Osteochondritis dissecans of the elbow: recent evolution of pathogenesis, imaging, and treatment modalities

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Background: The etiology and pathogenesis of osteochondritis dissecans (OCDs) lesions remain controversial.

Methods: This review presents the recent evolution about the healing, imaging, pathogenesis, and how to treat OCD of the capitellum in overhead athletes.

Results: Compressive and shear forces to the growing capitellum can cause subchondral separation, leading to OCD, composed of 3 layers: articular fragment, gap, and underlying bone. Subchondral separation can cause ossification arrest (stage IA), followed by cartilage degeneration (stage IB) or delayed ossification (stage IIA), occasionally leading to osteonecrosis (stage IIB) in the articular fragment. Articular cartilage fracture and gap reseparation make the articular fragment unstable. The mean tilting angle of capitellar OCD is 57.6 degrees in throwers. Anteroposterior radiography of the elbow at 45 degrees of flexion (APR45) can increase the diagnostic reliability, showing OCD healing stages, as follows: I) radiolucency, II) delayed ossification, and III) union. Coronal computed tomography and magnetic resonance imaging with an appropriate tilting angle can also increase the reliability. MRI is most useful to show the instability, although it occasionally underestimates. Sonography contributes to detection of early OCD in adolescent throwers on the field. OCD lesions in the central aspect of the capitellum can be more unstable and may not heal. Cast immobilization has a positive effect on healing for stable lesions. Arthroscopic removal provides early return to sports, although a large osteochondral defect is associated with a poor prognosis. Fragment fixation, osteochondral autograft transplantation, and their hybrid technique have provided better results.

Discussion: Further studies are needed to prevent problematic complications of capitellar OCD, such as osteoarthritis and chondrolysis.

Osteochondritis dissecans (OCDs) of the elbow is a localized disorder of the articular cartilage and subchondral bone, commonly arises in adolescent athletes, frequently forms detached articular fragment, and develops osteoarthritis.[46,57,61,91] Many authors have advocated that an accepted cause of OCD is either acute or repetitive microtrauma applied to the growing capitellum during sports, making the lesions unstable (Fig. 1).[7,10,11,12,16,28,32,55,57,80,84,85,91,99]

A variety of pathologic conditions associated with OCD have been reported, such as chondral injury,[2,3,38,39,82,91,93] cartilage degeneration,[17,32,79,82,91,102] ossification defects,[2-4,82,91] osteochondral fractures,[12,99] nonunion,[82,91,102] and avascular necrosis.[9,36,40,43,56,63,66,82,91,94,99] The etiology and pathogenesis of OCD lesions remain controversial. Recent histologic studies have revealed that osteonecrosis is observed not in the underlying bone but only in the articular fragments of the specimens less than half.[38,83,91,99,102]

The author reviewed the pathophysiology of OCD of the elbow, including its mode of progression, diagnostic imaging and other testing methods, and treatment methods for the condition, citing our experience and a large amount of supporting articles. The progression of the disease, the healing process, and the mechanism of progression are described based on the author's experience, and the diagnostic imaging methods to accurately diagnose the pathology, stage, and severity of the disease, as well as the treatment and surgical methods accordingly, are clarified, citing data and articles that support our findings.

Figure 1 Onset, healing, and progression of capitellar osteochondritis dissecans (OCD). Onset. Compressive and shear forces (pink arrow) from the radial head during elbow extension-valgus overload can cause separation between the cartilage and the bone, eventually leading to OCD. Subchondral separation is followed by 1) ossification arrest in the articular fragment, 2) fibrocartilage formation over the exposed bone surface, and 3) underlying bone erosion, as observed in early OCD. Case 1, a 10.8-year-old boy with nonoperative treatment (in blue box). Anteroposterior radiography of the elbow at 45 degrees of flexion (APR45) showing healing process of stable OCD through two-way ossification: i) enchondral ossification in the articular fragment and ii) membranous ossification in the gap from the underlying bone. Intact articular cartilage and fibrocartilage connection can help stabilize the articular fragment and to achieve ossification and union (blue arrows). In contrast, articular cartilage fracture and gap reseparation, caused by repetitive compressive and shear forces, can make the lesions unstable (pink arrows), leading to partial detachment (in red box) with gap widening and underlying bone depression, eventually leading to complete detachment (in black box). Case 2, a 12.5-year-old boy with surgical treatment, having nearly-normal-cartilaginous (IA), partially detached (−2) articular fragment in the central aspect. Case 3, a 14.6-year-old boy with surgical treatment, having deteriorated-cartilaginous (IB), partially detached (−2) articular fragment in the central aspect. Case 4, a 14.0-year-old boy with surgical treatment having cartilage-osteonecrotic (IIB), partially detached (−2) articular fragment in the central aspect. Case 5, a 14.2-year-old boy with surgical treatment having cartilage-ossifying (IIA), partially detached (−2) articular fragment in the central aspect. Case 6, a 16.6-year-old boy with surgical treatment, having cartilage-osteonecrotic (IIB), partially detached (−2) articular fragment in the central aspect. OCD, osteochondritis dissecans. APR45, anteroposterior radiography of the elbow at 45 degrees of flexion. I-I, II-I, II-II, III-II, and III-III show radiographic stages of the lateral-central aspects of the capitellum. Radiographic stages: I, flattening, radiolucency, or slight calcification with open physis; II, delayed ossification or bony fragment; and III, normal or bony union. Pathologic stages of the articular fragments in the central aspect: IA, nearly normal cartilaginous; IB, deteriorated-cartilaginous; IIA, cartilage-ossifying; and IIB, cartilage-osteonecrotic. Instability grading: −1, stable; −2, partially detached; and −3, completely detached.

Pathology/condition of the lesion

### Pathogenesis

Compressive and shear forces on the growing capitellum during sports may cause separation beneath the epiphyseal cartilage, which is programed to be replaced with bone (Fig. 1).[83,91] This theory that OCD begins with adolescent subchondral separation[83,91] has been supported by the following findings: 1) early OCD has already had separation between the intact cartilage and flattened bone with no evidence of osteonecrosis (Figs. 1 and 2)[82,87,89]; 2) histological studies of OCD have shown that separation is an early event (Fig. 3, D-F)[38,82,102]; 3) experimental studies have shown that OCD can arise as a sequel of cartilage fracture in the immature epiphysis[21,39,93]; 4) the mechanical properties of the

growing epiphysis demonstrate the weakness at the cartilage-bone interface[31,58]; 5) the adolescent tissue has a significant reduction in the fracture toughness of its cartilage-bone interface,[13,15] and 6) the mean age at onset of lateral elbow pain was 12.2 years (range, 10.0-15.8 years), and it was 11.8 years at stage IA.[91]

OCD begins with subchondral separation, ie, gap formation (Fig. 1).[82] Then, OCD lesions are divided into 3 layers (Fig. 3, A-C): 1) articular fragment (progeny fragment) composed of cartilage with or without bone, 2) intermediate layer (gap) including various degrees of separation and fibrocartilage, and 3) the underlying proximal epiphyseal bone (parent bone).[82,91,102]

