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Clinical cases presentation

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Three topics are discussed in this document:

- generalized tremors in growing pigs;
- atypical Glasser's Disease; and
- the impact of some diseases on gastric ulcers.

The first is a new clinical entity that, in Québec, was first diagnosed early in 1997. The second was chosen to remind us that *Haemophilus parasuis* can kill pigs without producing the classical fibrinous polyserositis lesions. Finally, the last is simply a topic on which—though there is little clear information in the scientific literature—there is a growing body of field evidence suggesting an association between debilitating diseases and a predisposition toward developing gastric ulcers.

Generalized tremors in growing pigs

This curious condition has now been diagnosed in more than 40 grow-finish herds in Québec. Interestingly it has so far been limited to a relatively small area of the province. Different companies using different genotypes have experienced the problem. In most cases, pigs between 45 and 125 lbs are suddenly seen with generalized tremors, and approximately 50% will die, usually within 24 to 48 hours. The morbidity is generally low (0.1–3%). The rectal temperature is often increased (40–42° C), and in some animals the tremors are markedly reduced or even stopped when they lie down and rest. The condition is usually observed in units where there are no other significant health problems, but occasionally mild to moderate respiratory signs are also noticed.

In most—but not all—cases, the affected pigs came from herds that had recently experienced PRRS (porcine reproductive and respiratory syndrome) problems, either at the time when these pigs were recently born, or still *in utero*. 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.

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. Blood samples were obtained from affected as well as unaffected pigs from the same pens. The enzymes AST and CK were higher on average in affected pigs, indicating muscular damage. The cerebrospinal fluid from all five affected pigs tested had an elevated concentration in proteins and the total number of nucleated cells was higher than normal. These results are compatible with the presence of a nonsuppurative meningoencephalitis.

Bacteriologic examination of various tissues from the pigs necropsied was inconclusive. The PRRS virus has been detected in some cases either by PCR or by viral isolation. The RFLP pattern and the sequence of ORF 5 of a strain isolated suggest that it was a field rather than a vaccine strain.

The etiology of this condition is still undetermined, although some data may suggest the involvement of the PRRS virus. Further work is in progress to investigate more thoroughly its possible etiology and pathogenesis.

Atypical Glasser's disease

Haemophilus parasuis (Hps) is the cause of a fibrinous polyserositis commonly referred to as Glasser's disease. It has also been involved, sporadically, in outbreaks of acute illness where fibrinous pleurisy, pericarditis, and peritonitis were not prominent features.[1,2] Riley and others described a septicemic disease with high mortality where the main changes in acutely affected pigs were skin discoloration, pulmonary edema, arthritis, meningitis, and renal glomerular thrombosis.[1] Peet and others reported an Hps-related septicemia in which the principal lesion noted was, again, renal glomerular thrombosis.[2] An earlier communication by Nordstoga and Fjolstad mentioned the association of *Haemophilus spp.* (reported as *Haemophilus parainfluenzae* and *Haemophilus suis*) infections with renal vascular lesions.[3] The authors stated that such lesions were occasionally seen in typical out-

breaks of Glasser's disease, but the most extensive vascular damage was observed with acute infections in the absence of the classical polyserositis lesions suggestive of this disease.

Reported here are the clinical and pathological observations made during an outbreak of atypical Glasser's disease, on a farrow-tofinish 200 sow farm. Seven 25–50kg pigs died suddenly within a week, in a recently established purebred herd selling animals destined to be breeeding stock. The sows and boars came from two different sources, free of most important swine diseases. The disease seemed initially limited to one of the nursery rooms. Some pigs were weak and trembling when forced to move, and two were unable to rise. Affected animals had fever (40.5–42.2° C); a few showed a mild dyspnea, and two out of 50 present in the section had warm swollen joints. All pigs were in good body condition. According to the manager, at no time were signs of central nervous system involvement like paddling and opisthotonos observed.

Blood samples were obtained from four clinically affected animals, as well as from two healthy pigs. Affected pigs had a degenarative shift to the left, defined as an increase in immature neutrophils (non-segmented and toxic) without any increase in the number of leucocytes.

An autopsy was performed on the seven pigs that died. Most carcases were cyanotic and in good condition. The following macroscopic lesions were observed for some or all the pigs:

- paleness of muscles;
- hydropericardium, hydrothorax, and hydroperitoneum;
- edema of the subcutaneous tissue, pancreas, and lungs;
- congestion of lymph nodes, lungs, kidneys, liver, spleen, and cerebral cortex vessels;
- presence of small filaments of fibrin in the abdominal cavity;
- petechial and ecchymotic haemorrhages on the epicardium and endocardium;
- hyperemia of the tonsils; and,
- enlargement and presence of small haemorrhages or white spots on the kidneys.

One pig had a meningitis and another a fibrinous polyarthritis.

