Clinical and postmortem examination of sows culled for lameness

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Sow lameness can be an important cause of economic loss for pig producers. The cost associated with purchasing additional gilts to replace lame sows can contribute significantly to the overall production cost of producing a weaned pig. The number of pigs produced per litter from first parity animals is less than older sows; therefore, premature culling of sows may reduce the number of pigs weaned per litter (1).

Veterinarians are frequently requested to investigate lameness problems in breeding-age swine. A herd study of lameness can be difficult. The average yearly culling rate due to lameness is approximately five percent, although in some herds the incidence may be five times this level (1,2). An investigation may require several months to allow time for the examination of a sufficient number of lame sows to determine the major cause of lameness on a herd basis.

Clinical examinations of the musculoskeletal system of swine is of limited usefulness, due to the uncooperative nature of the patient, and a postmortem examination may be necessary to differentiate the various causes of lameness in sows.

The differential diagnosis of lameness in breeding-age swine includes footrot, leg injuries, osteochondrosis (epiphysiolysis, osteochondritis dissecans), leg weakness, osteomalacia, fractures, and arthritis (3–5). Outbreaks of footrot occur in breeding herds, where up to 100% of the sows can be affected (4). In several studies, osteochondrosis, which is a generalized dyschondroplasia of growing pigs, was found to be the most common cause of lameness in culled breeding-stock animals (6–8). The definitive diagnosis of osteochondrosis is made by histological examination of affected joints (3,9,10).

The purposes of this study were to diagnose the causes of lameness in sows that were culled for lameness and to determine if the gross postmortem lesions found in these sows were also present in sows culled for reasons other than lameness. The relationships among the parity of the sows, the clinical lameness scores, and the cause of lameness were determined.

The annual culling rate due to lameness in one breeding stock herd and the 16 farms stocked by it ranged from 0% to 38% (2). The owners of these 17 farms were asked to submit all of the sows culled for lameness over a six-month period to the Ontario Veterinary College. Fifty sows from ten farms were included in this study. Five farms did not cull any sows for lameness, and two farms chose not to participate. Eleven sows that were culled for reasons other than lameness and matched by farm of origin, breed, and parity with the lame sows were also included. All sows were examined clinically and at postmortem to determine causes of lameness.

The sows examined clinically were graded zero to nine as follows: 0 — normal gait; 1, 2, 3 — stiff gait; mild, moderate, or severe; 4, 5, 6 — lame; mild, moderate, or severe; 7 — requires assistance to stand and then can walk; 8 — can stand with assistance but then falls; 9 — cannot stand with assistance.

The sows' hip, elbow, and stifle joints and feet were examined for gross postmortem lesions. A diagnosis of osteochondrosis, arthrosis, infectious arthritis, foot lameness or other was made for each sow after the clinical and gross postmortem examinations. If a specific diagnosis of the cause of lameness was made but additional lesions were found, these were also recorded. All evaluations were done by the first author.

For a diagnosis of infectious arthritis to be made, the presence of either a purulent or a mucopurulent discharge in the joint space was necessary. A diagnosis of foot lameness was based on the presence of torn dewclaws with secondary soft tissue swelling, hoof cracks, or sole ulcers. The lesions of arthrosis included fibrillation of the joint cartilage and ulceration of the articular surface on the central weight bearing region of the medial condyle of the femur or humerus and osteophyte production. The lesions of osteochondrosis were either osteochondritis dissecans or epiphysiolysis of the medial condyle of the femur or humerus, or the head of the femur.

The central weight bearing regions of the medial condyle of the femur and/or humerus were examined histologically in 48 of the 61 sows in the study. The bones of the other 13 sows were inadvertently discarded. Sections of cartilage and bone, approximately 1.5 mm by 1 mm by 4 mm, were fixed and decalcified for one month in a solution of formic acid (2.4 to 3.0 M) and formaldehyde (1 M) (CAL EXII, Fisher Diagnostics, Orangeburg, New York, USA). The sections were dehydrated in alcohol solutions, graded from 70% to 100%, and then embedded in paraffin, cut in 5 μm sections, and stained with hematoxylin and eosin. The sections were numbered and examined in a blind fashion for lesions consistent with osteochondrosis. These lesions were foci of metaphyseal dysplasia, eosinophilic streaks, intracartilaginous cavities, and protrusions of cartilage into the subchondral bone (3,6,9).

