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Osteochondral Lesion in Adults and Children

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Mini Review

Osteochondral lesions (OCL) are thought to be associated with osteochondral fractures within the area of avascular necrosis of the subchondral bone and the cartilage above it. Many possible causes have been suggested, including recurrent microtrauma and ischemia, as well as genetic effects. Acute trauma and abnormal ossification are also involved [1]. Of all these proposed causes, recurrent trauma is considered to be the major crime in most cases. For this reason, the term "osteochondritis dissecans" has become less popular, and in many cases "osteochondritis dissecans" or "osteochondritis dissecans" is the preferred term. Osteochondral lesions should be distinguished from osteochondral fractures, dysfunction and stress fractures, and subchondral cysts. Osteochondral fractures associated with appropriate medical history and MRI findings such as acute fracture surface, large joint effusion, and extensive bone marrow edema [2].

The chondral layer being the most superficial layer of the osteochondral unit is subjected to a greater number of force vectors. As with most of the osteochondral unit, the chondral layer must withstand compressive forces but in addition to this, it must also counter friction and shear forces generated cyclically during normal joint articulation. Chondral tissue is best described as being biphasic as it demonstrated features of both a fluid and solid phase substance [3]. Water and inorganic ions such as sodium, potassium, calcium and chloride are responsible for its fluid phase and ECM for its solid phase. With the presence of negatively charged proteoglycans and the porous permeable ECM interstitial fluids can move in and out of the tissue with the increasing and decreasing joint forces. This summarizes the flow-dependent mechanism which allows for the chondral tissue to exhibit a biphasic viscoelastic behavior. The flow independent mechanism is brought about by the viscoelastic behavior of the collagen-proteoglycan matrix. As the forces increase on the chondral tissue the tissue becomes stiffer and more resistant to the forces applied due to these mechanisms [4].

Dysfunction and stress fractures are characterized by diffuse bone marrow edema, usually without associated trauma, elderly patients with osteoporosis, usually associated ligament or meniscal pathology, and contour deformity is stated. Subchondral cysts show no localized edema, associated medical history, usually associated findings, contour deformities of any age or patient. Subchondral cysts also usually have overlapping cartilage disorders [5].

Osteochondral lesion (OCL) is thought to involve osteochondral fracture within an area of avascular necrosis in subchondral bone and overlying cartilage. A number of possible causes have been proposed, including repetitive microtrauma and ischemia, as well as genetic effects [6]. Acute trauma and ossification abnormalities are also implicated. Of all these proposed aetiologies, repetitive trauma is thought to be the primary insult in most cases. For this reason, “osteochondritis dissecans” is falling out of favour as a term and “osteochondral defect” or “osteochondral lesion” is the preferred terminology in many cases [7].

Osteochondral lesions should be differentiated from osteochondral fractures, insufficiency and stress fractures and subchondral cysts. Osteochondral fractures related to an appropriate history and MRI findings such as an acute fracture plane, large joint effusion, and extensive bone marrow oedema. Roemer et al. mentioned that insufficiency and stress fractures show the features of diffuse bone marrow oedema, usually no relevant trauma, elderly patient with osteoporosis, usually no associated ligamentous or meniscal pathology and no contour deformity [8]. Subchondral cysts show perifocal oedema, no relevant history, usually no associated finding, no contour deformity at any age and patient. Subchondral cysts typically have overlying chondrosis also [9].

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Osteochondral lesions (OCL) are thought to be associated with osteochondral fractures within the area of avascular necrosis of the subchondral bone and the cartilage above it. Many possible causes have been suggested, including recurrent microtrauma and ischemia, as well as genetic effects [12]. Acute trauma and abnormal ossification are also involved. Of all these proposed causes, recurrent trauma is considered to be the major crime in most cases. For this reason, the term "osteochondritis dissecans" has become less popular, and in many cases "osteochondritis dissecans" or "osteochondritis dissecans" is the preferred term [13]. Osteochondral lesions primarily affect the knee joint, especially the lateral surface of the medial femoral condyle (69%), the load-bearing portion of the lateral femoral condyle (15%), and the inferior medial pole of the patella (5%). Trochlear fove is the other most common places are the talus dome and skull. The OCL of the

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talus dome has been reported to have an incidence of 27 per 100,000. The skull is the most commonly affected area of the elbow, with the fastest increasing incidence. In children and young adults, OCL is a common cause of knee problems such as pain and dysfunction [14]. OCL is classically divided into two forms (juvenile and adult) based on the maturity of the skeleton. Juvenile OCL occurs when the growth plate is open, and adult OCL occurs in older adolescents and young adults when the growth plate is closed. These two forms are claimed to have different clinical courses. Juvenile OCL has been reported to have a good prognosis after conservative therapy and a high spontaneous healing rate. It has also been reported that juvenile OCL tends to be stable at onset, and juvenile and adult OCL tend to be unstable [15].

Conclusion

Differences between OCL presentation in children and adults have also been observed.

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