Indian Farming **63**(2): 23–26, May 2013

An insight into
classical swine fever

DB Shivaraj[1], S S Patil[2] and D Ratnamma[3]

Department of Microbiology, Veterinary College, KVAFSU, Hebbal, Bangalore 560 024

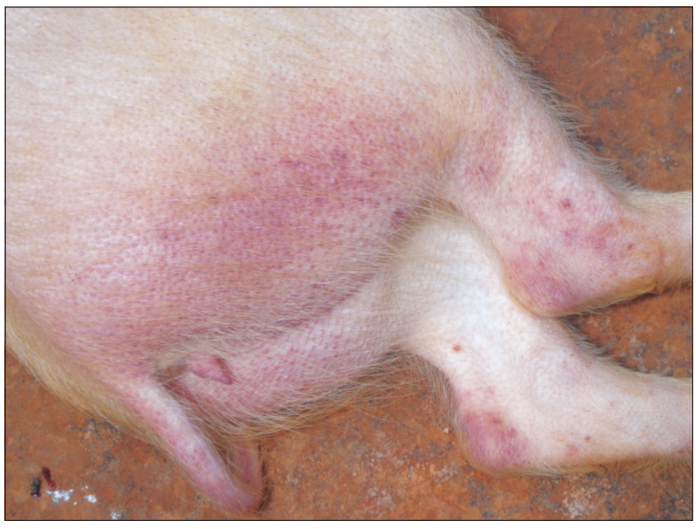
Pigs are the most potential source of meat production among various livestock species and more efficient feed converters after the broiler chicken. They are raised solely for meat production, as they are quick to multiply and can fit into diverse systems of management. Due to increasing trend of pork production and consumption in the country, it is necessary to control economically important disease in the swine population like Classical swine fever. This disease is a major constraint to the development of pig farming systems in India.

PIG farming can provide employment opportunities to seasonally employed rural farmers and supplementary income to improve their living standards. The pig population is around 11.1 million according to 2007 census and pork production stands at 4.02 lakh tonnes in 2011. In north-east India pig farming is a main source of livelihood since 80% of households rear pigs and pork is a key part of the local diet. Classical Swine Fever (CSF) is also known as hog cholera, pig plague, pig typhoid and swine pest. It is one of the most dreaded and devastating viral diseases of swine causing serious economic losses directly due to mortality, retardation of growth, reproductive problems and indirectly by bringing restrictions on exports of pork and pork products. The disease is enzootic in most of the pig producing states and particularly in the North Eastern states of India.

[1] PhD Scholar, [3] Professor and Head, Department of Microbiology, Veterinary College, Bangalore, [2] Senior Scientist, PDADMAS, Hebbal, Bangalore 560 024

Etiology

Classical swine fever is a contagious viral disease of pigs caused by a member of the genus Pestivirus of the family Flaviviridae. Only one serotype has been found, but minor antigenic variability has been demonstrated between viral strains.



Hemorrhagic lesions of the skin

Huddling together of diseased pigs

Resistance of Virus to Physical and Chemical Action

Virus is readily inactivated by cooking, heating meat to 65.5°C for 30 minutes or 71°C for one minute and survives months in refrigerated meat and years in frozen meat. Virus is stable at pH 5-10 and rapidly inactivated at pH <3.0 or pH >11.0. It is moderately fragile and does not persist in the environment and is sensitive to drying and ultraviolet light. It survives well in pens during cold conditions (up to 4 weeks in winter), and 7-15 days at 37°C. It survives well in meat during salt curing and smoking for 17 to >180 days depending on the process used. Virus inactivated by chlorine-based disinfectants, cresol (5%), sodium hydroxide (2%), formalin (1%), sodium carbonate (4% anhydrous or 10% crystalline, with 0.1% detergent), ionic and non-ionic detergents, and strong iodophors (1%) in phosphoric acid. It is also susceptible to ether, chloroform, ß-propiolactone (0.4%).

Transmission

Infected pigs are the only reservoir of virus. Virus shedding can begin before onset of clinical signs and occurs throughout the course of disease.

- Spreads mainly by the oral and oronasal routes, via direct or indirect contact.
- Direct contact between animals (secretions, excretions, urine, semen and blood).
- Most common means of entry into disease free areas is by insufficiently cooked waste food fed to pigs.
- Spread by farm visitors, pig traders.
- Indirect contact through premises, implements, vehicles, clothes, instruments and needles.
- In areas of high pig farm density, airborne transmission may occur over short distances (upto one kilometer).
- Transplacental infection may create inapparent carrier piglets or congenital abnormalities.

Clinical Symptoms

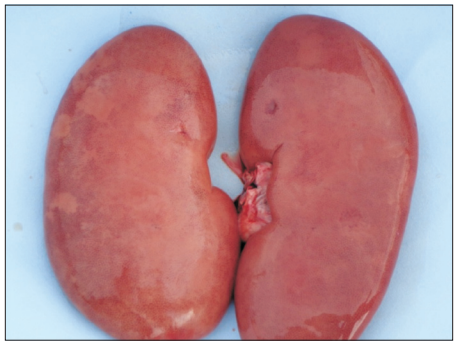
Incubation period is 2–14 days, depending on the virulence of the strain of virus, age/susceptibility of pigs and the herd health status. The disease may run an acute, sub acute, chronic, late onset or in apparent course, depending on a variety of viral and host factors. The most important factors include age of the animals, the virulence of the virus and the time of infection (pre or post-natal).

