that fed whey often resulted from torsion of the intestinal mass around the root of the mesentery.

According to Topel and Christian (1981) thirty five per cent hog producers in the United States have encountered the condition of porcine stress syndrome (PSS) and have experienced death losses from PSS.

Disorders of the heat regulatory mechanism, high ambient temperature, high acidity, inadequate ventilation, inadequate shade, transport, movement, handling animals, over crowding, advanced age, limited salt intake etc. might lead to heat stroke and sudden death in pigs (Casteel *et al.*, 1987).

2.3 EPIDEMIOLOGY

Vanvleet *et al.* (1970) stated that peak incidence of mulberry heart disease and hepatosis dietetica occurred during early spring, late summer and early fall.

Fontaine *et al.* (1977) observed a significant degree of muscular dystrophy in pigs at nine weeks of age.

According to Acland and Littlejohns (1981) younger pigs were more susceptible to encephalomyocarditis infection and in this case mortality approaches to 100 per cent.

Clostridium perfringense type C infection was first observed in England later it had been reported in many other countries (Bergeland, 1981).

Farrington (1981) reported that *Pasteurella multocida* was usually transmitted in swine by oral or nasal droplets or aerosol from infected animals.

An outbreak of Anthrax in Southern Ohio was reported by Ferguson (1981) and they found that bone meal as a source of infection.

According to Gustafson (1981) mortality due to pseudorabies virus infection was greatest in baby pigs and least in mature swine.

According to Harris and Glock (1981) swine dysentery by *Treponema hyodysenteriae* was most commonly observed in 15-70 kg pigs but it was also reported in suckling piglets and adults.

Oedema disease usually occurs in one to two weeks after weanling typically it was seen in rapidly growing apparently healthy animals, often the best animal in the litters were affected (Nielson 1981).

According to Nicolet and Scholl (1981) the *Hamophilus pleuropneumoniae* infection of pig was widely distributed. Its outbreaks were reported from India, England, Argentina, United States, Denmark, Norway, Canada, Japan and Taiwan. The disease was transmitted mainly by direct contact from pig to pig.

According to Stewart (1981), transmission of hog cholerae virus by contact was responsible for more infection than all other modes of transmission.

Wilcock (1981) reported that most outbreaks of Salmonellosis occurred in intensively reared weaned pigs.

Swine aged three months to three years were more susceptible to swine Erysepelas. Environmental and stress factors such as nutrition, ambient temperature and sudden changes in these conditions had long been linked to the appearance of swine erysipelas (Wood, 1981).

According to Schwarts (1991), *Salmonella choleraesuis* infection occurred in pigs stressed during the post weaning or grower phase at four to sixteen weeks of age or when exposed to environmental contamination or to carriers shedding

the bacteria. The author reported 32 per cent sudden death in septicaemic *Salmonella choleraesuis* infections.

Rodriguez-Buenfil *et al.* (2004) studied the incidence and identification of Salmonella species in pigs in two farm systems in Mexico and they found that the type of farm did not influence the incidence of salmonella species in fattening pigs. They reported *Salmonella typhimurium* as the serotype most frequently found.

Tarradas *et al.* (2004) reported isolation of 383 strains of *Streptococcus suis* from 99 farms in Spain between 1998 and 2002.

2.4 CLINICAL SIGNS

According to Farrington (1981) in septicaemic form of swine pasteurellosis death occurred within hours. High temperature, prostration, extreme depression, swelling of the pharyngeal region and diffuse bluish red discolouration in the body were the clinical signs noticed in these conditions.

According to Gustafson (1981) the cardinal signs of pseudo rabies were excessive salivation, fever, depression and convulsions.

According to Nielsen (1981) sudden death in one or more pigs in a susceptible group was the first indication of the oedema disease caused by *E coli* and the affected pigs showed dyspnoea, subcutaneous oedema, diarrhoea and shock.

According to Stewart (1981) clinical signs of hog cholerae include inactivity in early stage of the disease, fever, discharge from the eyes, constipation followed by diarrhoea, staggering gait followed by a posterior paresis.

According to Wilcock (1981) septicaemic salmonellosis caused sudden death in pigs and the clinical signs in affected pigs were restlessness, anorexia, fever and watery yellow diarrhoea.

Mc Carthy *et al.* (1986) reported that in the acute form of erysipelas characterised by high body temperature, enlarged lymph nodes and swollen spleen were common in pigs. External signs of the disease included red diamond shaped skin lesions and the animal died suddenly without displayed clinical signs. The author also reported that severe infection of *Pastereulla multocida* in pigs caused bronchopneumonia charecterised by laboured breathing, high fever and mucopurulant nasal discharge.

According to Gannon *et al.* (1988) verotoxigenic *E. coli* were associated with post weaning diarrhoea, bloody stools, sudden death and oedema disease.

According to Schwartz (1991) the clinical manifestation of *Salmonella choleraesuis* infection include fever, cyanosis of extremities, pneumonia, diarrhoea, icterus, emaciation, abortion, sudden death or a combination of any of these.

Mavenyengwa and Matope (1995) reported an outbreak of *Clostridium chauvoei* infection in pigs. The pigs showed swollen painful limbs, huddle in a corner, squeak in pain and death occurred within 24 hours.

Ravisanker *et al.* (2005) isolated *Streptococcus suis* from a fatal case of pneumonitis in a boar showing clinical signs such as respiratory distress, edema of the neck and brisket regions.

2.5 CLINICAL PATHOLOGY

2.5.1 Microbiological examination

Bacteriological examination of specimen assists in the field diagnosis of Salmonellosis in pigs (Belschner, 1972).

In a study about the drug resistant strains of the *E coli* Harne and Saxena (1976) reported that kanamycin, streptomycin and ampicillin were sensitive while chloramphenicol, tetracycline, terramycin and furadantin were less effective.

In a study about the sensitivity of different antibiotic agents towads different bacterial organisms Rhoades (1979) reported that the percentage of *E coli* specimen sensitive to streptomycin, tetracycline and triple sulpha was low in the specimens from pigs than from canine and bovine specimens.

According to Bergeland (1981) the isolation of *Clostridium perfringens* on blood agar plates, streaked directly with intestinal mucosa together with subsequent typing of isolated colonies was a reliable means of establishing an etiologic diagnosis.

According to Ferguson (1981) in case of Anthrax in pigs the impression smears were made by touching the freshly cut surface of lymph gland to a slide and stained with Loafflers alkaline methylene blue, the bacilli appeared blue in colour while the capsule stained light pink.

In case of *Haemophilus pleuropneumoniae* infection demonstration and isolation of the etiologic agent was easily achieved from bronchial and nasal exudates and pneumonic lesions. Gram stained smears in these cases show gram negative coccobacilli (Nicolet and Scholl, 1981).

Diagnosis of *Streptococcus suis* infection was achieved by isolation of alpha or beta haemolytic streptococci from lesions of suppurative meningitis and identification of the isolate by means of the Lance fields system (Ross, 1981).

For the culture of *Salmonella choleraesuis* Var.kunzendorf Brilliant green, Bismuth sulfite and Mc conkey agar was used (Wilcock, 1981).

Culture of *Erysepelothrix rusiopathiae* from tissue specimens was relatively simple using culture media such as tryptose or meat infusion media (Wood, 1981).

Casteel *et al.* (1987) opined that isolation of the organism from different specimen could be used as a laboratory diagnostic method in case of infectious causes of sudden death in swine.

Nielson *et al.* (1989) in their study about the antibiotic sensitivity of Salmonella sp. found that the different serotypes were sensitive to gentamicin and kanamycin and resistant to benzyl penicillin, cloxacillin, nalidixic acid and lincomycin.

Malorny *et al.* (2003) isolated nalidixic acid resistant strains of salmonella from pigs in Germany, between 1998 and 2001, the prevalence of these strains varied from 0.2 to 1.9 percent.

Merialdi *et al.* (2003) isolated 8.11 % *Escherichia coli* and 31.08 per cent salmonella from 74 farms with clinical colitis in growing and finishing pigs.

Actinobacills suis was a gram negative coccobacilli which grows well on blood and Mc conkey agar (Mauch and Bilkei, 2004).

Yang, *et al.* (2004) reported that multi antimicrobial resistant isolates of *E. coli* were susceptible to ceftiofur and ceftriazone.

2.5.2 Enzymology

Mahan *et al.* (1973) observed high serum glutamic oxaloacetic transaminase (SGOT) values in animals without supplementation of selenium when compared with those supplemented with Se.

Ruth and Vanvleet (1974) reported an increase in plasma enzymic activity of SGOT, lactate dehydrogenase (LDH) and creatine phosphokinase(CPK) of pigs fed vitamin E - Se deficient diet.

Fontain *et al.* (1977) reported that serum GOT and CPK levels were useful indices of tissue damage in association with vitamin E - Se deficiency in swine.

Bengtsson *et al.* (1978) reported that in case of vitamin E deficiency in pigs serum aspartate amino transferase level increases beyond the normal level.

Ullery (1981) reported an elevated level of aspartate amino transferase (AST) in myocardial lesions such as mulberry heart disease and also in hepatic injury like hepatosis dietetica.

According to Friendship *et al.* (1983) the normal level of AST was 16-67 U/L while that of creatine kinase was 91-1251 U/L.

Tolling and Jonsson (1983) measured the serum creatine kinase (CK) and lactic dehydrogenase (LDH) isoenzyme activities in pigs having myocardial damage and skeletal muscular lesions. They observed a pronounced elevation of CK-MM and LDH I and II.

Selenium deficiency caused an elevation of glutamic oxaloacetic transaminase and creatine kinase in serum (Koller and Exon, 1986).

Odink *et al.* (1990) reported the CK value of 11.43 kU/L, AST as 80.4 U/L and LDH 3.23kU/L in healthy swine.

Supplementation of vitamin E and Se to pigs were essential to control oxidative reactions, to maintain normal functioning of the immuno competent cells and consequently to improve resistance to infectious diseases (Lessard *et al.*, 1991).

Grandhi *et al.* (1993) reported a lower serum concentration of CPK, AST and LDH levels in pigs supplemented with vitamin E.

According to Vasudevan and Sreekumari (1995) CK, LDH and AST levels were used in the diagnosis of myocardial infarction.

Dubreuil and Lapierre (1997) reported that the normal serum concentration of aspartate amino transferase in pigs as 51.4 IU/L at eight weeks of age and serum creatine kinase as 294 U/L.

According to Kaneko *et al.* (1997) normal level of AST was 32-84U/L while that of creatine kinase was 2.4 to 22.5 U/L.

According to Radostits *et al.* (2000) the normal value of AST in pigs was 32-84 U/L.

According to Harapin *et al.* (2003) the normal level of creatine kinase for wild boar was between 455-1756 U/L and AST was 41-67 U/L.

2.5.3 Leukogram

Leukocytosis due to neutrophilia was seen in most bacterial infections or inflammatory lesions (Doxey, 1971).

According to Harris and Glock (1981) a transient increase in total leukocyte counts were observed in *Treponema hyodysenteriae* infection.

In case of hog cholerae severe leucopenia in the first or acute phase of illness noticed and it persists in the second phase. Leukocytosis was associated with terminal stage of the disease (Stewart, 1981).

According to Wood (1981) leukocytosis may occur in field cases of Erysepelas after several days of infection possibly from mixed bacterial infection but in uncomplicated acute swine erysipelas leucopenia occur.

The ability of the bone marrow to respond to a bacterial infection was measured by the total leukocyte count particularly neutrophils. The response to bacterial infection was neutrophilia with or without left shift. Most of the diseases caused by viruses lead to leucopenia. A variety of non infectious conditions like chemical intoxication stimulates release of indigenous corticosteroids and caused tissue destruction leading to neutrophilia (Jain, 1986).

According to Duncan and Prasse (1988) neutrophilia was a common finding in bacterial infections like Actinobacillosis, *Escherechia coli* infection, klebsiella infection, salmonellosis, pasteurellosis etc.

Leukocytosis due to an absolute increase in neutrophils was a common feature of inflammatory reaction especially in those induced by bacterial infection (Vegad, 1996).

2.6 NECROPSY FINDINGS AND HISTOPATHOLOGY

Necropsy of the pigs deficient in Vitamin E and selenium revealed hepatic necrosis, icterus, generalized oedema, anaemia, pale areas in cardiac and skeletal muscles and yellowish brown discolouration of body fat (Ewan *et al.* 1969).

Mulberry heart disease characterized by acute congestive heart failure with myocardial congestion, haemorrhage etc., while hepatosis dietetica characterised by massive haemorrhagic necrosis in liver. According to authors all the cases were presented with a history of the owner that most of these pigs found dead without observing premonitory signs, and it occurred in well-nourished rapidly growing pigs (Vanvleet *et al.* 1970).