Microscopic lesions consistent with osteochondrosis were seen in 45 of the 48 sows that were examined.

Can Vet J 1993; 34: 555–556

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This work was supported by a grant from the Ontario Pork Producers Marketing Board.

Table 1. The causes of lameness in 50 sows culled for lameness as determined by clinical and gross postmortem examination

Diagnosis	Number of sows	
	Primary cause	Additional lesions[a]
Osteochondrosis	17	4
Arthrosis	6	4
Infectious arthritis	11	2
Foot lesions	10	11
Other[b]	6	

[a]These sows had another diagnosis for the lameness but also had these lesions

[b]This group included one sow with a fractured femur, two sows with systemic diseases, and three sows whose lameness was not diagnosed

This percentage was comparable with that observed in Reiland's study of culled breeding-stock animals (6), where lesions of osteochondrosis or arthrosis were found in 96.3% of animals less than 18 months of age and 81.8% of animals more than 18 months of age. The diagnosis of either osteochondrosis or arthrosis was made after the clinical and gross postmortem examinations in 31 of these 45 sows. The three sows that did not have microscopic lesions of osteochondrosis also did not have gross postmortem lesions of osteochondrosis or arthrosis.

The average parity of sows and the average clinical lameness scores by cause of lameness were compared using Student's *t*-test.

The clinical and gross postmortem examinations of the 50 sows that were culled for lameness indicated that 17 had osteochondrosis, 6 had arthrosis, 11 had infectious arthritis, and 10 had foot problems (Table 1). The additional lesions found in sows were infectious arthritis, foot abrasions, and mild lesions of osteochondrosis or arthrosis (Table 1).

Of the 11 sows that were culled for reasons other than lameness, one was clinically lame due to arthrosis. The other ten were not lame, but two had foot lesions, and six had mild to severe lesions of arthrosis or osteochondrosis.

The combination of a clinical examination and a gross postmortem examination was determined to be a good method of diagnosing the cause of lameness for individual lame sows. In almost all cases, the primary cause of lameness was readily evident. However, several sows did present with more than one lesion, thus posing the question of which of the lesions was the most important cause of lameness (Table 1). Because gross postmortem lesions of osteochondrosis can be found in sows that are clinically sound, it should only be identified as the cause of lameness if the lesions are found in a joint of the clinically affected leg and only after other causes of lameness have been ruled out.

Osteochondrosis was the most important cause of culling due to lameness among the lame sows examined in the study. Lesions of osteochondrosis were bilateral and symmetrical and present in several joints in the same animal. The parities of the culled sows ranged from zero to nine with a mean of 2.67 (±2.45). The average parity of the sows that were culled with osteochondrosis was 1.3 (±2.9). This was less than 3.4 (±2.5), which was the average parity for sows culled with a lameness other than osteochondrosis ($p < 0.05$). This relationship would be expected, because clinical signs of osteochondrosis are concentrated in animals between 6 and 15 months of age (6,8). This culling of gilts and young sows is particularly costly, because they are removed from the herd before they have reached their peak level of performance (1).

The second two important causes were infectious arthritis and foot lesions. The latter included footrot, overgrown claws, or torn dewclaws. The average parity of the sows that were culled with foot lameness was 4.4 (±2.5), which was larger than the average parity of 2.4 (±2.5) for the sows culled for other causes of lameness ($p < 0.05$). Hence, older sows suffered from foot trouble more frequently than younger sows.

The clinical lameness grades of the sows ranged from zero to nine with a mean of 5.13 (±2.7). This average did not differ by cause of lameness.

Two of the ten farms each submitted 14 sows. In these herds, the causes of lameness were equally distributed among osteochondrosis, foot lameness, and arthritis. Presumably, in such herds where several causes of lameness occur, the examination of only one or two animals on a particular herd visit could be quite misleading. Therefore, it would appear from this study that a clinical and postmortem examination of a representative sample of animals over a period of six months to a year could be warranted as a herd diagnostic procedure.

Acknowledgments

We thank the pork producers for participating in the study. CVJ

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