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African swine fever: An update

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Abstract
One of the infectious diseases of livestock that has recently caused outbreaks in various regions of India is African Swine Fever (ASF). It is not only responsible for tremendous loss in the pig population and drastic economic consequences but has become a menace to food security worldwide. ASF is a haemorrhagic infectious viral disease of swine caused by the African swine fever virus, a DNA virus that gets transmitted through direct contact and infected soft ticks. As there is no vaccine available, widespread awareness is necessary for effective control of the disease. In this review, updated details about ASFV, epidemiology, pathogenesis and clinical forms of the disease, diagnostic approaches and prevention and control measures have been summarised.

Keywords: African swine fever, pigs, ASFV, *Asfarviridae*, *Ornithodoros* ticks

1. Introduction
In India, a large number of small pig holders are dwelling in rural as well as urban areas where pork is heavily consumed by people, mostly in the North Eastern region of India. According to the 20th Livestock Census, there are 09.06 million pigs in India accounting for 01.70% of the total livestock population of the country. Out of this, 46.85% of the pig population is confined to the north-eastern states of the country. Although commercial piggery is practised in some states, such as Assam, Arunachal Pradesh, Meghalaya, Uttar Pradesh, Kerala and Madhya Pradesh, it is the major source of income and nutrition for the local tribal population allover the country (Rajukumar *et al*., 2021) [24].

Infectious diseases tend to be the major cause of animal morbidity and mortality in India, resulting in a significant decrease in livestock production and increase in overall financial expenses. One such infectious disease which has recently led to an outbreak in the country is African Swine Fever (ASF). It is a highly transmissible viral disease that of both domestic and wild pigs that results in acute haemorrhages, high fever and significant mortality. In India, this disease has affected around 45% of the total pig population in recent years. Even though, it has limited host range and no zoonotic potential yet reported, its social and economic impact is very high. Hence, ASF is a notifiable disease to the World Organisation for Animal Health (Blome *et al*., 2020) [6].

The disease was first reported in pigs in Africa in the 1920s, and, therefore, it was named African swine fever. Gradually, it spread to countries like Georgia, Russia, and other European countries. On the Asian continent, China was the first to report an ASF outbreak, with more than 50% of the swine population affected. In India, the first case was reported in May 2020 from the north-eastern states, mainly Arunachal Pradesh and Assam, causing economic losses of around 60 crores through restrictions on the export of pork (Akand *et al*., 2020) [2]. Recently, an outbreak of the disease has been confirmed in Uttrakhand, Tripura, Uttar Pradesh, and Madhya Pradesh by the National Institute of High Security Animal Diseases (NIHSAD), Bhopal.

ASF is caused by the African swine fever virus (ASFV), which is a DNA virus belonging to the *Asfarviridae* family, under the genus *Asfivirus* (Alejo *et al*., 2018) [3]. Different strains of ASFV have been documented so far, which vary in their ability to cause disease. However, only one serotype of the virus has been recognised at present. Generally, the ASFV is transmitted through contact with infected animals (directly or via contaminated fomites) and by certain soft ticks (Zhang *et al*., 2022) [36].

Clinically, the disease manifests in a wide spectrum, ranging from per-acute to subclinical forms, depending on the pathogenic potential of the virus, the viral strain, and the features of the animal infected.

Both domestic and wild pigs show similar signs, including high fever, loss of appetite, leukopenia, haemorrhages in the skin, laboured breathing, swollen red eyes with type of discharge, vomiting, diarrhoea, and abortion in pregnant sows. Death may be seen within 4–15 days of the onset of symptoms in acute and sometimes in subacute cases (Blome *et al*., 2013) [7]. The leukopenia observed is due to massive destruction of the lymphoid cells, imparting a general state of immunodeficiency to the diseased animals. Typical lesions of ASF include haemorrhagic or hyperaemic splenomegaly along with pulmonary oedema (Salguero *et al*., 2005) [28].

