THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
July, 2023; 3(07), 1289-1295

Short Review

Classical Swine Fever: An Overview

Dr. Preet Shah[1], Dr. Meet Sharma[2] and Dr. Palak Vaidh[2]
[1]M. V. Sc. Scholar, Dept. of Veterinary extension education, College of veterinary science and A.H., Kamdhenu University, Anand.
[2]M. V. Sc. Scholar, Dept. of Animal Nutrition, College of veterinary science and A.H., Kamdhenu University, Anand.
https://doi.org/10.5281/zenodo.8147268

Abstract

Classical swine fever is one of the most significant diseases of domestic pigs and wild boars which has tremendous effects on pig industry. It is caused by Classical Swine Fever Virus of genus Pestivirus of family Togaviridae. It mainly transmits through infected pigs and its products along with inhalation and direct animal to animal contact. Disease is categorised into per acute, acute, and chronic stage according to its stage of occurrence and lesion which can be easily diagnosed by striking necropsy findings and along with latest sero-immunological tests. Being a viral disease, no standard treatment is available, where hyperimmune serum is the only available treatment source. Various control measures including vaccination, hygienic disposals and control of transmission along with the education in the stakeholders and the farmers need to be prioritized to eradicate the disease from the endemic areas.

Introduction

Classical swine fever (CSF) is one of the most significant viral diseases of domestic pigs and wild boar. It has tremendous effect on animal health and pig industry and is therefore notifiable to the World Organization for Animal Health (OIE). **(Edwards *et al*., 2000).** After application of strict control measures, several countries succeeded in eradicating classical swine fever. Nevertheless, in the countries with significant pig production, CSF is at least sporadically present. It is endemically present in several countries of South and Central America, parts of Eastern Europe and neighbouring countries, as well as Asia, including India.

For surveillance and control legal framework exists most countries. Integral parts of the control measures include, timely and prompt diagnosis, stamping out of infected herds, establishment

of restriction zones, movement restrictions, and tracing of possible contacts. vaccination and other treatments are often also strictly prohibited. However, in the areas of Europe, affected wild boar populations were shown to be an important reservoir for the virus, which acted as a source for reintroduction into the domestic pig population **(Fritzemeier *et al.*, 2000) (Rossi *et al.*, 2015)**, emergency vaccination of wild boar has been practiced to control the disease **(Rossi *et al.*, 2010) (Von Ruden *et al.*, 2008) (Kaden *et al.*, 2002) (Blome *et al.* 2011)**. Emergency vaccination is also one of the options to combat CSF in domestic animals. Furthermore, vaccination is still in use to reduce the disease burden in the countries where disease is present endemically.

Classical Swine Fever is an acute, febrile, highly infectious, and fatal disease of pigs characterized by fever, purplish discoloration of abdomen skin, blotching of ear, lacrimal discharged, nervous signs, and reproductive failure in pregnant sows (abortions, mummification, stillbirth, and birth of persistently infected pigs). In chronic disease, persistent congenital infection is occurred in newborn pigs infected during foetal life.

Etiology

Classical swine fever virus (CSFV) is a small, enveloped positive-sense, single stranded, RNA virus in the genus Pestivirus of the family Togaviridae. Most of viruses are non-cytopathogenic in culture, but there are some CSF and BVD that are cytopathogenic. It is antigenically and genetically diverse, with recombination possible between strains. There are four structural proteins (C, Erns, E1, and E2) and eight non-structural proteins. The E2 glycoprotein of CSF is a virulence determinant in swine. There are three major groups, Group-1(Russia and America), Group-2(Asia) and Group-3 (Asia).

Epidemiology

Diseases eradicated in many parts of the world, the countries that's always open to reinfection from illegal imports of fresh products, tourism, hunting, and illegal swill feeding. The pig is the only domestic animal species naturally infected by the CSFV. Wild boars are also affected. Outbreaks of acute hog cholera were quickly controlled by a rigorous policy of slaughter and quarantine. The disease usually occurs in epidemics, often with a morbidity of 100% and a case fatality rate approaching 100%, when a virulent strain of the virus infects a susceptible population.