Families with multiple OCDs[53,68,81] may have some genetic factors that predispose them to microtrauma in their growing epiphyses, leading to OCD. Medial elbow laxity or some forms of elbow deformity can increase the forces on the capitellum during
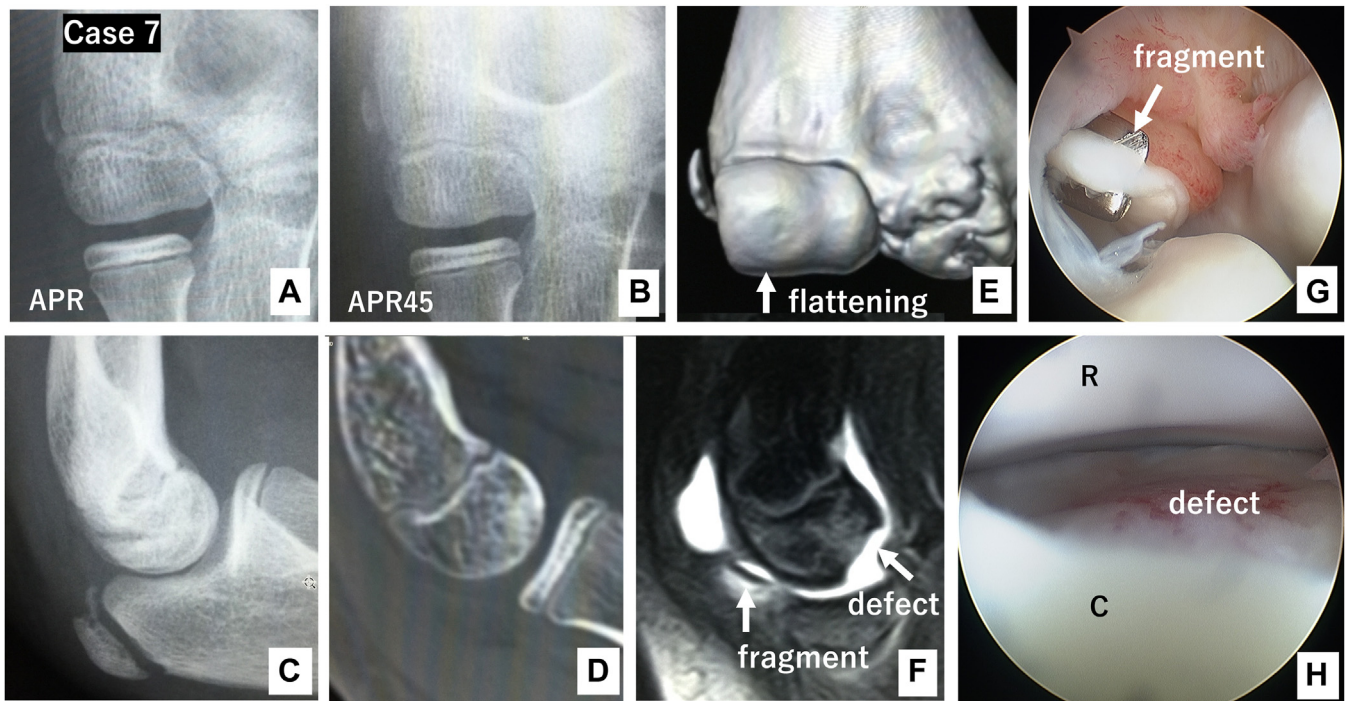
Figure 2 Case 7, a 9-year-old boy with symptom duration of 2 weeks underwent arthroscopic fragment removal. (**A-C**) Radiography showing partial sclerosis in the capitellum. Computed tomography (CT) showing sclerosis (**D**) and slight flattening of the capitellar bone surface (**E**). (**F**) Sagittal T2-weighted fat-suppressed magnetic resonance imaging (MRI) showing articular defect and completely detached fragment. (**G**) Arthroscopic removal of the cartilaginous articular fragment. (**G**) Articular defect of the capitellum. *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *CT*, computed tomography; *MRI*, magnetic resonance imaging; *C*, capitellum; *R*, radial head.

sports, thus increasing the risk of the OCD occurrence. It can be speculated that these OCD lesions develop through same pathogenetic process.

Location

The location of the capitellar OCD lesion is different between baseball players and gymnasts.[29] The tilting angle of capitellar OCD, defined as the angle between the long axis of the humerus and the line perpendicular to the lesion, was 57.6 ± 10.7 degrees in baseball players (Fig. 4) and 28.0 ± 10.7 degrees in gymnasts.[29] Anteroposterior radiography of the elbow at 45 degrees of flexion (anteroposterior radiography of the elbow at 45 degrees of flexion [APR45]) can clearly show the gap between the articular fragment and the underlying bone (Fig. 4). APR45 should always be used routinely to examine the elbow of athletes, and has high diagnostic reliability.[28,70,90] Occasionally, APR60 and APR30 may be optimal for some baseball players and gymnasts, respectively.

Early OCD

At the early stage of OCD, the following changes are observed in the separated three layers: 1) ossification arrest in the overlying cartilage, 2) fibrocartilage formation in the gap, and 3) the underlying bone erosion with the penetration of synovial fluid and fibrocartilage into the marrow cavity (Fig. 1). Early OCD lesions of the growing capitellum have the following findings: slight flattening and radiolucency of the subchondral bone (Figs. 1, 2, *A-E*, 3, *D*, 5, *A*, *E*, and *F*, 6, *A-C*, and 7, *A*), thickness of the cartilage with an intact articular contour (Fig. 5, *D*), separation of the cartilage from the bone (Fig. 2, *F-H*, 3, *D-F*, 5, *D*, *J*, and 6, *D-F*), and no histological evidence of osteonecrosis (Fig. 2, *H*, and 3, *F*).[82,87,89]

Healing and progression

APR45 shows the healing process of capitellar OCD as follows (Figs. 1 and 5): radiographic stage I, flattening, radiolucency, or slight calcification with open physis[28,51,57,90,84,88]; radiographic stage II, delayed ossification or bony fragment with separation[28,51,57,90,84,88]; and radiographic stage III, normal or bony union of the delayed ossified fragment with the underlying bone.[82,90,91] Radiographic stage I (radiolucency) and II (delayed ossification) correspond to pathologic OCD stage I (cartilaginous) and II (osteochondral), respectively.[82,91] OCD healing begins initially in the lateral aspect of the capitellum and delays in the central aspect (Figs. 1 and 5).[51,90,82,84,87,89,98] Union of the progeny fragment with the parent bone can be achieved through two-way ossification: i) delayed endochondral ossification in the progeny fragment (Figs. 1 and 5), which has a firm fibrocartilage connection to the parent bone, and ii) active ossification into the fibrocartilage from the parent bone (Figs. 1 and 5).[51,55,84,87,90,98]