Since, clinicopathological conditions of ASF are very much similar to Classical Swine Fever (CSF), which is caused by a different virus, it is difficult to differentiate the two conditions and laboratory testing should be done for the same (Salguero, 2020) [26]. Presumptive diagnosis of ASF can be made on the basis of mortality pattern, clinical signs and post-mortem examination of pigs vaccinated against CSF. However, for confirmatory diagnosis of ASF, diagnostic procedures such as isolation of the virus, detection of viral antigen by direct fluorescent antibody test (FAT) and haemadsorption, and detection of viral DNA by polymerase chain reaction (PCR) should be used (Aguero *et al*., 2003) [1]. Definitive treatment and effective vaccine are not available for ASF till date and therefore, prevention has a pivotal role in the control strategy of the disease (Bellini *et al*., 2016) [4].

2. Aetiology

ASFV is an enveloped virus with double-stranded DNA as genome, formerly included in the family *Iridoviridae*. The ASF virus is the only member of the *Asfivirus* genus within the *Asfarviridae* family and, currently, the only documented DNA arbovirus. The ASF virus has a large and complex structure with an overall diameter of 175–215 nm. The outermost covering of the virus is made up of a lipid-containing envelope that surrounds the complex icosahedral capsid. The viral capsid contains 1892–2172 capsomers and p72 capsid protein. Beneath the capsid, the virion has a nucleoprotein core of 70–100 nm in diameter, surrounded by an internal lipid layer. ASFV consists of around 50 proteins, involving various structural proteins (pp220, pp62, p72, p54, p30, and CD2v) and several enzymes required for transcription and post-translational modification of mRNA (Wang *et al*., 2019) [33].

The genome of ASFV consists of a single molecule of linear double-stranded DNA that varies in length between approximately 170 and 193 kbp, mainly due to the loss or gain of multigene family members. The genomic organization of ASFV (Fig. 1) consists of internal terminal repeats (ITRs) at both the ends followed by 38-47 kbp long variable region (VR) with multigene families and around 125 kbp long central conserved region (CCR). It comprises of all the essential genes required for cytoplasmic replication in the target host cell. Following replication, the virus is released either by budding through the plasma membrane or following cellular disintegration. The mutation rate in the DNA of ASFV is very low (Zhao *et al*., 2019) [37]. This is ensured by the accurate proof-reading mechanism of DNA polymerase and the virus-encoded base-excision DNA repair system. Additionally, due to the lack of related viruses, recombination with other viruses is very unlikely. Thus, the probability of ASFV crossing the species barrier is negligible (Brown and Bevins, 2018) [8].

Different genotypes of ASF virus have been recognized by genomic sequencing techniques. Based on the presence of restriction endonuclease in viral DNA and major capsid protein gene sequencing, 5 and 23 genotypes have been described, respectively. For instance, the ASF virus endemic in Sardinia, Italy belongs to genotype I whereas the ASF infection observed in 2007 was caused by genotype II ASF virus (Salas and Andres, 2013) [25]. Depending on the presence of genes associated with host immune suppression and apoptosis, ASFV isolates exhibit varying degrees of virulence. The standardized nomenclature of genotypic isolates includes name of the city or country where it was isolated, followed by last two digits of the year of isolation, such as DR ’78, Lisbon ’60, *etc.* (OIE, 2019) [35].

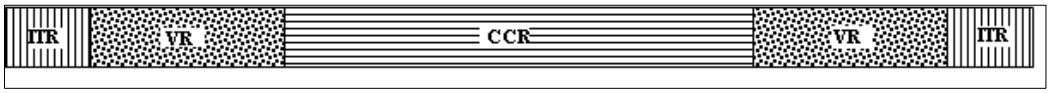

Fig 1: Genomic organization of African swine fever virus.

ASFV is stable in the environment, even at low temperatures and its heat inactivation can be done at 56 °C for 70 minutes or 60 °C for 20 minutes. It is susceptible to ether and chloroform and is inactivated by 8/1000 sodium hydroxide (30 minutes), hypochlorites-2.3% chlorine (3 minutes), 3/1000 formalin (30 minutes), 3% orthophenylphenol (30 minutes) and iodine compounds. As the virus is highly stable in a proteinaceous environment, it remains viable in blood, faeces and tissues for longer periods, especially in uncooked or under-cooked pork products (Brown and Bevins, 2018) [8].

