# **Osteochondral Lesions of Major Joints**

## Büyük Eklemlerin Osteokondral Lezyonları

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## Abstract

This paper provides information about osteochondral lesions (OCL) and example cases of OCL occurring in major joints, some of which are rarely seen. This simple tutorial is presented in question and answer format.

**Keywords:** Major joint, magnetic resonance imaging, osteochondral lesion

#### Özet

Bu yazıda, osteokondral lezyonlar, büyük eklemlerde görülen ve bazıları nadir örnekleri eşliğinde tartışılmaktadır. Sunum öğretici soru ve cevap formatında yapılmaktadır.

Anahtar Kelimeler: Büyük eklem, manyetik rezonans görüntüleme, osteokondral lezyon

## Introduction

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. Acute trauma and ossification abnormalities are also implicated [1-4]. Of all these proposed aetiologies, repetitive trauma is thought to be the primary insult in most cases [5]. 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.

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 [6]. Roemer et al. [7] 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. Subchondral cysts show perifocal oedema, no relevant history, usually no associated finding, no contour deformity at any age and patient [7]. Subchondral cysts typically have overlying chondrosis also.

This paper aims to answer some of the most commonly

asked questions about OCL and what physicians should consider when confronted with a possible case.

#### What Areas Does OCL Involve?

Osteochondral lesion mostly affects the knee joint, especially the lateral aspect of the medial femoral condyle (69%), the weight-bearing portion of the lateral femoral condyle (15%), and the inferomedial pole of the patella (5%) and trochlear fossa (1%) [8, 9]. The other most common sites are talar dome and capitellum. For OCL of talar dome, an incidence of 27 per 100,000 people has been reported [10]. Capitellum is the most commonly involved area in the elbow and is increasing in incidence at the most rapid rate [11].

In children and young adults, OCL is a common source of knee problems such as pain or dysfunction [4]. Below we present a selection of cases of OCL involving major joints.

**Shoulder.** Osteochondral lesion rarely involves the shoulder; however, some cases have been reported it has mainly been shown to affect males on the dominant side [12]. Pitching has been reported to be an etiologic factor. The antero-superior aspect of the humeral head is the most frequently affected area, followed by the superior and postero-superior aspects. The glenoid can also be involved [13].

**Elbow.** Osteochondral lesion can affect the elbow and most commonly the lateral distal aspect of the humerus and capitellar convex surface that is most susceptible to OCL [6, 14]. It is related to throwing activities. The trochlea, radial

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head (Figure 1) and the olecranon are other sites that can be involved [15, 16]. The capitellar pseudodefect and trochlear groove, along with a focal area devoid of cartilage at the junction of the coronoid and olecranon articular surfaces of the ulna in the trochlear notch should not be confused with OCL. Features such as smooth contours, absence of bone marrow oedema, and the posterior location can help to distinguish OCL from other features found on scans [14, 17-19]. Panner disease has also been considered. It is an osteochondrosis of the capitellum and produces changes similar to those observed in Legg Calve Perthes disease. Chronic repetitive trauma, congenital and hereditary factors, fat embolism, and endocrine disturbances have been accused. In contrast to OCL. Panner disease heals spontaneously with little if any residual deformity and does not typically lead to intra articular loose bodies. It occurs in a younger age group than OCL [14, 20]. It classically affects the entire capitellum and demonstrates low T1 signal and high T2 signal.

**Wrist.** OCL relating to the wrist is rarely reported in the English literature [21]. Etiologic factors have been reported to be acute trauma, repetitive microtrauma, and disturbance of local blood supply. All reported cases involved the scaphoid. To our knowledge ours is the first case of capitate OCL (Figure 2).

**Hip.** Osteochondral lesion of the femoral head (Figure 3) or acetabulum are relatively rare [22, 23]. For hip joint the normal structure and variant have to be known. The femoral head is entirely lined by articular cartilage, apart from a small depressed central portion termed the fovea capitis. This is the site of the ligamentum teres insertion. The acetabular roof variants such as the stellate crease, superior acetabular notch, and supraacetabular fossa, have



**Figure 1.** Grade II OCL of the radial head of a 13-year-old boy. Coronal proton density image (Skyra, Siemens, Enlargen, Germany) shows hypointense interface between the fragment and the parent bone (arrow). There is an intense bone marrow oedema.