Modes of transmission

The source of virus is always an infected pig or its products, and the infection is usually acquired by ingestion & inhalation is also a possible portal of entry. Direct animal-to animal contact

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
*Published:*13.07.2023

is the most important method of spread. Blood is a high risk for spreading infection from pig to pig. Infected boars can shed the virus in semen. Severely affected animals could play a prominent role in CSF transmission. The resistance and high infectivity of the virus make spread of the disease by inert materials, especially uncooked meat, a major problem. In the disease-free areas, introduction is usually by the importation of infected pigs or the feeding of garbage containing uncooked pork scraps. *(Figure 1 and 2)*

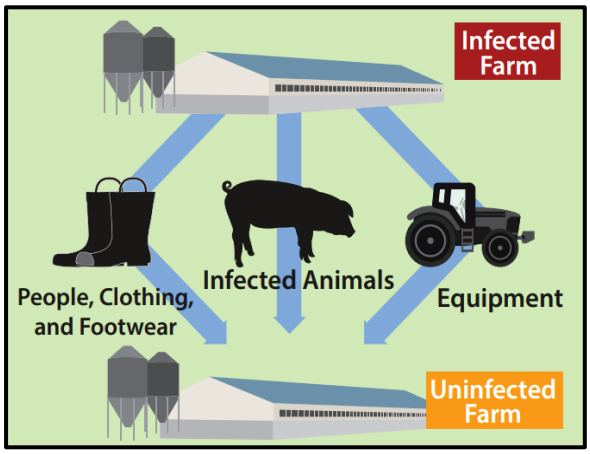

Figure 1: Transmission of virus through fomites (inanimate objects)

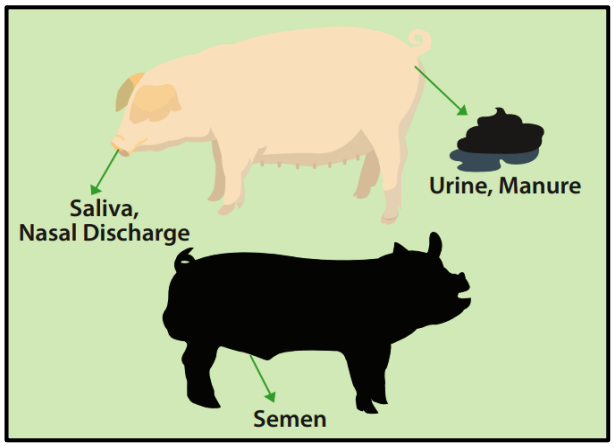

Figure 2: Routes of virus excretion

Photo credit: www.securepork.com

Pathogenesis

After ingestion, tonsil is the primary site of virus invasion. Primary multiplication occurs in tonsils, and it will spread to peripheral lymph nodes. Which eventually results in viremia, then virus moves through lymphatic vessels and enters blood capillaries, spleen, and other sites such as peripheral and visceral lymph nodes, bone marrow, and peyer's patches. The virus predominantly exerts its pathogenetic effect on endothelial cells, lymphoreticular cells and macrophages, and epithelial cells. In particular, B-lymphocytes, T-helper cells, and cytotoxic T cells are affected.

Most of the lesions are produced by hydropic degeneration and proliferation of vascular endothelium, which results in the occlusion of blood vessels. This will produce characteristic lesions of congestion, haemorrhage, and infarction from changes in arterioles, venules, and capillaries. Vascular changes are most severe in the lymph nodes, spleen, kidneys, and gastrointestinal tract. Leukopenia is common in the early stages, followed by leukocytosis in some animals, and anaemia and thrombocytopenia occur. Disseminated intravascular coagulation is common with microthrombi in small vessels, particularly of the kidney, liver, spleen, lymph nodes, lung, intestine, and intestinal

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
*Published:*13.07.2023

lymph nodes. Macrophage activation, and subsequent release of proinflammatory cytokines, plays an important role in the development of the classical signs of CSF. This is particularly true for the pulmonary intravascular macrophages.

Clinical findings

Per-acute and Acute disease

Incubation period ranges up to 35 days. In per acute cases, young pigs die without evidence of clinical signs. In the acute form the disease is characterized by anorexia, lethargy, conjunctivitis, respiratory signs, and constipation. Pyrexia (40.5–41.5° C) major clinical sign (biphasic temperature, 2 and 6th days).

In acute cases, affected pigs are depressed, do not eat, stand in a drooped position with their tails hanging, disinclined to move and, when forced, do so with a swaying movement of the hindquarters, lie down and burrow into the bedding and often piled one on top of the other. Constipation followed by diarrhoea and vomiting also occurs. Later, a diffuse purplish discoloration of the abdominal skin occurs. Small areas of necrosis are sometimes seen on the edges of the ears, tail, and lips of the vulva. A degree of conjunctivitis is usual, and in some pigs the eyelids are stuck together by dried, purulent exudate.

