Osteochondrosis or Leg Weakness Syndrome in Swine

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Introduction

Osteochondrosis or swine leg weakness syndrome is a lameness condition in hogs that is becoming a major concern to swine producers. This paper will attempt to describe the lesions, occurrence, pathogenesis, and suggest means to cope with the condition once diagnosed.

By definition osteochondrosis is a disturbance in endochondral ossification, disruption of the normal ossification process of cartilage which leads to cartilage necrosis and disturbed bone growth. In swine disturbed bone growth occurs in both epiphyseal growth plates and joint cartilages as well as other sites which have less clinical significance.

Lesion Description

Occurrence of osteochondrosis lesions is not sex or breed related (there are “lines” of pigs within a particular breed which have a higher incidence of lesions than other lines) but clinical lameness is more common in the boar due to more strenuous usage. Any lameness in boars is especially important because it hinders their ability to service sows.

The lesions are generally bilateral and any resultant lameness is generally a shifting one. Also, lesions can and often do occur at more than one site simultaneously in a given animal, although the severity of lesions at different sites usually varies.

Lesions first appear at approximately four months of age. However, not all lesions become clinically manifested later on in life. Subsequent lameness depends upon further progression of the lesions caused by various environmental factors. Lameness won’t begin to occur until six to eight months of age, therefore osteochondrosis is a condition only important to breeding stock.

Osteochondrosis has classically been known to occur only in a few individuals in a herd, although this is not always the case.

As stated earlier osteochondrosis is a disturbance in endochondral ossification. Microscopically and grossly, focal thickenings in joint or growth plate cartilage is seen. This is the result of normal ossification of surrounding cartilage causing the bone to grow around the islands of abnormal cartilage “toward” the epiphyseal plate. The normal columnar arrangement of the chondrocytes is absent in these thickenings. Vascularization of the base of these metaphyseal islands is thought to fail to occur as it does in normal ossification. This causes ischemic necrosis of the tips of the cartilage islands.

In the joint cartilage this can progress to a yellowing of the overlying cartilage at four to five months of age. At six to eight months of age the joint surface visibly flattens. From six to twelve months of age fissures with hyperemic bases start to form in the joint cartilage. These fissures are not to be confused with synovial fossae which appear at five months of age in the semilunar notch of the ulna. If the fissures communicate with the necrotic cartilage space below or if cracks from the necrotic area reach the joint surface, synovium can then leak into that space causing an inflammatory response. This inflammatory response loosens the overlying cartilage further. It is thought that if a stress is placed directly on this area of loosened cartilage it will tear, forming a cartilage flap. This cartilage flap is termed osteochondritis dessicans. This lesion causes the severe
lameness. The osteochondritis dessicans lesion can only heal if the cartilage flap tears loose so the defect can fill with fibrocartilage. The loose cartilage piece eventually is resorbed. The pig will become sound if there are not other secondary changes such as erosion of the opposite joint cartilage, osteoarthritis, osteophytes, or even ankylosis. These joint cartilage lesions occur on the medial or lateral femoral condyles, central caudal humeral head, and the medial humeral condyle.1,2,3

If the focal disruption of cartilage ossification occurs in a growth plate, irregular widening of the growth plate will be seen both grossly and on radiographs. This may degenerate further causing premature closing of the growth plate, and shortening and angulation of the bone. These changes are seen most often in the growth plates of the femoral head, distal femur, humeral head, and distal ulna.1,2,3 If the ulna is shortened the radius will be bowed. If necrosis of the tips of the cartilage islands occurs, epiphyseolysis or fracturing along the growth plate can occur. This is most often seen in the growth plate of the femoral head.

Case Report

Two farms north of Marion, Iowa operated by the same farmer were visited by Dr. Terry Mangold.

Three groups of gilts totaling approximately 200 head six to ten months of age were observed. Both groups were progeny of a Duroc-Yorkshire cross.

Approximately 35% of the ten month old bred gilts were showing some reluctance to move, exhibited a stiff gait and very straight pasterns. 50% of the six month old gilts showed more severe lameness with some animals walking on their knees. In both groups swelling in the carpal area was noted.

Upon initial examination it was noted none of the animals were febrile or depressed and that they were still eating normally. This, coupled with the fact that the animals had been vaccinated for erysipelas, helped place erysipelas as a low probability cause. Also, the numbers involved and the older age of the pigs did not support a diagnosis of streptococcal or Corynebacterium pyogenes purulent arthritis.

Since there was such a high incidence of lameness it was decided to have the gilts' ration analyzed to rule out the possibility of a nutritional imbalance. The results showed there was a slight calcium: phosphorus imbalance but not enough to produce a nutritionally related lameness.

