

Indian Farmer Volume 9, Issue 01, 2022, Pp. 43-47. Available online at: www.indianfarmer.net ISSN: 2394-1227 (Online)

ORIGINAL PAPER

African swine fever - a threat to indian pig industry

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Article Received: 24 January 2022

Published Date: 27 January 2022

A frican Swine Fever is caused by African Swine Fever Virus (ASFV) belonging to the genus *Asfivirus* of the family *Asfarviridae*. The disease was first reported in 1921 when European pigs were introduced in Kenya. Since then, this disease has shown its global presence including Europe, Russia, China, India and other Asian countries. India has a total population of 9.06 million pigs (20th livestock census, 2018) and North Eastern states account for 45% of the total pig population. Recently a devastating outbreak nationally and worldwide has given this disease a greater importance in the current situation. A total of 160 new outbreaks worldwide from August-September of 2020 and 11 major outbreaks in May 2020 in the Indian states of Assam and Arunachal Pradesh has caused a devastating effect on the economy of swine industry and pig farmers (Patil*et.al.* 2020) So, clear insights in its etiology, epidemiology, distribution, life cycle, host range, pathogenesis, diagnosis, prevention and control should be understood clearly to avoid further outbreak and its aftermath economic consequences.

African Swine Fever Virus structure and etiology

The etiological agent responsible for African Swine Fever is African Swine Fever Virus belonging to the genus *Asfivirus* of the family *Asfarviridae* (OIE 2020). It is an enveloped double stranded DNA virus with a linear genome of 170-190kbp. The ASFV has a

central DNA, thick protein layer (Core), inner lipoprotein envelope, and capsid. The variability of the genome is determined by genes encoding the variable regions. It has an icosahedral capsid symmetry with a diameter of 200 nm. There are 24 distinct genotypes classified based on p72 protein and only one serotype of ASFV identified by antibody test.

Epidemiology

Globally, there are three different cycles such as sylvatic, tick-pig and domestic cycle responsible for epidemiological and transmission dynamics of ASFV. Soft *Ornithodoros spp.* of ticks, warthogs (wild african pigs), domestic pigs and pig derived products are the main players in its transmission. In the sylvatic cycle, virus is circulated and maintained between soft ticks and warthogs. In the tick pig cycle, the soft tick is a biological reservoir that transmits to domestic pigs. In the domestic cycle, the infection is transmitted between domestic herds. Apart from these cycles, a fourth cycle called the wild boar-habitat cycle is also reported in some European countries (Gallardo *et.al.* 2019)

Wild boar-habitat cycle - This cycle has both direct transmission between infected and susceptible wild boars and indirect transmission by its carcasses in its habitat. The carcasses can be infectious if these carcasses are scavenged by other domestic/wild pigs which becomes a loci of transmission. Thus, careful monitoring is essentially needed in junctional interface between wild and domestic pigs which can be a critical area for its transmission.

Host range and Transmission

ASFV infects members of the swine family which includes domestic and wild boar, feral pigs, bush pigs, warthogs, forest hogs, and Ornithodoros ticks. However, all wild pigs: Warthogs (*Phacochoerusaethiopicus*), bushpigs (*Potamochoerusporcus*), and ticks of Ornithodoros genus act as reservoir hosts, maintaining the virus thereby transmitting to the domestic pigs in Africa. The most important cause for the transmission is the unrestricted movement of men and materials including pigs in the porous border of the Indian sub-continent. Other factors include the thick and densely populated pig farm, stys, improper disposal of carcass, sale of sick pig, feeding of swill, unrestricted movement of men and pigs between farms are some of the reasons.

Clinical signs & Gross pathology

The clinical presentation of the disease can be either peracute, acute, sub-acute and chronic which is associated with the virulence of the virus. The incubation period of the virus varies from 4-19 days. In peracute form, it causes death with 4 days of infection without gross lesion. Infected animals show pyrexia (upto 42C), anorexia, lethargy and sudden death.