The OCD lesion often remains unhealed in the central aspect (articular side) of the capitellum even after the lateral aspect (lateral wall side) has normalized (Figs. 1 and 5, *B* and *C*).[51,82,84,87,89,90,98] Until the whole of the articular fragment heals, repetitive compressive and shear forces can cause fracture of the articular cartilage and reseparation of the gap, leading to partial detachment with gap widening and underlying bone depression Figs. 1, 5, *C*, 7, *A* and *B*, and 8), eventually to complete detachment of all or part of the ununited articular fragment (Figs. 1 and 2). Thus, instability of the articular fragments is grossly divided into 3 grades: 1) stable, intact lesions with no evidence of displacement from its normal site or of fracture of the articular cartilage; 2) partially detached, unstable in situ fragments with fracture or fissure of the articular cartilage; and 3) completely detached, unstable loose fragments lying free in the joint (Fig. 1).[75,90,82,87]
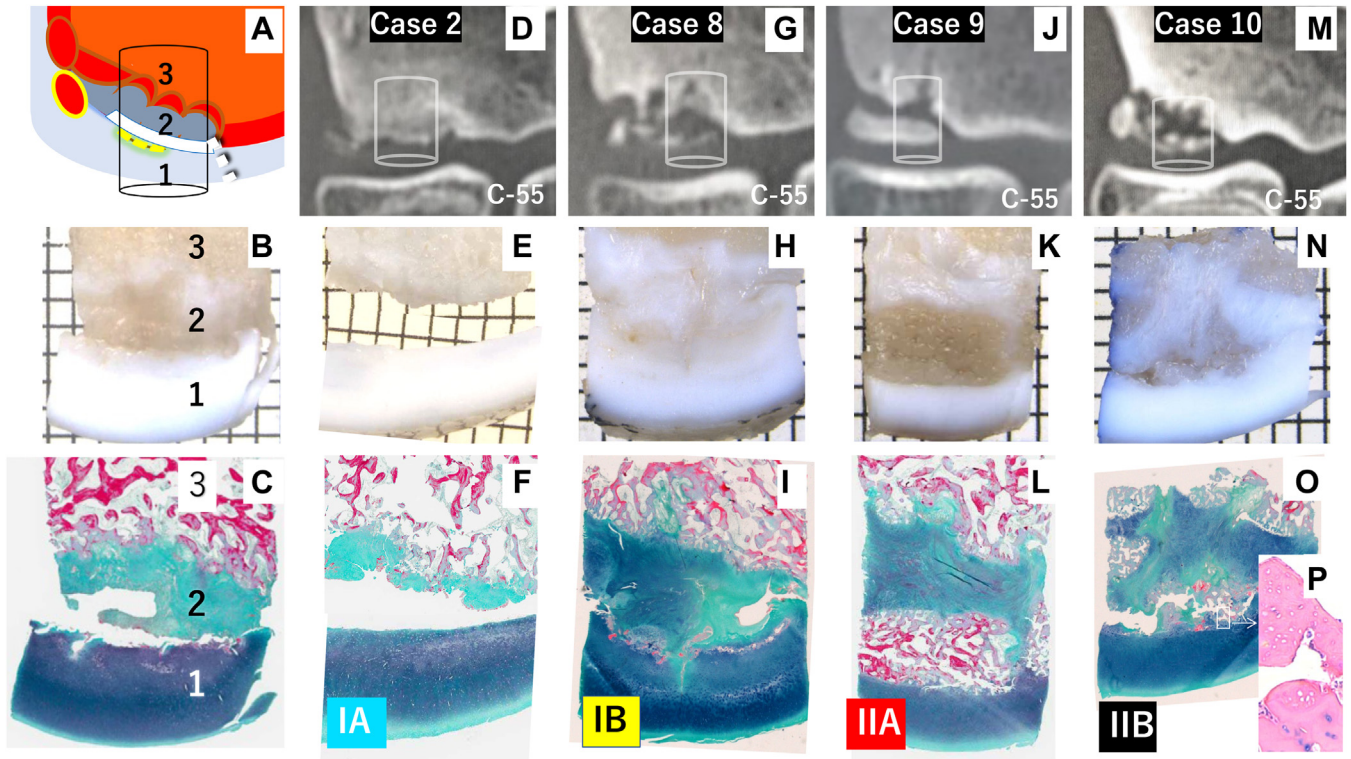
Figure 3 (**A-C**) 3 layers of OCD lesions: 1) articular fragment, 2) intermediate layer, and 3) underlying bone. (**D-F**) Case 2, a 12-year-old boy, (**G-I**) Case 8: a 15-year-old boy, (**J-L**) Case 9: a 12-year-old boy, and (**M-P**) Case 10, a 13-year-old boy. (**D, G, J,** and **M**) Coronal CT images of capitellar OCD, taken along a plane with a tilting angle of 55 degrees in flexion (C-55). (**A, D, G, J,** and **M**) Cylinder indicating the osteochondral plug to be harvested from the central aspect of the capitellum. (**B, E, H, K,** and **N**) Longitudinal section in half of the harvested cylindrical osteochondral plugs. (**C, F, I, L,** and **O**) Histological photographs with Elastica−Masson staining. (**P**) Hematoxylin-eosin staining showing empty lacunae in bone matrix of the articular fragment. Pathologic staging of articular fragments: (**F**) IA, nearly normal cartilaginous; (**I**) IB, deteriorated cartilaginous; (**L**) IIA, cartilage-ossifying; and (**O**) IIB, cartilage-osteonecrotic. (**C, F, I, L,** and **O**) The underlying bone surfaces are covered by fibrocartilage, and have several depressions with the fibrocartilage penetration. There is no bone plate between fibrocartilage and marrow cavity in several places of the underlying bone surface. *OCD,* osteochondritis dissecans; *CT,* computed tomography.
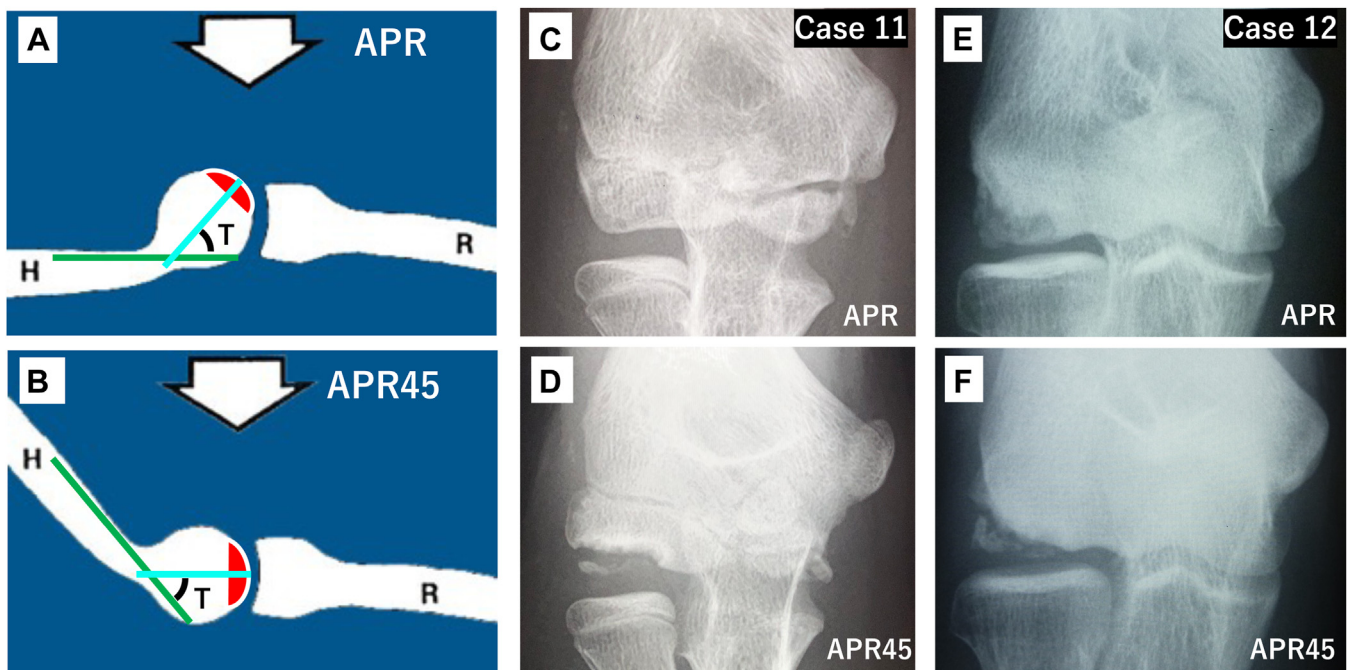
Figure 4 (**A** and **B**) Anteroposterior radiography of the elbow. (**C** and **D**) Case 11, a 10-year-old boy. (**E** and **F**) Case 12, a 14-year-old boy. Because the mean tilting angle of capitellar OCD is 57.6 degrees in baseball players, APR45 more clearly showing the OCD lesions (**B, D** and **F**) rather than APR with the elbow extended (**A, C,** and **E**). APR45 should always be used routinely to examine the elbow of athletes. *OCD,* osteochondritis dissecans; *APR45,* anteroposterior radiography of the elbow at 45 degrees of flexion; *H,* humerus; *R,* radius; *T,* tilting angle of capitellar OCD, green line, a long axis of the humerus; blue line, a line perpendicular to the lesion.
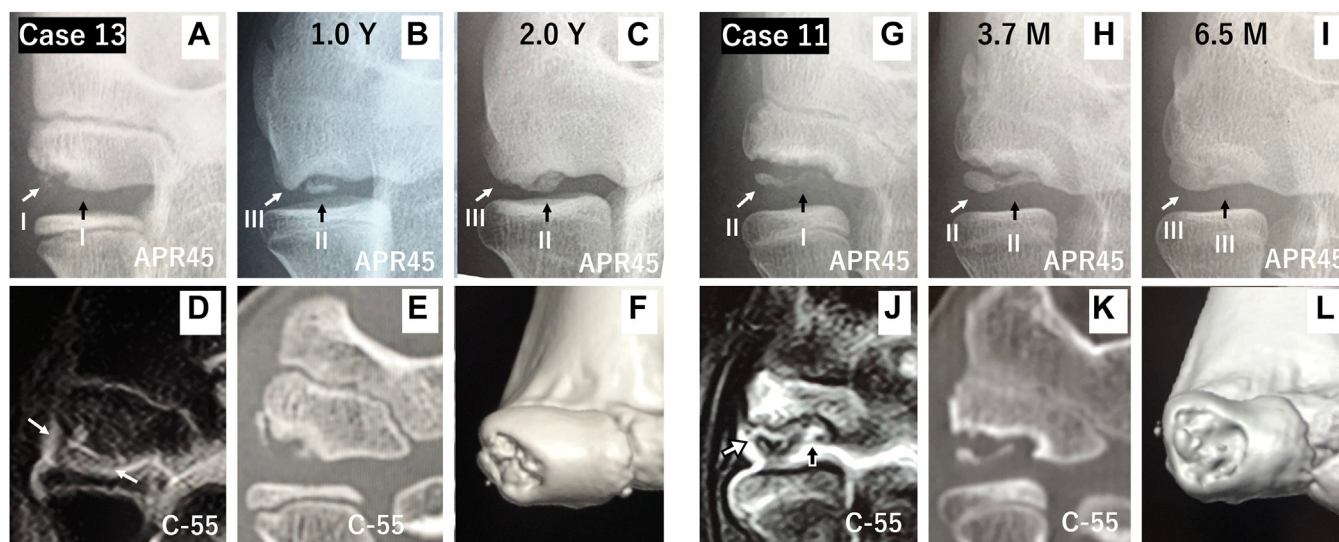
Figure 5 Nonoperative treatment of capitellar OCD. Case 13, a 11.5-year-old with activity restriction (AR). (**A-C**) APR45 shows the healing of the lateral aspect, delayed ossified fragment (**B**), and its displacement (**C**). (**D**) Coronal MR image, taken along a plane with a tilting angle of 55 degrees in flexion (C-55), showing high-signal-intensity line (white arrows) with no evidence of displacement from its normal site or of fracture of the articular cartilage. (**E** and **F**) CT at initial examination. Case 11, a 10-year-old boy with cast and splint. (**G-I**) APR45 shows ossification and complete union. (**J**) Coronal MR image, taken along a plane with a tilting angle of 55 degrees in flexion (C-55), showing clear high-signal-intensity line (white arrow) with fracture or fissure of the articular cartilage (black arrow). (**K** and **L**) CT at initial examination. Radiographic OCD stages of the lateral (white arrow) and central (black arrow) aspects of the capitellum: I, flattening, radiolucency, or slight calcification with open physis; II, delayed ossification or bony fragment; and III, normal or bony union. *OCD*, osteochondritis dissecans; *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *MR* image, magnetic resonance image; *CT,* computed tomogrpahy.

