A Gastric-Ulcer Condition of Swine

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A Gastric-Ulcer Condition Of Swine

John N. Berg

A GASTRIC-ULCER CONDITION OF swine was first recognized as a definite disease entity at the Boar Testing Station at Ames, Iowa in early 1957 in pigs that had been farrowed in the fall of 1956. The condition was at first thought to be bloody dysentery, but the feces were not typical in that no apparent break of free blood occurred. Since the condition has been recognized as a separate entity, cases have been reported from the field.

Occurrence

As was previously stated most of the first cases have been reported from the Boar Testing Station in Ames. The other boar testing stations in Iowa have reported similar occurrences. At these stations it has involved only males since these are the only swine on test, but in the field it has been reported in females. At the station its nature is sporadic. The pigs affected are picked out one at a time from a pen (each pen consisting of four pigs) with the pens seldom being side by side and the disease not involving more than one or two pigs at any given time.

Investigations of the sources of some of the affected boars has shown no apparent incidence of the disease in the home herds or area.

The field incidence of this disease is unknown. Where veterinarians have made special efforts to necropsy pigs which have died a sudden death, some gastric ulcers have been found to have caused this death. Wider publicity should result in a greater recognition of the disease.

Etiology

This is largely an unknown factor at the present time. Multiple factors may be involved. At the boar testing stations the pigs are placed in pens with a very limited space. They are on a high energy ration with a very low fiber content to gain as fast as possible. As far as scientific nutrition can determine, the rations contain all essential nutrients that the pigs may need. Because of the conditions under which they are raised, the stress on the digestive tract is probably at a very high level.

If an infective agent is the cause, it has a very low infectivity. As was pre-
viously stated it occurs sporadically with swine on both sides of the affected ones apparently normal and the pigs in the same pen also being unaffected.

There is a possibility of it being an infective agent which, in combination with the predisposing factors, causes the disease. The most likely agent would be a virus, though bacterial and fungal agents cannot be ruled out because an adequate examination for these, especially the fungus, has not as yet been made.

Further complicating the picture is the fact that a diagnosis is made only when chocolate colored feces appear or death occurs which indicates severe bleeding has occurred. How many pigs have the ulcers without showing these apparent symptoms is unknown.

Symptoms

The most characteristic sign of this disease is the color of the feces. They have the dark and fetid appearance that indicates bleeding is occurring high in the digestive tract. Most of these pigs have some looseness of the bowels with the stools resembling chocolate malted milk in color, consistency and homogeneous mixing. About 25 per cent of the pigs feces have this same color but the consistency varies from normal to more firm than normal.

The pigs rapidly become gaunt with a ruffled, dull hair coat and show a general dejection. The visible mucous membranes become pale to almost white if the symptoms last longer than three days. Some of the pigs may show some respiratory distress for eight to twelve hours prior to death. There is no significant rise in temperature, but some pigs may show a subnormal temperature.

Sudden deaths will occur fairly often in affected swine which have not shown symptoms. These animals apparently bleed to death very rapidly into their stomach and small intestines.

The usual outcome of this disease is death, although recovery has occurred in a few cases. Some animals have shown symptoms two or three times before eventual death occurred. Only one boar and one barrow have shown complete recovery of those animals showing severe symptoms.

Pathology

The most characteristic lesions of this condition are gastric ulcers. There are usually one or two very large ulcers of the gastric mucosa, often in the cardiac region, although multiple small ones may occur over the entire gastric mucosa. The small intestine may also have multiple small ulcers scattered over the mucosa but it is more commonly a diffuse necrotic hemorrhagic ileitis.

The stomach may be filled with a well organized fibrin clot or just have free blood with considerable mucus present. The mucosa that isn't covered with blood may be covered with a thick layer of mucus. The stomach may be full of food. The intestine more commonly has blood in a more fluid state, mixed into the mucus. Some blood may spill over into the cecum and colon but no ulceration with bleeding has been found in these organs.

No other significant gross lesions are present.

Diagnosis

The diagnosis of this condition as a separate entity from hemorrhagic dysentery is fairly easily made due to its sporadic nature, the fact that the feces do not contain pure blood, and the typical gastric ulcers which are present.

Treatment

A specific treatment for this condition is unknown at the present time. Bowel astringents, furacins, sulfonamides, antibiotics and Vitamin K have been tried in pigs showing symptoms with no significant results. The injection of clotting agents and the feeding of a bland diet may be indicated. Blood transfusions might be successful except for the difficulty of administering them.

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ing of the glans penis, making it impossible to draw this organ back through the naturally small preputial orifice. If this condition receives attention before extensive lacerations of the exposed tissues occur or necrosis of the glans penis develops, the prognosis is surprisingly good. The treatment should be directed toward the prevention of further trauma to the exposed penis and prepuce. After cleansing the tissues, the penis should be treated with an oil base, non-irritating dressing. The exposed penis is then wrapped in a layer of cotton and covered with a loosely applied bandage. The penis should then be supported against the ventral abdominal wall by a many tailed bagbandage or similar device. The dressing on the penis should be changed once or twice daily until the swelling subsides to the point where the penis can be drawn back into the sheath. It may be advisable to retain the penis in the sheath by sutures across the orifice until tone returns to the retractor penis muscles. Again, the surgical shortening of the retractor muscles may be necessary to prevent recurrence of the condition.

REFERENCES


Part II of this article will appear in the next issue of The Iowa State University Veterinarian.

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Conclusions

Practitioners in the field should be on the alert for this condition. It has probably been largely overlooked as a cause of sudden death in swine. A routine autopsy of all sudden deaths in swine would probably disclose a much higher incidence than is now reported. While the large gastric ulcers are the most characteristic lesion, smaller ulcers of the gastric mucosa or of the small intestine should not be missed. Also, the ulcers may in some cases involve only the small intestine.

Acknowledgment

A NTI-HOG CHOLERA HYPERIMMUNE SERUM INTERFERENCE. Immunity against swine erysipelas was less solid in areas of Yugoslavia where pigs were vaccinated simultaneously against erysipelas and cholera when antihog cholera serum was used.

Tests proved that ten of eleven commercial lots (91%) of anti-hog cholera serum examined contained antibodies against swine erysipelas. One lot (9%) had a titer of 4.65 immunity units and showed ability to lower the immunizing capacity of commercial absorbed bacterin by 47 per cent.

Since anti-hog cholera serum, being homologous, is eliminated slowly, vaccination for erysipelas should either be postponed more than two weeks after its use, or a slowly absorbed erysipelas vaccine should be used.