In acute form, it is characterized by high fever with 100% mortality by 4-9days post infection. The infected animal shows hemorrhagic spots on the ears, abdomen, generalized

reddening of the skin, blood from nose/mouth. In dead animals, pulmonary edema, hyperemic splenomegaly, intensive necrosis of lymphoid tissue. Multifocal hemorrhagic lymphadenitis is the most pathognomonic lesion in the acute form.

In sub-acute form, it is generally produced by moderately virulent strain with mild clinical signs than acute form. Illness may last up to 30-45days (Kouam*et.al.* 2020). Mortality may vary between 30-70% and pigs may die 20 days after infection. Death may occur due to intense thrombocytopenia or leukopenia. In the chronic form, it is produced by low virulent strains with low mortality and no vascular lesions. Weight loss, growth retardation, joint swelling and mild respiratory signs will occur occasionally.

Pathogenesis

It is a disease causing severe immunodeficiency in affected pigs. Tonsils and regional lymph nodes are the primary organs after entry of virus through the oro-nasal route (Salguero*et.al.* 2005). Virus spreads to secondary organs through blood and lymph within 2-3 days post infection and mainly affects the monocytes and macrophages causing apoptosis and necrosis. In acute form, lymphoid organs like spleen, lymph node, thymus are destroyed along with the majority of B cells, T cells. Additionally a marked edema, ascites, hydropericardium can be observed in sub-acute conditions.

Diagnosis

Blood, serum, and tissues such as spleen, lymph nodes, bone marrow, lung, tonsil and kidney can be the sample of choice for the detection of the agent and immune response by serological test

Detection of the agent

- 1. Virus isolation Primary leukocyte culture and porcine bone marrow are commonly used
- 2. HAD (Hemadsorption test) It is the gold standard for the identification of ASFV in the primary outbreak areas. The 8DR protein of ASFV is responsible for HAD property wherein viruses in macrophages bind to pig erythrocytes causing rosette. (Gallardo *et.al.* 2019)
- 3. FAT (Fluorescent Antibody Test) It serves as a presumptive diagnostic test along with the clinical signs and typical lesion of ASFV.
- 4. Conventional and Real Time PCR OIE recommends to follow quantitative real time PCR using Real time primers and probes.

Serological Test

1. Indirect ELISA - Suitable for testing serum and plasma for the detection of antibodies against ASFV infection.

- 2. IPT (Immunoperoxidase Test) and IFAT (indirect FAT) OIE recommended confirmatory test for the sera from areas that are free of ASFV.
- 3. Immunoblotting can be used as an alternative to IPT & IFAT. It has the advantage of detecting weak positive samples for ASFV antibodies. (Pastor *et.al.* 1989)

PREVENTION AND CONTROL

Antiviral drugs

Sodium Phenylbutyrate has shown inhibition of ASFV replication and viral protein synthesis *in vitro* (Frouco*et.al.* 2017). Resveratrol and oxyresveratrol have shown an antiviral effect on ASFV in cell culture. All these drugs are results from *in vitro* which should be validated in host animals.

Vaccines

At present, there are no vaccines available commercially. But a DNA vaccine containing the ASFV genome devoid of CD2v, p54 and p30 was tried and found to have limited protection in the population. But vaccine development for ASFV is difficult because of its complex nature of the virus and viral proteins. The most important obstacle in developing a vaccine is the non-availability of a permanent cell line which can sustain its multiplicity and production on a large scale.

Biosecurity measures

Biosafety at farm level is to be practiced. Persons/laborers handling the infected pigs should take all bio- safety precautions such as wearing protective equipment such as aprons, spectacles, gloves, and gumboots. Isolation, movement restriction and sanitation has controlled ASFV (Kouam*et.al.* 2020). Rapid culling of all infected and in-contact pigs and proper dis- posal of cadavers, litter, and waste food is essential. All these things should be buried deeply in the vicinity over layered with lime and salt, not to transport to distant places to avoid spillage. These are some of the biosecurity measures one can adopt for the successful prevention and control of ASFV.

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