3. Epidemology

3.1 Geographical Distribution

The epidemiology of ASF is complicated, with diverse patterns of infection in Africa, Europe, and Asia. This depends on the environment, pig husbandry, the presence/absence of relevant tick vectors, the presence/absence of wild pigs and human behaviours. The disease was reported in Kenya, East Africa for the first time (1920) (Lubisi *et al*., 2005) [18]. The disease remained confined to the African subcontinent until the middle of the 20th century when it spread to Europe and later to South America and the Caribbean nations. Gradually, the disease was reported in countries like Georgia, Russia and other European countries. In Asia, ASF disease was primarily reported in the North-eastern region of China in 2018 (Zhang *et al*., 2022) [36].

In India, the first outbreak of ASF was reported in North-eastern region, specifically, Arunachal Pradesh. Within one month of this outbreak, seven more outbreaks have been reported in the North-eastern states of India between February 24, 2020, and April 10, 2020. It was revealed that ASF had caused death of pigs exceeding 2.5 thousand in numbers in about 306 villages of Assam belonging to Biswanath, Dhemaji, Dibrugarh, Jorhat, Lakhimpur, and Sivasagar districts. It was observed that the spread of the disease to different parts was much faster in India than in other countries. This may be due to the extensive sale and/or

purchase of pigs at lower prices by alarmed and unaware farmers. Further, it was concluded that all 11 outbreaks occurred in the form of one cluster within 3 months period (Subedi *et al*., 2021) [32].

3.2 Host Species Affected

All subspecies and strains of domestic and wild (*Sus scrofaferous*) pigs are susceptible to the pathogenic effects of ASFV. However, some wild pig species such as warthogs (*Phacochoerus aethiopicus*), bush pigs (*Potamochoerus porcus*) and giant forest hogs (*Hylochoerus meinertzhageni*) are asympotamically affected and thus, act as reservoir hosts. The ASFV is naturally present in *Ornithodoros* ticks, under family *Argasidae* and thus, they act as both reservoirs as well as biological vectors. It has been documented that adult warthogs donot develop clinical forms of the disease, however, the ASFV can be isolated from their lymph nodes. In rare occasions, adult warthog may also develop viraemia. Contrastingly, viraemia along with clinical disease is observed in young warthogs which act as an important source of ASF virus for the ticks (Jori *et al*., 2013) [17].

3.3 Transmission

There are two common modes of transmission of ASF virus to the susceptible host, namely direct and indirect contact. Direct transmission occurs when a healthy susceptible host come in direct contact with the infected animal itself, or its secretions, bedding materials, etc. Indirect transmission of infection occurs either by contact with contaminated fomites or via biological vectors (soft ticks). Rarely, mechanical transmission through biting flies such as stable fly (*Stomoxys calcitrans*) can also be observed (Gallardo *et al*., 2015) [13].

Common sources of infection include infected pigs, contaminated feed, unprocessed infected pig meat or meat products, biting flies, and ticks. Other sources of infection include consumption and contact with contaminated bedding, feed, equipment, clothes, footwear, and vehicles of infected pigs. The virus can be transmitted to a very long distance through ASF-infected meat products, including both frozen and salted products (Wilkinson *et al*., 1977) [34].

Domestic pigs generally become infected by direct contact with infected pigs or by ingestion of contaminated feed. Even though wild warthogs act as reservoir of the virus, they are unable to transmit infection to domestic pigs directly. Spread from this reservoir is via the soft tick of the genus *Ornithodoros*. However, some bush pigs have been able to spread the virus to domestic pigs only under experimental conditions (Costard *et al*., 2009 and Jori and Bastos, 2009) [11, 16].

Ticks ingest the virus while taking a blood meal and then pass it on to the susceptible animals following subsequent feeding or blood meal. Additionally, transstadial (transmission between stages), transovarian (transmission from one generation to other via infected ova) and sexual transmissions in *Ornithodoro*s ticks have been documented. In some regions of Africa, a cycle of ASFV transmission can be seen between young warthogs and the soft ticks, which are inhabitants of their burrows. Surprisingly, no infected soft tick has been recorded in the north-eastern region of India (Dixon *et al*., 2020) [12]. Guinat *et al*. (2016) [15] reported three transmission cycles in endemic areas, namely