In an outbreak of swine fever in Barbados, Huston (1974) observed necropsy findings like peripheral haemorrhage in the lymph nodes, petecheal haemorhage in the renal cortices, larynx, stomach, urinary bladder, gall bladder and lungs, button ulcers in caecum and colon. The prominent histopathological lesions were damage to the blood vessels. Fibrinoid necrosis of the vessels of the brain and its meninges.

Necropsy of pigs died of vitamin E - Se deficiency revealed small pale streaks in ventricular myocardium and hepatic lobes, accumulation fluid in the thoracic and abdominal cavities and eosophago gastric ulcer (Ruth and Vanvleet 1974).

Necropsy of pigs fed diet deficient in Vitamin E and selenium revealed hepatosis dietetica, skeletal muscle degeneration, mulberry heart, cutaneous microangioapthy, gastric parakeratosis and ulcers and congestion of liver, kidneys and intestine (Bengtsson *et al.* 1978).

Postmortem examination of the pigs died from mulberry heart disease revealed distension of pericardical sac with clear fluid and opaque discolouration of the pericardium, necrosis and echymotic and suffusive haemorhages on the myocardium, hyperaemia in kidneys and hyperaemia and ulcers in stomach and intestine. The histopathology of these cases revealed degenerative changes in the heart, liver, kidney and brain and emphysematous changes in the lungs (Kwatra *et al.*, 1980).

Acland and Little johns (1981) observed blood stained fluid in the abdominal cavity, myocardial pallor and they found more lesions in the right ventricle during the post mortem examination of the pigs died of encephlomyocarditis.

No specific findings in carbamate and organoposphate poisoning but in some cases fluid accumulation in respiratory tree and gastro intestinal tract could be detected. A heavy set of lungs reflects pulmonary oedema (Carson and Lloyd, 1981).

Complete necropsy of the carcass of the anthrax suspected cases should be discouraged to prevent spread of infection. On necropsy tonsils covered with fibrinous exudates, red swollen pharyngeal mucosa, edematous cervical region, enlarged, red or gray yellow coloured, mandibular and superpharyngeal lymph nodes were observed (Ferguson, 1981).

Lobar consolidation and edema of the lungs, usually in dependant portions, pleural effusions, fibrinous pleuritis, pericarditis and purulent arthritis was the postmortem lesions in pasteurellosis (Farrington, 1981).

Fibrinous meningitis, pleuritis, pericarditis, peritonitis and arthritis were found during the post mortem examination of pigs having *Haemophilus parasuis* infection (Nicolet and Scholl, 1981).

Peripheral haemorrhage at the lymph nodes, occurrence of numerous echymotic turkey egg haemorrhages in the kidney, infarctions of the spleen, button ulcers in the large intestine were the major lesions observed in necropsy of pigs died of hog cholerae (Stewart, 1981).

Cutaneous haemostasis in snout, ears, jowls, throat, abdomen and thighs, petechiae and echymosis in kidneys, epicardium and atrial muscles, catarrhal haemorrhagic gastritis, spleenomegaly were the necropsy findings in erysipelas (Wood, 1981).

Anderson (1985) observed hydropericardium and severe necrosis of heart muscle on necropsy of affected pigs in a study to reveal the causes of acute death in feeder pigs. The author also noted cessation of mortality when the pigs were injected with a vitamin E / Se product.

In case of heat stroke necropsy findings included foamy discharge with blood from nostrils, edema, congestion and hemorrhage of lungs and other viscera. (Casteel *et al.*, 1987).

On postmortem examination pale subcutaneous tissue, dilated flabby, round shaped heart and hydropericardium were observed by Dhanya *et al.* (2003) during their study about the swill fed pigs.

At necropsy of pigs died of pasteurellosis gelatinization of the fat, serous fluid in the pericardial sac, fibrinous deposits on the pericardium, peticheial and echymotic haemarhages in the epicardium and endocardium, congestion and oedema of lungs, serous fluid in abdominal cavity, engorgement and severe congestion of intestinal mucosa were observed by Sujatha *et al.* (2003).

The postmortem lesions observed in *Streptococcus suis* infection by Ravishankar *et al.* (2005) were severe congestion of lungs, liver, stomach and kidneys.

On gross pathology of pigs died of *Clostridium difficile* infection revealed mesocolonic oedema, hydrothorax, and ascites (Silvapru and Bilkei, 2005).

2.7 TREATMENT AND CONTROL

Vanvleet *et al.* (1970) reported that supplementation of small amount of Se or vitamin E or both were necessary to prevent field cases of hepatosis dietetica or mulberry heart disease in pigs.

Whole cereals, especially the germ part and green forages are good sources of vitamin E (Whitehair, 1970).

Tocopherols were found almost exclusively in plants and only to a minimal degree in animal tissues (Schudel *et al.* 1972)

Vanvleet *et al.* (1973) recommended 20,000 to 30,000 IU of vitamin E /ton as dietary supplementation in pregnant, lactating, starting and growing pigs.

Herrick (1975) reported that successful treatment of deficiency of Se and vitamin E in farm animal had been achieved through the use of Se and tocopherol.

Vitamin E was one of the most important vitamins for pigs and it occurs naturally in green forages and high quality cereals, which had been satisfactorily stored (Whittemore and Elsley, 1979).

According to Ullery (1981) when pigs were fed corn soybean diet deficient in vitamin E - Se it should provided with Se supplements at a rate of 0.1mg/kg, vitamin E supplements should include at least 10 – 20 IU/kg and 30 IU/kg in problem herds.

Chloramphenicol and sulpha trimethoprim was the antimicrobial drug of choice for initial treatment of septicaemia caused by *Salmonella choleraesuis* in swine (Smith, 1986).

According to Gillespie (1987) good dietary source of vitamin E includes whole cereal grains and green forages. The author reported that the vitamin E level in the feed declined on storage.

Vitamin E was widely distributed in green feeds and cereal grains (Ranjhan, 1997).

Abrahm (2000) reported that vitamin E was present in varying degrees in all green leafy foods.

Materials and methods

3. MATERIALS & METHODS

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy over a period of three semesters from February, 2005 to April, 2006. Animals from various organized pig farms in Kerala where sudden deaths were reported had been utilized for the investigation.

3.1 OUTLINE OF THE STUDY

During the period of study more than 50 animals were reported to have died suddenly and out of this six animals were subjected to post mortem examination. Fortifive live animals those were in contact with pigs died suddenly were utilized for the present investigations. Six apparently healthy animals which were maintained on identical mangemental condition in other farms where no sudden death reported so far were utilized as control animals. Based on the clinical signs manifested by animals in contact they were grouped three

Group I Healthy controls

Group II Contact animals without any obvious clinical signs of disease

Group III Contact animals with clinical signs of illness

3.2 DATA COLLECTION

The history pertaining to the feeding and management practices and clinical signs of the ailing pigs, were collected using a questionnaire by interaction with farmers.

3.6 EVALUATION OF THE SAMPLES COLLECTED

3.6.1 Wet film examination

Wet film examination was conducted in live animals which were in contact with those pigs which died suddenly and in control animals.

3.6.2 Leukogram

3.6.2.1 Total leucocyte count (TLC)

Total WBC count was estimated using Thoma's fluid as per Coles (1986) and value expressed as total cells/mm^3 of blood.

3.6.2.2 Differential leucocyte count (DLC)

Blood smear was stained by Leishman's stain and 100 leucocytes were counted under oil immersion objective and differential counts were expressed as percentage (Benjamin, 1985).

3.6.3 Serum enzyme analysis

The serum samples were analysed for enzymes such as asparate amino transferase, creatine kinase and lactic dehydrogenase using Microlab 200 with commercially available kits of AGAPPE.

3.6.4 Microbiological examination

The blood smears obtained from the pigs were examined under oil immersion objective of the microscope. The blood collected under sterile conditions inoculated into brain heart infusion (BHI) agar and incubated at 37°C for 24 hours. In cases where growth obtained gram's staining was done and examined microscopically for the morphology of the organism. In vitro antibiotic sensitivity of the organisms was studied using Disc Diffusion

Technique (Barry, 1976). Antibiotic disc used were ceftriaxone, cefotaxime, enrofloxacin, gentamicin, oxytetracycline, furazolidone and cotrimoxazole. The zone of inhibition of bacterial growth around each disc was measured and interpreted as sensitive(S), Moderately Sensitive (M), or Resistant (R) by comparing the ranges given by the manufacturer.

3.6.5 Histopathological examination

The tissues were processed by routine paraffin embedding techniques (Sheehan and Hrapchack, 1980). Sections were cut at 4 micron thickness and stained with routine Haematoxylin and Eosin (Bancroft and Cook, 1984) for histopathological studies. The stained sections were examined in detail under light microscope.

3.7 TREATMENT

All the animals in the pig farm are advised to give a mineral and vitamin mixture containing vitamin E and Selenium @ 10g / animals daily. The animals with clinical signs of illness were treated with antibiotics. The treatment started with oxy tetracycline @ 10 mg/kg then changed the treatment according to the result of culture and sensitivity result. Cefotaxime were given to the animals in the group III @ 50 mg / kg for five days.

3.8 STATISTICAL ANALYSIS

Statistical analysis was done according to the method of Snedecor and Cochran (1994). The means of all groups were compared with that of the control using analysis of variance (ANOVA).

Results

4. RESULTS

In the present study forty five pigs from six different pig farms in various parts of Kerala were taken as study material. These pigs were in contact with the pigs which died suddenly. Six apparently healthy animals were taken as controls. Based on the clinical signs these animals were classified as

Group I Healthy controls

Group II Contact animals without any obvious clinical signs of disease

Group III Contact animals with clinical signs of illness

These animals were subjected to detailed clinical examination and all parameters under study such as signalment, history, physical examination, and leukogram, serum biochemical assay and microbiological examination were carried out.

In the present investigation 29 samples were collected from pigs in contact with the animal which died suddenly, but without any clinical signs of systemic disease (Group II), 16 samples were collected from pigs in contact with the animal which died suddenly, with clinical signs of systemic disease (Group III) and six samples were collected from the healthy control animals (Group I) from the farms where sudden death had not been reported so far. Whenever possible the postmortem examinations of the animals were conducted, gross and histopathological lesions were studied in detail.

4.1 OCCURRENCE OF SUDDEN DEATH

Eight animals out of 45 animals died in one farm. In another farm out of 500 pigs 60 pigs were died during the period of study due to this sudden death

problem. In the other four farms 3-4 animals died suddenly out of 25 animals in each farm. The occurrence of the sudden death was around 12.4 per cent.

4.2 HISTORY

In all the six farms from where the samples collected the feed given were chicken waste and hotel wastes without cooking. Sudden death occurs without showing any premonitory signs in these cases. Apparently healthy animals in good condition were found dead.

4.3 AGE

Age of the animals, which were died all on a sudden, was within the range of 3-6 months. Out of the six farms from where the death reported, the age groups of the affected animals were within 3-4 months, while in one farm it was 5-6 month.

4.4 SEASON

Incidence of sudden death occurred in one farm during the months of August – September while in other farms it occurred during the months of January-March.

4.5 BREED

Duroc and Large White Yorkshire cross breeds were most commonly found dead. About 60 percent Large white Yorkshire cross breeds and 40 percent Duroc cross breeds were affected by this problem.

4.6 SEX

Prevalence of the sudden death was equal in both sexes.

4.7 CLINICAL SIGNS

There was no clinical sign noticed in most of the pigs. Some animals in group III showed dullness, anorexia, and staggering gait before death. The visible mucous membrane of the group I and group II animals were pale pink while most of the animals in the group III showed congested mucous membrane. In case of the group III animals (Fig.7) the early clinical signs reported were reduced feed intake. Varying degrees of loss of appetite was reported by farmers in these cases. In some of the cases (group III) diarrhoea were also reported by the farmers.

4.8 RESPIRATION, PULSE AND TEMPERATURE

Values of respiration, pulse and temperature are given in the table (Table 1). Clinical parameters like pulse and respiration did not show much difference from the control group while animals in group III showed a significant increase ($P<0.05$) in temperature (104.95 ± 0.17^0F).

Table1. Clinical data of animals of group I, II and III (Mean ± SE)

Parameters	Group I (6)	Group II (29)	Group III (16)
Respiration rate (per min.)	16 ± 0.58^a	15.37 ± 0.27^a	14.915 ± 0.40^a
Pulse rate (per min.)	80.3 ± 1.15^a	79.55 ± 0.49^a	80.25 ± 1.15^a
Temperature (^0F)	102.13 ± 0.07^a	102.17 ± 0.04^a	104.95 ± 0.17^b

Number in the parenthesis shows number of animals examined.

