Osteochondritis Dissecans Development

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KEYWORDS

• OCD • Cartilage • Osteochondrosis • Osteochondritis dissecans • Pathogenesis • Development

KEY POINTS

- Blood supply to developing cartilage is very important because the articular-epiphyseal cartilage thickness far exceeds diffusion limits of synovial fluid.
- Anastomoses of cartilage canals and associated vessels along the ossification front are vulnerable to injury and may be an inciting factor in osteochondrosis (OC) development.
- Abnormal cartilage development and biomechanical trauma to cartilage canals or the osteochondral junction are etiologic factors for developing OC.
- Early OC lesions have the potential to heal intrinsically or develop into more advanced osteochondritis dissecans (OCD) lesions.
- Restricting exercise during the healing phase may help to prevent early OC lesions from becoming OCD flaps or fragments.

INTRODUCTION

Osteochondritis dissecans (OCD) and osteochondrosis (OC) are often interchangeably used to describe a defect in endochondral ossification leading to osteochondral fragmentation and/or cysts in joints of foals; however, they actually describe different stages of disease. OC represents the initial disease process, whereas OCD reflects secondary changes resulting in cartilage flap or osteochondral fragment formation (**Fig. 1**). This disease has been extensively studied in horses during the past 20 years, with more than 150 studies published during that time. The focus of this article is to provide a review of current knowledge of OCD development in horses, including normal cartilage development, early OC pathogenesis, and factors that result in healing or advancement to OCD fragments.

NORMAL CARTILAGE DEVELOPMENT

Articular cartilage covers the ends of long bones, creating a frictionless surface for normal movement of joints. In the postnatal period, dramatic changes occur in

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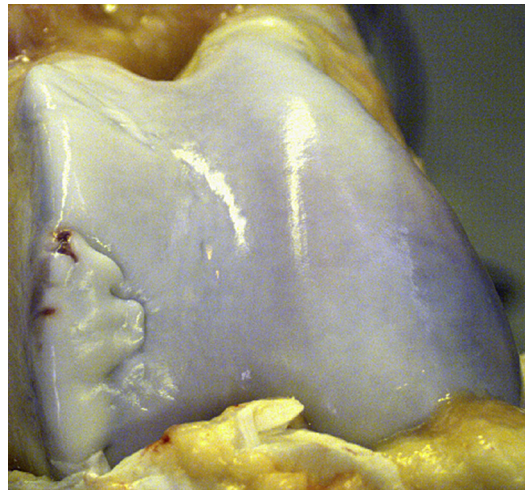

Fig. 1. OCD lesion on the lateral trochlear ridge of the distal femur in a yearling quarter horse filly.

articular cartilage and the underlying epiphyseal cartilage (so-called articular-epiphyseal [A-E] complex). The epiphyseal cartilage must be transformed into bone, whereas articular cartilage remains cartilage throughout the horse's life. Processes critical to normal cartilage development include endochondral ossification and cartilage canal formation and regression.

HOW DOES CARTILAGE TURN INTO BONE?

Endochondral Ossification Process

Endochondral ossification is an intricate process whereby a cartilage template is transformed into bone. This process occurs in physeal growth plates, A-E cartilage, and during fracture healing. Coordination of cell-to-cell signaling, cellular differentiation, and matrix modifications make this a highly orchestrated process.[1–3] Most of the literature describes the endochondral ossification process in growth plates but equally important is the transformation of A-E cartilage into subchondral bone.

In both the growth plate and A-E cartilage, several zones of cartilage cells (chondrocytes) are apparent, representing different stages of differentiation (**Fig. 2**). The resting zone contains round stem-like chondrocytes in a relatively quiescent state.[1] Resting chondrocytes produce parathyroid hormone-related protein (PTH-rP), which diffuses to nearby chondrocytes and helps to delay the differentiation of chondrocytes through its negative feedback on another protein called Indian hedgehog (Ihh).[3] Resting chondrocytes nearest the proliferative zone are recruited to enter the proliferative phase, where they develop a flattened appearance and line up in columns. As cells stop proliferation, they become rounder in appearance (prehypertrophic chondrocytes) and begin producing Ihh.[1] Ihh regulates cartilage differentiation and stimulates production of PTH-rP by resting chondrocytes. The classic feedback loop of Ihh and PTH-rP controls the pace of chondrocyte differentiation. Regulation of cartilage maturation is also coordinated by multiple signaling pathways including Wnts, bone morphogenetic proteins, retinoic acid, fibroblast growth factors, and many others.[2,4–6] Following the prehypertrophic phase, chondrocytes enter the hypertrophic phase, which is characterized by cytoplasmic enlargement and secretion