CLASSICAL SWINE FEVER - AN EMERGING THREAT

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Classical swine fever is a highly infectious viral disease of pigs, which is clinically characterized by a high rise in temperature, diarrhea, purple discolouration of the skin over abdomen and finally death. It may occur as acute, chronic or inapparent infection.

The disease was first reported in Ohio, USA in 1833. In India, first case was reported from West Bengal in 1951. The disease is prevalent in many parts of India; but not reported from Kerala until isolated cases were identified at Wayanad and Kozhikode districts of Kerala recently. Hence, hereafter, veterinarians in Kerala must have in their mind the complete picture of classical swine fever too while differentially diagnosing diseases of pigs.

Etiology

This disease is caused by an enveloped RNA virus in the pesti group of the family Toga viridae. Although there is only one antigenic type, the virus exists as a number of strains of variable virulence and antigenicity. This virus is antigenically related to Bovine Viral Diarrhoea virus.

The virus is killed at 60-70^0 C and by disinfectants like 5% phenol, 2% sodium hydroxide and 1% formalin. Hypochlorite (bleaching powder) containing 1.66% available chlorine will destroy the virus in 15 minutes. The organism can survive at room temperature for about a month and in frozen pork for upto 4 years. The virus can also survive in meat preserved by salting and smoking. The virus persists for 3 to 4 days in decomposing organs and for 15 days in decomposing blood and bone marrow.

Epidemiology

The disease is world wide in distribution. Australia, New Zealand, Canada and United States are free of the disease. It is currently endemic in some of the countries of Western Europe, Central and South America, Asia and Africa. Domestic pigs and wild boars are the animals that are naturally infected. Pigs of all breeds and age are equally susceptible. Outbreaks due to virulent virus cause nearly 100% morbidity and mortality.

Transmission

The source of the virus is always infected pigs or uncooked pig products. Infective pens and pastures aid in transmission of the disease. Infection occurs mainly by ingestion and less commonly by inhalation. Importation of infected pigs and feeding of garbage containing uncooked pork scraps are the main causes of swine fever outbreaks. Direct contact with infected animals, their excretions, semen and blood and indirect contact through visitors, veterinarians, pytroderm, vehicles, implements, clothes etc. aids in rapid spread of the disease. Lung worms may serve as a reservoir of swine fever virus. Pregnant sows transplacentally transmit the disease to its offsprings.

Pathogenesis

The virus enters the host mainly through the digestive tract and occasionally through the respiratory tract. Tonsil is the primary site of virus invasion where the multiplication first occurs. The virus is carried through the lymphatics to the blood capillaries and the initial stage of viraemia sets in. It invades the endothelial cells and erythrocytes and causes hydropic degeneration and proliferation of vascular endothelium. This results in the characteristic lesions of congestion, haemorrhage and infarctions. Such vascular changes are most severe in lymphoid organs, spleen, kidney, lungs and intestine. In many cases secondary bacterial infection occurs; especially with *Salmonella cholerae suis*. Virus will be excreted through faeces, urine and through pulmonary exudates.

Symptoms and clinical findings

Clinical signs usually appear 5 to 10 days after infection. In peracute cases, pigs die without any clinical signs. Acute cases are characterized by high temperature (105 to 107F), conjunctivitis, depressed appearance, unwillingness to take food, drooping of tail, hyperaemia of skin and constipation followed by severe watery yellowish gray diarrheoa and vomiting. In the later stages, a diffused purplish discolouration occurs over the abdominal skin and small areas of necrosis appear on the edges of ears, on tail and lips of vulva. Nervous signs such as circling, in-co-ordination, muscle tremors and convulsions may appear at any stage of the disease. During the terminal stages, sick pigs show a particularly noticeable weaving, staggering gait, which is usually followed by posterior paresis. Leucopenia followed by leukocytosis, anemia and thrombocytopenia occur. Death usually occurs in 5 to 7 days time after the commencement of the illness.

In chronic form of the disease with less virulent strains, incubation period is longer. The characteristic skin lesions in chronic cases include alopecia, dermatitis, blotching of ears and in the final stages purplish discolouration of the skin over the abdomen. Pigs may survive mild infection and there is a phase of apparent recovery, then relapse and death occurs.

Another indirect evidence of swine fever infection is reproductive failure. Abortion, low litter size, mummification, still birth and anomalies of piglets are observed. In-utero infection with strains of the virus of moderate or low virulence can result in what is referred to as the 'carrier sow' syndrome follwed by pre-

natal or early post-natal death, birth of diseased piglets or apparently healthy but infected litter. Affected piglets excrete the virus continuously until death.