Means bearing the same superscript in a row do not differ significantly ($P< 0.05$)

4.9 MICROBIOLOGICAL EXAMINATION

4.9.1 Microscopical examination of the blood smear and wet film

On the microscopical examination of the stained blood smear no organism could be detected. No moving parasitic organism could be detected on wet film examination.

4.9.2 Cultural examination of the blood

From the six farms where the present investigations were carried out, three representative samples from each farm were collected for microbiological examination. Bacterial growths in nutrient agar were obtained from the two farms out of the six farms studied, on 24 hour incubation at 37^0C (Fig.8), while the samples from other four farms were negative for bacterial growth even after 48 hours of incubation. On staining gram negative coccobacilli (Fig.9) were obtained from one farm and gram negative bacilli were obtained from the other farm.

4.9.3 Antibiotic sensitivity test

Gram negative bacilli were obtained by cultural examination of the six samples, three from each farm. The isolated gram negative bacilli were sensitive to cefotaxime and ceftrixone, moderately sensitive to gentamicin and resistant to oxytetracycline, enrofloxacin, furazolidone and cotrimoxazole. In other group gram negative coccobacilli were isolated and sensitive to enrofloxacin, cefotaxime and ceftriaxone, moderatively sensitive to gentamicin and resistant to cotrimoxazole, oxytetracycline and furazolidone. The antibiotic sensitivity of organisms are given in the table (2)

Table2. Antibiotic sensitivity of the gram negative organisms

Antibiotic disc used	Sensitivity of the organisms	
	Gram negative bacilli	Gram negative coccobacilli
Cefotaxime	S	S
Ceftriaxone	S	S
Enrofloxacin	R	S
Gentamicin	M	M
Oxytetracycline	R	R
Furazolidone	R	R
Cotrimoxazole	R	R

S – Sensitive, M – Moderately sensitive, R – Resistant

4.10 LEUKOGRAM

Values of the leukogram are given in the table (3)

4.10.1 Total leukocyte count

The mean Leukocyte count of animals of group I was 16386±682.64 cells/mm^3. In animals of group II and III the values were 17577.24±310.5 cells/mm^3 and 24370±528.77 cells/mm^3 respectively. There is no significant change in the mean value of TLC in group II animals. Statistically significant ($P< 0.05$) increase in total leukocyte count was obtained in group III animals. Comparison of the mean values of the total leukocyte count were given in the Fig.1

Table 3 Leukogram of group I, group II and group III animals (Mean ± SE)

PARAMETERS	GROUP I (6)	GROUP II (29)	GROUP III (16)
Total WBC count (per mm^3)	16386±682.64 [a]	17577.24±310.5 [a]	24370±528.77 [b]
Neutrophil (per cent)	37.50±3.75 [a]	36.79±1.71 [a]	72.1±2.90 [b]
Lymphocyte (per cent)	57.50±2.29 [a]	58.24±1.04 [a]	19.60±1.78 [b]
Monocyte (per cent)	4.67±0.21 [a]	4.21±0.10 [a]	2.9±0.16 [a]

Number in the parenthesis shows number of animals examined.

Means bearing the same superscript in a row did not differ significantly ($P< 0.05$)

4.10.2 Differential count

The mean value of the percentages of the neutrophils, lymphocytes, and monocytes were 37.5±3.75, 57.5±2.29 and 4.67±0.21 respectively in case of group I animals. The mean value of the percentages of the neutrophils, lymphocytes, and monocytes were 36.79±1.71, 58.24±1.04 and 4.21±0.10 respectively in case of group II animals. The mean value of the percentages of the neutrophils, lymphocytes, and monocytes were72.1±2.9, 19.6±1.78 and 2.9±0.16 respectively in case of group III animals.

Statistically no significant difference between the mean value of the lymphocyte percentage of group II animals when compared to the control animals. The mean value of the neutrophil percentage between the animals in the group III and that of the group I differed significantly ($P< 0.05$). There was an increase in

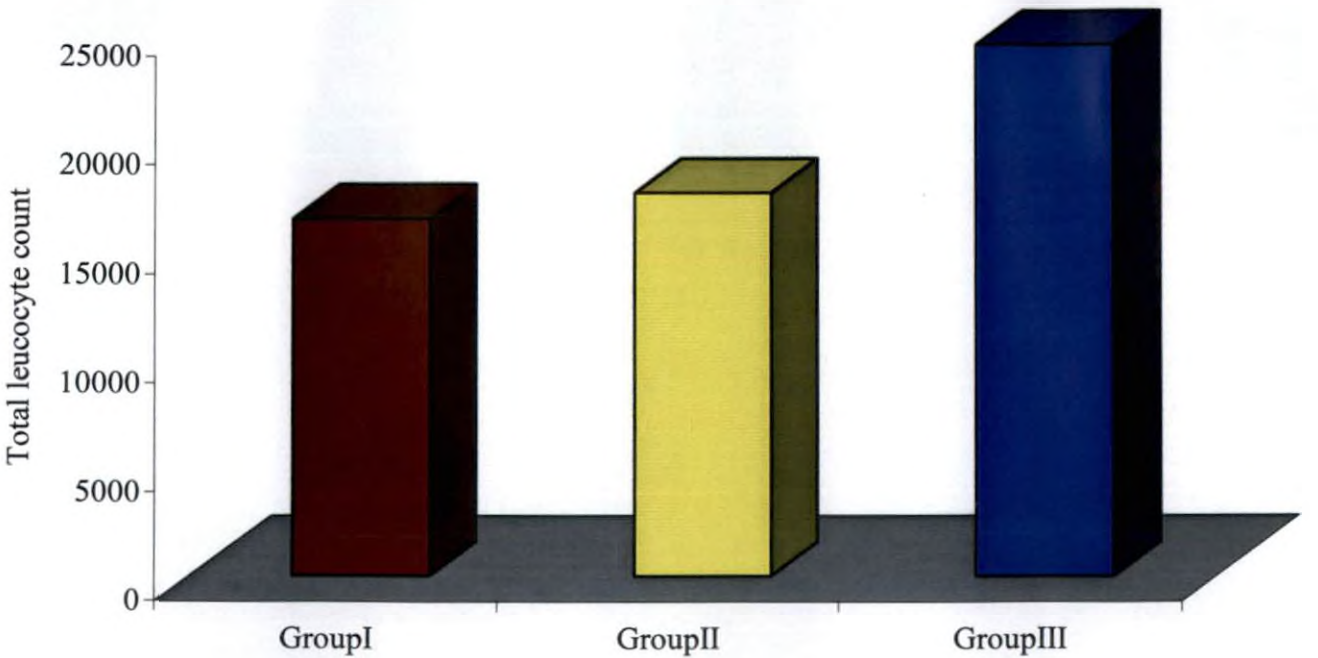

Fig. 1 Comparison of mean values of TLC of group I, II and III animals

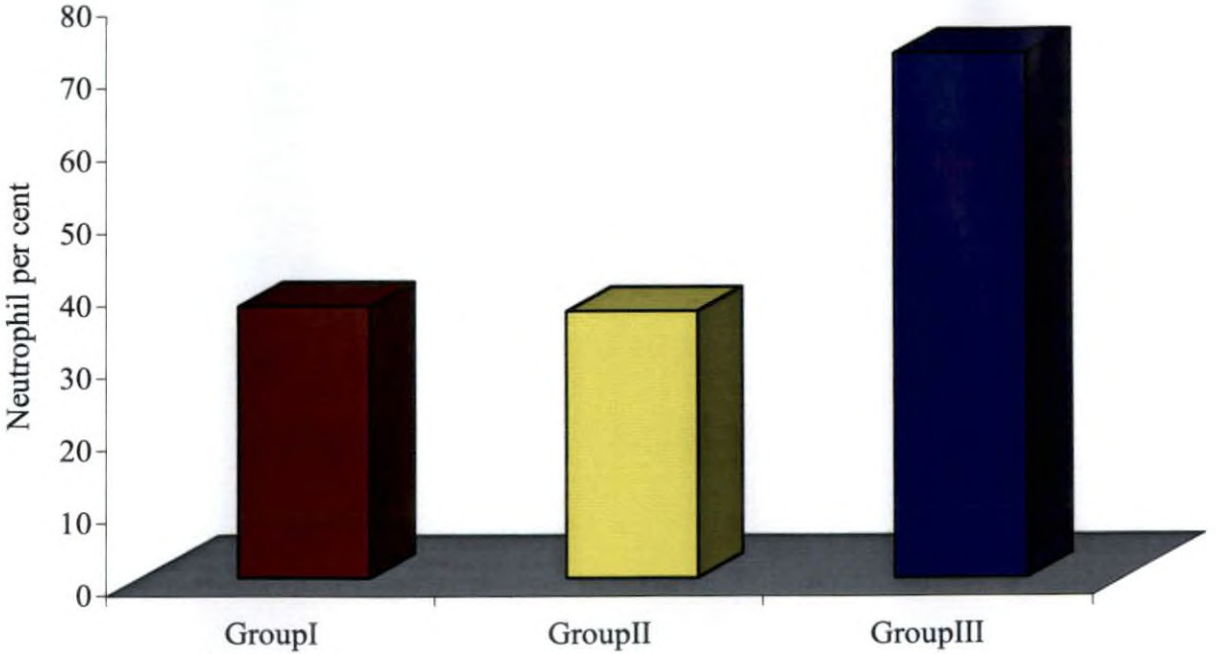

Fig. 2 comparison of mean values of neutrophil per cent of group I, II and III animals

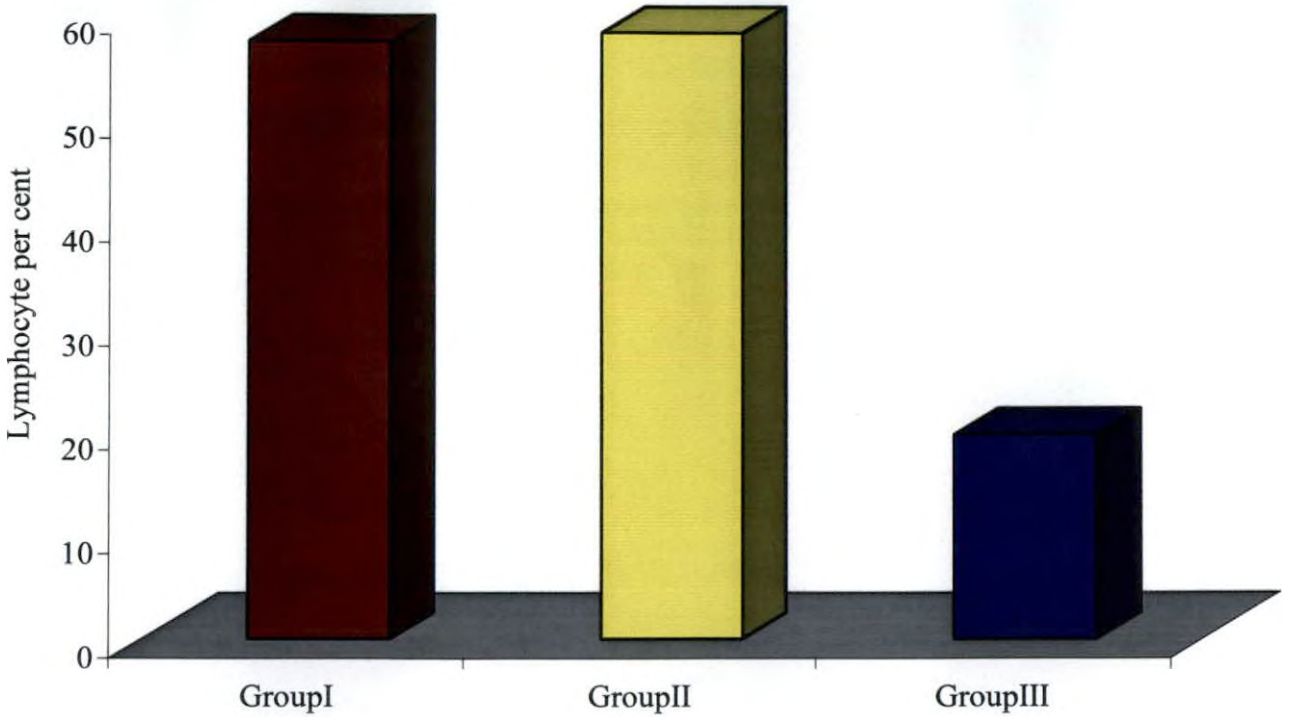

Fig. 3 Comparison of mean values of lymphocyte per cent of group I, II and III animals

the mean value of the neutrophils of these contact animals when compared to the control animals in the group I (Fig.2).

