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Review

Articular osteochondrosis: a comparison of naturally-occurring human and animal disease

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SUMMARY

Background: Osteochondrosis (OC) is a common developmental orthopedic disease affecting both humans and animals. Despite increasing recognition of this disease among children and adolescents, its pathogenesis is incompletely understood because clinical signs are often not apparent until lesions have progressed to end-stage, and examination of cadaveric early lesions is not feasible. In contrast, both naturally-occurring and surgically-induced animal models of disease have been extensively studied, most notably in horses and swine, species in which OC is recognized to have profound health and economic implications. The potential for a translational model of human OC has not been recognized in the existing human literature.

Objective: The purpose of this review is to highlight the similarities in signalment, predilection sites and clinical presentation of naturally-occurring OC in humans and animals and to propose a common pathogenesis for this condition across species.

Study design: Review.

Methods: The published human and veterinary literature for the various manifestations of OC was reviewed. Peer-reviewed original scientific articles and species-specific review articles accessible in PubMed (US National Library of Medicine) were eligible for inclusion.

Results: A broad range of similarities exists between OC affecting humans and animals, including predilection sites, clinical presentation, radiographic/MRI changes, and histological appearance of the end-stage lesion, suggesting a shared pathogenesis across species.

Conclusion: This proposed shared pathogenesis for OC between species implies that naturally-occurring and surgically-induced models of OC in animals may be useful in determining risk factors and for testing new diagnostic and therapeutic interventions that can be used in humans.

Introduction

Osteochondrosis (OC) is a developmental orthopedic disease characterized by clinical signs of joint pain, effusion, and dysfunction caused by the formation of clefts extending through the articular cartilage into the subchondral bone. Extensive studies evaluating the clinical aspects of this condition are available in both human and veterinary medicine; however, there is limited information available regarding the similarities and differences between OC in humans and animals.

The majority of studies aimed at describing the etiologic factors and pathogenesis of OC in humans focus on osteochondral fragments removed surgically from adolescents or adults presenting with clinical symptoms of OC[1]. By this time, the fragments have been present for months to years. Understandably, it is nearly impossible to determine the pathogenesis of the disease from examination of these end-stage tissues. Obtaining osteochondral samples from juvenile human cadavers is difficult, and currently

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there is no established method for screening asymptomatic children or adolescents for OC. Both of these factors have hampered the understanding of the pathogenesis of naturally-occurring human disease. In contrast, in the veterinary literature, OC is defined as a focal disturbance of endochondral ossification[2], the process by which a cartilage template ossifies in the appendicular skeleton of a growing individual. Extensive studies performed in young growing animals of several species have demonstrated early, developing lesions at predilection sites well before the age at which clinical disease manifests[3–5]. We believe that naturally-occurring and surgically-induced OC in animals may provide valuable translational models to help understand the etiology and pathogenesis of human disease. Our review, therefore, aims to highlight the similarities in signalment, predilection sites and clinical presentation of naturally-occurring OC in humans and animals, and by doing so, propose a common pathogenesis for this condition across species.

Disease terminology

Evaluation of the literature pertaining to OC is complicated by the variety of terminologies used. In 1887, König proposed the term "osteochondritis dissecans" for an underlying lesion in the joint cartilage facilitating formation of loose bodies in the absence of significant trauma[6]. Subsequent histological studies have not supported a primary inflammatory etiology for the condition, making "osteochondrosis" the more accurate term, as suggested by Howald in 1942[7,8]. However, the original phrase has persisted, and in fact, "osteochondrosis" and "osteochondritis" are often used interchangeably. In the clinical literature, when a fissure or fracture in the overlying articular cartilage is present, the condition is nearly universally referred to as osteochondritis dissecans (OCD), although osteochondrosis dissecans would be more appropriate. In the veterinary medical field, focal abnormalities of endochondral ossification involving the articular–epiphyseal cartilage complex (AECC) are referred to as OC (or OCD, as appropriate) regardless of anatomical location. Conversely, in the human literature, manifestations of OC at various anatomical sites are given different names (Table I). Additionally, the phrase "the osteochondroses" includes conditions affecting the AECC, the physis, and various apophyseal locations. This general phrase has also been used to describe diseases of primary osteonecrotic etiology, such as Legg–Calvé–Perthes disease[9]. The present article will specifically focus on articular manifestations of OC.

