Porcine Reproductive and Respiratory Syndrome (PRRS)

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Adapted from:


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DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis of PRRS is complicated by the variability of the clinical signs among age groups and the fact that once the disease has become endemic in herds no clinical signs may be apparent. In these herds the only evidence of the presence of PRRS, apart from serological evidence, may be failure to reach production targets due to increased susceptibility to other diseases, in particular those affecting the respiratory tract. Laboratory confirmation of any suspected case is therefore essential.

Clinical signs and pathology

Clinical signs depend on the virus strain, host’s immune status, concurrent infections and how the pigs are managed. There are no pathognomonic or even consistent clinical signs. The presentation of PRRS ranges from a subclinical to a severe and sometimes highly fatal disease.
PRRS epidemics in naïve herds typically commence with pigs of all ages developing fever indicated by anorexia and lethargy.

Some pigs may show reddening of the skin and/or cyanosis of the extremities; the latter gave rise to the name ‘blue ear disease’. Morbidity can vary from 5 – 75% and spread throughout the herd is rapid.
Blue discolouration of the ears may occur in PRRS

Feverish pigs showing skin flushing and huddling together

Purple blotches on the extremities of a pig with PRRS
Sows of any parity may abort litters, or farrow litters close to, at or a little after their due date that may include normal piglets, weak small piglets, stillborn fresh piglets, and autolysed and partially to completely mummified piglets.

Abortions are the main indication of PRRS in adult sows

Sows may show no clinical signs apart from negative effects on reproduction or may develop agalactia, nervous signs, pneumonia or exacerbation of existing conditions such as mange and cystitis/pyelonephritis. Mortality in sows is generally less than 5% but sows that recover may show delayed return to oestrus and/or low conception rates in the next cycle. Boars, in addition to fever and respiratory signs, may have reduced libido and semen quality; however, shedding of virus in semen has been demonstrated to be a potent source of infection.
Pre-weaning mortality may increase to as much as 60%, and a high proportion of the piglets may show various abnormalities such as periocular oedema, splay legs, failure to suckle resulting in starvation and emaciation, severe dyspnoea and anaemia. Watery diarrhoea has been described but is not consistent.

Pigs with PRRS may develop severe diarrhoea

Weaner and grower pigs show mainly signs of fever with anorexia, lethargy, skin discolouration and rough hair coats, with varying degrees of respiratory distress, and up to 20% of pigs may die. The incidence of other infectious diseases is increased and these may include meningitis caused by *Streptococcus suis*, bacterial bronchopneumonia, Glässer’s disease, salmonellosis, and mange.

PRRS usually becomes endemic in infected herds, and clinical disease is usually only observed in highly susceptible groups like weaned pigs in which passive immunity has waned and naïve pigs introduced into the herd like gilts and young boars. However, exposure to a different strain of virus may result in epidemic PRRS as the degree of cross-protection between strains is variable. PRRS has also been incriminated as the major cause of porcine respiratory disease complex (PRDC) which manifests as slow growth, decreased feed efficiency, anorexia, fever, coughing and dyspnoea in weaning and finishing pigs. Reproductive failure in sows and temporary infertility in boars may also been seen in affected herds. A wide range of respiratory pathogens as well as porcine circovirus-2 have been implicated in PRDC but co-infection with swine influenza virus and *Mycoplasma hyopneumoniae* appears to be frequent (Dorr *et al.* 2007).

The highly virulent form of PRRS that emerged in China in 2006 is characterised by high fever with up to 100% of pigs affected and with 20 – 50% mortality (Zhou *et al.* 2010).

There are no pathognomonic pathological lesions but enlarged lymph nodes and interstitial pneumonia affecting the cranial lobes or the entire lungs are consistently seen in pigs that die of PRRS, usually sucklers and weaners up to 70 days of age. Affected lungs are mottled, do not collapse, and may be
variably rubbery, red and oedematous, depending on the severity of the pneumonia. Lymph nodes are moderately to greatly enlarged, tan-coloured and oedematous, progressing to becoming paler and firm. Microscopic lesions consist of round cell infiltrates in alveolar septa of the lung and in multiple organs around blood vessels (perivascular cuffing). In cases of longer duration in older pigs lesions of secondary infections may be present in the lungs, sometimes obscuring the lesions caused by PRRS.

