

TIBIAL DYSCHONDROPLASIA

IN DOMESTIC POULTRY

SLIDE STUDY SET #6

A CONTINUING EDUCATION PROGRAM PREPARED

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INTRODUCTION

Tibial dyschondroplasia characterized by an abnormal mass of cartilage in the proximal tibiotarsus has been described in commercial broiler chickens in many countries of the world (1,2,6,7,8). A similar lesion has been described in ducks (9) and in turkeys in which species it has been called osteochondrodystrophy (5,9). An identical lesion was described as a cartilage abnormality in four week old meat type chickens fed a purified diet by Leach and Nesheim (3) who demonstrated that the defect was influenced by genotype and diet. In spite of extensive studies no specific nutritional factor was identified by addition of natural ingredients to the purified diet and changes in the acid-base balance of the diet altered the incidence and severity of the defect (4).

CLINICAL SIGNS

Severely affected birds tend to squat and are often reluctant to move. These birds do not stand properly and have an awkward gait which is associated with an enlargement of the proximal tibia and marked outward bowing in an anterior and lateral direction of this bone. This deformity is often most noticeable at processing and can result in downgrading of broiler chickens. It should be noted that many birds with less severe lesions have a normal gait and no obvious external bone deformation. Subclinical tibial dyschondroplasia has been reported to be widespread in broiler chicken flock (1,7). The defect is readily seen on radiographs.

SLIDE 1. 9-week-old broiler chicken. The bird had severe tibial dyschondroplasia with characteristic bowing of the tibia.

SLIDE 2. 8-week-old broiler chicken carcass. Swelling and bowing at the proximal end of the tibia due to dyschondroplasia is present and will result in downgrading of the carcass.

SLIDE 3. A bowed leg from an eight-week-old broiler chicken with tibial dyschondroplasia compared to a leg from a normal chicken.

SLIDE 4 AND 5. Lateral and anterior posterior radiographs of tibias from eight-week-old broiler chickens. These radiographs compare a leg with severe tibial dyschondroplasia with a normal leg. The abnormal leg has a well-defined radiolucent area within the proximal metaphysis of the tibia. Proximal to this area the normal line of density underlying the cartilaginous epiphysis is

missing. The tibia is bowed anteriorly and laterally.

SLIDE 6. Oblique radiograph of the tibia of an eight-week-old broiler chicken. The tibia has a minor defect restricted to the medial posterior portion of the metaphysis. This radiograph was taken with the bird alive. It is possible to screen live birds for tibial dyschondroplasia using radiography.

GROSS PATHOLOGY

The defect is most readily observed in a routine necropsy by an oblique cut through the posterior-medial aspect of the femoral-tibial joint. Minor lesions are generally restricted to small tongues of abnormal cartilage in the posterior medial aspect of the metaphysis of the proximal tibia. More severe cartilaginous lesions will occupy the whole metaphysis of the proximal tibia and are generally associated with bulging of the metaphysis and a lack of cortical bone around the defect. When bending of the tibia occurs the cortical bone is very thin on the convex surface and thickened on the concave surface. Pathological fractures through the tibia below the cartilaginous mass have been observed. The lesion is generally bilateral affecting both legs. In approximately 10 percent of affected birds similar abnormal cartilage will be found in the proximal metatarsi but no abnormalities have been observed associated with any other growth plates. The restriction of the lesion to these two locations, normal bone strength except adjacent to the lesion and normal parathyroid glands are useful gross criteria to differentiate this

condition from rickets.

SLIDE 7. Sagittal sections of tibias from eight-week-old broiler chickens. The tibia on the right has a tongue of abnormal cartilage in the posterior medial portion of the metaphysis. This tibia is not bowed and the bird was clinically normal. The tibia on the left has a mass of abnormal cartilage in the metaphysis. The tibia is bent and the bird was reluctant to move.

SLIDE 8. Frontal sections of a tibia and a metatarsus from an eight-week-old broiler chicken. Both tibia and metatarsus have abnormal masses of cartilage in the proximal metaphysis and both are bowed.

SLIDE 9. Sagittal sections of tibias from two, twelve-week-old broiler type chickens. Severe bending associated with pathological fractures is present below the abnormal cartilage in the proximal shaft.

HISTOPATHOLOGY

In histological sections of abnormal growth plates the zone of proliferation appears normal but may be thickened. The zone of prehypertrophy where the chondrocytes start to become round in shape, the lacunae enlarge and an increase in matrix occurs also appears normal but complete hypertrophy does not occur. This interruption in hypertrophy of chondrocytes is associated with a lack of calcification of the matrix and a lack of vascular invasion from the metaphysis. Without these processes no removal of cartilage occurs and prehypertrophic cartilage persists. In the center of this persistent cartilage many of the lacunae are empty

and others contain small shrunken chondrocytes with eosinophilic granular cytoplasm and pyknotic nuclei. At the distal end of this persistent cartilage there is no orderly vascular invasion or orderly arrangement of bony trabeculae.

SLIDE 10 AND 11. Histological sections of the growth plate from a normal broiler chicken compared with the same from a broiler chicken with tibial dyschondroplasia. In slide 10 the zone of proliferation in the normal growth plate is underlain by a thin fainter staining zone of prehypertrophy only a few cells thick. Below the zone of prehypertrophy is the zone of hypertrophy slightly darker staining and irregularly invaded by vessels from the metaphysis. In the abnormal growth plate the zone of proliferation is slightly thicker and underlain by a fainter staining zone of prehypertrophy which appears to persist. There is no vascular invasion from the metaphysis. Slide 11 is a higher magnification of the zone of prehypertrophy. In the normal growth plate on the left the flattened cells in the zone of proliferation (at the top) enlarge and become round in shape as they progress through the zone of prehypertrophy. The lacunae also enlarge and are largest in the zone of hypertrophy where the thinning matrix between the lacunae becomes calcified and is invaded by blood vessels from the metaphysis. In the abnormal growth plate the chondrocytes enlarge and become round below the zone of proliferation but the lacunae never become as large as in a normal growth plate. The matrix is correspondingly more abundant and does not become calcified. No vascular invasion occurs.

SLIDE 12. Histological section from the center of the abnormal mass of cartilage from a bird with tibial dyschondroplasia. Cartilaginous matrix is abundant. Many lacunae are empty or contain very small shrunken eosinophilic chondrocytes.

SLIDE 13. Histological section from the distal end of the abnormal mass of cartilage from a bird with tibial dyschondroplasia. There is a lack of orderly vascular invasion or arrangement of the underlying bone trabeculae.

RESOLUTION OF THE DEFECT

The structure of the growth plate of affected birds will return to normal by 14-16 weeks of age. In some severely affected birds the abnormal cartilage will persist as a sequestrum surrounded by bone trabeculae in the diaphysis until at least 30 weeks of age.

SLIDE 14. Sagittal section of the tibia of a 12-week-old broiler type bird. The growth plate is almost normal but an abnormal mass of cartilage persists in the shaft of tibia only connected to the growth plate by a thin thread of cartilage in the posterior metaphysis.

SLIDE 15. Sagittal sections of tibias from two, 30-week-old broiler type chickens. The growth plates have closed in these birds but sequestra of cartilage persist in the shaft of the tibia as a sequela to tibial dyschondroplasia.

SLIDE 16. Radiographs of the tibias shown in slide 15. The areas of sclerosis in the shafts of these tibias corresponds to the

bone trabeculae surrounding the sequestra of cartilage.

SLIDE 17. Histological section of a persistent sequestrum of cartilage showing an enclosing network of bone trabeculae.

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