A 210-kg Duroc boar was donated by a Saskatchewan commercial herd on November 3, 2005, to the Western College of Veterinary Medicine for evaluation of a grade II–III left front leg lameness for a suspected case of osteochondrosis desicans (OCD). The boar was treated with isoflupredone acetate sterile injectable suspension (PreDef 2X; Pfizer Animal Health, Kirkland, Quebec), 0.04 mg/kg bodyweight (BW), IM, q24h for 3 d to reduce any musculoskeletal inflammation. The boar’s lameness resolved temporarily, but returned 8 d later. The boar was treated with ketoprofen injectable solution (Anafen; Merial, Victoriaville, Quebec), 3 mg/kg BW, IM, q24h for 3 d. The usage of ketoprofen in this context in swine is extra-label, since it is being used as an analgesic for pain associated with lameness in pigs instead of in cats, dogs, cattle, and horses — as per label. Ketoprofen therapy was continued, 3 mg/kg BW, IM, q48h for 5 wk and the lameness resolved completely. However, the boar became recumbent the day following the withdrawal of the treatment and was euthanized by captive bolt on January 4, 2006.

Necropsy revealed several lesions, including a mild hyperkeratosis of the pars oesophagus, bilateral front leg cartilaginous degeneration, and bilateral erosion of the medial humeral condyles, with secondary fissure formation and fibrosis (Figure 1). A focal erosion was present in the articular cartilage of the right femoral head. There was marked synovial villous hypertrophy of the left shoulder joint and mild synovial villous hypertrophy of both stifle joints. Pulmonary parenchyma was congested and subpleural hemorrhage was identified; both were thought to be sequelae to the euthanasia. Histopathological examination of the left front radiohumeral joint revealed hypertrophied chondrocytes interspersed by large irregular areas of fibrous connective tissue as eosinophilic granular streaks — a pattern typical of osteochondrosis.

Osteochondrosis is a focal failure of endochondral ossification in physeal and epiphyseal growth cartilage, and is more appropriately referred to as dychondroplasia. Macroscopically, the major sites of growth cartilage dysplasia in the epiphyses are the medial humeral and femoral condyles, and the intervertebral synovial joints, whereas in the physeal, they are in the distal parts

Abstract — A 2-year-old, 210-kg, Duroc boar manifested with a grade II–III left front lameness. The boar was treated systemically with isolfupredone acetate and a 5-week course of ketoprofen. The lameness resolved and the ketoprofen was discontinued; however, the lameness returned and the boar was euthanized humanely. Postmortem examination was consistent with osteochondrosis desicans.

Résumé — Traitement palliative de l’ostéochondrite disséquante chez un verrat Duroc. Un verrat Duroc de deux ans pesez 210 kg a manifesté une claudication de la catégorie II–III de ça jambe avant gauche. Le verrat a été traité avec de l’acétate d’isoflupredone suivi par un cours de 5 semaines de ketoprofen. La claudication a résolue et la ketoprofen a été cessé, seulement d’avoir la claudication retourner après la cessation de la thérapie. Le verrat était euthanized avec humanité sous peu ensuite. Examination post-mortem à vérifié l’osteochondrosis desicans.

(Traduit par l’auteur)
of the ulnas and femurs and costochondral junctions. Lesions are more common among the medial than the lateral physeal condyles for both the humerus and femur (1). Lesions are almost always bilateral. The lesions manifest clinically as lameness and depression, without a concurrent febrile state.

Dyschondroplasia, which can cause a weight bearing or non-weight bearing lameness, has an insidious onset and often results in a pig either refusing to move or walking with a swaying gait. A shortened stride and partial flexion of the affected limbs occur and may be due to pain. Atypical posturing is thought to accommodate for this. Pigs with extensive lesions in the humeroulnar joints are reported to either walk on the tips of their hooves or crawl, using the dorsal aspect of their carpii to propel themselves (2). Apart from being a serious animal welfare concern, the lameness results in significant economic loss related to the stipulation that severely lame pigs cannot be shipped to market or slaughter. Fortunately, there exists clinical evidence to the stipulation that severely lame pigs cannot be shipped to market or slaughter. Fortunately, there exists clinical evidence to the stipulation that severely lame pigs cannot be shipped to market or slaughter. Fortunately, there exists clinical evidence to the stipulation that severely lame pigs cannot be shipped to market or slaughter. Fortunately, there exists clinical evidence to the stipulation that severely lame pigs cannot be shipped to market or slaughter.

The recommended treatment for dyschondroplasia in swine is glucocorticoids, such as isoﬂupredone acetate. Several studies have shown that corticosteroids possess osteolytic properties in some species (5,6). However, glucocorticoids may also possess the potential to alter the healing capacity of pigs affected with OCD. Dexamethasone administration in piglets (7) induces an overall reduction in bone turnover. Osteocalcin (OC) is an indicator of osteoblastic activity, and tartrate-resistant acid phosphatase (TRAP) is an indicator of osteoclastic activity. Their relative activities provide a quantitative measurement of bone turnover. Isoﬂupredone acetate reduces OC synthesis by mature osteoblasts, as well as lowering TRAP activity (8). The drop in OC synthesis is theorized to occur via 3 different mechanisms: OC reduction by activation of osteoblastic glucocorticoid receptors decreases its transcription; the inhibition of type I collagen mRNA decreases the matrix synthesis vital to osteogenesis; a decrease in IGF-1 secretion, which accentuates bone catabolism and increases osteoclast apoptosis, following glucocorticoid administration (9,10).

Extended periods of rest may be superior to medical therapy for young pigs with mild dyschondroplasia, as the lesions may heal (2). Dyschondroplastic lesions are less common than in pigs older than 1.5 y than in those younger than 1.5 y due to spontaneous repair of OCD lesions (1). The highest occurrence of dyschondroplastic lesions coincides with the period of puberty. If the lesions acquired during puberty are superficial and do not affect the articular surface, they may resolve spontaneously. Conversely, lesions acquired during puberty that form cracks and fissures in the articular surface do not heal and often progress to arthritis (1). Repair was observed more often in pigs housed on pasture following development of dyschondroplasia (2).

The spontaneous repair of a dyschondroplastic joint in a pig is dependent upon a balance between reduction of excessive mechanical joint forces with sufficient weight bearing to prevent muscular atrophy, yet promote healing. The recommended conservative management involves a 6-week period of rest within a $8 \times 4.6$ m pen that provides secure footing (11). The prevention of mounting by cohorts, and the provision of good traction and access to some limited exercise may promote healing (2,12). A minimum of 6 wk rest is recommended, since fighting and copulation may induce separation of the joint epiphysis (3,4). Other management techniques employed to address the development of OCD lesions include selective breeding for better joint conformation in particular swine breeds. The Yorkshire breed has a lower incidence of OCD compared with Norwegian Landrace pigs, mostly due to their skeletal conformation (13). In contrast, crossing wild boar with Swedish Yorkshire pigs surprisingly did not alter the heritability of OCD (12).

The treatment of the boar in this case report is typical of nonresponsive osteochondrosis dessicans. Treatment with glucocorticoids initially may have contributed to the progression of the dyschondroplastic lesion. Clinical resolution of the lameness was achieved with systemic NSAID’s.

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References