1. Domestic pig-to-domestic pig cycle, which does not involve other vertebrate or invertebrate hosts
2. A domestic pig-to-tick-to-wild pig cycle (Sylvatic cycle)
3. A domestic pig-to-tick cycle, without the involvement of wild pigs

Several studies delineate that airborne transmission of the ASF virus can also be a possible route over short distances, not exceeding 2 meters (Mellor *et al*., 1987) [19]. The viral transmission through water sources is unlikely because in water, the concentration/titer of the virus instantly gets diluted to less than the concentration required to produce infection in susceptible animals. Transplacental transmission of ASFV from infected sows to fetuses during pregnancy is not sufficiently validated. Furthermore, sexual transmission of the virus in pigs has not yet been reported, but its presence in genital secretions has been observed. Therefore, it is always advised to examine the semen samples for before using then for artificial insemination (Penrith *et al*., 2004) [23].

4. Pathogenesis

The ASF virus enters the bodies of domestic pigs either via contact, ingestion, or an infected tick bite. After entering via oral route, the virus replicates initially in the pharyngeal mucous membrane and tonsils, followed by spread to the draining lymph nodes. Infection then extends via the bloodstream to the target organs, including spleen, lung, liver, kidney, bone marrow, and other lymph nodes of the body, within 2-3 days post-infection. The virus replicates in the cytoplasm of monocytes and macrophages in these organs, and thus, they are the main sites of secondary replication, which gives rise to prolonged viraemia. The cellular receptors and virus ligands required for receptor-mediated endocytosis of the virus still remain elusive. The virions are released by budding and can be observed free in the blood, lymph, and interstitial spaces (Patil *et al*., 2020) [21].

Apoptosis induced by the ASFV results in the destruction of monocytes and macrophages. In acute form, a major population of lymphocytes and macrophages is destroyed, resulting in marked leukopenia and immunosuppression (Carrasco *et al*., 1996) [9]. Thrombocytopenia is observed in the final stage of acute infection and the initial stage of subacute infection. This thrombocytopenia is described by the following mechanisms: (1) reduced production of thrombocytes; (2) ASFV-induced apoptosis/necrosis of megakaryocytes; and (3) exhaustion of thrombocytes due to disseminated intravascular coagulation (Sanchez-Corden, 2021) [29].

The incubation period variable depending upon the clinical forms of disease, but is typically 5 to 7 days in acute cases. Lesions produced in the acute case of the disease are mainly related to widespread haemorrhages in multiple organs. The development of disseminated intravascular coagulation and the degeneration of megakaryocytes are considered to be major factors in the pathogenesis of the haemorrhages. The mechanisms responsible for recovery from infection and immune response generation are not well understood. Cell-mediated immunity has been proposed to play a significant role in the defense mechanism. It is currently not possible to demonstrate the presence of antibodies which can fully neutralize the virus in the serum of recovered animals (Patil *et al*., 2020) [21].

5. Clinical Signs

Clinical disease can be manifested in several forms, ranging from death with no signs (peracute) to acute, subacute, and chronic forms. Sometimes, an asymptomatic infection can

also be observed. However, most isolates of ASFV cause acute hemorrhagic fever in domestic pigs and result in mortality around 100%. The duration of the disease usually depends on the virulence of the respective virus/ viral isotope, however, more than one form can also be caused by a particular virus (CFSPH, 2019) [10]. Various forms of the disease have been described below.

5.1 Peracute Form
In the peracute type of ASF, pigs die within 4 days without showing any signs. The rate of mortality is 100% in this form of disease. Sudden deaths may be the first indication of infection in some herds. However, very small doses of the virus can also lead to infection in runted animals causing some clinical signs, such as fever, before death. Clinical signs in wild boar experimentally inoculated with a highly virulent isolate of the virus are in correspondence with the clinical signs observed in infected naturally domesticated pigs. Warthogs and bush pigs generally carry asymptomatic infection and sometimes, may show mild symptoms (Salguero, 2020) [26].

