OSSIFICATION VARIANTS IN THE FEMORAL CONDYLES AND TROCHLEA ARE CAUSED BY SUBCLINICAL OCD IN CHILDREN

Cathy S. Carlson, DVM, PhD1; Kevin Shea, MD2; Ferenc Tóth, DVM, PhD3; Stina Ekman, DVM, PhD4; Bjornar Ytrehus, DVM, PhD5; Peter Cannamela, MD6; John Polousky, MD6; and Kristin Olstad, BVSc, PhD7

1University of Minnesota, Saint Paul, Minnesota, USA; 2St. Luke’s Sports Medicine, Boise, Idaho, USA; 3University of Minnesota, Minneapolis, Minnesota, USA; 4Swedish University of Agricultural Sciences, Uppsala, Sweden; 5Norweign Institute for Nature Research, Trondheim, Norway; 6Children's Health Andrews Institute, Plano, TX, USA; 7Norweign University of Life Sciences, As, Norway

Introduction
Over the last 50 years, the genesis of OCD lesions has been thought secondary to a primary bone necrosis origin. This theory has been challenged recently, as histological studies performed on specimens obtained from asymptomatic mammals, have revealed that ischemic necrosis of epiphyseal cartilage (rather than bone) is the precursor lesion of osteochondrosis dissecans (OCD) in several animal species. The earliest lesion identified histologically is osteochondrosis (OC) latens, in which the area of necrosis is confined to epiphyseal cartilage. As the ossification front advances, the area of necrosis causes a delay/failure in endochondral ossification that is visible radiographically and by CT and is termed OC manifesta. Juvenile osteochondritis dissecans (JOCD) in children has many similarities to OCD in animals; however, subclinical disease in children has not been studied due to the invasive nature of this work and the difficulty in obtaining appropriate cadaveric specimens. Lesions of OC latens are not recognized in children, and lesions resembling OC manifesta identified by CT are often considered to be normal ossification variants. Previous work by our group identified 32 suspected OC manifesta lesions in the medial and lateral femoral condyles (MFC and LFC) and the lateral trochlear ridge (LTR) in 14 cadaveric specimens from children ranging in age from 7 to 11 years.

Our aim was to determine if skeletally immature human knees contained histological evidence of delayed endochondral ossification occurring secondary to ischemic necrosis of epiphyseal cartilage.

METHODS
Eleven sites (4 MFC, 4 LFC, 3 LTR) containing suspect OC manifesta lesions identified on CT from five male children (age range 7-11 years) were decalcified in 10% EDTA. Areas corresponding to the CT lesions were trimmed into 3-mm thick slabs (n=2-4 slabs/site), processed into paraffin, sectioned at 5 µm, and stained with hematoxylin and eosin. All sections (n= 30 total) were examined by four veterinary pathologists/radiologists with extensive experience in the study of OC in pigs and horses.

RESULTS
All sites examined contained at least one section containing histological evidence of one or more areas of OC manifesta. Lesions were evidenced by focal failure of endochondral ossification accompanied by remnants of necrotic blood vessels, chondrocyte necrosis with matrix degeneration. Some lesions were accompanied by evidence of repair/healing response, including chondrocyte clusters, proliferating blood vessels, and fibrous connective tissue.

CONCLUSIONS
Similar to other mammals, human OCD lesions appear to develop as a primary defect secondary to avascular necrosis of epiphyseal cartilage, rather than primary avascular necrosis in bone. Our findings strongly support a common pathogenesis of OCD in humans and mammals. Historically, ossification variants have been considered a developmental anomaly with a benign clinical course. Our histologic findings suggest that ‘ossification variants’ may in fact be delayed presentations of a continuum of OCD lesion development that starts years earlier due to primary epiphyseal cartilage necrosis. Some of these ‘variants’ may progress to healing, but many may progress to frank ‘osteochondritis dissecans’, suggesting that these lesions that are identified on x-ray or with MRI sequences require close follow-up.

Peter C. Cannamela
Email: pcannamela@sandiego.edu

References