There is no significant difference between the mean value of the percentage of the lymphocyte of the group II animals and that of the group I animals. A statistically significant ($P< 0.05$) decrease in the mean value of the percentage of lymphocyte in group III animals were obtained when compared to the group I animals (Fig.3).

There was no statistically significant difference in the monocyte percentages. The mean value of the monocyte percent in different group of animals remained within the normal range.

4.11 SERUM BIOCHEMISTRY

Serum biochemical values are presented in the table (4).

4.11.1 Creatine Kinase (CK)

The mean values of CK concentration in animals in group I, II and III were 135.83±20.11 U/L, 281.62±9.15 U/L and 162±15.58 U/L respectively. The elevation of mean value of the group II were statistically significant ($P < 0.05$) when compared to the group I. There is no significant difference between the values of group I animals and group III animals. The comparison of the mean values of the different groups were given in the Fig. 4

4.11.2 Lactic Dehydrogenase (LDH)

The mean values of LDH concentration in animals in group I, II and III were 274.17 ± .08 U/L, 443.38±. 04 U/L and 437.30 ±. 06 U/L respectively. The mean values of the group II were significantly ($P< 0.05$) higher when compared to the group I. There is no significant difference between the values of group II animals and group III animals. There was a significant increase in the mean values

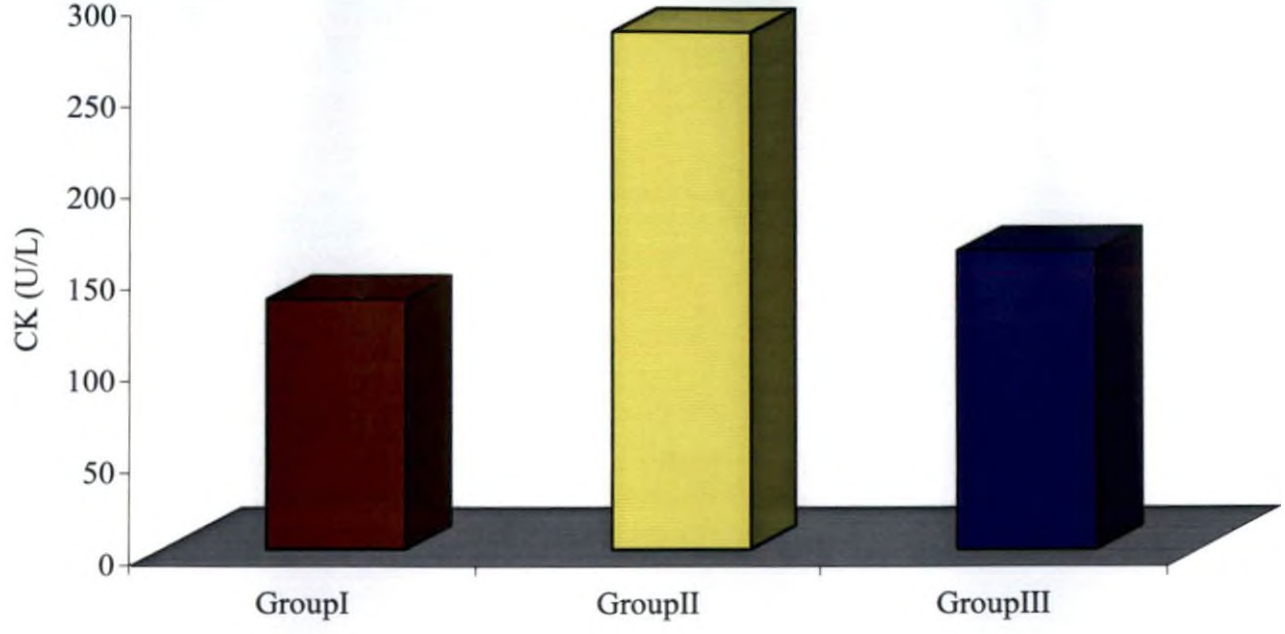

Fig. 4 Comparison of mean values of CK of group I, II and III animals

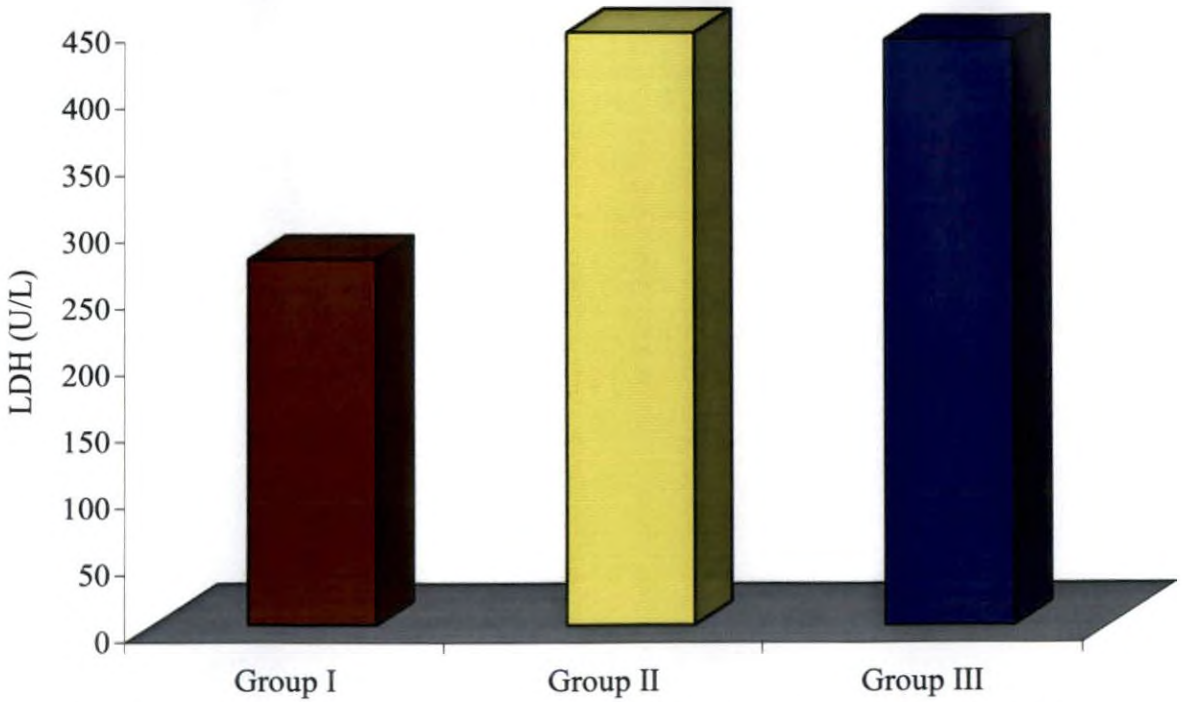

Fig. 5 comparison of mean values of LDH of group I, II and III animals

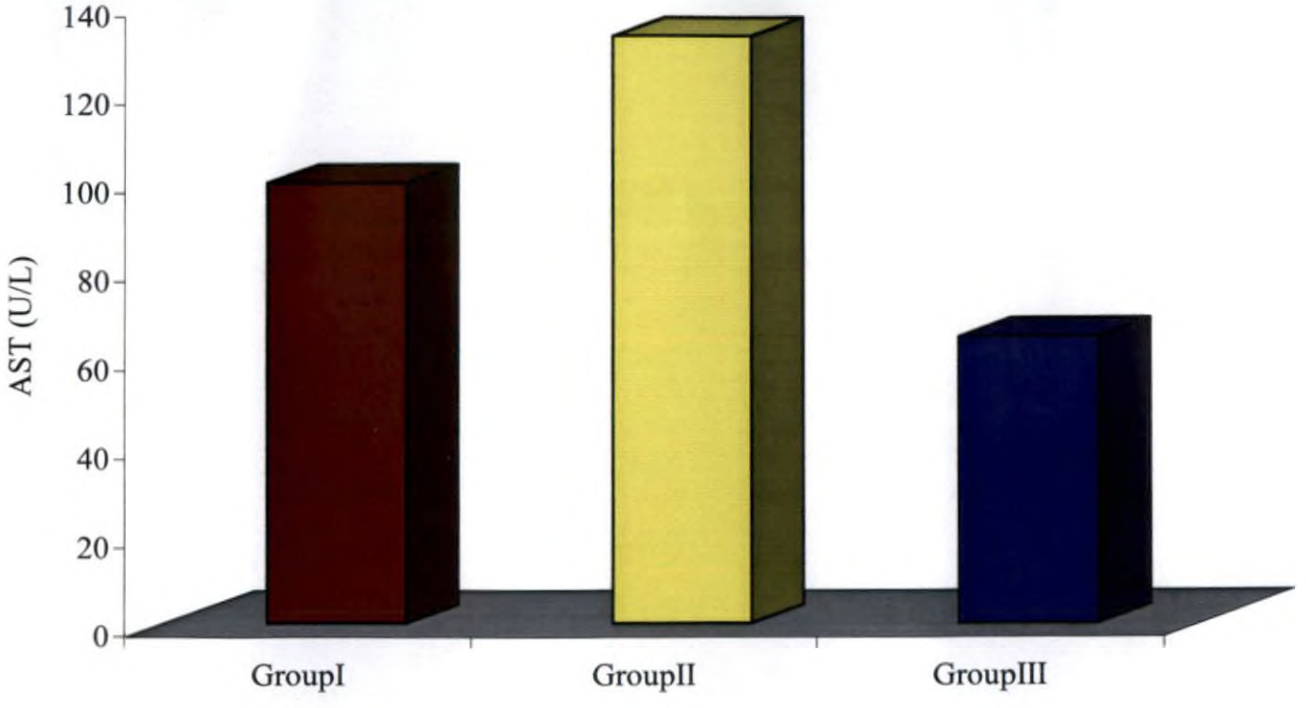

Fig. 6 Comparison of mean values of AST of group I, II and III animals

LDH level of group III animals also when compared to the group I animals. The comparison of the mean values of the different groups were given in the Fig.5

4.11.3 Aspartate Amino Transferase (AST)

The mean values of AST concentration in animals in group I, II and III were 99.17±.07 U/L,132.38±.03U/L and 64.40± .05U/L respectively. The increase in the mean value of the group II were statistically significant ($P < 0.05$) when compared to the group I. Group III animals show a lower value when compared to the group I animals. Comparison of the mean values of the different groups were given in the Fig. 6

Table 4 Biochemical values of group I, group II and group III animals (Mean ± SE)

Parameters	Group I(6)	Group II(29)	Group III(16)
CK (U/L)	135.83±20.11 [a]	281.621±9.15 [b]	162±15.58 [a]
LDH(U/L)	274.17±. 08 [a]	443.38±. 04 [b]	437.30 ±. 06 [b]
AST(U/L)	99.17±. 07[a]	132.38±.03[b]	64.40±. 05[c]

Number in the parenthesis shows number of animals examined.

Means bearing the same superscript in a row did not differ significantly ($P< 0.05$)

4.12 PATHOLOGICAL CHANGES

4.12.1 Gross pathology

The postmortem examination of the six animals from three different farms was conducted out of the six farms studied. In cases where no systemic signs of disease were noticed, hydropericardium (Fig.10), degenerative changes in heart, liver and presence of gastric ulcer (Fig.13) were the prominent lesions. One case

the post mortem examination revealed petecheal to echymotic hemorrhages in the heart, degenerative and congestive changes in brain, liver and lung. Gastric mucosa revealed ulcers. The lymph nodes showed mild hemorrhages. Kidneys showed mild whitish patchy areas in the cortex. In most cases heart showed pale and necrotic areas. Dilated, flabby, gelatinized round heart was also observed (Fig.11). Hemorrhages in the heart and congestion in the gastric mucosa were also noticed (Fig.12) in some cases.

4.12.2 Histopathology

Prominent histopathological lesions were seen in the brain and heart in case of sudden death in pigs.

Histopathological examination of the heart revealed varying degrees of pathological changes varying from mild inflammation, degeneration, and necrosis. Hyaline degenerative changes, fragmented muscle fibres and proliferation of the fibroblasts were also observed (Fig.14). The cardiac muscle fibres showed disagregation of the cardiac myofibrils. The cytoplasms of the cardiac muscle were granular. The muscle fibers were thin and widely separated with a wavy appearance (Fig.15). Focal necrotic areas were also evident in some of the areas. Proliferation of the fibroblast type of the cells and mild to moderate degree of infiltration of mononuclear inflammatory cells were evident in the interstitial spaces. Fragmentation of the cardiac muscle with hemorrhage and mild degree of inflammatory changes were also observed during post mortem examination (Fig.17). The inflammatory changes were noticed in the sections from group III animals while degenerative necrotic changes were prominent in group II animals. In severely affected cases, fragmentation of the hyalinised cardiac muscles was very prominent with haemorrhages and necrotic changes (Fig.16).