Postmortem lesions

In peracute cases, gross changes are limited. In acute cases, submucosal and subserosal petechial haemorrhages occur; usually under the capsule of kidney, at ileocaecal valve, lymph nodes, bladder, epiglottis, larynx and lungs. Similar petechial haemorrhages appear on skin as well. Haemorrhage is the most significant gross lesion in acute infection. Lymph nodes show peripheral haemorrhages, often described as 'mottled' or 'strawberry like'. Small and numerous petechiae on the capsular surface of the kidney gives it a 'turkey egg appearance'. Lymphomegaly and congestion of liver, bone marrow and lungs are seen. Infarctions in the mucosa of gall bladder and spleen is a common finding. Fibrinous exudate is seen on gastric mucosa. Necrotic circular raised 'button ulcers' in the mucosa of colon is a pathagnomonic lesion; especially in chronic cases. Abscess like lesions are observed in lungs and tonsillar tissue.

Histologically, important changes are hydropic degeneration and proliferation of vascular endothelium and non-suppurative encephalitis with or without vascular cuffing.

Aborted foetus show petechial haemorrhages and malformation like microcephaly, hypoplasia, pulmonary hypogenesis and joint deformity.

Diagnosis

Much can be contributed to diagnosis by close observation of the herd. Significant symptoms are high temperature, typical lesions on skin, diarrhea and high morbidity and mortality. On post mortem examination, typical lesions like petechiae on kidneys (turkey egg appearance), petechiae on lungs and epiglottis, peripheral haemorrhage in lymph nodes, haemorrhagic infarctions of spleen and 'button ulcers' in intestine are of diagnostic value. Leucopenia in the early stage of the disease can also aid in diagnosis. However, confirmation of the disease is possible only by laboratory diagnosis. The laboratory tests that can be employed in the diagnosis include Fluorescent Antibody Test (FAT), Virus Neutralization Test, Agar Gel Precipitation Test (AGPT), Complement Fixation Test (CFT), Haemagglutination-Inhibition Test (HI) and ELISA.

At present none of the laboratories in Kerala is equipped with facilities for swine fever antigen/antibody detection. AGID for diagnosis of swine fever is being done at Centre for Animal Disease Research and Diagnosis (CADRAD) under Indian Veterinary Research Institute (IVRI), Izatnagar. Serum samples or tissue samples should be dispatched for detection of antibody or antigen respectively. The samples should be maintained in cold chain until it reaches the laboratory. Detection of antibodies is particularly useful in herds suspected of being infected at least 30 days previously with the virus. Tissue samples to be collected include brain, intestine and other internal organs in 10% formalin for histopathological study and tonsils, lymph nodes especially mesenteric lymph nodes and pancreas without preservatives under cold conditions but not frozen in sealed containers for antigen detection.

Differential diagnosis: The disease should be differentially diagnosed from African Swine Fever, Swine pastuerellosis, Salmonellosis, Acute Erysipelas, *Haemophilus suis* infection, salt poisoning and purpura haemorrhagica.

Treatment

Hyper immune serum is the only available treatment and can be administered in the early stage of the disease and in in-contact animals in doses of 50-150ml.

Prevention and Control

Swine fever can be eradicated by adopting slaughter policy or can be controlled by regular vaccination. For eradication, slaughter must be supported with effective barriers to prevent reintroduction of the virus such as restriction to movement of pigs. If eradication policy cannot be adopted, due to limitations of any kind, control by vaccination must be resorted to.

In swine fever free areas, slaughter policy is the best and all infected and in-contact animals must be slaughtered and carcasses destroyed.

In enzootic areas, the disease can be controlled by immunizing the in-contact or susceptible pigs. Live viral vaccines produce solid immunity but have the potential to produce the disease if 'vaccine breaks' occur. Attenuated tissue culture vaccines give protection within 5 to 10 days of vaccination and the immunity persists for three years.

The general measures to be adopted for prevention and control of the disease are:

1 Proper disposal of the carcass; preferably by burning.
2 Restriction to movement of pigs around the area of outbreak
3 Hyper immune antiserum administration to pigs in the quarantine area
4 Disinfection of pen and premises, utensils, equipments etc with 5 %phenol and boiling of contaminated clothings
5 Proper cooking of garbage before feeding or avoidance of garbage feeding
6 Introduction of new pigs only from farms known to be free from the disease
7 Education to farmers so as to avoid taking ill animals to markets

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3. Thereafter animals should not be dewormed for more than two times per year unless they are found infected with worms.

4. Strategic treatments in adult animals should be restricted to two times per year preferably (a) at the end of the dry season and (b) at the end of the rainy season.

This is to reduce the development of anthelmintic resistance and prolong the usefulness of anthelmintics. It must be remembered that developing new anthelmintic to be used in the food chain is not remunerative and hence pharmaceuticals are not willing to invest huge amounts in to it. So it is our responsibility to conserve the existing anthelmintics for the future.

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