*Figure 2. a, b.* Grade II OCL of the capitate at the proximal pole. Coronal T1-weighted (a) and proton density (b) images (Skyra, Siemens, Enlargen, Germany) demonstrate hypointense interface and intense bone marrow oedema (arrow).



*Figure 3. a, b.* Grade I OCL of the femoral head. Transverse T1-weighted (a) and proton density (b) images reveal subchondral bone oedema at the posterior aspect of femoral head (arrow) (Avanto, Siemens, Enlargen, Germany). Joint effusion is also evident.



**Figure 4.** *a*, *b*. Grade III OCL of the posterior aspect of the lateral femoral condyle with complete discontinuity but not dislocation (arrow). Sagittal T1-weighted (a) and proton density (b) images (Skyra, Siemens, Enlargen, Germany) show fluid intensity dissecting the fragment and the parent bone.

been described as mimickers of acetabular cartilage defects [24]. Stellate crease is a stellate-appearing zone lacking of hyaline cartilage and located above the acetabular notch. Superior acetabular notch is an anatomic variant in the development of the acetabulum without clinical relevance [25]. The supraacetabular fossa is usually located in the acetabular roof and can mimic a cartilage defect.

**Knee.** Osteochondral lesion frequently involves the medial femoral condyle, lateral femoral condyle, patella and femoral trochlear sulcus (Figure 4). The most commonly reported location is the lateral aspect of the medial femoral



*Figure 5. a, b.* Grade II OCL of the talar dome. The transverse T1-weighted turbo spin echo image (a) reveals a peripheral low-intensity contour (arrow). The T2-weighted turbo spin echo image (b). demonstrates bone oedema (arrow) at the medial talar dome (Avanto, Siemens, Enlargen, Germany).



*Figure 6. a, b.* Grade IV OCL of the posteroinferior aspect of the bilateral lateral femoral condyles in a 13-year-old boy. Plain radiography (a) shows widening of the joint space at the lateral compartment. Concavity, lucency and irregularity are seen at the lateral condyles (arrows). The sagittal T1-weighted turbo spin echo image (b) reveals a dislocated fragment lying within the bed (Skyra, Siemens, Enlargen, Germany).

condyle [26]. Current theories on aetiology favour repetitive microtrauma with microfracturing of subchondral bone, subsequent ischemia, and altered local growth.

**Ankle.** Osteochondral lesion may also involve the talar dome (Figure 5), most frequently the medial aspect. It is relatively prevalent and are an important cause of ankle morbidity [27].

## **Does OCL Differ for Children and Adults?**

OCL is classically divided into two forms - juvenile and adult - according to skeletal maturity. Juvenile OCL occurs when growth plates are open (Figure 6), while adult OCL occurs in older adolescents and young adults when growth plates are closed [28, 29]. It has been claimed that these two forms have different clinical courses. Juvenile OCL is reported to have a better prognosis and higher rates of spontaneous healing after conservative therapy [4, 30]. Juvenile OCL was also reported to be more likely to be stable at presentation, whereas adolescent and adult OCL tend to be unstable [31].

## How Can Magnetic Resonance Imaging Help with OCL?

Magnetic resonance imaging (MRI) confirms the diagnosis, characterizes and assesses the stage of the lesion [26, 32]. It also contributes to differential diagnosis from osteochondral fractures, insufficiency and stress fractures and subchondral cysts. According to the MRI staging system, lesions are described as follows [33] (arthroscopic grading is also given for comparison):

Stage I lesions are stable with a continuous softened area covered by intact cartilage. No interface is seen and the cartilage surface is smooth (partially soft however intact cartilage on arthroscopy).