Histologically interesting features included necrosis of tonsillar lymphoid tissues (2/3), of splenic nodules (1/1), and of lymph node germinative centres (1/1); a fibrinopurulent meningitis (2/6), and the presence of numerous fibrinous thrombi in glomerular capillaries (3/5).

Hps was isolated from one out of seven pigs and only from the meninges. In the following weeks the disease spreaded to other nursery rooms, sows, and suckling piglets. Hps was isolated from the liver and joints of a 17-day-old piglet with polyaarthritis. One sow died with lesions very suggestive of classical Glasser's disease, but no organism could be isolated.

The sows' and piglets' feeds were medicated with oxytetracycline. Losses stopped when all pigs in the affected room were injected with penicillin three times in a period of six days.

Two SPF herds had been used to populate that farm. These herds were screened, using nasal swabs, to detect if they were positive or not to Hps. Although the sensitivity of this technique may be questioned, one was found positive and the other negative.

Debilitating diseases and gastric ulcers

Many risk factors have been proposed as predisposing to gastric ulcers. Among the most significant, let us mention finely ground feed (resulting in small particle size), the pelleting process, and feed withdrawal or restriction like the one that occurs before slaughter. The impact of other diseases on the development of gastric ulcers is sometimes mentioned in the literature, but usually as a factor playing only a minor—if any—role. Several conditions, though, appear to be associated with an increase in the prevalence of gastric ulcers and the following lines will briefly look at the author's personal experience with some of them, as well as at a few documents from the literature.

Proliferative and necrotizing pneumonia (PNP) was initially described in Québec, and it was first thought that atypical influenza strains were responsible for that condition. Affected pigs show dyspnea, fever, wasting; and many eventually die or remain poor doers. Most people now believe that this condition is actually associated in one way or another with the PRRS virus. The author had to deal with many cases of that condition within a specific integrated organization. Within that system, pigs in all grow-finish herds would eat exactly the same feed. It was thus very simple to compare, for example, the number of pigs that would die of gastric ulcers in grow-finish herds that had problems with that particular pneumonia, to the one in herds not experiencing these problems. Very quickly it became quite obvious that in herds with PNP problems, a lot more pigs were dying of gastric ulcers. In herds with a regular health status, the overall mortality rate would be in the area of 2%, with 0.0–0.4% (approxi-

mately) dying of gastric ulcers. In herds with serious PNP problems, the mortality rate frequently rose to 10% or higher, and sometimes up to 3–4% of the pigs would die with gastric ulcers.

The following example illustrates this situation. A farm had seven identical finishing units on the same site. In one of the barns, 550 piglets from four sources were introduced and, following an outbreak of PNP, 13% (72) of them died. About half of the pigs were autopsied and at least 10 had gastric ulcers. In the next barn, only three of the sources were introduced, no PNP problem was diagnosed and only one pig died (0.2%), of what appeared to be a heart failure. In these two identical barns, the same piglets were introduced (except for one source), they were managed by the same people, and, finally, the same pelleted feeds and medication programs were used. Yet, the incidence of pigs dying with gastric ulcers was completely different. The only difference appeared to be the serious PNP outbreak that occurred in one barn, but not in the other.

Most people have heard about the 18-week wall. In the author's opinion, the most important organism involved in this condition is *Mycoplasma hyopneumoniae*. It usually starts with a gradual increase in coughing that eventually develops into a more acute respiratory problem with dyspnea, fever, and an increased mortality rate. While working in North Carolina with finishing units experiencing this problem, it became apparent that many of the dead pigs were actually dying of gastric ulcers.

PMWS (post-weaning multisystemic wasting syndrome) is a disease that was first described in western Canada and that has now been reported from several countries, including the US. The most common and consistent clinical signs are wasting and dyspnea. According to Dr. Edward Clark, a specialist on that disease, one of the frequent observation made in these cases is that many pigs are found to have gastric ulcers.

In Norway, Dr. J. H. Jansen described a membranoproliferative glomerulonephritis that was diagnosed in ten Yorkshire piglets, aged between 33 and 73 days, submitted for routine autopsy.[4] Eight of the piglets (80%) had a gastric ulcer. Similarly, another study conducted in Québec on 32 cases of glomerulonephritis concluded that one of the most frequent extra-renal lesions observed in these cases was the presence of gastric ulcers.[5]

Finally Straw and others found that 22% of sick pigs had erosion or an ulcer of the *pars esophagea*, though it was only 6% for normal pigs evaluated at a slaughter house.[6]

In the light of this information, one might ask if all diseases may in fact increase the incidence of pigs that die of gastric ulcers? It has been suggested that acute respiratory problems, like swine influenza, were much more ulcerogenic than chronic illnesses.[7] There may be a need to define chronic and acute illnesses, but in the author's personal experience the diseases that tend to be associated with an increase in gastric ulcers seem to be of the debilitating type—like PNE, PMWS, and others—where it takes some time before the animals die. Porcine pleuropneumonia, for example, can be a very severe and acute disease, but it has rarely been associated with significant increases in gastric ulcers in the author's practice.

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