Gastric ulceration in swine

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Gastric ulceration is a cause of economic loss and a source of welfare concern worldwide. Lesions are almost exclusively limited to the *pars oesophagea*, the region of stratified squamous epithelial tissue surrounding the oesophageal opening. This part of the stomach is nonglandular and, unlike the rest of the stomach, is unprotected by a mucus coating. Ulceration of the *pars oesophagea* does not appear to have been a problem until modern animal husbandry practices were adopted. Surveys from abattoirs reveal that approximately 20% of pigs have erosive lesions, and an additional 60% have pre-ulcerative parakeratosis lesions.[1–3]

The major economic concern associated with gastric ulceration is mortality. On some farms, sudden death from bleeding gastric ulcers is the most common cause of mortality during the grower-finisher stage.[4] Less acute blood loss may result in pigs becoming anemic and unthrifty. Scar formation during healing may result in occlusion of the oesophageal opening into the stomach. This may be so extensive that the passage of feed becomes difficult, but, in addition, there may be leakage from the stomach into the oesophagus causing oesophagitis. Grower-finisher pigs from 3 to 6 months of age are most commonly affected by gastric ulceration, but adult animals are also affected.[5]

Monitoring gastric ulcers

Losses associated with gastric ulceration might warrant an ongoing monitoring program. The first step of a monitoring program is to ensure that the herdsmen are familiar with the clinical signs of gastric ulceration. Blood loss into the gastrointestinal tract, the main clinical event in this condition, may cause anemia and melena. Anemia may be severe, resulting in a very pale, weak pig with rapid breathing. The feces are generally scant, black, and tarry. A number of infectious agents (eg, *Lawsonia intracellularis*, *Salmonella* serovars, and *Brachyspira hyodysenteriae*) may induce lesions resulting in blood loss into the intestines, but these conditions are associated with diarrhea, which is not the case with gastric ulcers.

Pigs with extensive and severe erosive lesions of the *pars oesophagea* may continue to appear healthy if blood loss is minimal. Damaged tissue heals rapidly. If the opening of the oesophagus into the stomach is restricted because of scar tissue formation, pigs may be observed to eat and then vomit shortly afterwards. This process may be repeated many times, and pigs with this type of lesion are likely to grow more slowly than pen mates.

Frequently, blood loss is so severe and acute that the pig is found dead before clinical signs have been detected. Not all pigs that die suddenly and are pale have gastric ulcers. Acute haemorrhagic enteritis (caused by *L intracellularis*) and intestinal torsion are two rule-outs that need to be considered. Gross postmortem examination is generally all that is necessary to verify gastric ulcer mortality. Frequently, the stomach is filled with clotted or partially digested blood. If blood isn't present in the lumen of the stomach, the organ should be further examined. If the stomach is opened along the greater curvature and inverted, the *pars oesophagea* can be easily visualized. The entire area of the *pars oesophagea* may be eroded, leaving a deep ridge at its margin with the cardiac portion of the stomach. However, a focal lesion (often in the area of the margin of the *pars oesophagea* and the cardia) may be very deep and sufficient to cause severe blood loss.

A gastric ulcer death may be secondary to another clinical problem.[6] Frequently, pigs die from ulcers during an outbreak of respiratory disease. It is likely that anorexia caused by the infectious disease is the event that triggers gastric ulceration. In a monitoring program to determine the prevalence and severity of an ulcer problem, it is important to note other diseases present in the animal at the time of necropsy, in order to keep a proper perspective regarding the significance of gastric ulceration relative to other disease conditions.

