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Generalized tremors in grower pigs: Preliminary observations

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Since the beginning of 1997, a new clinical entity has been observed in approximately 30 growing-finishing units of Québec, all within the same geographical area. The condition is characterized by generalized tremors that develop suddenly. Affected pigs are of different breeds and sexes and originate from unrelated integrated organizations. The present report describes the clinical, pathologic, epidemiologic, and laboratory findings observed in this newly recognized condition.

Clinical findings

Typically, one or a few pigs within a unit are observed with a sudden onset of generalized tremors. These tremors vary in intensity from barely detectable to violent, and are often aggravated by external stimuli like noises or movements around the animals. Some pigs are hyperesthetic and make strange cries when approached or manipulated. The rectal temperature is increased (40–42°C). In some animals, the tremors reduce markedly or even stop when they lie down and rest. Affected pigs usually have good body condition and do not present other obvious clinical signs. The condition is generally observed in units where there are no other significant health problems. Occasionally mild to moderate respiratory signs are also noticed.

To date only animals between approximately 20–55 kg (44–120 lb) have been affected. In most cases, the morbidity rate is low (0.1%–3.0%) and the case fatality rate is high (≥50%). The disease usually evolves rapidly, with many pigs dying within 24–48 hours. Other than providing a quiet and uncrowded environment with easily accessible water and feed, no treatment (antibiotics, corticosteroids, or sedatives) has yet been successful. Time of recovery in surviving animals varies from a few days to a few weeks.

Epidemiologic findings

In many, but not in all cases, animals with tremors originated from herds that had experienced problems with porcine reproductive and respiratory syndrome (PRRS) either when these pigs were neonates or when still *in utero*. In one organization, it was observed that when pigs from different sources were grouped in growing-finishing units, pigs from only one of these sources were usually affected. If grouping took place at weaning (about 18 days), the condition was seen mainly but not exclusively in pigs from one source. At least two sow herds have produced affected pigs at two different periods, separated by intervals of several months.

Since the genetic lines used by the various organizations are different, and since the condition appeared in these systems at approximately the same time, heredity as the sole cause of the condition seems unlikely. Similarly, nutritional factors can probably be excluded because of the low morbidity within farms where pigs are all fed the same ration, and also because this same ration is fed in many other herds that have no such problem.

Pathologic findings

The gross lesions found in these animals are nonspecific and inconsistent, but several pigs necropsied had pneumonia and generalized lymphadenopathy. Microscopically, almost all pigs had an interstitial pneumonia and a reactive hyperplasia of the lymph nodes. The presence of mononuclear cells around blood vessels (perivascular cuffing) was noted in many organs, including brain and spinal cord in which this lesion was mild and with no specific distribution. A mild and multifocal nonsuppurative interstitial nephritis was present in about 50% of the submissions. The skeletal muscles of nearly half of the pigs examined had mild lesions of polyphasic myonecrosis.

Laboratory findings

Blood samples were obtained from affected as well as unaffected pigs from the same pens. The enzymes AST (aspartate aminotransferase) and CK (creatinine kinase) were higher on average in affected pigs, indicating muscular damage. Serum calcium and potassium concentrations were increased significantly. The cerebrospinal fluid from all five affected pigs tested had an elevated concentration in proteins and the total number of nucleated cells (mainly small and large monocytes) was higher than normal. These results are compatible with the presence of a nonsuppurative meningoencephalitis.

Whole blood from two affected pigs to detect the halothane gene (Hal) mutation, associated with porcine stress syndrome (PSS) were found to be homozygous negative.

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Diagnostic notes are not refereed.

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Bacteriologic examination of various tissues from the pigs necropsied was inconclusive. Although the number of submissions for virology has been limited, PRRSV has been detected either by polymerase chain reaction (PCR) or by viral isolation in some cases. Restriction fragment length polymorphism (RFLP) pattern (using Hinc II, Mlu 1, and Sac II) and the sequence of open reading frame 5 of a strain isolated suggest that it was a field rather than a vaccine strain.

Discussion

The etiology of this condition is still undetermined. Although some epidemiologic, pathologic, and virologic data may suggest the involvement of PRRSV, it is still not clear whether or not it is the actual cause. This virus has been present in Québec for the last 10 years and the first cases of generalized tremors were observed in early 1997. The lesions present in animals with tremors, particularly at the level of the central nervous system, are not different in type or severity from those seen in pigs affected with PRRS but not showing this particular clinical sign. Since PRRSV is known to mutate easily, one could hypothesize that mutations have produced strains with a different tropism and, thus, a different clinical picture. This organism might also simply increase the susceptibility of animals to other pathogens or disorders. Finally, it is possible that the relation between this prevalent agent and the condition described herein is only coincidental. Further work is in progress to investigate more thoroughly the possible etiology and pathogenesis of this condition.

In Québec, these cases of generalized tremors have to date been observed in a relatively limited geographical area of the province. A condition very similar to what is described in this paper has recently been seen in one herd from Iowa.[1]

Reference

1. Engle MJ. Personal communication. 1998.

PRACTICE TIP

Ear necrosis

Ear necrosis in nursery pigs is caused by one or more of a combination of:

- unpalatable feed,
- no feed (feeders either left empty for some time or set too tight), or
- overcrowding (usually toward the end of the nursery feeding period).

One or more of these conditions, along with high humidity in the room, retard healing of ear suckling lesions because of secondary infection predisposed by the high humidity. Correct this by:

- never letting feeders go empty;
- adjusting feeders properly;
- feeding the correct diet;
- not letting nursery feed become stale;
- allowing at least 1 square foot of pen space per 25 lb (11 kg) bodyweight; and
- keeping the relative humidity below 50%.

Even though pigs may sneeze (from dust) and utility bills will be higher when the humidity is low, it is worth reducing humidity by increasing airflow. High humidity is a predisposing factor to greasy skin disease and pneumonia. In my opinion, the reduction in humidity is the reason wean-to-finish building pigs generally outperform conventionally housed nursery pigs.

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