Acute Form

It is by more virulent virus strains and/or younger pigs. Pigs exhibit fever (41°C), anorexia, lethargy, severe leucopenia, multifocal hyperemia, hemorrhagic lesions of the skin, conjunctivitis, swollen lymph nodes, cyanosis of the skin especially of extremities (ears, limbs, tail, snout), transient constipation followed by diarrhea, dyspnoea, coughing, ataxia, paresis, convulsions, and huddle together. Death occurs 5–25 days after onset of illness. Mortality in young pigs can approach 100%.

Subacute Form

It is by moderately virulent strains. In this form clinical signs are similar to acute form but symptoms are less severe and death occurs usually within a month.



Turkey egg (petechial hemorrhages) appearance of kidney

Chronic Form

It is by less virulent virus strains or partially immune herds. Dullness, capricious appetite, pyrexia, diarrhea for up to 1 month, ruffled appearance of pigs, growth retardation, apparent recovery with eventual relapse and death within 3 months.

Congenital Form

Its outcome depends on virulence of virus strain and stage of gestation. In pregnant sows, the virus may cross the placental barrier and reach the fetuses. In-utero infection with strains of the virus of moderate or low virulence can results in the 'carrier sow' syndrome. It causes prenatal or early post-natal death, the birth of diseased piglets or an apparently 'healthy' but persistently infected litter. Due to transplacental transmission it results in fetal death, resorption, mummification, stillbirth, abortion, congenital tremor, weakness, runting and poor growth over a period of weeks or months leading to death. Piglets which are clinically normal are persistently viraemic and important intermittent shedders of virus until dying in 6–12 months (late onset form).

Mild Form

Usually in older animals, causes transient pyrexia and inappetence. Sometime, pigs may recover and get lifelong immunity.

POSTMORTEM LESIONS

The lesions of Classical swine fever are highly variable. During outbreaks, the likelihood of observing characteristic necropsy lesions is better if four or five pigs are examined.

Acute Form

In acute disease, most common lesion is haemorrhage.

- Leucopoenia and thrombocytopenia, skin may be purple discolored.

- Enlarged hemorrhagic lymph nodes are common.
- Widespread petechiae and ecchymoses, especially in the skin, lymph nodes, epiglottis, bladder, kidney (Turkey egg appearance of kidney) and rectum.
- Severe tonsillitis with necrotic foci occurs commonly.
- Multifocal infarction of the margin of the spleen is characteristic or pathognomonic.
- Lungs may be congested and haemorrhagic.

Lesions are usually complicated by secondary infections. In some cases, lesions may be absent or inconspicuous, if death of pigs occurs immediately after infection.

Chronic form

Lesions are usually complicated by secondary infections

- 'Button' ulcers in the caecum and large intestine mucosa
- Generalised depletion of lymphoid tissue
- Transverse striations of cartilage at costochondral junctions in growing pigs
- Haemorrhagic and inflammatory lesions are often absent.

Morbidity and Mortality

The morbidity and mortality rates are high during acute infections and the case fatality rate can approach 100 per cent. Morbidity and mortality are lower in sub acute disease. Chronic infections are always fatal, but may affect only a few animals in a herd. The age and immune status of the animals also affects the course of the disease, with lower mortality rates in adult pigs than younger animals.

Public Health

Classical swine fever does not affect humans.

Laboratory diagnosis

Classical swine fever should be suspected in pigs with signs of septicemia and a high fever, particularly if uncooked swill (hotel waste) has been fed or new animals have been introduced into the herd. This disease may also be considered in herds with poor reproductive performance and disease in piglets. The variability of the clinical signs and post-mortem lesions do not provide firm evidence for unequivocal diagnosis. A tentative diagnosis based on clinical signs and post-mortem lesions must therefore be confirmed by laboratory investigation.

Detection of virus or viral nucleic acid in whole blood and of antibodies in serum are the methods of choice for diagnosing CSF in live pigs, whereas detection of virus, viral nucleic acid or antigen in organ samples is most suitable when the pig is dead .

Samples to be collected from:

- Blood in EDTA or Heparin (live cases)
- Tissue from tonsils, lymph nodes (pharyngeal, mesenteric), spleen, kidney, distal ileum.(death cases)
- Serum from recovered and affected animals

Refrigerate and ship to laboratory as quickly as possible

Diagnostic Techniques

Serological tests: Due to the immunosuppressive effect of virus, antibodies cannot be detected with certainty until 21 days post-infection. Submit sera from convalescent pigs and from contact herds when more than 3 weeks have elapsed since suspected contact took place. Serum should also be tested from sows with suspected congenitally infected litters. Antibodies persist for life in recorded pigs. The following may be used for serological diagnosis or surveillance;

- Neutralisation peroxidase-linked assay
- Fluorescent antibody virus neutralization
- ELISA

Molecular techniques: Reverse transcription polymerase chain reaction (RT-PCR) or real time RT-PCR more suitable for pre clinical diagnosis.