The brain revealed perineural oedema and vacuolar degeneration in most of the cases (Fig.18). Focal area of liquefaction necrosis was noticed in one case of group III animals (Fig.19).

The prominent lesions in the lung of group III animals were mild to moderate degree of peribronchiolar infiltration of mononuclear inflammatory cells, along with thickening of the interalveolar septa. Emphysematous changes were also noticed in some cases (Fig.20).

In one case, the hepatic cells revealed fatty degenerative changes with mild disruption of the hepatic cord (Fig.21). Mononuclear cell infiltration was noticed in the portal area in one case from the group III animals.

Kidney did not reveal any significant pathological change except for mild degeneration of the epithelial cells of the renal tubules in case of group III animals.

The stomach and intestine showed mild to moderate degree of congestion and inflammatory changes with infiltration of mononuclear cells in the sub mucosa in both group II and III animals (Fig.22). Hyperplasia of the parietal cells of the stomach was seen in one case.

4.13 RESPONSE TO TREATMENT

For all the animals under study in the present investigation supplementation of a mineral and vitamin mixture containing both vitamin E and selenium were advised and a good response were obtained within fifteen days of treatment. There was drastic reduction in the mortality of the animals when included this supplementation in their diet. In animals where bacterial organisms were obtained on culture appropriate antibiotic therapy was also done. The treatment was started with oxytetracycline and then changed according to the antibiotic sensitivity test (cefotaxime). Good responses were obtained in these animals. In addition to this antibiotic, fluids and injections of phenyl butazone were also given to these animals.

Fig. 7 Animals with clinical signs of disease

Fig. 8 Microbial growth in BHI Agar

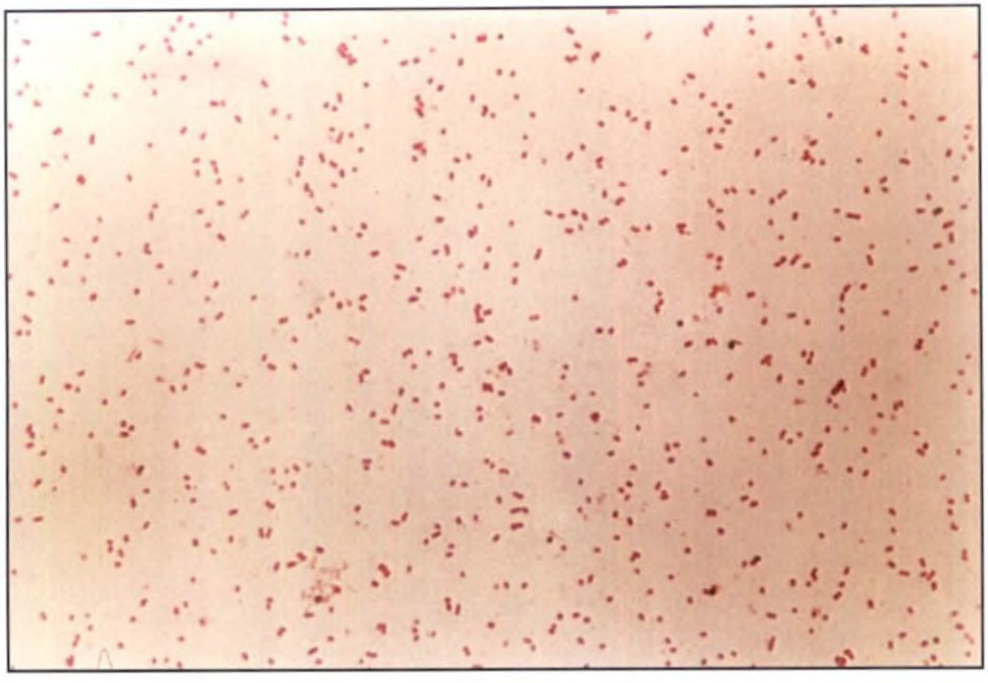

Fig. 9 Gram negative coccobacilli (X 1000)

GROSS PATHOLOGY

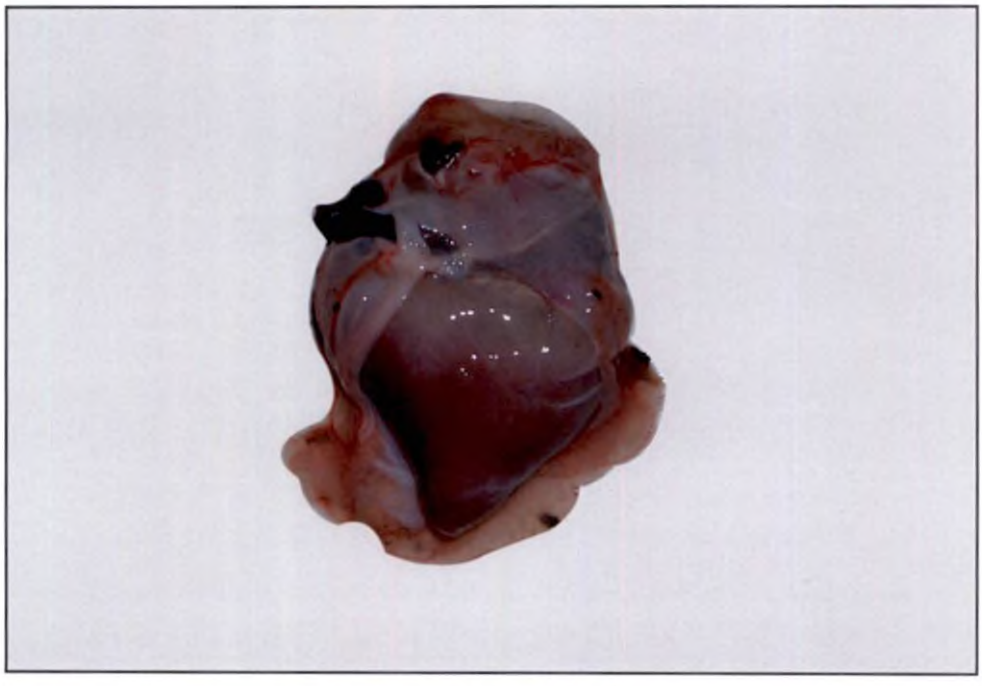

Fig. 10 Hydropericardium

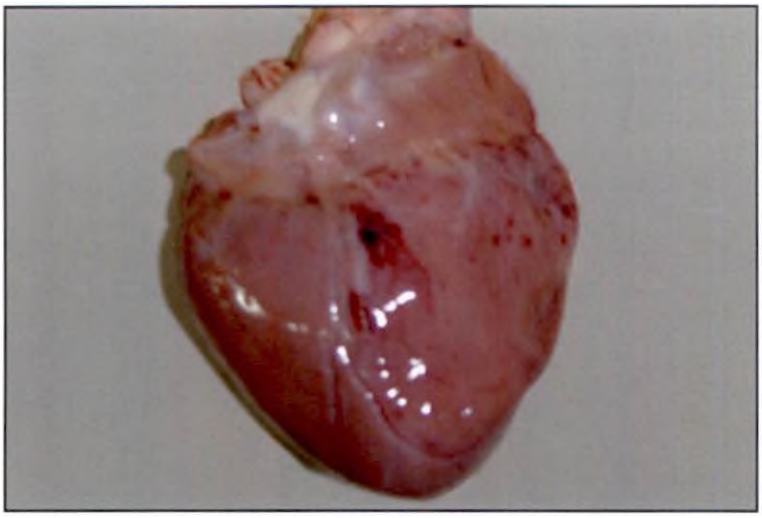

Fig. 11 Heart :- Dilated, flabby gelatinized round heart

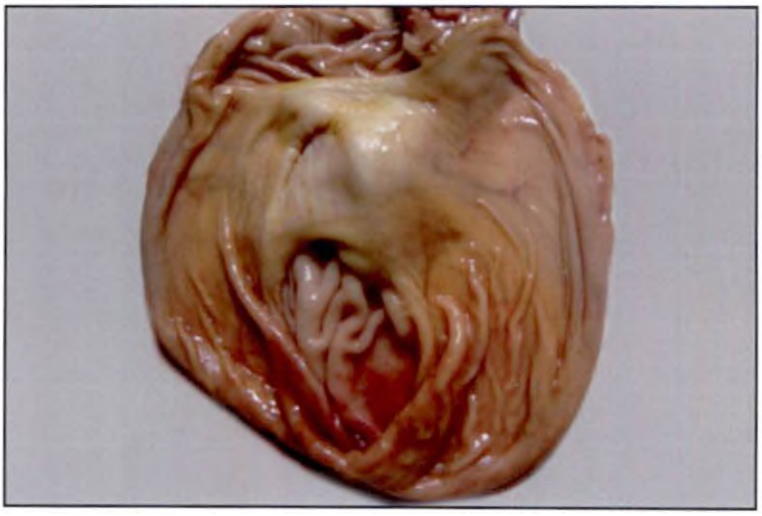

Fig. 12 Gastric mucosa with congestion & ulcer

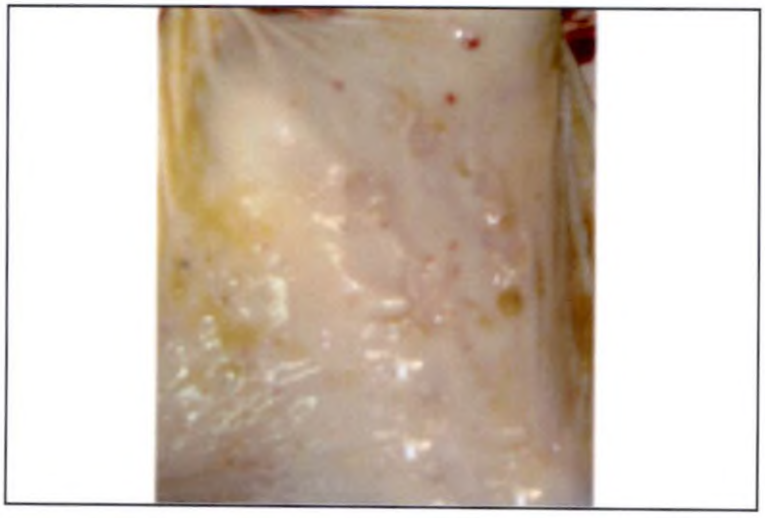

Fig. 13 Gastric mucosa with ulcer

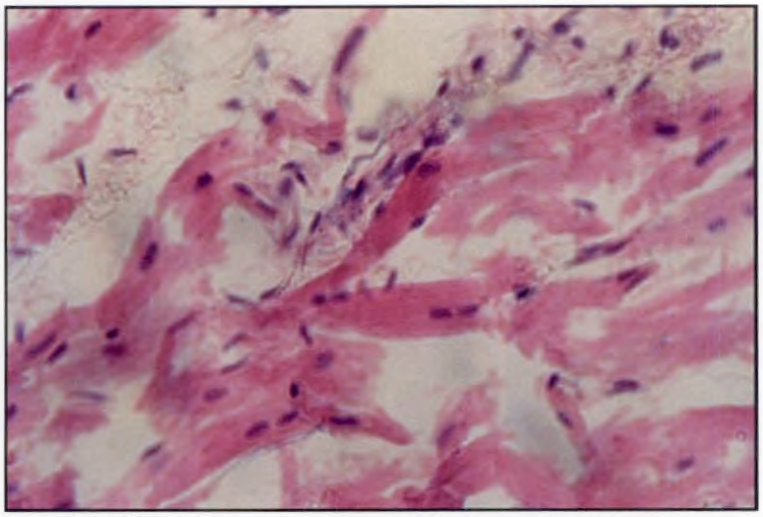

Fig. 14 Heart :- Hyaline degenerative changes and fragmented cardiac muscle fibres, proliferation of the fibroblasts - H&E X 400