5.2 Acute Form
The characterized signs observed in acute form of the disease include high fever (>41 °C), anorexia, lethargy, weakness, and recumbency. Erythema can be seen in white or light-skinned pigs, which develop cyanotic discoloration of the skin particularly on the ears, lower legs, tail, and ventral body surfaces. Additionally, diarrhea, vomition, constipation, and abdominal pain are observed in the affected pigs. Initially, mucoid diarrhea is seen in affected animals which turns bloody in later stages. Respiratory signs including dyspnea, epistaxis, nasal and conjunctival discharges, along with neurological signs such as ataxia (24 - 48 hours before death), have also been reported in affected pigs. Abortion is frequently observed in pregnant sows. Leukopenia and thrombocytopenia of varying severity may be detected in haematological examination. Death of the affected animals is reported within 7-10 days post infection (CFSPH, 2019 and Blome *et al*., 2020) [10, 6].

5.3 Subacute Form
The subacute form of ASF is similar to the acute form but with milder clinical signs. Fever, leukopenia and thrombocytopenia may be transient. However, hemorrhages are observed when the number of thrombocytes get reduced. Abortion is commonly the earliest sign in case of an outbreak caused in subacute form. Affected pigs usually die but can recover within 3 to 4 weeks post infection. The mortality rate in the subacute type of ASF ranges from 30% to 70% (Beltran-Alcrudo *et al*., 2017 and OIE, 2019) [5, 35].

5.4 Chronic Form
Animals affected with the chronic form of ASF show nonspecific clinical signs such as an intermittent/transient fever of low degrees, depression, reduced feed intake and emaciation. However, some affected animals may develop respiratory distress and coughing with swelling in joints. Diarrhoea have also been observed in some pigs along with occasional vomiting. Small and red elevated areas may appear on the skin followed by necrosis and ulceration specially at the sites of physical trauma. Chronic African Swine Fever can also be fatal after prolonged emaciated condition (Beltran-Alcrudo *et al*., 2017) [5].

6. Lesions
6.1 Gross Lesions
The gross lesions in ASF affected pigs are highly variable depending upon viral virulence and form of the disease. In acute and subacute forms of ASF, multiple organs get affected at variable degrees. The carcasses of those animals which die in an acute condition are usually in good condition. There may be bluish-purple discoloration *i.e.* hemorrhages in the skin, signs of bloody diarrhea and other internal hemorrhages in acute cases.

Typical gross findings observed at the post-mortem examination are haemorrhagic lesions in multiple organs such as spleen, kidney and heart including diffuse hemorrhages in superficial inguinal, mandibular, pulmonary, gastro-hepatic, renal and mesenteric lymph nodes (Salguero *et al.*, 2002) [27].

Animals infected with highly virulent isolates show hyperaemic splenomegaly with friable consistency and dark red to black discoloration. In other cases, splenomegaly may be observed with minor changes in colour and no friability. Petechial haemorrhages in the renal cortex, diffuse haemorrhage in the renal pelvis and perirenal oedema are traditionally reported in ASF. Pin point to extensive hemorrhages can also be detected in other organs of the body such as urinary bladder, lungs, stomach and intestines (Mozos *et al*., 2003) [20].

Congestion and oedema in the lungs can be predominant in some pigs. Congestion of the liver along with oedematous bile duct and wall of the gall bladder may also be observed. Blood stained or straw-coloured fluid may be present in the peritoneal, pleural, and/or pericardial cavities, along with congestion, oedema and haemorrhages in the brain and meninges. Other lesions include focal necrosis and ulceration of skin, swollen joints, caseous infiltrate in lungs with consolidation, fibrinous pericarditis, adhesions in pleura and generalized lymphadenopathy which have been reported in subacute and chronic cases. Furthermore, aborted fetuses show generalized swelling (anasarca), multiple hemorrhages in the skin and a mottled appearance in the liver (Salguero, 2020) [26].

6.2 Microscopic Lesions
The microscopic lesions observed in various organs of the affected animals are more or less associated with the degree of vascular alterations and form of the disease. Mild hyperaemia and slight deposition of karyorrhectic macrophages in and around inflamed blood vessels (vasculitis) are seen in dermis in acute infection. Contrastingly, severe dermal lesions are observed in subacute form due to the occurrence of oedema and infiltration of lymphocytes, macrophages, mast cells and plasma cells in perivascular spaces of skin (Blome *et al*., 2020) [6].