While a cartilaginous articular fragment has remained partially detached (Fig. 1, IA-2) for a prolonged period, the epiphyseal cartilage could not be followed by endochondral ossification, resulting in deterioration and degeneration with excessive calcification (Fig. 1, IB, 3, *G-I*, and 8). While the osteochondral articular fragment (Figs. 1, IIA-2, and 3, *J-L*) remains ununited, increased instability can cause to disturb the blood supply to the bony fragment, resulting in osteonecrosis (Fig. 1, IIB, 3, *M-P*, and 7).

Imaging

Radiography

APR45 occasionally shows displacement of the articular fragment and widening of the gap, suggesting the unstable OCD lesions.[28,87] However, radiography sometimes underestimates the actual degree of instability,[84] and may not show whether there is displacement of the cartilaginous articular fragment (Figs. 2 and 6). Further imaging is necessary to reveal any instability and determine the optimal choice of treatment.[71,84,88] There are many cases in which the radiographic stage and instability grade are quite different between the lateral and central aspects.[82,90,91] The OCD lesions are usually more unstable and remain unhealed in the central aspect of the capitellum (Figs. 1, 5, *B* and *C*, and 7).[82,90,91] The radiographic stages should be assessed in the lateral and central aspects of the capitellum separately, as shown in Figures 1 and 5.[82,90,91]

Sonography

The author has used sonography to assess capitellar OCD since 1988. Sonography of the elbow is advantageous,[88] as it 1) provides sagittal and coronal images of both the cartilage and subchondral bone, 2) clearly demonstrates the cartilage-rich fragment, which lacks any osseous content,[89] 3) reveals the instability of the lesions that might have been underestimated by radiography, 4) provides dynamic images, and 5) is handy and usable anywhere.[19]

Sonography has recently contributed to the detection of many early and silent OCD lesions in adolescent baseball players on the field, especially in Japan.[19,30,52,69,88,89,92]

On the basis of sonographic appearance, capitellar OCD lesions have been divided into 4 types: A) localized subchondral bony flattening and normal articular surface, B) lesion with nondisplaced osteochondral fragment, C) lesion with slightly displaced fragment, D) and capitellar osteochondral defect.[88] The former two (A, B) are assessed as probably stable lesions, while the latter two (C, D) are considered to be unequivocally unstable.

Computed tomography (CT)

Coronal, sagittal, and 3D CT images are very useful to show the location of the free bodies, the size and displacement of the articular fragment, the degree of gap widening, and the area of the underlying capitellar bone and any cyst formation, and the size and location of any bony spur formation (Figs. 3, 5-9).[71]

Although the mean tilting angle of capitellar OCD is 57.6 degrees in throwing athletes, the coronal images of the elbow show cross sections targeting not OCD lesions but the distal humerus. We have routinely obtained the coronal images of the OCD lesions along a plane with a tilting angle of 55 degrees in flexion (C-55, Fig. 7, *D*) and sagittal images along a plane with a tilting angle of 10 degrees in valgus (S-10, Fig. 7, *C*). This imaging of OCD lesions allows precise measurement of the articular fragment, gap, and underlying bone, thus increasing the diagnostic reliability (Figs. 3, 5-9).

Magnetic resonance imaging (MRI)