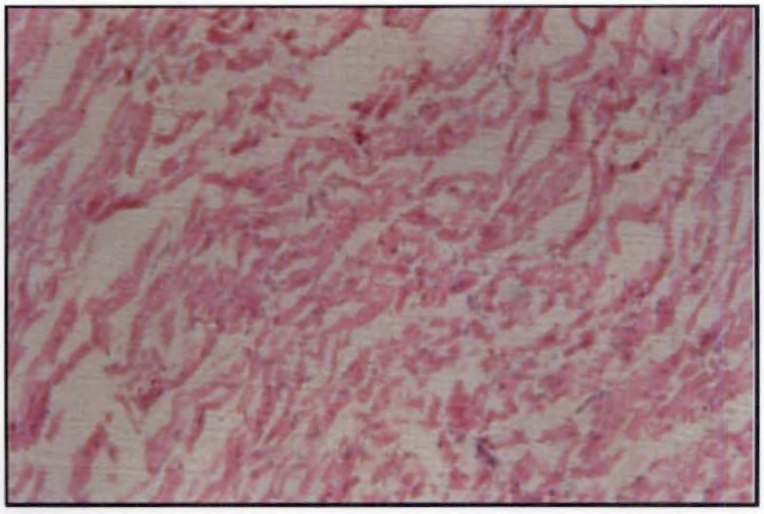

Fig. 15 Heart :- Thinning and separation of the cardiac muscle fibres, H&E X 100

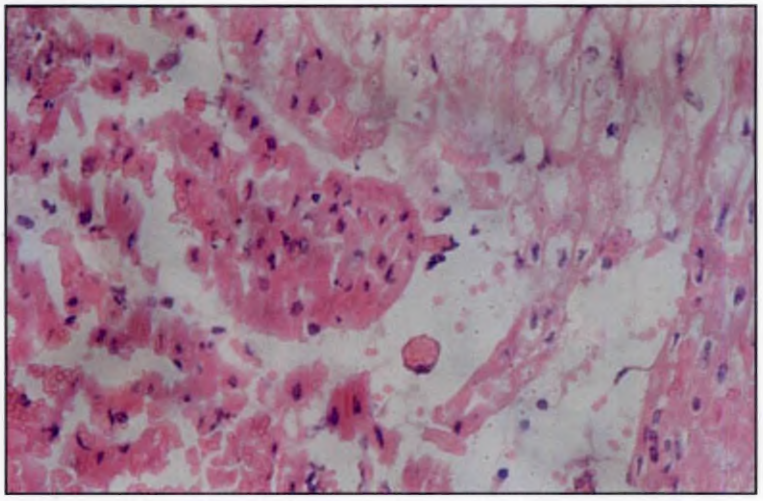

Fig. 16 Heart :- Hemorrhage and necrotic changes, H&E X 400

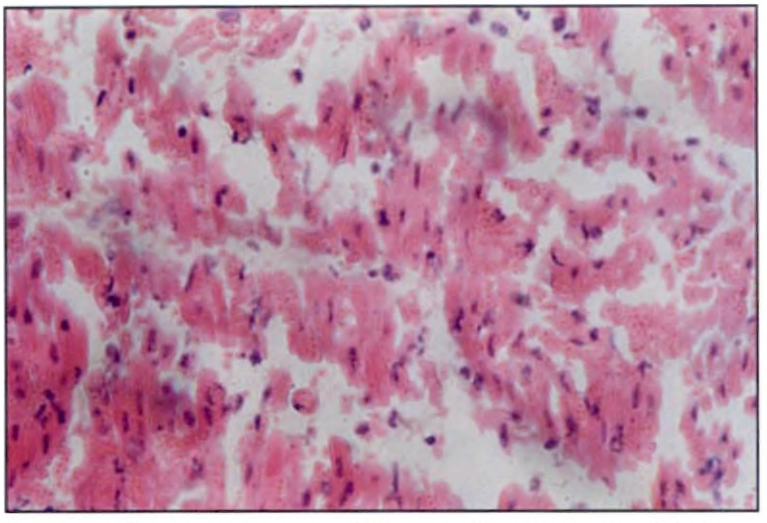

Fig. 17 Heart :- Fragmentation of the cardiac muscle with hemorrhage and mild degree of infiltration of inflammatory cells, H&E X 400

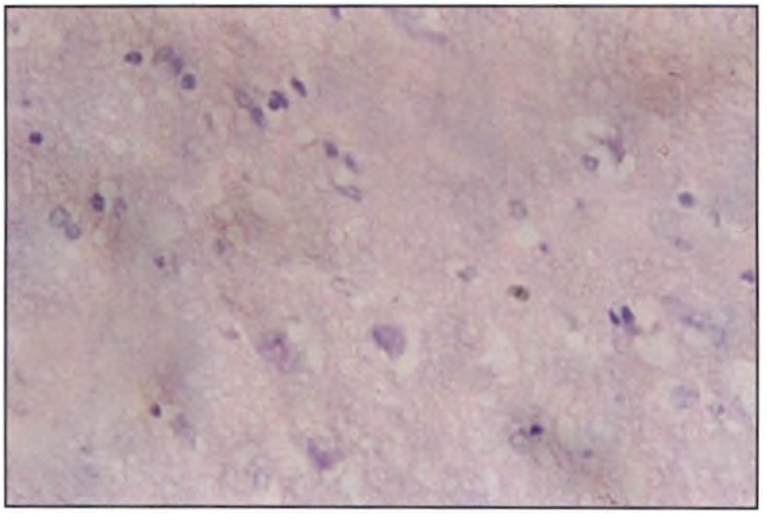

Fig. 18 Brain :- Showing oedematous changes, H&E X 400

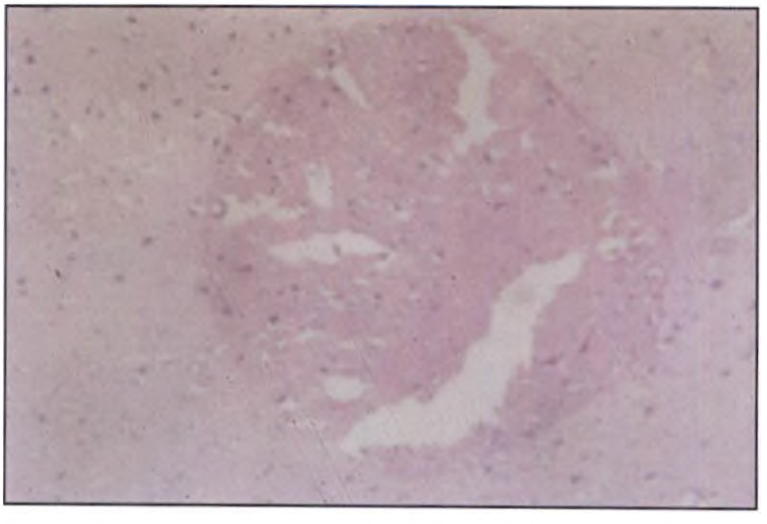

Fig. 19 Brain :- Showing focal area of liquefaction necrosis, H&E X 100

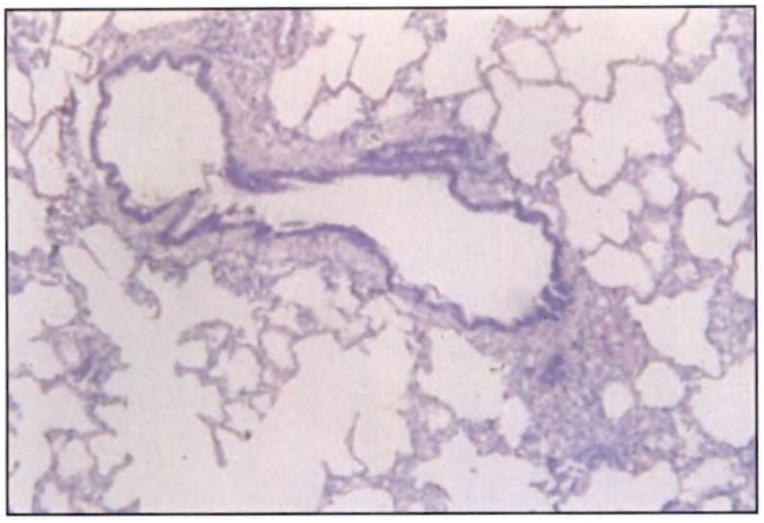

Fig. 20 Lungs :- Emphysematous changes, H&E X 100

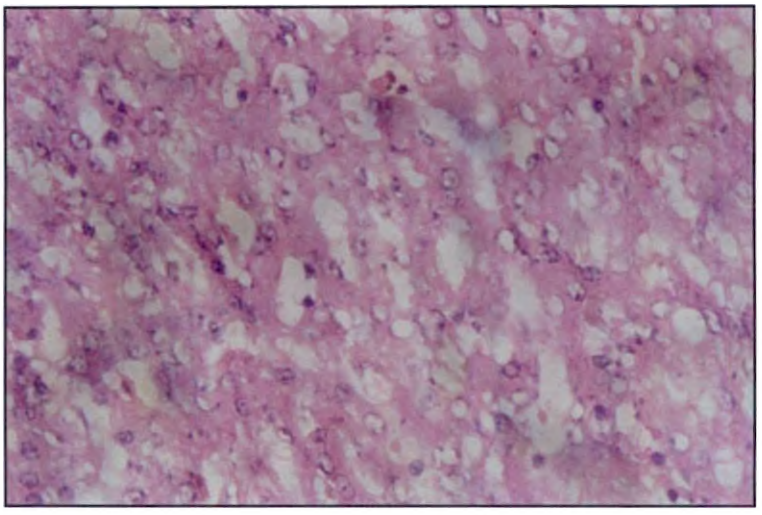

Fig. 21 Liver :- Showing severe degree of fatty degenerative changes, H&E X 400

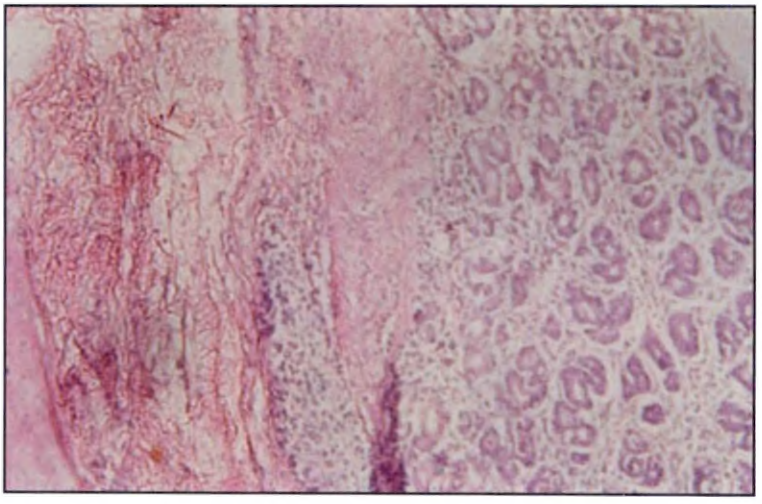

Fig. 22 Intestine :- Infiltration of inflammatory cells in the submucosa, H&E X 400

Discussion

5. DISCUSSION

Sudden death is an important problem encountered by the pig farmers in Kerala. Many infectious and non-infectious causes may be involved in the sudden death of pigs. The present study envisages an insight into sudden death in pigs and the different probable etiological factors leading to sudden death in pigs.

5.1 OCCURRENCE OF SUDDEN DEATH

The occurrence of 12.4 per cent of sudden death was reported from six farms. Occurrence of mulberry heart disease and sudden death in pigs of Assam were reported by Kwatra *et al.* (1980). Dhanya *et al.* (2003) recorded sudden death in swill fed pigs of Kerala.

5.2 HISTORY

In all the farms under the present study, swill was the sole source of feed and the pigs were reared under confinement condition. In most of the cases there were no clinical signs of illness preceding death. More and more pigs were being raised in the complete confinement, without access to pasture. So the entire swine life was dependent upon the nutrients provided to them in the swill feeds. Swill feed contains a greater concentration of poly unsaturated fatty acids. The rancidity and peroxidation of fatty acids deplete the diet of vitamin E or produce specific toxins that precipitate the deficiency (Whitehair, 1970).

In the present investigation the farmers were not providing any concentrates or mineral mixture to their pigs. Hence there are more chances for developing nutritional deficiencies especially the deficiency of micronutrients. Dhanya *et al.* (2003) also reported similar observations.

According to Vanvleet *et al.* (1970) sudden death of pigs which were apparently healthy and well nourished occurs in vitamin E - Selenium deficiency. This is in accordance with the present study where no clinical signs were reported and the animals were apparently healthy (group II).

According to Schudel e*t al.* (1972) various tocopherols were found almost exclusively in plants and only to a minimal degree in animal tissues. Green forages and cereals were good sources of vitamin E (Whitehair, 1970, Whittemore and Elsley, 1979, Gillespie, 1987, Ranjhan, 1997). In the present study chicken waste was the main source of food and no green forages were supplemented to the pigs. This increases the chances for deficiency of vitamin E in these animals.