Necrosis in walls of blood vessels with enlarged endothelial cells, thrombi and fibrin deposition may be seen in acute form of ASF. Whereas, multiple necrotizing ulcers are frequently observed in the dermis and cornea of the animals bearing subacute or chronic infection. Furthermore, viral replication and antigens can be noticed in the endothelial cells, intravascular monocytes and perivascular macrophages in dermis of affected animals using electron microscopy and immunohistochemistry. The histopathological alterations observed in the eye of ASF affected animals include oedema, hemorrhages, macrophagic infiltration and thrombosis in cornea, ciliary body and subretinal tissues (Mozos *et al*., 2003) [20].

ASF viruses primarily target organs where macrophage population is abundant, such as lymph nodes, tonsils, thymus, spleen, and other lymphoid tissues causing extensive tissue necrosis, reduction in lymphocytic population and large number of ASFV infected cells. The percentage of lymphocyte depletion is in direct proportion with the degree of ASFV infected macrophages present in the respective organ. Extensive accumulation of erythrocytes, fibrosis, cellular infiltration and debris due to massive destruction of splenic macrophages are documented in the spleen of acutely infected animals (Sanchez-Cordon *et al*., 2021) [29].

Common histopathological alterations observed in liver lobules include multiple hemorrhages, congestion, oedema, and mononuclear cells infiltration. Sinusoidal leucocytosis and presence of giant Kupffer cells engorged with cellular debris displaying rounded nuclei and chromatin margination are characteristically observed in liver. Oedema is observed in the wall of gall bladder separating connective tissue and muscle layers of the wall. In subacute form, such alterations are noticed with increased severity along haemorrhagic and necrotic mucosa of gall bladder. Hyperplasia in bile duct is characteristically evident in the chronic form of ASF (Sehl *et al.*, 2020) [31].

Histopathology of stomach reveals cellular infiltration in and around blood vessels along with hyperaemia and oedema multiple layers. Mucosal necrotic ulcers may also be seen in the stomach in severe cases. Additionally, short and thickened villi with cellular infiltration is generally observed in small intestinal mucosa (Salguero, 2020) [26]. Presence of hyperaemia, pericardial sac thickening and multiple hemorrhages can be detected in microscopic examination of heart.

Affected lungs microscopically show typical vascular alterations along with hypertrophied endothelial cells and characteristic proteinaceous exudate accumulation in bronchial, alveolar and interstitial regions. Haemorrhages, cellular infiltration, specifically, macrophages, deposition of fibrinous material in alveolar lumen and thickening of alveolar septa are some other common observations. Pulmonary intravascular macrophages are reported to be the primary target for replication of ASF virus in the lungs during early infection. In the kidneys, desquamation of tubular epithelium, intratubular protein casts, and focal aggregations of mononuclear inflammatory cells are usually seen (Sanchez-Cordon *et al*., 2021) [29].

7. Diagnosis

The clinical signs and lesions observed in ASF are not very specific since similar signs can also be observed in CSF, Erysipelas, Salmonellosis, and other diseases of swine. Therefore, laboratory tests become essential for confirmatory diagnosis of the disease. The samples favourably collected for laboratory diagnosis of ASF include bloodsamples, impression smears and tissue samples from tonsil, lymph nodes and spleen. The diagnostic echniques commonly used for ASFV detection consists of direct immunofluorescence (DIF), haemadsorption, and polymerase chain reaction (PCR) (Sanchez-Vizcaino, 2015) [30].

Direct immunofluorescence is a rapid and cost-effective test that can be conducted using tissue impression smears to detect presence of viral antigen. Thus, it can be used in field conditions where quick diagnosis and immediate control actions are required. The limitation of DIF is its low sensitivity (around 40%) for subacute and chronic cases of ASF. This low sensitivity is attributed by the blocking action of antibody which are bound in antigen- antibody complexes already present in the body. Haemadsorption is an important property observed in various strains of ASF virus. It is the phenomenon in which red blood cells get adhered to surface of virus infected monocytes and macrophages giving characteristic rosette appearance. The haemadsorption test is conducted by adding primary WBC cultures with blood samples or homogenized tissue samples from suspected animals (Beltran-Alcrudo *et al*., 2017) [5].