Stage II lesions are also stable but exhibit partial discontinuity (Figure 7). On T2-weighted and 3D gradient echo images, hypointense interface can be seen between the fragment and parent bone and partial cartilage tear (overlying cartilage fissure on arthroscopy). Stage IIa shows surrounding bony oedema while IIb without oedema.

Stage III lesions show complete discontinuity but are not dislocated. Hyperintense interface on T2-weighted and 3D gradient echo images and partial/complete cartilage tears are seen. These lesions are unstable (exposed bone or attached fragment on arthroscopy).

Stage IV lesions have an empty defect or a dislocated fragment lying within the burrow (Figure 8). Hyperintense interface on T2-weighted and hypo- or isointense interface on 3D gradient echo images (fluid at interface) with complete cartilage tear are seen (partially detached fragment on arthroscopy).

Stage V lesions are osteochondral defects and contain loose bodies (craters with loose bodies on arthroscopy) [33] (Figure 9).

#### Is the Lesion Stable or Unstable? Is It Viable?

Stage III-V lesions suggest instability and therefore may be an indication for orthopaedic intervention. Kijowski et al. [4] reported the instability criteria as the presence of a high T2 signal intensity rim, cysts surrounding an OCL lesion (Figure 10), a high T2 signal intensity fracture line extending through the articular cartilage overlying an OCL lesion, or a fluid-filled osteochondral defect. In addition, the presence of intraarticular loose bodies is also indicative of instability [4, 33]. However, for juvenile OCL the presence of a high T2 signal intensity rim or cysts surrounding the OCL lesion is not found to be indicative of instability [4, 34].

Viability can be assessed by the administration of contrast material during MRI. Fragment enhancement suggests adequate blood supply and bone viability [14, 35]. Enhancement of the zone between the fragment and parent bone corresponds to histologic evidence of loose fragment and subjacent granulation tissue.

Magnetic resonance arthrography is reported to be more accurate than conventional MRI in the evaluation of articular cartilage, the assessment of stability of OCL and the detection



**Figure 7.** Grade II OCL of the anteroinferior aspect of the lateral femoral condyle. On the T2-weighted image a hypointense interface between the fragment and the parent bone is demonstrated (arrow). A slight depression is seen at the cortex (Avanto, Siemens, Enlargen, Germany).



*Figure 8.* Grade IV OCL of the posteroinferior aspect of the medial femoral condyle. An empty defect (arrow) is seen on the sagittal T2-weighted image (Avanto, Siemens, Enlargen, Germany).

of intra-articular bodies [36, 37]. With direct arthrography performed by administrating the contrast material to the joint space, a lesion is thought to be unstable if there is a



**Figure 9.** Grade V OCL of the inferior aspect of the medial femoral condyle (arrow). The lesion contains an osteonecrotic fragment (star) on the sagittal T1- weighted image (Skyra, Siemens, Enlargen, Germany).



*Figure 10.* Sagittal T2-weighted image revealing cysts (arrow) surrounding a talar OCL lesion (Skyra, Siemens, Enlargen, Germany).

direct insinuation of contrast material between the lesion and parent bone [6].

Treatment choices for OCL are non-operative and operative treatment. Skeletal maturity of the patient; size, location, and stability of the lesion have to be considered when deciding. Non-operative approaches include activity modification, cast immobilization, and brace treatment. Indications for operative treatment are failure of non-operative management, stable lesions with physeal closure within 6 months, unstable lesions, detached lesions (loose bodies), and fullthickness loss of overlying articular cartilage identified by MRI [9]. Operative choices are arthroscopic drilling, debridement of fibrous tissue and bone grafting, internal fixation, salvage procedures including microfracture to help promote the filling of the defect with pluripotent cells, autologous chondrocyte implantation, osteochondral autograft, and allograft [9, 31, 38].

## Conclusion

As a conclusion, in this paper we have reviewed OCL and noted which areas may be affected. Differences between OCL presentation in children and adults have also been explored. We discussed the role of MRI as a valuable method of identification, characterization, staging, viability determination and follow-up, showing how a grading system can be used to assess the degree of damage, the stability and viability of the lesion.

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