OSTEOCHONDROSIS IN PIGS FROM 4-24 WEEKS OLD.

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Osteochondrosis (OC) is a dyschondroplasia occurring in epiphyseal and physeal growth cartilages (4,6,2,7). Although it is a generalized disease, foci of abnormal cartilage develop and tend to be bilaterally symmetrical (2,7). Degenerative joint disease (osteoarthrosis, arthrosis) at predilection sites for OC is considered secondary to OC in pigs of 4 months and older and development of arthrosis probably coincides with onset of lameness (5,7). Reiland (1978) described OC in 4-5 months-old pigs and Grondalen (1974) found lesions in pigs weighing less than 50 kg (7,2). In a microangiographic and histologic study, changes were noted in physes of 3 days and 3 weeks-old piglets, but limited material was available (1).

Objectives of the following study were to determine the age at which lesions first developed and to describe their pathogenesis.

Sixty mixed crossbred and purebred boars were obtained from one farm and randomly assigned to 6 groups of 10 so that the chronological development of OC could be studied.

Beginning at 4 weeks of age, 10 animals were killed and dissected. Subsequently 10 pigs at 4 weekly age increments up to 24 weeks were used.

Long bones were dissected out and serial slab sections, 5 mm thick, were cut from the proximal and distal ends of selected bones such that each segment included the articular epiphyseal (A-E) complex, epiphysis, metaphysis and part of the diaphysis. Slabs were radiographed, decalcified and processed for light microscopy.

Lesions were found in the physes of 25 to 30 days-old pigs, whereas in the A-E complex the earliest lesions were detected at 111 days. For all age groups lesions were seen most frequently in the proximal and distal femur and humerus. They were also common in one or more costo-chondral junctions. In A-E complexes distal femoral, distal humeral and tibial tarsal bone lesions were most frequent. Changes were typical of those described previously for OC (2,7). Characteristic changes in the physis consisted of focal zones of failure of endochondral ossification that left cartilagenous wedges or trapezoids projecting from the physis into the metaphysis. These consisted mainly of rows of hypertrophied chondrocytes in apparently normal matrix (H & E section). In some lesions eosinophilic streaks, possibly remnants of cartilage canals, were present in the zones of widened physis, but these streaks were frequently in areas of physis regarded as normal.

Eosinophilic granular material similar to the streaks was sometimes at the chondro-osseous interface. Lesions that were considered to be more advanced had peripheral areas with pale eosinophilic matrix, unmasked fibrils and shrunken, red chondrocytes, but morphologically normal chondrocytes were sometimes present. Fibrils stained blue with picromallory stain stain consistent with collagen or reticulin. Focal areas with secondary spongiosum, instead of the usual primary spongiosum, abutting the zone of hypertrophied chondrocytes, were in most physeal lesions. Giant cells and clusters of macrophages or osteoblasts were frequently at the chondro-osseous junction in lesions.

Most A-E complex lesions involved the epiphyseal zone of chondrocyte proliferation and hypertrophy. They consisted of bands or irregular triangular and rectangular areas of pale eosinophilic, fibrillar cartilage matrix containing shrunken, red chondrocytes. Nests of chondrocytes were often in normal matrix next to the lesions and were occasionally within the lesions. Osteoarthrosis was not observed.

Most histologic lesions corresponded in size and position with areas of radiolucency on radiographs of the bone slabs.

Radiography on live animals was not a useful diagnostic indicator of the extent or number of lesions in the physis or articular epiphyseal cartilage, but on careful scrutiny of chronologic radiographs most of the lesions were seen. Some developed and apparently resolved by 5-6 months.

As lesions occurred in the youngest pigs examined, pathogenesis of lesions is speculative. The smallest areas with simplest changes consisted of wedges of persistent hypertrophied cells projecting into primary spongiosum. In older lesions (based on lesion length and bone growth during the preceding month) changes consistent with chondrocyte death and matrix degeneration were observed near the tip of the lesion. Apparently normal endochondral ossification occurred in the center of a few lesions.

OC has been associated with rapid growth at puberty (7) and overloading of growth cartilage (3,8), but it is apparent from this study that neither puberty nor a heavy load (normal body growth or artificial loading in maturing swine) were necessary for onset of lesions.

The authors concluded that OC developed in the physis as early as 25 days of age, but 111 days was the earliest age at which the A-E complex was involved. Further studies will be directed at fetuses, neonatal pigs and pigs up to 1 month old.

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