The farmer said the gilts were exceptionally fast growing.

At this time osteochondrosis lameness was tentatively diagnosed. Mycoplasma hyosynoviae was also considered, but since mycoplasma infection doesn't generally produce carpal swelling it was doubtful that it was the sole cause of the lameness.

Blood samples were taken for paired serum analysis for Mycoplasma hyosynoviae.

Radiographs of the carpal joints of two lame gilts, one from each age group, were taken. Examination of the radiographs revealed widening and irregularity of the ulnar growth plates.

The same gilts were slaughtered and the humeri, ulnas, and femurs were recovered and examined. A spectrum of lesions identical to those seen in osteochondrosis was found. They ranged from a flattened yellow area on a humeral head to osteochondritis dessicans, or a cartilage flap, on a medial femoral condyle.

A second set of blood samples were drawn two weeks after the first samples. Comparing the titers taken from both lame and sound pigs, two out of seven hogs had significant titers, one to M. hyosynoviae and the other to M. hyorhinis on the first sampling. When the second sample was taken, six of the seven hogs showed significant titers to M. hyosynoviae or M. hyorhinis. One of the two pigs that had a titer on the first sampling showed a rise from its initial level.

These results indicate the herd had undergone joint changes comparable to those seen in the osteochondrosis leg weakness syndrome with a concurrent infection caused by M. hyosynoviae and/or M. hyorhinis. Since the antigen composition of the different strains of M. hyosynoviae and M. hyorhinis are not always exclusive, infection with just one could have caused the titer rises seen for both. The mycoplasma infection was considered to be secondary since the titer rises occurred after severe clinical lameness was observed.

Tentative diagnosis of osteochondrosis antemortem must be done by radiographing the distal ulnar growth plate (see Fig. 1).
proximal ulnar growth plate or other areas exhibiting lameness, along with ruling out other possible causes of lameness. It must be noted that ulnar growth plate lesions can be found without concurrent lameness. Therefore definitive diagnosis radiographically is impossible unless you observe island shaped bony defects in the stifle or elbow joints (see Fig. 2).

Definitive diagnosis of osteochondrosis can only be made post mortem by observing the aforementioned joints or growth plates for changes of osteochondrosis. These findings must be coupled with the exclusion of other causes of lameness.

**Treatment and Prevention**

Osteochondrosis, as stated earlier, is considered to be strongly related to genetics with production of clinical lameness depending on such environmental factors as rapid growth rate and various types of joint stress. The only treatment possible is rest in a stress free environment. Even then recovery depends upon the absence of secondary degenerative joint changes.

Prevention is the best course, although it is not absolute. Short term measures to decrease clinical occurrence of osteochondrosis in swine directly related to other swine known to have osteochondrosis include:

1. Restricted feeding of potential breeding stock. This has the obvious disadvantage of not being able to use growth rate in the selection of breeding stock.
2. Routinely allow hogs out on a lot for exercise.
3. Keep to a minimum transportation of potential breeding stock.
4. Decrease the number of hogs per pen and provide nonslip flooring.
5. Make sure hooves are kept healthy.
6. Keep adequate levels of vitamins and minerals in the feed.

Long term breeding program improvement measures include:

1. Select stringently from a large number of potential breeding stock.
2. Select pigs that don't develop osteochondrosis after being fed ad lib.
3. Use good individuals as breeding stock as long as possible.

Osteochondrosis can occur in large numbers of related pigs thus making it a severe economic problem for seed stock or purebred breeders. Its occurrence and therefore importance is likely to increase in the coming years until more people become aware of the problem and attempt to eliminate the predisposing causes, both environmental and genetic.

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**Bibliography**

Figure 2: Note the condylar articular cartilage defect at the arrow.

The staff of the ISU Veterinarian would like to thank the Southern Minnesota—Northern Iowa Veterinary Medical Society on behalf of the student chapter for their generous contribution of $100 in support of our publication. The money will help defray printing costs for this year. The society also contributed the same amount last year.

Errata

The ISU Veterinarian would like to apologize for a mistaken identity on page 138 of volume 41, no. 3 (Fall 1979). The Norden Distinguished Teacher Award was presented to Dr. Brian L. Hill as correctly reported in the article. Unfortunately, the picture inserted is of Dr. Phillip T. Pearson, Dean of the Veterinary College who is presenting the plaque to Dr. Hill. The entire picture is shown above to avoid confusion.

A second error occurred on page 115 of the same issue. The author credit at the bottom of the left column should acknowledge Mr. Wilmot as a third year student, not Doctor.