In nervine signs, the incubation period is often shorter. Nervous signs often occur in the early stages of illness and include circling, incoordination, muscle tremor, apparent blindness, stumbling and convulsions. Pigs in lateral recumbence show a tetanic convulsion for 10 to 15 seconds followed by a clonic convulsion of 30 to 40 seconds. The convulsion may be accompanied by loud squealing and may occur constantly or at intervals of several hours, often being followed by a period of terminal coma. Infection with *Salmonella Choleraesuis* may also be potentiated by hog cholera infection, and the two diseases in combination can result in high mortality.

Chronic diseases

Incubation period is longer than normal, and there is depression, anorexia and persistent mild fever. Appearance of characteristic skin lesions, including alopecia, dermatitis, blotching of the ears, and a terminal, deep-purple coloration of the abdominal skin. In the reproductive failure significant feature of disease infection of the sow can occur at any stage of pregnancy and may result in no clinical signs other than a mild pyrexia, but it may be followed by a high incidence of abortion, low litter size, and mummification, stillbirth, and anomalies of piglets. Persistent congenital infection is characterized by persistent viremia, continuous virus excretion, and late onset of disease, with death

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
*Published:*13.07.2023

occurring 2 to 11 months after birth. In experimentally, a high incidence of myoclonic congenital (congenital trembles) associated with cerebellar hypoplasia has been observed in prenatal infection with hog cholera virus outbreaks.

Clinical pathology

Haematology

A valuable ante mortem diagnostic test is the total and differential leukocyte count. In the early stages of the disease there is marked leukopenia, with the total count falling from a normal range of 14,000 to 24,000 cells/µl to 4000 to 9000 cells/µl. This is specifically granulocytopenia caused by bone-marrow atrophy Leukocyte count gives the earliest pointer to CSF infection but of course does not confirm the disease. Apoptosis or necrosis occurred, result of cytokine interaction.

Necropsy findings

In per-acute cases, there may be no gross changes at necropsy.

In acute form (more common), there are many submucosal and subserosal haemorrhages. The haemorrhage results from erythrodiapedesis and increased vascular permeability, probably aided by mast cell degranulation. The haemorrhages are usually petechial and rarely ecchymotic. The petechial haemorrhages are most noticeable under the capsule of the kidney (turkey egg kidney) *(Figure 3)*, near the ileocecal valve, in the cortical sinuses of the lymph nodes, and in the bladder and larynx. The lymph nodes are enlarged, and the spleen may contain marginal infarcts. Infarction in the mucosa of the gallbladder is a common but not constant finding and appears to be an almost pathognomonic lesion. Circular, raised button ulcers in the colonic mucosa *(Figure 4)* are usual but cannot be distinguished from those of salmonellosis. A recent study found that the lymph nodes had the highest score for lesions and that the fewest lesions were found in the spleen and tonsil because infection of these organs was also rare. The most common lesions were also in the lymph nodes, around the ileocecocolic junction, and around the blood vessels of the brain.

In the chronic form of the disease, ulceration of the mucosa of the large intestine is usual. Secondary pneumonia and enteritis commonly accompany the primary lesions of hog cholera. Malformations such as microcephaly, cerebellar hypoplasia, pulmonary hypogenesis, and joint deformity appear as a result of inhibition of cell division and function in these areas. In pigs showing signs of myoclonic congenita, cerebellar hypoplasia is highly suggestive of hog cholera infection. Immune complex glomerulonephritis also evident.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
*Published:*13.07.2023