Virus isolation: Virus isolation in cell culture, with virus detection by immunofluorescence or Immunoperoxidase.

Prevention and Control

No treatment is possible. Affected pigs must be slaughtered and the carcasses buried or incinerated, since treatment is not possible.

Sanitary prophylaxis

- Effective communication between veterinary authorities, veterinary practitioners and pig farmers.
- Effective disease reporting system.
- Strict import policy for live pigs, pig semen, and fresh and cured pig meat.
- Quarantine of pigs before admission into herd.
- Efficient sterilization (or prohibition) of waste food fed to pigs.
- Serological surveillance targeted to breeding sows and boars.
- Effective pig identification and recording system.
- Effective hygiene measures protecting domestic pigs from contact with wild boar.

Prophylaxis by vaccination

Vaccination with modified live virus strains is effective in preventing losses where classical swine fever is enzootic.

Lapinized vaccine: It is developed by serial passage of CSF virus in rabbits, and the vaccine is processed from the spleen and other lymphoid tissues of the infected rabbits. The biggest limitation in the production of this vaccine is its dependence on the availability and continuous supply of rabbits. This vaccine is currently produced by;

- Institute of Veterinary Biologicals at Guwahati, Kolkata, and Lucknow.
- Institute of Animal Health and

Veterinary Biologicals, Mhow ,Madhya Pradesh.
- Indian Veterinary Research Institute, Izatnagar.

Lapinized cell culture vaccine: The lapinized vaccine strain is used to produce vaccine in cell culture. Production of this vaccine does not depend on availability of rabbits and can therefore be made in large quantities. This vaccine is currently produced on 'trial basis' by Institute of Animal Health and Veterinary Biologicals (IAH & VB) in Bangalore. Its commercial production is expected soon after licensing is received from government.

Indian Veterinary Research Institute, Izatnagar also has developed this technology and is working towards its commercial production. Production and use of this vaccine appears to be the best available choice. India requires a total of 22.26 million doses of CSF vaccine per year, with north-east India alone requiring 7.64 million doses. Currently, the country has about 1.2 million doses of the vaccine.

Policy Recommendations for Prevention and Control of CSF

- To meet the immediate demand of vaccine, the lapinized vaccine production facilities at the Institute of Veterinary Biologicals (Guwahati, Kolkata, Lucknow) and at the Institute of Animal Health and Veterinary Biologicals, Mhow, should be strengthened with infrastructure and manpower support. This can increase production level from the current 1.2 million doses to 5 million doses within one year.
- To meet the country's entire demand, commercialization of the lapinized cell culture vaccine technology through public–private partnership is a best option.
- Strengthening of laboratory infrastructure for rapid CSF diagnosis and initiation of disease control measures by the state veterinary authorities.
- Strengthen the cold chain facilities and the vaccine storage capacity at the headquarters of each state.
- Develop a strategy for vaccination at key border points/markets/trade routes and develop quarantine facilities.
- State CSF surveillance units should be set up in each states directorate of Animal Husbandry and Veterinary Services and the state epidemiologist should independently investigate all disease outbreaks supported by laboratory investigations.
- The government may consider launching a national CSF control/ eradication program with particular focus on north-east India under 12th Plan Period.

Response to Outbreaks

Immediate intimation has to be sent to nearest veterinary officers, working at veterinary hospitals.

- Slaughter of all pigs in affected farms
- Safe disposal of carcasses, bedding, etc.
- Thorough disinfection; Sodium hypocholorite and phenolic compounds are effective disinfectants.
- Designation of infected zone, with control of pig movements.
- Detailed epidemiological investigation, with tracing of possible sources (up-stream) and possible spread (down-stream) of infection.
- Surveillance of infected zone, and surrounding area.

SUMMARY

In north-east India, pig farming is a main source of livelihood since 80% of households rear pigs and pork is a key part of the local diet. The classical swine fever disease may run as acute, sub acute, chronic, late onset or in apparent course, depending on a variety of viral and host factors. The morbidity and mortality rates are high during acute infections and the case fatality rate can approach 100 per cent. In chronic form, pigs which are clinically normal are persistently viraemic and important intermittent shedders of virus until dying in 6–12 months. In such cases, treatment is not possible and affected pigs must be slaughtered and the carcasses buried or incinerated. The variability of the clinical signs and post-mortem lesions do not provide firm evidence for unequivocal diagnosis. A tentative diagnosis based on clinical signs and post-mortem lesions must therefore be confirmed by laboratory investigation. By sanitary prophylaxis and vaccination with modified live virus strains is very effective in preventing losses where classical swine fever is enzootic. This information will help the farmers in generating awareness to prevent spread of disease.