CLASSICAL SWINE FEVER

(also commonly known as **Hog Cholera**, **Swine Fever**, **Swine Plague**)

Swine fever, also known as hog cholera, is an acute, febrile, highly contagious and often fatal disease of pigs, caused by a **Pestivirus** of family **Flaviviridae**. Chronic or inapparent infection also occurs, including persistent congenital infection in newborn pigs, infected during foetal life. The disease in nature is limited to domestic and wild pigs. The virus of swine fever has an antigenic relationship to that causing bovine virus diarrhoea.

SPREAD

The pig is the only domestic animal which is naturally infected by the virus. The infection is usually acquired by ingestion or inhalation. In case of infection with a virulent virus, all excretions, secretions and body tissues of affected pigs contain the virus.

PATHOGENESIS

The tonsil is the primary site of virus invasion following oral exposure. Primary multiplication of the virus occurs in the tonsils within a few hours after infection. The virus is then transported through lymphatics, and enters blood capillaries resulting in an initial viraemia by about 24 hours. At this time, the virus can be found in the spleen and other sites, such as peripheral and visceral lymph nodes, bone marrow, and Peyer's patches. The virus exerts its pathogenic effect on endothelial cells, lymphoreticular cells, macrophages, and epithelial cells.

Most of the lesions are produced by hydropic degeneration and proliferation of vascular endothelium which result in the **occlusion of blood vessels**. This effect on the vascular system results in the characteristic lesions of congestion, **haemorrhage and infarction** from changes in arterioles, venules, and capillaries. **Thrombosis** of small and medium-sized arteries is another feature. **Leukopaenia** is common in the early stages, followed by a **Leukocytosis, Anaemia, and Thrombocytopaenia**. Leukopaenia in the early stages is pronounced, the total count falling from a normal range of 14,000-24,000/ µl to 4,000-9,000/ µl. This can be of value in differentiation from bacterial septicaemias.

SIGNS

Incubation period of 7 days. Depression and **high fever** (106° F) are accompanied by severe **Leukopaenia** in which the total leukocyte count may be less than **4,000/µl**. Besides weakness and inappetence, nervous symptoms are also observed. These include lethargy (lack of energy or dullness), occasional convulsions, grinding of the teeth, and difficulty in locomotion. In pigs with light skin, erythematous lesions appear, on the skin of the abdomen,

axillae and inner surface of the legs. Most animals die within 10 days after the onset of signs. A few that live longer, show intestinal and pulmonary involvement.

Infection with a virus of reduced virulence can result in a longer course, which has been called "chronic" swine fever. Following an apparent remission (lessening of severity), there is an aggravation of the disease, resulting in death as late as 40-70 days after the initial signs of infection. Pigs with chronic disease play an important role in the dissemination of swine fever, because they excrete virus intermittently throughout the entire course of the infection.

Virus crosses the placenta and invades foetal tissues, and can be demonstrated or isolated from piglets. Infection *in utero* can also lead to lesions resembling swine fever in adult pigs, or to the birth of persistently infected pigs which shed the virus. A variety of foetal and neonatal abnormalities have been attributed to exposure to the virus during pregnancy. The most critical period of exposure appears to be **after 20 days of gestation.** Abnormalities include mummification, anasarca, ascites, still-birth, cerebellar hypoplasia, and neonatal death.

LESIONS

The virus of swine fever **exerts a direct effect on the vascular system**, and the signs and lesions result from changes in the capillaries. For this reason, the gross lesions appear as areas of congestion, haemorrhage, or infarction.

In acute cases of longer duration, lesions are found in a variety of organs and are seen in this order of frequency kidney > lymph nodes > urinary bladder > skin (in white-skinned pigs) > spleen > larynx > lungs > and large intestine. With less frequency, lesions occur in the heart, liver, small intestine, and stomach.

In the VASCULAR SYSTEM, the specific action of the virus is manifested by **petechiae and ecchymoses**. The earliest and most pronounced microscopic lesions are found in the capillaries and pre-capillaries. The most constant change is **swelling and proliferation of endothelial cells**. The capillary wall may become completely hyalinized, resulting in partial or complete occlusion. Fat droplets may be seen in the capillary walls.

In the CENTRAL NERVOUS SYSTEM, the lesions are related to the vasculature. Apart from congestion of vessels, gross changes are not seen in the brain. The most striking microscopic lesion is **nonsuppurative meningo-encephalomyelitis** that occurs with accumulation of lymphocytes in the **perivascular (Virchow-Robin) spaces around arteries and veins**. This perivascular cuffing comprises lymphocytes, mononuclear cells, plasma cells, and occasionally, eosinophils. Neutrophils are not a part of the inflammatory exudate. **Small nodules of proliferated microglia are present in both white and grey matter**.

In the SPLEEN, **INFARCTS** are seen that are sharply outlined, red, irregular in shape, and elevated or definitely wedge-shaped. Microscopically, degenerative changes in the wall of

follicular or trabecular arteries are characterized by proliferation of endothelium, hyalinization, and necrosis in the media and adventitia with resultant thrombosis. Haemorrhagic infarction is seen as sharply demarcated areas of necrosis.

In LYMPH NODES, swelling and hyperaemia with bright red subcapsular haemorrhages to dense dark-coloured haemorrhages obscuring the entire nodal architecture are seen.

In SKIN, **Erythematous Areas** (**Red to Violet spots; Purple discoloration**) resulting from cyanosis are the most common gross lesions. They usually appear as areas of violet / purplish discoloration, on the ventral surface of the abdomen and thorax, the medial surface of the thigh and leg, **EARS**, skin of the perineum, and the snout. The cyanotic changes can be readily detected in white-skinned pigs. The typical changes in the vascular system are also responsible for these cutaneous lesions.

In the KIDNEYS, **subcapsular pin-point hemorrhages** - sharply demarcated petechiae, 1 - 5 mm in diameter are visible grossly just beneath the capsule, and deep in the renal cortex. These petechiae give the kidney a characteristic appearance, known as "**Turkey Egg Kidney**". Microscopically, haemorrhages are found in the interstitial stroma and in Bowman's spaces.

The digestive system is usually affected in pigs dying after a more prolonged course. The characteristic lesion is a **spherical ulcer (Button Ulcers) in the mucosa, particularly in the COLON**. These ulcers are sharply circumscribed, single or multiple and develop into encrusted button-shaped foci ("**button ulcers**"). This lesion develops after occlusion of a small artery by swelling and hydropic changes in its endothelium. Thus, the **"button ulcer" is the result of infarction**.

DIAGNOSIS

- On the basis of clinical signs, and gross and microscopic lesions
- Fluorescent Antibody Staining for demonstrating presence of the virus
- The agar gel precipitation test (AGPT) and enzyme-linked immunosorbent assay test (ELISA) is also useful for large-scale testing of herds.

In differential diagnosis, swine fever has to be differentiated from **salmonellosis, acute erysipelas, and acute pasteurellosis**. Salmonellosis is usually accompanied by enteritis and dyspnoea; and in acute erysipelas and acute pasteurellosis subserous haemorrhages are ecchymotic rather than petechial