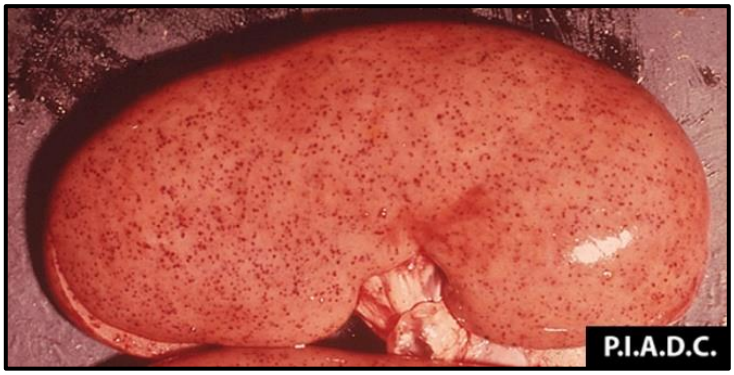

Figure 3: Turkey Egg Kidney

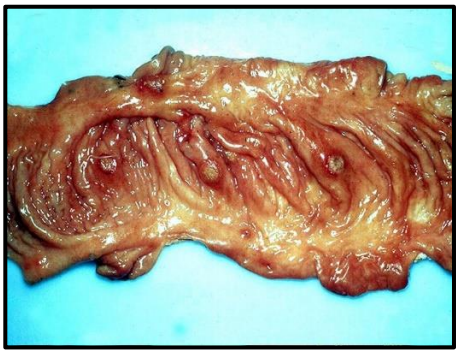

Figure 4: Button Shaped Ulcers

Photo credits: Plum Island Animal Disease Center, Orient Point, New York

Diagnosis

Diagnosis is made based on the history, clinical signs, clinical pathology, necropsy finding and virus detection. The most characteristic lesions were found in the lymph nodes, petechial haemorrhages on capsule of the kidney, followed by necrotic lesions in the ileum and hyperaemia of the brain. Splenic infarction and necrotic tonsils. The qRT-PCR can also be used to differentiate between virus species BD, BVD and CSF. It can also be used to differentiate infected from vaccinated animals (DIVA).

Treatment

Hyperimmune serum is the only available treatment and may be of value in the very early stages of the illness if given in doses of 50 to 150 ml and can be repeated after 24 hrs. It has more general use in the protection of in-contact animals.

There is also the future possibility of imidazolepyridines, which have a potent in vitro activity against CSFV, being used for treatment. The reduction of CSFV transmission to untreated pigs has been shown by the pestivirus inhibitor BPIP.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
*Published:*13.07.2023

Control

Control method for CSF include eradication and control by vaccination. For removal of the source of infection necessitates, isolation of infected animals, suitable hygienic precautions to prevent the spread of infections on boots, clothing, and utensils, disposal of carcasses by burning and disinfection of pens.

The choice of serum or vaccine may depend on local legislation and will depend on circumstances. Pigs in the affected pen should receive serum (20 to 75 mL, depending on size), and pigs in unaffected pens should be vaccinated.

Vaccine is comprising of two sorts: The first group is the classical live group (C-strain) containing attenuated virus, and recently developed second group of live vaccines aimed as marker vaccines based on the E2 protein. Vaccination: primary; 2 weeks of age, booster; 8-10 weeks and 6-7 months of age (route; I/M)

References

Blome, S.; Gabriel, C.; Staubach, C.; Leifer, I.; Strebelow, G.; Beer, M. (2011) Genetic differentiation of infected from vaccinated animals after implementation of an emergency vaccination strategy against classical swine fever in wild boar. *Vet. Microbiol.*, *153*, 373–376

Blood, D.C., Radostits, O. M., and Handerson J. A., Veternary Medicine: A textbook of the diseases of cattle, Sheep, Pigs and Horses, Bailliere Tindall, Paris.

Chakrabarti A. (1988) Textbook of preventive veterinary medicine, Kalyani publishers, New Delhi.

Edwards, S., Fukusho, A., Lefevre, P.C., Lipowski, A., Pejsak, Z., Roehe, P., Westergaard, J. (2000) Classical swine fever: The global situation. *Vet. Microbiol. 73*, 103–119.

Fritzemeier, J., Teuffert, J., Greiser-Wilke, I., Staubach, C., Schlüter, H., Moennig, V. (2000) Epidemiology of classical swine fever in germany in the 1990s. *Vet. Microbiol. 77*, 29–41.

Melinda D. Merck (2019) The merck veterinary manual, 11th edition, Wiley-blackwell publisher, United Kingdom.

Kaden, V.; Heyne, H.; Kiupel, H.; Letz, W.; Kern, B.; Lemmer, U.; Gossger, K.; Rothe, A.; Böhme, H.; Tyrpe, P. (2002) Oral immunisation of wild boar against classical swine fever: Concluding analysis of the recent field trials in Germany. *Berl. Munch. Tierarztl. Wochenschr.*, *115*, 179–185.

Rossi, S.; Pol, F.; Forot, B.; Masse-Provin, N.; Rigaux, S.; Bronner, A.; Le Potier, M.F. (2010) Preventive vaccination contributes to control classical swine fever in wild boar (*Sus scrofa* sp.). *Vet. Microbiol. 142*, 99–107.

Rossi, S.; Staubach, C.; Blome, S.; Guberti, V.; Thulke, H.H.; Vos, A.; Koenen, F.; Le Potier, M.F. (2015) Controlling of csfv in european wild boar using oral vaccination: A review. *Front. Microbiol. 6*, 1141.

Von Rüden, S.; Staubach, C.; Kaden, V.; Hess, R.G.; Blicke, J.; Kühne, S.; Sonnenburg, J.; Fröhlich, A.; Teuffert, J.; Moennig, V. (2008) Retrospective analysis of the oral immunisation of wild boar populations against classical swine fever virus (csfv) in region Eifel of Rhineland-Palatinate. *Vet. Microbiol.*, *132*, 29–38.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com

***Published:*13.07.2023**