Classical Swine Fever
(CSF)

Community Reference Laboratory for Classical Swine Fever
Institute of Virology, School of Veterinary Medicine Hannover
Bünteweg 17, D-30559 Hannover
e-mail: crl@tiho-hannover.de
Etiology

Family: *Flaviviridae*

Genus: Pestivirus

Genome: ssRNA, +Polarity, ~ 12 KB, 1 ORF, 2 UTRs

Proteins: 11 (5 structural, 6 nonstructural)
Classical Swine Fever
Courses of disease
- postnatal -

Horizontal transmission, general infection

INFECTION

Acute illness
<4 weeks

death
Chronic course of disease
>4 weeks
reconvalescence
In incubation period lasting for approximately seven to ten days, first clinical symptoms develop. Animals may show atypical signs of illness such as loss of appetite, fever, swollen lymphnodes and lethargy as well as “classical” symptoms of CSF, e.g. haemorrhages of the skin, conjunctivitis and/or neurological signs. Viraemia already starts during the incubation period and can last for several weeks post infection. Antibodies against CSFV can be detected from two to three weeks post infection onwards. Pigs suffering from the acute form of CSF either die or recover. The severity of the disease depends mainly on the animals’s age at the time of infection, its immunological reactivity as well as the infecting dose and virulence of the virus.
“Classical” CSF, skin haemorrhages
CSF - Acute Course

Petechiae on the epiglottis
CSF - Acute Course

Petechiae in the kidney
CSF - Acute Course

Haemorrhages - Lung and Lymphnodes
Petechiae on the peritoneum and the intestines
“Typical” skin cyanosis on the ears and nose; 
Terminal stage of the acute form of CSFV-infection
Map-like confluent haemorrhages of different grades on the hind-limbs
Skin lesions resembling an infection with poxvirus
Mild form of CSF in a fattening pig; generalised erythema
CSF - Acute Course

Skin haemorrhages predominantly over bone protuberances
CSF - Acute Course

Swollen inguinal lymphnodes; haemorrhages on the prepuce
CSF - Acute Course

Weakness of the hindlegs, staggering gait, often resulting in posterior paresis
The chronic form of CSF occurs when pigs are not able to develop an effective immune response against CSFV. Antibodies can be detected in the serum at approximately one month post infection, but the production is not sufficient to neutralise the virus. Initial signs of infection resemble those of the acute form of CSF (“early acute reaction”), later predominantly non-specific signs can be observed, “typical” haemorrhages are often missing. Animals suffer from intermittent fever and chronic enteritis and also show growth retardation (period of partial recovery). They may survive two to three months before they eventually die (period of relapse and death). The chronic form of CSF is relatively rarely seen under field conditions, but of great importance for the epidemiology of the infection, since chronically infected animals constantly shed the virus from the onset of clinical symptoms until death.
CSF - Chronic Course

Often animals appear normal and show good appetite for a longer period of time, however, growth retardation and wasting are the most evident signs.
CSF - Chronic Course

Diffuse dermatitis
Confluent skin haemorrhages and necroses on the ear edges are seldom present in chronic CSF, however, they can occasionally be found.
CSF - Chronic Course

Diffuse dermatitis in pigs suffering from the chronic course of infection with CSFV
CSF - Chronic Course

Diarrhoea
Button ulcers in the large intestine
The CSF virus has the ability to cross the placental barrier and infect the foetuses. The infection in the sow is often subclinical. The outcome of transplacental infection is dependent on the stage of gestation and on the virulence of the virus involved. Infection during early pregnancy can lead to abortion, stillbirth, mummification, neonatal death or malformations. The fertility index in the diseased holding is therefore reduced.

Infection of sows at up to 90 days of pregnancy can result in the birth of persistently viraemic piglets, which may appear clinically "normal" at birth and may survive for several months before they die as a consequence of the infection.
Time of intrauterine infection is the crucial factor. When the foetuses are not yet immunocompetent, they become immunotolerant against the virus and persistently viraemic. Only persistently viraemic foetuses show the late onset course of disease which is not necessarily clinically evident. Post-mortem findings are unspecific.
During the first weeks after birth maternal antibodies can be detected in persistently viraemic as well as in non-viraemic piglets. Later on non-viraemic offspring is not immunotolerant against CSFV and antibodies are produced, whereas no more antibodies can be detected in persistently viraemic piglets.
Aborted and mumified fetuses.

Detection of CSF in pig breeding operations might be particularly difficult to detect, since the symptoms in adult pigs may be very mild and can be caused by many other pathogens. Thus an investigation for CSFV should be carried out in any case of reduced fertility index, when any other risk factors for CSF (e.g. area where CSF occurs in wild boar) are present and/or other diseases of the reproductive tract have been excluded.
Persistently infected wild boar piglet ('late onset') showing growth retardation when compared to the litter mates (left).
Epidemiological links between cases of CSF in wild boar and domestic pigs have frequently been reported. Thus, in areas where CSFV infection occurs in the wild boar population, it has to be considered as a possible source of infection for domestic pigs. Clinical and pathological findings in wild boar and domestic pigs are comparable and it is assumed that both are equally susceptible to the virus.
CSF in wild boar

Multiple petechial haemorrhages on different organs and swollen, haemorrhagic lymphnodes in a wild boar (acute form of CSF)
Petechiae on the epiglottis of a wild boar (acute form of CSF)
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