Ullery (1981) reported an increased vitamin E - Selenium requirement for the pigs under a variety of stress. Complete confinement, overcrowding, higher or lower atmospheric temperature and unhygienic environment were some of the factors causing stress to the pigs. Many of the infectious organisms flare up during the stress condition of the animals.

Increased incidences of sudden death in swill fed animals might be due to variety of factors like confinement system of swine rearing and decrease in use of pasture make them deficient of vitamin E. Increased amount of poly unsaturated fatty acid in the diet were another cause of sudden death in pigs. The rancidity and peroxidation of the fatty acids may either deplete the animal of vitamin E or produce toxins that precipitate the deficiency. In the present study also confinement system of rearing of pigs were practised and the farmers were not providing any pasture to the animal. They were providing swills which mainly include chicken waste and hotel waste without cooking. This may act as a source of infection and pigs under stress are more prone to infection. Swill may act as a source of many bacterial organisms like *Salmonella* spp., *Escherechia coli* etc. Opportunistic pathogens like

Pasteurella spp. may flare up under stress conditions. In the present study also these may be the reason for the infection in group III animals.

5.3 AGE

Occurrence of sudden death was more in three to six month old age groups. According to Dhanya *et al.* (2003) the age of piglets brought for autopsy after sudden death were between four to six months. Vanvleet (1970) reported sudden death due to mulberry heart disease and hepatosis dietetica in two to four month old piglets. According to Koller and Exon (1986) in young animals selenium deficiency caused sudden death due to myocardial dystrophy. Sivenesan (2005) reported sudden death of pigs in Kerala between the age group three to nine months. All these findings were in accordance with the present study.

According to Schwarts (1991) most commonly pigs develop Salmonellosis when stressed during the post weaning or grower phase of four to sixteen weeks of age. In the present study gram negative organisms were identified in two farms (group III), in pigs aged between three to six months.

Kwatra *et al.* (1980) reported sudden death of pigs having mulberry heart disease without showing any clinical signs between the age group of two to three months. Fontaine *et al.* (1977) in their studies about the vitamin E- Se deficiency in pigs found that an appreciable amount of muscular damage occurred at nine weeks of age. In the present study all the pigs affected were in growing stage. During this growing phase animals were in great demand for the nutrients and at many times the demand were not met through the diet provided to them. At post weaning stage animals will be under stress and susceptible to many diseases.

Moreira and Mahan (2002) reported a decline in the serum α- tocopherol concentration of pigs after weaning. In the present study also most of the

affected pigs were at post weaning stage and there is an increased chance for the vitamin E - Selenium deficiency.

5.4 SEASON

In the present study reports of sudden death occurred during the month of August to September and January to March. Kwatra *et al.* (1980) also reported similar observations where as Pathak *et al.* (2004) reported maximum mortality of piglets during the June to September period and then March to May. They reported maximum death due to septicaemia during December to February. Vanvleet (1970) reported peak incidence of mulberry heart disease and hepatosis dietetica in early spring, late summer and early fall.

Increased incidence of sudden death during this period might be due to their limited ability to cope with the environmental stressors like high and low temperatures, humidity, disease and limited nutrition predisposes piglets to high rates of mortality.

5.5 BREED

In the present study cross bred Large White Yorkshire and cross bred Duroc were affected mainly. Kwatra *et al.* (1981) reported mulberry heart disease and sudden death in Landrace pigs in Assam.

The population of cross breds of Large White Yorkshire and Duroc were more in these areas compared to other cross breds. Most of the cross breeds were fast growing and there is an increased demand for the nutrients. But many times this increased demands were not met, and the animal may suffer from deficiency of nutrients. In the present study no reports of sudden death in desi pigs were obtained from any farm. The probable reason for this may be that these pigs were more adapted to the environmental condition prevalent in Kerala. Hence they can adjust with the various environmental stressors and become more resistant to various disease conditions.

5.6 SEX

In the present investigation mortality of both sexes were almost equal. Lay *et al.* (2002) and Sujatha *et al.* (2003) reported a higher percentage of mortality among the males compared to the females. But Prasad *et al.* (1987) observed no significant effect due to sex in piglet mortality. This finding was in accordance with the present study. The similar pattern of mortality among both sexes may be due to equal distribution of males and females in the population.

5.7 CLINICAL SIGNS

In the present study the animals died suddenly without any premonitory clinical signs (group II). Some of the animals (group III) showed increased temperature, staggering gait, dullness and diarrhoea. According to Belschner (1972) in Pasteurellosis high temperature, lack of appetite, depression was noticed. The author reported similar symptoms in Salmonellosis also. In some of the animals in group III diarrhoea were also noticed. *E. coli* were one of the most common pathogen responsible for diarrhoea in pigs (Johnson *et al.* 1992). Fitzgerald (1988) also reported *E. coli* as an important pathogen causing pre weaning and post weaning diarrhoea in pigs. According to Tubbs (1988) salmonellosis caused mucohaeomorrhagic diarrhoea in growing pigs. In the present study also diarrhoea may be caused by bacterial infections as revealed in cultural examinations.

5.7.1 RESPIRATION, PULSE AND TEMPERATURE

In the present study respiration, pulse and temperature of the all contact animals were noted. Sujatha *et al.* (2003) reported an increased temperature of 104-105° F in case of pasteurellosis in pigs. In the present study respiration, pulse and temperatures were normal in animals from most of the farms. Animals from two farms (group III) showed a higher degree of temperature. This may be due to bacterial infections as revealed in microbiological examinations. In all other farms temperature were within the normal range as mentioned by Kelly (1974).

5.8 MICROBIOLOGICAL EXAMINATION

5.8.1 Blood smear and wet film examination

No organism could be detected in blood smear and wet film.

5.8.2 Cultural examination of blood

In the present investigation representative samples from the two farms (Group III) were positive for bacterial growth in culture media. Gram negative organisms were identified in these animals. These gram negative organisms may be *Pasteurella* spp., *Salmonella* spp. or *E. coli*. Chances for flaring up of the opportunistic pathogens like *Pasteurella* spp. were more in farms under study. They were gram negative bipolar organisms. Here another chance is for Salmonellosis since the pigs were being fed with swill. The swill may act as a source of infection. *E. coli* being an environmental pathogen, when animals were in stress it can cause infection. Contaminated environment may act as a source of this infection.

Members of the genus *Pasteurella* were small gram negative rods or coccobacilli (Farrington, 1981). In the present study also gram negative coccobacilli were obtained. *Salmonella* spp. was another gram negative organism commonly seen in piglets and they are gram negative bacilli (Wilcock, 1981). *E. coli* was another group of gram negative bacilli causing sudden death by oedema disease (Wilson, 1981). In the present study also gram negative bacilli were obtained.

Trapp *et al.* (1970) in their study about the mulberry heart disease found that results of microbiological examination were negative in many of the cases. In the present study results of microbiological examinations from the four farms (Group II) out of the six farms were negative.

5.8.3 Antibiotic sensitivity test

One isolate of gram negative bacilli were sensitive to cefotaxime and ceftriazone while another group of gram negative coccobacilli organisms were sensitive to enrofloxacin, cefotaxime and ceftriazone and moderately sensitive to gentamicin. Malorny *et al.* (2003) reported incidence of quinolone resistance in *Salmonella* spp., but here one group of organisms (coccobacilli) were sensitive to enrofloxacin. In this study all organisms isolated were sensitive to cefotaxime and ceftriazone. Similar observations were reported by Yang *et al* (2004). Here fifty per cent organisms might not have developed resistance to enrofloxacin group of drugs. According to Bentley (1983) oxytetracycline was an effective drug against salmonellosis and pasteurellosis. But in these cases oxytetracycline was found to be not very effective. These groups of organisms might have developed resistance to these antibiotics.

According to Smith (1986) gentamicin were effective against gram negative septicaemias by *E coli*, *Salmonella* spp., *Enterobactor* spp., *Pasteurella* spp. etc. Here in this study also moderate sensitivity of the organisms against gentamicin discs were obtained. Gannon *et al.* (1988) recorded that gram negative *E coli* were sensitive to gentamicin. Falade *et al.* (1989) in their study about the antibiotic sensitivity found that many of the gram negative organisms were sensitive to gentamicin. Paul and Soman (1989) reported sensitivity of gentamicin and streptomycin in case of *Pseudomonas aeruginosa* infection. They also observed resistance of these organisms towards kanamycin, tetracycline, ampicillin etc.

Indiscriminate uses of antibiotics invariably result in the development of antibiotic resistant strains of bacteria and renders treatment more difficult. In the present study also resistance towards many antibiotics like oxytetracycline, cotrimoxazole, furazolidone were noticed.

5.9 LEUKOGRAM

5.9.1 Total leukocyte count

The animals in the Group II showed TLC within normal range (17577.24). Trapp *et al.* (1970) reported normal level of haematological values in vitamin E - Se deficient pigs. Fontaine *et al.* (1977) also reported that total leukocyte counts were not affected by vitamin E - Se deficiency.

In case of animals in the group III there is an increase in the total leukocyte count compared to the control animals. According to Jain *et al.* (1986) there is an increase in the total leukocyte counts in bacterial infections. This was in accordance with the present findings. Doxely (1971) and Vegad (1996) also reported leucocytosis in bacterial infections.

5.9.2 Differential leukocyte count

There is an increased neutrophil percentage in animals in the Group III while in other animals (group II) the percentage of the neutrophils was within the normal range. According to Duncan and Prasse (1988) neutrophilia was a common finding in bacterial infections like *E coli*, *Klebsiella*, *Salmonella*, and *Pasteurella* spp.. According to Doxely (1971) leukocytosis due to neutrophilia occurs in bacterial infections or inflammatory lesions. Vegad (1996) also reported leucocytosis due to an absolute increase in neutrophils in bacterial infections.

The decrease in the lymphocyte percentage in animals in the Group III was recorded. This may be a relative lowering of the lymphocyte percentage due to the increase in neutrophil percentage. In the present study the percentage of neutrophils were within the normal range in group II animals.

Friendship *et al.* (1984) recorded the mean value of the neutrophils (32.2 per cent), lymphocyte (49.8 per cent) and mononcytes (6.7 per cent) in healthy swine. This finding was almost similar to the differential leukocyte count of the

animals in the Group I and II. There is no significant difference in the monocyte percentage.

5.10 SERUM BIOCHEMISTRY

5.10.1 Creatine kinase

The mean value of serum creatine kinase level was significantly elevated in animals of the group II. The normal level of creatine kinase value ranges from 2.4 to 22.5 U/L (Kaneko *et al.* (1997). Friendship *et al.* (1984) reported the normal range as 91 to 1251 U/L. Dubriel and Lapierre (1997) reported it as 297 U/L. There is a great variation reported with normal value of CK in healthy pigs. Creatine kinase was an enzyme specific for muscle tissue, an increased level of CK indicate muscle damage which caused leakage of enzyme into the extra cellular space (Harapin, *et al.* 2003). According to Tolling and Jonsson (1983) there is an increase in CK-MM acivities in pigs having myocardial and skeletal muscle necrosis. Elevated serum CK activity was a useful indicator of subclinical muscular dystrophy in vitamin E and Se deficiency in swine (Fontaine *et al.*1977). In the present study also the contact animal in the Group II showed an elevated level of serum creatine kinase activity compared to the control animals. Nutritional deficiency of vitamin E – Se resulted in muscle membrane instability and release of CK into serum (Kramer and Hoffman, 1997). Ruth and Vanvleet (1974) reported an increase in the CPK activities in pigs fed a diet low in vitamin E and Selenium. In the present study also there is no supplementation of any vitamin E or Se containing product in diet of pigs. Hence there is a chance for the occurrence of vitamin E - Se deficiency in these animals and the sudden death could be attributed to the deficiency of vitamin E and Selenium in these animals. Animals in the group III also showed a higher level of CK when compared to the control animals but not to the significant level. This may be due to the fact that these animals might be in the initial phase of the vitamin E – Se deficiency and in these group of animals the major pathology were associated with infection with

micro organisms suspected to be *E. coli, Salmonella* spp., *Pasteurella* spp etc. Hence the sudden death reported could be due to infection.