Apart from the abnormal composition of aborted to full term litters, which can consist of normal, freshly dead, autolysed and mummified piglets, there are no foetal lesions that are useful for diagnosis of PRRS.

Laboratory confirmation

It has been emphasised previously that laboratory confirmation of PRRS is absolutely essential. As for all diseases, laboratory confirmation depends upon the suitability and quality of the samples submitted. The limited pH and temperature tolerance of PRRS virus makes careful handling of samples important and they should be kept at 4°C and transported to the laboratory as soon as possible. Serum, tonsils, lymph nodes, and lung are the preferred samples in an acute outbreak, while in older cases tonsils and lymph nodes may be the only tissues that still contain sufficient virus for detection. From aborted foetuses, lung, liver, kidney and spleen should be collected to prepare 20% tissue homogenates for viral isolation attempts. Thoracic fluids have also been used for viral isolation.

The gold standard for diagnosis is viral isolation with confirmation by a fluorescent antibody test (FAT) or immunohistochemistry (IHC) to detect viral antigen. PRRS virus only grows in a limited number of cell lines, and in particular the European viruses have a strong preference for pig pulmonary alveolar macrophages. It is recommended that culture should be attempted in more than one cell line. Because of the difficulties encountered in viral isolation a pig bioassay is considered the most sensitive assay for detection of PRRSV. Seroconversion or detection of replicating virus in a young pig injected with a homogenate of the sample indicates that it contained PRRSV.

Viral antigen may be detected in frozen tissues (lung tissue is usually used) by means of an FAT or in formalin-fixed tissues (lung, lymph nodes, spleen, thymus, tonsil) by IHC. FA tests are more rapid but IHC is more sensitive and is the only option for formalin-fixed tissues. Both techniques require skilled performance and interpretation.

For detection in foetuses, live-born or freshly dead foetuses from affected litters are preferred because autolysed and mummified foetuses rarely yield virus.

PCR-based assays to detect viral RNA are more sensitive and specific than viral isolation, FA or IHC, the results are available within 1 – 3 days and detection is possible in samples like faeces, autolysed foetuses and semen. A positive PCR does not necessarily mean that the virus is infectious, but it can be followed by culture if the sample is suitable and it provides proof that the pig was infected with PRRS virus. Infection of pigs with material that was positive for PRRSV by means of polymerase chain reaction
(PCR) assays but from which virus could not be isolated has been reported, and this was interpreted as indicating that the infective dose for pigs might be very low. However, another, more probable interpretation is that culture failed because of the fastidiousness of the virus.

Antibody detection is not the ideal way to diagnose an outbreak of PRRS, particularly in a previously infected or vaccinated herd, unless paired samples (acute and convalescent) are taken to demonstrate a rising titre, because the available tests do not distinguish between infected and vaccinated animals. Demonstration of the agent is always preferable, since antibodies also do not necessarily indicate current infection. In the case of PRRS, in persistently infected pigs the virus may be present in tonsils and lymph nodes when antibodies are no longer detectable, and the seronegative pigs will be assumed to be uninfected. The standard serological test for antibodies is a commercial ELISA with good sensitivity and specificity and potentially able to detect antibodies to both the North American and European-type viruses. Suspicious positive results may be confirmed by an indirect fluorescent antibody test (IFA) or by a blocking ELISA. Serological testing for antibodies to PRRS is used for herd monitoring but is usually combined with screening by PCR. In herds without clinical signs pigs of 9 – 16 weeks old are the best group to sample to detect circulating virus (Duinhof et al. 2011).

The diagnostic tests used for PRRS are described in Chapter 2.08.07 of the OIE Terrestrial Manual for Diagnostics and Vaccines available as a pdf file on the OIE website (OIE 2011).
Differential diagnoses for PRRS include diseases that result in reproductive failure, namely porcine parvovirus, Aujeszky’s disease, porcine enteroviruses, classical and African swine fever, haemagglutinating encephalomyelitis virus and leptospirosis, and respiratory infections like swine influenza and cytomegalovirus. The enlarged lymph nodes are also suggestive of post-weaning multisystemic wasting syndrome caused by porcine circovirus-2. Any of these infections and many others may be concurrent with and potentiated by PRRS infection and therefore diagnosing one or more of them does not necessarily rule out PRRS.

Skin lesions on the hind legs can resemble classical swine fever or porcine dermatitis and nephropathy syndrome