The coronal (C-55) and sagittal (S-10) magnetic resonance images clearly show the three layers of OCD lesions. Fat-suppression T2-weighted MRI is most useful for revealing signs of any instability,[23,24,35,46,71,84,103] such as separation beneath the articular fragment (a high-signal-intensity line; Figs. 5, *D, J*, 6, *D*, and 7, *E* and *F*), fracture of the articular cartilage (high-signal-intensity through the articular cartilage; Fig. 5, *J*, 6, *D*, and 7, *E*), widening of the gap
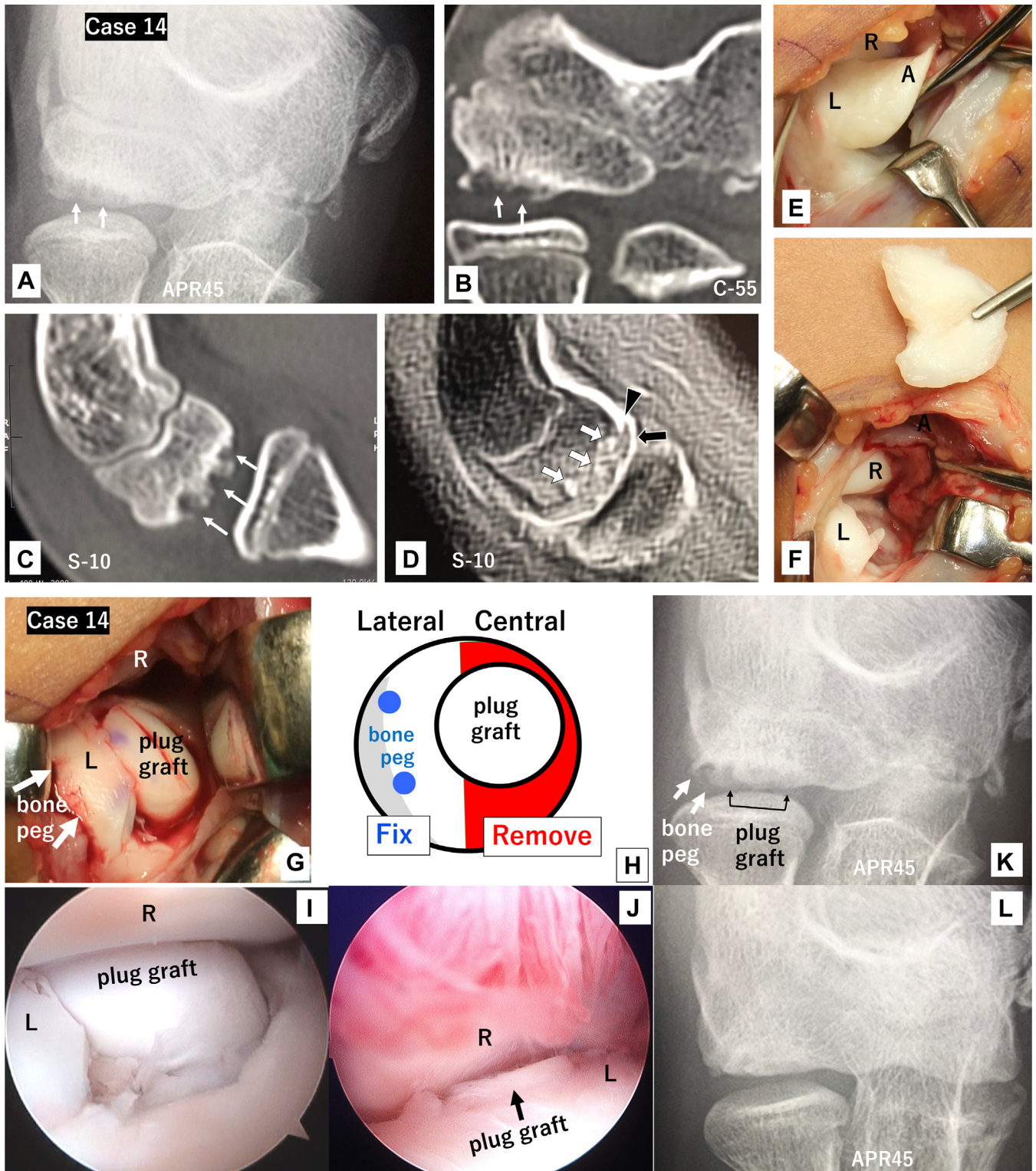
Figure 6 Case 14, a 12-year-old boy with symptom duration of 0.2 years underwent a hybrid OAT technique with bone-peg fixation. (**A-C**) Preoperative APR45, coronal and sagittal reconstruction CT images showing radiolucency and bone erosion (arrows) in the central aspect of the capitellum. (**D**) Sagittal T2-weighted fat-suppressed MRI showing slight displacement of the articular fragment (black arrow), fracture of the articular cartilage (high-signal intensity through the articular cartilage: black arrowhead) and widening of the gap (cyst-like lesion: white arrow). (**E** and **F**) Articular fragment in the central aspect of the capitellum (A) being more unstable than in the lateral (L) and was excised (**F**). Excised articular fragment was nearly normal-cartilaginous. (**G**-**J**) Hybrid OAT (plug graft) technique with bone-peg fixation (arrows). (**I**) Arthroscopy through soft spot portal showing plug graft to mainly bear the load from the radial head (R). (**J**) Arthroscopy through proximal medial portal showing plug graft with the surface height of plus 0.5-1 mm. (**K**) APR45, taken 5 weeks postoperatively, shows OAT (black arrows) and bone pegs (white arrows). (**L**) APR45, taken 6 months postoperatively, shows almost normal appearance of the capitellum. He completely returned to sports 6 months postoperatively. *OAT*, osteochondral autograft transplantation; *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *C-55*, coronal images of OCD lesions taken along a plane with a tilting angle of 55 degrees in flexion; *S-10*, sagittal images of OCD lesions taken along a plane with a tilting angle of 10 degrees in valgus; *CT*, computed tomography; *MRI*, magnetic resonance imaging; *OCD*, osteochondritis dissecans; *A*, articular fragment of the central aspect of the capitellum; *L*, articular fragment of the lateral aspect of the capitellum; *R*, radial head.
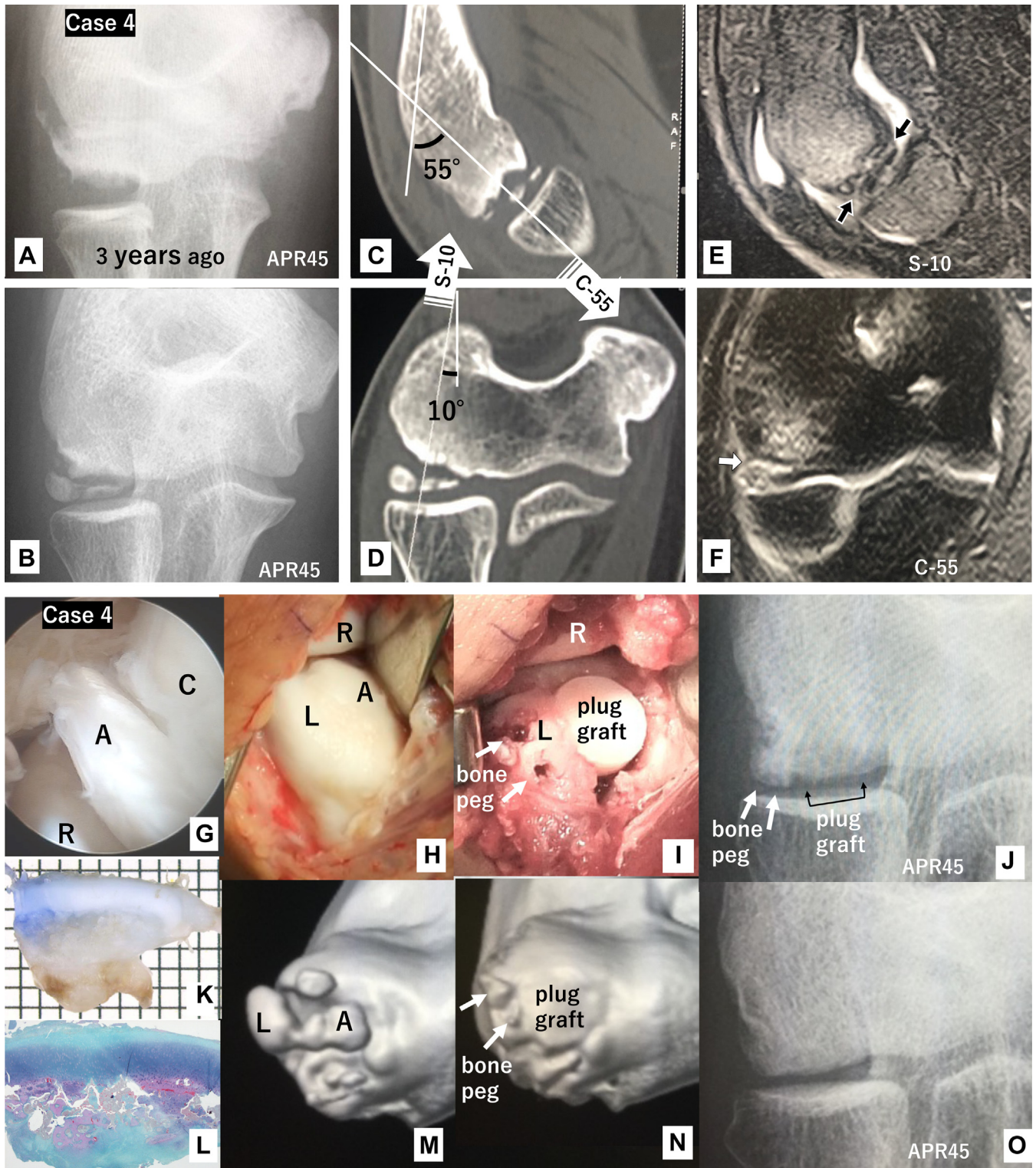
Figure 7 Case 4, a 14-year-old boy with a hybrid OAT technique with fixation. (**A**) APR45 taken at 3 years ago. He was treated nonoperatively and dropped out. (**B**) APR45, taken preoperatively, shows displaced bony fragments. (**C-F**) Coronal and sagittal images of OCD lesions routinely taken along a plane with a tilting angle of 55 degrees in flexion (C-55) and 10 degrees in valgus (S-10), respectively. Coronal and sagittal T2-weighted fat-suppressed MRI of OCD lesion showing articular cartilage fracture (high-signal intensity through the articular cartilage: **E**, black arrows) and separation of the articular fragment (clear high-signal intensity line: **F**, white arrow). (**G**) Arthroscopy showing dynamically unstable articular fragment in the central aspect of the capitellum. (**H**) Articular fragment of the capitellum being more unstable in the central aspect (A) than in the lateral (L). (**I** and **J**) Hybrid OAT (plug graft) technique with bone-peg fixation (arrows). (**K** and **L**) Excised articular fragment. (**K**) Transverse section. (**L**) Histological photograph with Elastica–Masson staining shows OCD at stage II–B with collapsed bone with osteonecrosis. (**M** and **N**) Preoperative and postoperative 3D-CT images. (**J**) APR45, taken 2 weeks postoperatively, showing osteochondral autograft (black arrows) and bone pegs (white arrows). He completely returned to sports 6 months postoperatively. (**O**) APR45, taken 1.4 years post-operatively, showing almost normal appearance of the capitellum. *OAT*, osteochondral autograft transplantation; *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *CT*, computed tomography; *MRI*, magnetic resonance imaging; *OCD*, osteochondritis dissecans; *A*, articular fragment of the central aspect in the capitellum (C); *L*, articular fragment of the lateral aspect of the capitellum; *R*, radial head.
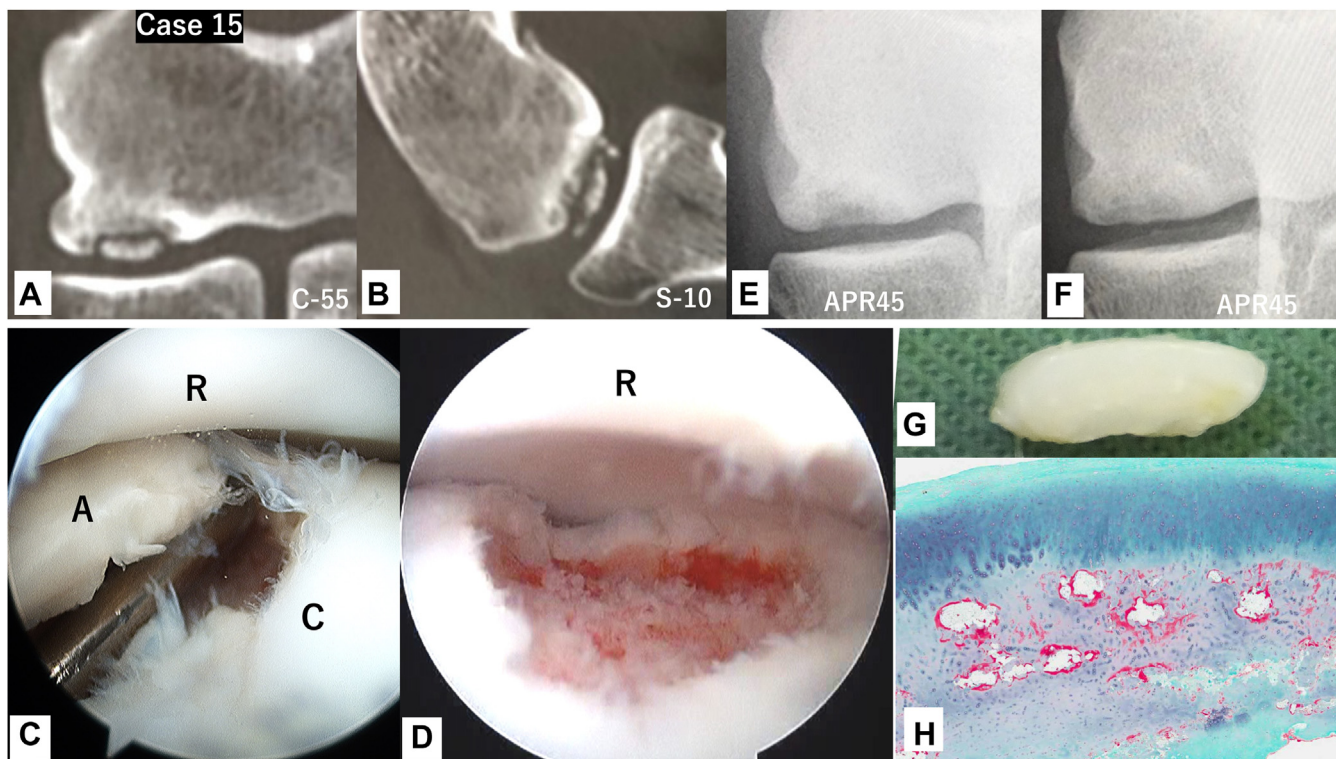
Figure 8 Case 15, a 15-year-old boy with symptom duration of 4.0 years treated with arthroscopic fragment removal. (**A** and **B**) CT images shows displaced fragments in the central aspect of the capitellum. (**C**) Arthroscopic through soft spot portal. Articular fragment (A) was elevated to be removed. (**D**) After fragment removal and abrasion over the underlying bone, the radio-capitellar joint being assessed as "Cartilage on Cartilage". (**E** and **F**) APR45, taken one week (**E**) and one year (**F**) postoperatively, shows bone remodeling of the defect. (**G** and **H**) Excised articular fragment. (**G**) Transverse section. (**H**) Histological photograph with Elastica−Masson staining showing OCD at stage I-B, deteriorated cartilage with chondrolysis and excessive calcification. *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *C-55*, coronal images of OCD lesions taken along a plane with a tilting angle of 55 degrees in flexion; *S-10*, sagittal images of OCD lesions taken along a plane with a tilting angle of 10 degrees in valgus; *CT*, computed tomography; *OCD*, osteochondritis dissecans; *C*, capitellum; *A*, articular fragment of the central aspect; *R*, radial head.