5.10.2 Lactate dehydrogenase

In the present study there is a significant increase in the mean value of LDH in Group II animals when compared to the control group. Ruth and Vanvleet (1974) reported an increase in the plasma LDH activity in pigs fed a diet low in vitamin E and Selenium. According to Tolling and Jonsson (1983) there was an increase in the LDH I and II activity in pigs having myocardial and skeletal muscle necrosis. In case of vitamin E - Se deficiency heart is the primary organ affected and there is an increased chance for the elevation of LDH level in these cases. Grandhi *et al.* (1993) reported low level of LDH in pigs fed vitamin E and Se supplement. An increase in LDH level was an indication of myocardial necrosis (Vasudevan and Sreekumari, 1995). There is significant difference between the control animals LDH level and that of the animals in group III. Serum LDH activities increased as a result of muscle injuries but they may also increases as a result of injury to many other organs (Thrall, 2004). This may be the reason for the increased LDH value in the group III also.

5.10.3 Aspartate amino transferase(AST)

In the present study there is significant increase in the mean value of AST in the animals of Group II from that of the control animals (Group I). Although CK was more specific marker of muscle damage than AST, it was frequently used to complement creatine kinase changes (Kramer and Hoffman (1997). Vanvleet *et al.* (1970) reported an increased AST level in pigs with hepatosis dietetica, caused by the deficiency of the vitamin E and Seleniium. Vanvleet (1973) reported an increased AST level in animals fed a diet low in vitamin E and Se. Grandhi *et al.*(1993) reported a low level of AST in piglets when fed diet containing vitamin E and Se. Fontaine *et al.* (1977) reported a transient increase in the AST level in cases of piglets with Vitamin E - Se deficiency. These when correlated with the findings of the present investigation

it can be presumed that deficiency of vitamin E- Se could be the reason for the elevation of AST in these piglets. In case of animal in the Group III the cause of death may due to infectious organisms. Here in these animals there is a decrease in the AST level when compared to the control animals but it comes under the normal range.

Elevation of creatine kinase, lactate dehydrogenase and aspartate amino transferase together with response to supplementation of vitamin E and selenium, necropsy and histopathological findings are suggestive of vitamin E and Selenium deficiency in these animals.

5.11 PATHOLOGICAL CHANGES

5.11.1 Gross pathology

In gross pathology, haemorrhagic lesions in heart were one of the major findings. According to Robinson and Maxie (1993) linear and echymotic haemorrhages in the heart were major findings in vitamin E - Se deficiency in pigs. Petecheal and echymotic haemorrhages in heart and hydropericardium were also noticed. Similar observations were made by Anderson (1985) and Vanvleet *et al.* (1970) in case of pigs with mulberry heart disease. Vitamin E is an important factor in maintaining the stability and integrity of membranes. Its deficiency caused increased permiability of small vessels and capillaries resulting in transudation and at times haemorrhage (Whitehair, 1970). In Pasteurellosis blood spots may be observed in the serous and mucous membranes of the heart sac and on the heart itself (Belschner, 1972). Epicardial and endocardial haemorrhages and excess fluid in the pericardial sac were also noticed in the *E coli* infection (Neilson, 1981).

Cogestion of the brain was another gross lesion observed. Venous congestion and brain oedema was a finding in Vitamin E and Se deficiency (Robinson and Maxie 1993). Oedema of brain may be present in the *E coli* infection in pigs (Neilson, 1981).

Gastric ulcer was also noted in the present study. Gastric ulcer was a common finding in many of the cases of vitamin E - Se deficiency (Trapp *et al.* 1970, Vanvleet *et al.*1970 and Bengston *et al.* 1978).

The post mortem findings in present investigation was not pathognomonic for any particular disease of pigs but some of the lesions in heart indicate that the cause of death was suggestive of the vitamin E - Se deficiency and / or some bacterial infections.

5.11.2 Hisopathology

The major histopathological lesions were noticed in heart. The heart showed varying degrees of pathological changes ranging from mild inflammation to degeneration and necrosis. According to Robinson and Maxie (1993) myocardial degeneration was the most common manifestation of vitamin E - Se deficiency in growing weaned piglets six to twenty weeks of age. They also suggested that circumstance of sudden death combined with gross and microscopic lesion strongly suggest that the affected pigs die of congestive heart failure following the development of ventricular dysrhythmias. They also reported extensive areas of haemorrhage in vitamin E - Se deficiency. In the present study as there were haemorrhagic lesions in heart in most of the cases, it can be presumed that deficiency of vitamin E and Selenium could be a reason for the development of sudden death in these cases. Hyalin degenerative changes were noticed in the present study. In case of mulberry heart disease these types of changes were observed (Vanvleet *et al.*1970, Bengston *et al.* 1978, Anderson, 1985 and Degritz *et al.* 1994).

Histopathology of brain, liver and kidney revealed degenerative changes. Emphysematous changes were observed in lungs. Kwatra *et al.* (1980) reported similar lesions in pigs with mulberry heart disease.

5.12 RESPONSE TO TREATMENT

In the present study the supplementation of a preparation containing vitamin E and Se could prevent the sudden death in these farms. Vanvleet *et al.* (1970) reported that only relatively small amount of Se or Vitamin E or both were necessary to prevent the field cases of hepatosis dietetica and mulberry heart disease. Trapp *et al.* (1970) reported that the acute death losses due to hepatic necrosis and muscular degeneration were reduced following supplementation of diet containing vitamin E and Se or by the injection of Vitamin E or Se or both in the herds.

In the cases where bacterial growth obtained on culture, the pigs were treated with antibiotics and observed a good response. According to Smith (1986) treatment of gram negative septicaemias should include antimicrobials, fluids and anti prostaglandin drugs like aspirin, phenyl butazone etc. and the auther also suggested cephalosporin group of drugs as an effective antimicrobial in many gram negative septicaemias. In the present study also cefotaxime was used as the antibiotic.

In many cases disease prevention is more important than treatment. The main factors requiring attention are proper nutrition at all time, provision of adequate green feed, clean water supply, avoid overcrowding, hygienic feeding system to avoid contamination of the food, good sanitation in and around piggery, no mud holes or stagnant water, the isolation of the purchased pigs until they are found to be free of disease, the prompt isolation of the sick animals, the burning or deep burriel of the pigs which died of the disease and disinfection of the infected sites and yards, vaccination of the animals for the diseases prevalent in the areas. Good mangemental practices itself can prevent many of the diseases prevalent in pigs.

As the present study involving sudden death of pigs which was suspected to be caused by pathology of heart as evident in the necropsy

findings and elevation of CK, LDH and AST it can be presumed that vitamin E - Se deficiency could be a factor involved in the development of these problem.

The present study can be summarised as follows

1. Sudden death was recorded in farms where swill feed was given which is rich in poly unsaturated fatty acids and deficient in vitamins and minerals.

2. From the necropsy findings and elevated CK it is presumed that sudden death may be associated with cardiac problem which is presumed to be due to vitamin E - Selenium deficiency. As the animals are fed with swill feed there was chance for contamination and bacterial infection as revealed by the culture and sensitivity testing.

Summary

6. SUMMARY

Sudden death in pigs is an important problem encountered by many pig farmers in Kerala. It causes heavy economic loss to the farmers. This unexpected death is mainly found in pigs in apparently healthy condition. In the present study titled "Investigation on sudden death in pigs", around fifty animals were reported to have died suddenly and six animals were subjected to post mortem examination. Forty-five animals that were in contact with the pigs died suddenly were utilized for the study. Six apparently healthy animals, which were maintained on identical managemental conditions in other farms where there is no sudden death reported so far, were utilized as controls. Animals under study were grouped based on the clinical signs as, group I healthy control (6 animals), group II contact animals without any obvious clinical signs of illness (29 animals) and group III contact animals with obvious clinical signs of illness (16 animals). Data pertaining to history, feeding, managemental practices and clinical signs were collected by interaction with the farmers. Detailed clinical examinations of the contact animals were done. Blood samples were collected for haematological, microbiological and biochemical examination. Wet film examinations were conducted in live contact animals.

Detailed post-mortem examinations were conducted in six animals and the gross pathological lesions were noted. Tissues from the carcasses presented for necropsy after sudden death were collected for histopathological examination.

During the period of study sudden death of more than fifty animals were reported. In all the farms chicken waste and hotel waste were fed without cooking. Ages of the affected animals were between three to six months. Occurrence of sudden death reported mainly during the months of January to March and then August to September. There is no sex predisposition in sudden death cases. In most of the animals there was no obvious clinical signs noticed before death. While the

contact animals in group III showed some clinical signs like increased temperature, reduced feed intake, congested mucous membrane and diarrhoea.

During the microscopical examinations of the stained blood smear no organisms could be detected. No moving parasites could be detected on wet film examination. On cultural examination of three representative blood samples from six farms, samples from two farms were positive for microbial growth. Gram-negative organisms were obtained in both cases.

On antibiotic sensitivity testing of the three samples from one farm cefotaxime and ceftriaxone were found to be sensitive while the samples from other farms showed sensitivity towards enrofloxacine, cefotaxime and ceftriaxone, moderately sensitive to gentamicin and resistant to cotrimoxazole, oxytetracycline and furazolidone.

The mean values of total leucocytes count in group II animals were with in the normal range with no significant difference from the animals of group I, while animals of group III showed a significant elevation. The mean values of per cent of neutrophils in animals of group III were significantly higher ($P<0.05$) when compared to the group I animals while there was no significant difference in the neutrophil per cent of group II animals. There is decrease in the value of lymphocyte per cent in group III animals when compared to the group I animals. There is no significant difference noticed in monocyte per cent.

There was a significant elevation in the mean values of creatine kinase in-group II animals (281.62±9.15U/L) when compared to the group I animals. There is no significant difference between the mean values of CK in animals of group III and I. The mean value of LDH concentration in animals of group II (443.38±0.04 U/L) and III (437.3±0.06 U/L) were significantly higher ($P<0.05$) when compared to the controls. The AST values of group II animals (132.39±0.05 U/L) were significantly higher than the group I animals. The mean values of AST of group III animals were within the normal range.

The major gross pathological findings were hydropericardium, degenerative changes and hemorrhages in heart, degenerative changes in liver and gastric ulcers. In group III animals, petecheal haemorrhages in heart, hemorrhagic lymph nodes, gastric ulcer, congestion of brain and mild nephritis were noticed. The prominent histopathological lesions were noticed in brain and heart. Heart showed varying degrees of degeneration, necrosis and inflammation. The brain revealed perineuronal edema and vacoulation.

All the animals under study were supplemented with a mineral mixture containing vitamin E and Selenium @ 10 gram/ animals. Animals in the group III were treated with cefotaxime @ 50mg/kg for five days. This helped in the prevention of further sudden death of pigs in these farms.

From the present study it can be concluded that occurrence of sudden death were observed mainly in farms where swill was the only feed given. Swill is rich in polyunsaturated fatty acids and this increases the demand for the vitamin E – Selenium, which is not met from diet of the animals under confinement. These affects mainly heart as indicated by the elevation of CK, LDH and AST and necropsy findings and histopathology. These swills could act as a source of infection as it was given without cooking.

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INVESTIGATION ON SUDDEN DEATH IN PIGS

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ABSTRACT

Study of "Investigation on sudden death in pigs" was conducted in pigs from various pig farms in Kerala, where cases of sudden death were reported. In these farms around 12.4 per cent of the total pigs were lost due to sudden death. Post mortem examination were carried out in six animals. Clinical materials were collected from the forty-five contact animals and six apparently healthy control animals.

Occurrences of sudden death were common in three to six month old piglets. Most of the animals died without any premonitory clinical signs. All these animals were fed with uncooked swill mainly containing chicken waste. Some of the contact animals showed clinical signs like elevated temperature, congested mucous membranes and diarrhoea. On cultural examination of the samples from the two farms growth of the gram-negative organisms were obtained. Transmission of these organisms to these pigs may be from the uncooked swill fed to them. On antibiotic sensitivity testing cefotaxime and ceftriaxone were found to be effective against these organisms.

Leukocytoses with neutrophilia were observed in animals of group III where as normal leukogram observed in animals of group II.

Serum biochemical studies revealed an elevated level of creatine kinase, lactate dehydrogenase and aspartate aminotransaminase in animals without any obvious clinical sign of illness, indicating involvement of heart in the pathological condition. Animals with obvious clinical signs of illness revealed an elevated level of lactate dehydragenase, indicating damage of the tissues.

Gross pathology on post mortem revealed lesions in heart, liver, brain, lymph nodes and kidney. Haemorrhages in the heart were the major findings. Prominent

histopathological changes were noticed in heart and brain. Heart showed varying degrees of inflammation, degeneration and necrosis. Stomach, intestine, lung, liver and kidney also showed pathological changes.

For all the animals under study supplementation of mineral mixture containing vitamin E and Selenium were advised. Animals with clinical signs of illness were treated with cefotaxime. It prevented further mortality in these farms.