Many researchers have attempted to monitor the herd prevalence and severity of gastric ulcers by performing slaughter-house checks.[7–9] There are limitations in the interpretation of data obtained at the abattoir as it pertains to gastric ulcers. Ulcers may occur quickly (within 12 hours) and healing occurs relatively quickly as well. Researchers have shown that the prevalence of ulcers was much higher, and their severity was much greater, in pigs held overnight compared to pigs from the same source that were slaughtered on the day that they arrived.[8]

Judging the severity of a lesion is subjective. Most researchers attempt to record severity using a scale with the following gradations: Normal (*pars oesophagea* is smooth and glistening); Parakeratosis (rough surface and possibly bile stained); Small focal erosive lesions; More extensive lesions involving a large proportion of the *pars oesophagea;* Entire surface of *pars oesophagea* eroded. Most studies have found a moderate to poor correlation between gross postmortem examination scores and histological lesions.[10,11] This is partly because histological examination measures depth of lesions, whereas gross examination takes into account how extensive the lesion is. However, gross examination at slaughter is generally done quickly out of necessity, and it is performed on a dead animal that has been exsanguinated. The observer tries to evaluate a white lesion on a white background, and frequently mild to moderate erosions are missed. The other major limitation of an abattoir survey is that it is a snapshot of the situation at the time of

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slaughter. If the problems have occurred several months earlier, there may be little evidence to be seen when the pigs reach market weight.

Endoscopic examination has been used to evaluate the health of the *pars oesophagea* in the live pig. The advantage of this technique is that erosions are easily visualized, and, likewise, small bleeding blood vessels may be noted.[12,13] The technique is simple and easily mastered. The disadvantage is that the pig must have an empty stomach to allow visualization of the *pars oesophagea,* and anesthesia is required for good restraint. When feed intake is restricted to perform the endoscopic examination, it becomes more likely that gastric acids and bile will move into the proximal part of the stomach. In other words, the monitoring procedure will increase the likelihood of the pigs developing ulcers. This may not be an acceptable risk, especially in a herd which already has a significant ulcer problem.

Monitoring risk factors

The cause of gastric ulceration is not fully understood; however, a number of important risk factors are well recognized. In a program to control losses due to gastric ulceration, it is important to monitor these risk factors and reduce their impact if possible. The *pars oesophagea* is protected from the harmful effects of gastric acid, enzymes, and bile while the stomach is full. Anything that causes an empty stomach is a risk factor. Finely ground pelleted feed is associated with a high prevalence of gastric ulcers.[14] This is at least partly a result of rapid stomach emptying when pigs are fed rations with fine particle size.[15] The standard technique for measuring feed particle size is performed by placing a 100-gram sample of mash feed in a shaker consisting of a series of sieves with descending screen size. All material left on each screen is weighed, and the weights are entered into a logarithmic equation.[16] It is generally recognized that when the mean particle size is smaller than about 700 µm, the prevalence of stomach lesions may become a concern.

Disruptions in feed delivery to a pen or a barn may potentially cause an increase in ulcers. Management records should note the occurrence of such problems. Hot weather or outbreaks of infectious diseases are also recognized as triggering factors. Information regarding management or environment changes that might result in pigs with empty stomachs should be considered in the investigation of a gastric ulcer outbreak.

Some reports link the presence of *Helicobacter*-like organisms in the stomachs of pigs to a higher likelihood of ulceration of the *pars oesophagea.*[17] The significance of these bacteria is not certain. Investigation of their presence may be warranted as part of an investigation of gastric ulceration. These spiral-shaped microorganisms may be readily demonstrated in the glandular region of the stomach by histological methods if special stains are used (for example Warthin Starry silver stain).[18] *Helicobacter* spp produce urease, and researchers have used this characteristic to develop a quick screening test. Pig stomachs are coated with a urea gel containing a color indicator sensitive to pH change. If urease-producing bacteria are present in large numbers, the urea is broken down to ammonia, causing pH to rise and a color change to occur.[18] Surveys suggest that *Helicobacter*-like bacteria are widespread in the pig population, and possibly certain strains are more pathogenic than others.

Conclusions

Economics and welfare concerns justify the monitoring of swine populations to determine the prevalence and severity of stomach lesions. In order to reduce gastric ulcer losses, the monitoring of major risk factors, such as feed disruption and feed particle size, should be encouraged.

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