between the articular fragment and the underlying bone (cyst-like lesion; Fig. 6, *D*), displacement of the articular fragment (Figs. 2, *F*, and 6, *D*), and any articular defect (Figs. 2, *F*, and 9, *J*). MRI also reveals any bone edema of the capitellum (Figs. 5, *J*, and 7, *F*) and synovitis with the joint effusion (Fig. 2, *F*, and 9, *J*). Furthermore, MRI has the potential to show bone edema of the capitellum in very early OCDs before APR45 shows slight changes.[89] Although MRI has been recommended as most useful tool to show the instability, some OCD lesions may be underestimated irrespective of the imaging modality employed.[24,35,71,84,88,103]

Histological findings

Articular fragment (progeny fragment)

The pathologic OCD stages of the articular fragments were grossly divided into four (Fig. 3)[82,91] Pathologic stage IA (nearly normal cartilaginous): nearly normal-cartilaginous with or without slight calcification in the epiphyseal cartilage (Fig. 3, *D-F*). Pathologic stage IB (deteriorated-cartilaginous): deteriorated-cartilaginous with degradation or degeneration in the epiphyseal cartilage, such as local chondrolysis and excessive calcification (Fig. 3, *G-I*). Pathologic stage IIA (cartilage-ossifying): cartilage-ossifying with endochondral ossification at the cartilage-bone interface (Fig. 3, *J-L*). Pathologic stage IIB (cartilage-osteonecrotic): cartilage-osteonecrotic with empty lacunae of the bone matrix and deterioration of the bone matrix and marrow (Fig. 3, *M-P*).[82,91] There are some articular fragments including osteonecrosis

in the newly formed bone, suggesting that osteonecrosis can be secondary to ossification.

Pathologic stage IA (nearly normal cartilaginous) articular fragment is caused by separation beneath the immature epiphyseal cartilage, and the stage IA can result in stage IB (deteriorated-cartilaginous) with prolonged ossification arrest. On the other hand, delayed endochondral ossification (stage IIA) appears in the stage IA articular fragment (Figs. 1, 5, and 7, *A* and B), which has a firm fibrocartilage connection to the underlying bone. Osteonecrosis (stage IIB) in the articular fragment is a last event following detachment of the stage IIA fragment (Figs. 1, 3, and 7).[82,91]

Intermediate Layer (Gap)

In the gap, there are various degrees of separation and fibrocartilage formation (Fig. 3).[82] The fibrocartilage formation is a common reaction to exposure of the bone.[82] The fibrocartilage connection can help to stabilize the articular fragment. In contrast, prolonged instability of the articular fragment may lead to gap widening (Figs. 1 and 3).

Underlying Bone (Parent Bone)

The surface of the underlying bone is always covered by the fibrocartilage. The underlying bone is composed of trabecular bone and marrow, and there is no structure like subchondral bone plate (Fig. 3, *C, F, I, L* and *O*). The viability of the underlying bone is nearly normal.[38,82,99,102] There is no evidence of osteonecrosis in the
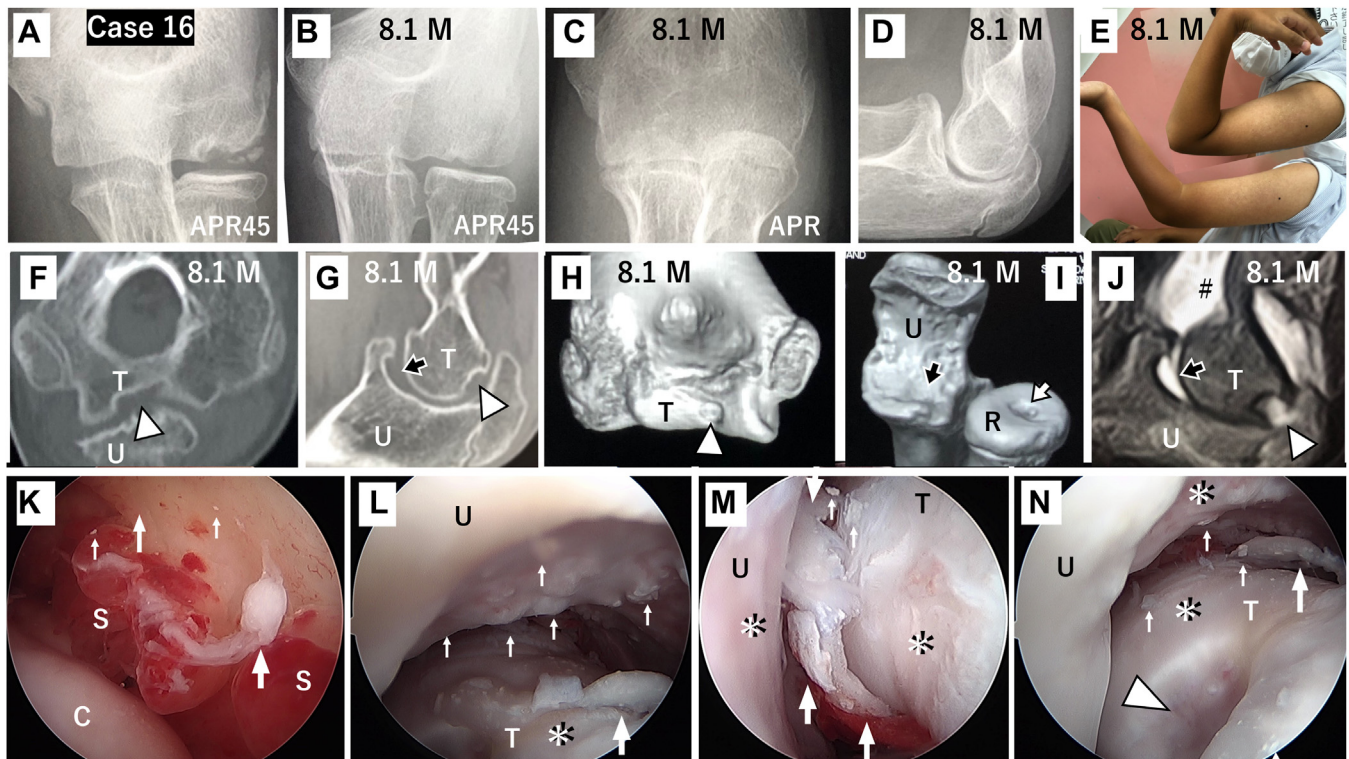
Figure 9 Case 16, a 12-year-old boy developing chondrolysis after nonoperative treatment with a cast and splint. Radiographs (**A**, 5 weeks after initial presentation; **B** and **D**, 8.1 months later) shows complete healing of the OCD lesion although the joint space became narrow with subchondral bone irregularity. (**E**) Restricted active range of elbow motion at 8.1 months after initial presentation. CT (**F-I**) and (**J**) MRI, taken 8.1 months after initial presentation, shows focal defects of the articular surface of the ulno-humeral joint (arrow and arrowhead) and arthritis with severe effusion (**#**). (**K-M** and **N**) Arthroscopic photographs, taken 8.1 months after the initial presentation, show numerous cartilaginous particles (small white arrows), cartilaginous loose fragments (large white arrow), synovitis (*S*), softening of articular cartilage, cartilage ulceration (*) and focal bony defect (arrowhead). *APR45*, anteroposterior radiography of the elbow at 45 degrees of flexion; *CT*, computed tomography; *MRI*, magnetic resonance imaging; *OCD*, osteochondritis dissecans; *C*, capitellum; *R*, radial head; *T*, trochlea; *U*, ulna.