Molecular techniques such as PCR are used for detection of viral DNA from ASFV, usually in those tissue samples which are unfavourable for isolation of virus or detection of viral antigen. As it is reported that the antibodies against ASFV remain in the recovered animals for longer durations even when infected with less virulent strains. Therefore, serological detection can be considered most significant method for diagnosis of such strains of ASFV. Serological detection of antibodies against ASFV can be done using ELISA, immunoblotting, indirect immunofluorescence test, complement fixation test and radioimmunoassay (Aguero *et al*., 2003) [1].

8. Prevention and Control

Presently, vaccine is unavailable for the efficient control of ASF (Subedi *et al.*, 2021) [32]. Owing to its ability to remain alive in meat, contaminated fomites and natural reservoirs for longer time period, elimination of ASFV from area with established infection is very tricky. Culling all the suspected animals can be useful in controlling the transmission of the virus. However, one has to face various environmental, financial and ethical challenges. Using strict biosecurity actions is considered as the most effective method of prevention of ASF. Restricting the entry of personnel and vehicles to piggeries; use of separate clothing and boots inside piggeries; and frequent disinfection with use of foot baths can reduce virus transmission (Penrith and Vosloo, 2009) [22].

Various other effective measures to be followed in order to prevent and/or during an outbreak include restriction of movement of pigs from infected area, prevention of any contact between wild and domestic pigs, avoiding feeding contaminated garbage from international airports and docks to pigs so such waste should be incinerated, etc. Stake holders should be made aware of the biosecurity measures by initiating easily assessible awareness programmes and providing compensatory inducements. Emergency planning and preparedness should be done according to the geographical conditions, financial status, epidemiology and level of ASF infection in neighboring areas (Gavier-Widen *et al*., 2015) [14].

9. Differential Diagnosis

Confirmatory diagnosis of African Swine Fever can be done after laboratory detection, particularly in chronic form of the disease. The following disease must be considered under differential diagnosis.

- **Classical swine fever (CSF):** Caused by *Pestivirus* (a RNA virus with no arthropod associated transmission). The presence of encephalomyelitis with perivascular cuffing and neutralizing antibodies in CSF affected pigs is the main differentiating feature.
- **Salmonellosis:** Usually accompanied by enteritis and dyspnoea.
- **Swine Erysipelas:** In which there are characteristic

diamond skin lesions and the subserous hemorrhages are likely to be echymotic rather than petechial.

- **Colibacillosis:** Enteritis produced by *E. coli* during first week of life and causes high temperature and death within 48 hours due to dehydration and toxaemia.
- **Mulberry heart disease:** This disease usually affects the best healthy pigs. There is no temperature reaction and lesions are more confined to heart.
- **Necrotic enteritis:** Main seat of predilections are on the caecum and colon. Button ulcers are not present.
- **Streptococcal meningitis:** The disease may have resemblance to nervous manifestation of swine fever. Piglets of age 2-6 weeks may be affected. Diagnosis is done by isolation of the bacteria.
- **Salt poisoning:** This is an afebrile condition which sets in suddenly and affects large numbers of pigs in a pen. Respond to therapy with *ad libitum* water.
- Porcine reproductive and respiratory syndrome
- Aujeszky's disease (or pseudorabies) in younger swine
- Pasteurellosis and other septicemic conditions (Salguero, 2020) [26].

10. Conclusion

Pork meat is one of the primary and cheap sources of animal protein, having the highest dressing percentage and easy availability, especially for the tribal population of India. Therefore, the diseases of swine pose a serious threat to food security worldwide. ASF is one such highly contagious viral disease of pigs and wild boars, causing high mortality and responsible for massive losses in the pig population along with drastic economic consequences. It is caused by ASFV of the *Asfarviridae* family and biologically transmitted by *Orinthodoros* ticks. This is highly resistant in the environment, aiding in the spread of the disease through contaminated fomites and pork products. The clinical form of the disease and its associated lesions may vary depending on the geographical conditions, source of infection and immune status of the animals. Therefore, farmers and veterinarians must always be cautious. There is no effective vaccine against ASF available at present and thus, rapid diagnosis, slaughter policies and strict biosecurity measures should be used for controlling of the disease transmission.

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