Clinical aspects of OC in humans and animals

Human OC is typically not recognized in children or adolescents until the onset of clinical symptoms, at which point the disease is advanced[10]. In many cases, a lag time of months to years may exist between the onset of symptoms and diagnosis of the disease[11]. OC diagnosed prior to the age at which physeal closure occurs is known as juvenile OC; however, lesions diagnosed in adulthood also most likely developed prior to physeal closure[12]. Common presenting clinical complaints include joint pain, especially with extreme flexion or extension, swelling, and catching or locking of the joint. These symptoms may be intermittent, especially early in the course of disease, and may be associated with athletic activity. Continuous or more severe symptoms may be indicative of a loose osteochondral fragment within the joint[12,13]. Bilateral disease is not uncommon, although clinical symptoms are typically worse in one joint than the other[14]. Diagnosis is typically made by radiologic and/or magnetic resonance imaging (MRI) examination of the affected joint. MRI more closely aligns with arthroscopic findings[15] and is also more sensitive for identification of subtle cartilage abnormalities (i.e., prior to formation of overt osteochondral fragments), suggesting that this may be the better imaging modality for OC, especially for early lesions[12]. The preferred initial treatment for OC when the articular surface is intact is non-surgical management, including a combination of non-steroidal anti-inflammatory drugs, physical therapy, and modification of activity, typically with some form of joint immobilization. If conservative therapy fails, or if partially or completely detached osteochondral fragments are present at the time of diagnosis, then surgical intervention via arthroscopy is pursued[12,14,16]. Although removal of the fragment/flap followed by debridement is most common, reattachment of large osteochondral flaps using internal fixation has also been described[17]. Lesions that are not treated adequately may lead to development of degenerative joint disease with long-term debilitative consequences for the individual; thus, early intervention is recommended[10,17].

In horses, asymptomatic OC is usually identified at an early age due to extensive radiographic screening aimed at facilitating sale of racehorses as yearlings (before 2 years of age). In more slowly-maturing breeds that usually do not undergo early radiographic screening, OC is most often identified after 3 years of age as clinical signs, including subtle lameness and joint effusion, develop after the commencement of regular training. This latter presentation is strikingly similar to that noted in cases of juvenile OC in humans, which most frequently affects young athletes and usually presents with poorly localized pain that is exacerbated with exercise[8,14,18]. In horses, OC lesions are most often treated with arthroscopic removal of loose fragments followed by debridement of the fragment bed with or without microfracture[19]. Although many horses go on to perform in their intended capacity after treatment, the prognosis for future athletic career following surgical debridement of OC lesions diminishes as the size of the lesion increases[20]. Novel treatment modalities attempting to salvage and reattach large osteochondral flaps have recently been introduced to address this concern[19].

In commercially bred pigs, OC is considered to be an important cause of lameness with profound economic implications[5,21]. Clinical signs consistent with OC have been associated with decreased

Table I
Disease names for manifestations of OC at specific anatomical locations as reported in the human literature. For comparison, location of predilection sites in pigs and horses is also presented

Disease name (human)	Location	Pig	Horse
Articular			
Theimann's disease	Proximal and distal interphalangeal joints (fingers and toes)		
Panner's disease	Elbow (humeral capitellum)	X	
OCD	Elbow (humeral capitellum), knee (medial or lateral femoral condyle), ankle (medial talus)	X	X
Freiberg's disease	Metatarsophalangeal joint (head of second metatarsal)		X
Non-articular/apophyseal			
Sinding-Larsen-Johansson disease	Knee (inferior pole of patella)		X
Osgood–Schlatter disease	Knee (proximal tibia)		
Sever's disease	Ankle (proximal calcaneus)		
Köhler's disease	Ankle (tarsal navicular bone)		
Iselin disease	Ankle (base of fifth metatarsal)		
Medial epicondyle apophysitis	Elbow (medial epicondyle)		
Physeal			
Blount disease (tibia vara)	Proximal tibial physis		
Scheuermann's Disease	Vertebrae	X	X

Human[9,12,16,99].
Pig[21].
Horse[91,100].