underlying bone irrespective of the stages of the overlying articular fragments.[38,82,99,102] Active ossification is observed at the interface of the fibrocartilage and the bone.[82] These observations indicate that there are no causative conditions of OCD in the underlying bone.

APR45 and CT often show several depressions of the underlying bone surface (Cases. 1-6, and 8-16), and MRI shows cyst-like lesions (Fig. 6, *D*). The depression in the underlying bone surface is filled with the overlying fibrocartilage (Fig. 3 *C*, *F*, *I*, *L*, and *O*). There is no bone plate between fibrocartilage and marrow cavity in several places of the underlying bone surface (Fig. 3 *C*, *F*, *I*, *L*, and *O*). Repetitive micromovement of the overlying articular fragment can allow the penetration of synovial fluid and fibrocartilage into the underlying marrow cavity, leading to erosion of the underlying bone surface.

Classification

OCD lesions in the central aspect of the capitellum are classified according to the pathologic staging of the articular fragment (IA, IB, IIA, and IIB) while also taking into account instability (−1, −2, and −3), as shown in Figure 1.[91] This classification system includes all of OCD lesions and demonstrates the individual backgrounds. It is helpful for understanding and treating this problematic condition. Stable OCD, including IA-1 (nearly normal cartilaginous - stable) and IIA-1 (cartilage-ossifying - stable), are treated nonoperatively. IIA-2 (cartilage-ossifying - partially detached) could be successfully treated with reattachment. It is appropriate to remove

the articular fragments of stage IA-2,3 (nearly normal cartilaginous - partially, completely detached), stage IB (deteriorated cartilaginous), and stage IIB (cartilage-osteonecrotic), because such fragments could not unite with the underlying bone.[91] Further studies are needed to derive a diagnosis algorithm capable of reliably differentiating the actual conditions included in the classification system (Fig. 1).
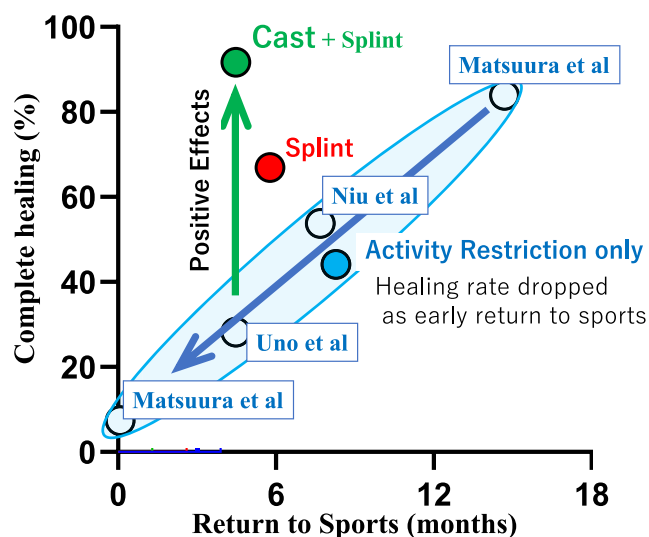
Treatment

Conservative treatment

Nonoperative treatment has been commonly performed for stable OCD lesions, characterized by silent or slight symptoms, subtle radiographic changes, narrow gap between the progeny fragment and the parent bone, and intact articular contour with no evidence of displacement from its normal site or of fracture of the articular cartilage (Case 1 and Fig. 5, *A*, *D*, and 7, *A*).[46,51,55,62,71,74,84,85,90,98] These characters suggest that the lesions are stable at stage IA-1 and stage IIA-1.[46,51,55,62,71,74,84,85,90,98] Sports activities such as throwing and swinging a racket were prohibited[51,55,62,90] as well as avoiding heavy use of the elbow, including actions such as arm wrestling, push-ups, and weightlifting. Running and fielding (only catching the ball) are approved. General stretching, such as the sleeper stretch or core and hip stretches, is performed to resolve general tightness.[6,67]

The healing rates with nonoperative treatment are higher in patients with a stage I lesion, a stable lesion, a smaller lesion

Healing (%) and Return to Sports (months)

Figure 10 OCD healing and return to sports. In the previous nonoperative treatment with AR only, the rate of complete healing and the mean period required for return to sports were 7% and no fixed period by Matsuura et al,[51] 28% and 4.4 months by Uno et al,[98] 53.8% and 7.6 months by Niu et al,[62] and 84.2% and 14.6 months by Matsuura el al, respectively.[51] Healing rate dropped with an early return to sports. In our results, those were 41% and 8.2 months (blue spot) in AR group, 67% and 5.7 months (red spot) in splint (8.8 weeks) group, and 92% and 4.4 months (green spot) in cast (3.7 weeks) + splint (7.3 weeks) group, respectively.[90] Immobilization showing positive effects on both complete healing and an early return to sports. *OCD*, osteochondritis dissecans.

without a cyst-like lesion, an open physis, a short symptom duration, progression of ossification within the first three months, and no enlargement of the radial head and who are compliant with activity restriction (AR).[14,20,37,51,55,62,90,98,100] The prior studies have shown that the complete healing of OCD lesions requires 1 or 2 years and that the rates of healing dropped dramatically as the period of AR was shortened: 84.2% for a mean of 14.6 months,[51] 53.8% for a mean of 7.6 months,[62] 28% for a mean of 4.4 months,[98] and 7% for no fixed period[51] (Fig. 10). Even if an OCD lesion has more factors predictive of healing, success is mainly dependent on whether the patient is compliant with AR for a given period.[28,51] Nonoperative treatment with AR rarely achieves a successful outcome within six months.[51,55,62,90,98] In contrast, elbow immobilization with a cast (mean: 3.7 weeks) and a splint (mean: 7.3 weeks) was shown to significantly accelerate ossification (mean: 1.9 months) in the central aspect of the capitellum, enabling both an early return to sports (mean: 4.4 months) and complete healing (92%) within a shorter period (mean: 5.5 months).[90] Cast immobilization is therefore strongly recommended as a first choice of nonoperative treatment for a stable OCD lesion of the elbow before epiphyseal closure. Immobilization has a positive effect on forming firm fibrocartilaginous connection between the articular fragment and the underlying bone.[90] The firm fibrocartilaginous connection can play the following important roles: i) as a stabilizer of the overlying articular fragment, ii) as a vascular pathway from the underlying bone to the articular fragment, and iii) as a scaffold for ossification from the underlying bone (Fig. 5, *G-I*).[90]

The author treats stable OCD lesions of the elbow as if minimally displaced, cartilage-rich, intraarticular fractures. For the first 1 month, the author recommends cast immobilization of the elbow, forearm, and wrist, followed by splinting for two months. For the second 1 month, the patients are permitted intermittent active motions of the elbow, forearm, and wrist only at rehabilitation room or in bath. For the third 1 month, the patients are permitted to take off the splint only in their home. Surgery should be considered for lesions without the progression of ossification during the first 3 months.[98]

Little is known about the optimal timing of return to sports. Uno, et al proposed both an OCD lesion width of <8.0 mm and a lateral normal width of >2.0 mm as radiographic landmarks of the timing of the return to sports.[98] However, APR45 occasionally shows worsening of the OCD lesions after return to sports before radiographic complete healing. It is clearly optimal for the patients to start throwing after complete healing of the lesion.[51] Within the first six months, the author recommends elbow rest until APR45 shows complete healing of the OCD lesions.

Surgical treatment

Surgical treatment is principally recommended to the patients who have persistent pain, locking phenomenon, severe restriction of elbow motion, fragment displacement, wide gap between the progeny fragment and the parent bone, irregular contour or defect of the articular surface, and T2 high signal interface through the articular cartilage.[46,71,84] Surgical procedures include arthroscopic removal of the lesion with or without drilling and microfracture,[5,8,34,50,97] fragment fixation,[18,22,44,65,64,95,101] autologous chondrocyte implantation,[27] osteochondral autograft transplantation (OAT),[1,15,25,42,45,48,62,72-74,76-78,96] wedge osteotomy,[33] osteochondral allograft, and various novel techniques.[47,59,60] Candidate donor sites for OAT include the non-weight-bearing area of the lateral femoral condyle and the transitional area between the rib and its associated cartilage.[1,15,25,42,45,48,60,73,74,76-78,96,101]

Arthroscopic removal can provide an early return to sports and yield satisfactory results.[5,8,34,50,97] However, the long-term outcomes of removal are not necessarily good.[5,86,96,97] A large residual osteochondral defect in the capitellum has been associated with a poor outcome.[86] Although OAT can provide satisfactory results,[1,15,25,42,45,46,48,60,73,74,76-78,96] the clinical and radiographic outcomes tend to be relatively inferior when lesions are extensive and affect the lateral and central aspects of the capitellum.[15,41,48,96] Based on the Lysholm knee score and International Knee Documentation Committee evaluation form, no adverse effects on donor knee function were found after mosaicplasty for capitellar OCD in young athletes.[26] On the other hand, the donor site mobility rate was 2.3% with the usual criterion (persistent symptoms for >1 year or the need for subsequent intervention) and 12.8% with the stricter criterion (the presence of any symptoms, such as effusion, patellofemoral complaints, crepitation, unspecified disturbance, stiffness, pain/instability during activities, and osteoarthritic change).[49] MRIs of the knee showed an abnormal signal intensity and hypertrophy in some cases.[26,49]

Cylindrical or block-shaped costal osteochondral autograft can reconstruct the large, wide-range articular cartilage defects affecting the lateral wall of the capitellum.[60,73,74,76,77] Donor site no longer causes pain 2 or 3 days after surgery.[60] Costal osteochondral autograft successfully achieved anatomical and biological reconstruction in the treatment of advanced OCD of the humeral capitellum, although 10%-20% of the patients postoperatively needed additional minor surgical procedures.[73,74]

A new surgical procedure has been reported including a bone marrow stimulation technique augmented by implantation of ultrapurified alginate (UPAL) gel. Ultrapurified alginategel is a novel biomaterial that enhances hyaline-like cartilage repair for articular defects.[59] This novel technique can be a useful, minimally invasive approach for treating small cartilaginous lesions in athletes.

Author's preferred methods of surgical treatment

Arthroscopic removal

In principle, arthroscopic removal is the first choice of treatment when the postoperative osteochondral defect appears to be small, ie, within a width of 12 mm as one of the landmarks used by the author.[83] The partially detached articular fragment is elevated and removed through a soft spot portal (Fig. 8, *C*). Abrasion of the fibrocartilage is performed over the underlying bone until bleeding appears (Fig. 8, *D*), suggesting that no further drilling or microfracture is necessary. After removal, the radio-capitellar joint is arthroscopically assessed during passive elbow motion as follows: 1) Cartilage on cartilage: both the lateral and medial edges of the radial head are always supported on the normal articular cartilage of the capitellum (Fig. 8, *D*); 2) Cartilage on borderline: Either the lateral or medial edge of the radial head passes on the borderline between the normal articular cartilage and the defect; and 3) Cartilage in defect: either or both edges of the radial head drop into the defect. Cartilage on cartilage after removal can predict a successful outcome. For the other conditions, articular reconstruction of the defect should be considered. It is important to reconfirm that no fragments exist anywhere in the elbow joint, because occasionally they are missing.

Postoperatively, the mean required period for resumption of throwing is one month, and three months for full return to sports. Excluding cases involving a large osteochondral defect or osteoarthritis, most patients return to preoperative sports without any problems. Postoperative APR45 shows osseous remodeling of the defect, and about half of patients have an almost normal capitellum with or without slight flattening (Fig. 8, *E* and *F*), suggesting that the articular surface may have been covered with fibrocartilage proliferation.

Hybrid OAT technique with fixation

For a large OCD lesion more than 12 mm in width,[82,83] the first choice of treatments is a hybrid OAT technique with fixation, ie, OAT in the central aspect of the capitellum followed by preservation of the lateral articular fragment with or without bone-peg fixation (Fig. 6, *G-J*, and 7, *I*).[91] This technique is a hybrid version of procedures previously reported.[44,45,54,83,101] One osteochondral plug 8 mm in diameter and 10-14 mm in length is harvested from the nonweight-bearing articular surface of the lateral femoral condyle and inserted in the central aspect of the capitellum by press fit.[83] Care must be taken to reconstruct the height and congruity of the articular surface. Although the authors described that over-reconstruction should be avoided in previous reports,[83] a surface height of plus 0.5-1 mm, relative to the capitellar surface, is now considered optimal for this hybrid technique so that the grafted plug can mainly bear compressive and shear forces from the radial head (Fig. 6, *G-J*). It is occasionally necessary to minimally slice the higher edge of the reconstructed articular surface over which the radial head smoothly pass during elbow motion. The lateral articular fragment mostly has ossifying bone, its instability is less severe, and its healing potential is relatively higher, rather than the central fragment.[90] To accelerate union of the lateral articular fragment, a few small bony chips are inserted beforehand in the underlying gap through the recipient hole created for OAT. The lateral articular fragment is mostly stabilized by OAT. If insufficient, bone-peg grafts or one more OAT should be considered. Two or three bone-pegs 3 or 4 mm in diameter and 15 mm in length are harvested from the olecranon and inserted at the lateral corner of the lateral articular fragment (Figs. 6 and 7). While the grafted plug mainly bears compressive and shear forces from the radial head, the OCD lesions surrounding OAT can heal with the decreased forces from the radial head (Figs. 6 and 7).[91] Hybrid OAT technique with fixation has several benefits: 1) it is simple and easy for reconstructing the nearly original contour of the capitellum, 2) the reconstruction is appropriate for bearing load from the radial head, and 3) donor site mobility is minimized.

Postoperatively, the mean required period is three months for resumption of throwing and six months for full a return to sports. More than 90% of patients return to their previous sports without problem. There is no tendency for outcomes to be inferior for the large lesions affecting both the lateral and central aspects of the capitellum. Poor results may be associated with under-reconstruction of the articular surface. The author believes that the key to success is to reconstruct the central articular surface with a height of plus 0.5-1 mm rather than the original height, so that the grafted plug bears mainly compressive and shear forces from the radial head.

Complications

Osteoarthritis is the most problematic complication of capitellar OCD. Whereas the patients with capitellar OCD are adolescent osteoarthritis is often seen at initial presentation, and this may subacutely progress during treatment. Osteoarthritis usually appears in not only the radio-capitellar joint but also the ulno-humeral joint, ie, it can affect the whole of the elbow joint. Osteoarthritis associated with capitellar OCD is characterized by aggressive osteophyte formation, narrowing of the distal humeral fossa, and enlargement of the radial head, resulting in a different shape from the original contour of the elbow with severe restriction of motion. In contrast, there are rare cases of capitellar OCD combined with sporadic chondrolysis on the whole articular surface of the elbow (Fig. 9).[90] Arthroscopic débridement of the cartilaginous fragments seems to be useful for reducing subacute sporadic chondrolysis, although we have experienced only a small number of such cases.[80] Both aggressive osteophyte formation and subacute chondrolysis can appear anywhere in the elbow joint, suggesting the influence of joint fluid containing some various growth factors and cytokines. Further studies are needed to address and prevent these problematic conditions.

Conclusion

Compressive and shear forces on the growing capitellum can cause subchondral separation, leading to OCD, which is composed of 3 layers: the articular fragment, the gap, and the underlying bone. Subchondral separation can cause ossification arrest (stage IA), followed by cartilage degeneration or delayed ossification, occasionally leading to osteonecrosis in the articular fragment. Articular cartilage fracture and gap reseparation make the articular fragment unstable. APR45 can increase the diagnostic reliability. Coronal CT and MR imaging with an appropriate tilting angle can also increase reliability. Sonography contributes to the detection of early OCD in adolescent throwers on the field. OCD lesions are more unstable and remain unhealed in the central aspect of the capitellum. Cast immobilization has a positive effect on healing for stable lesions. Arthroscopic removal provides early return to sports, although a large osteochondral defect is associated with a poor prognosis. Fragment fixation, OAT, and their hybrid technique have provided better results. Further studies are needed to prevent problematic complications of capitellar OCD, such as osteoarthritis and chondrolysis.

Acknowledgment

Disclaimers:

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