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Juvenile and adolescent elbow injuries in sports

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The treatment of elbow injuries in the skeletally immature athlete continues to increase as participation in organized athletics expands for this group. Juvenile and adolescent athletes are participating and competing at earlier ages and with greater intensity. Specialization or year-round focus in specific sports at younger ages has led to a shift in the etiology of many elbow injuries within this cohort from macrotrauma (eg, fractures and dislocations) to repetitive microtrauma. As a result, the spectrum of injuries commonly seen in skeletally immature athletes has increased at a time when long-term outcomes and less invasive interventions with biologic principles are gaining greater attention. Optimal treatment of elbow injuries in the skeletally immature athlete requires a knowledge of the complex developmental and radiographic anatomy, an understanding of the pathophysiology and natural history of its disorders, and a knowledge of the indications and expected outcomes for conservative and operative management.

Physeal anatomy

The development and growth of the human skeleton can be divided into three stages. Childhood terminates with the appearance of all secondary centers of ossification. Adolescent development terminates with fusion of the secondary ossification centers to their respective long bones, and is followed by young adulthood, which is terminated with the completion of new bone growth and achievement of the final adult skeletal form [1]. Specific elbow injury patterns are commonly observed in association with each stage of growth and development. This finding is most directly due to the skeletal developmental stage of the growing athlete's elbow defining the weakest link in its anatomy. In addition,

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injury patterns are also influenced by sport-specific factors that reflect characteristic forces applied to the elbow and its surrounding structures. Knowledge of the skeletal developmental anatomy of the elbow is critical to identifying and treating frequently encountered injury patterns in juvenile and adolescent athletes.

Skeletal growth at the elbow occurs via six secondary ossification centers that appear at characteristic times in male and female development [1]. At birth, the distal humerus is a single cartilaginous epiphysis encompassing both condyles and epicondyles with only one physis. This epiphysis differentiates into two epiphyses (the capitellum and trochlea) during the first decade, and two apophyses (the medial and lateral epicondyles). In predictable synchrony with the development of these structures, the radial head and olecranon epiphyses develop.

The appearance of the secondary ossification centers about the elbow begins with the capitellum at 1 to 2 years of age. The mnemonic "CRITOE" is often used to remember the order of appearance, with each letter representing an ossification center as follows: C, capitellum; R, radial head; I, internal (medial) epicondyle; T, trochlea; O, olecranon; and E, external (lateral) epicondyle. Appearance of the capitellum at age 1 to 2 is followed by the radial head at approximately age 3, the medial epicondyle at age 5, the trochlea at age 7, the olecranon at age 9, and finally, the lateral epicondyle at approximately age 10 in girls and age 11 in boys. Fusion of these secondary ossification centers also occurs in a sequential, age-dependent order, and the trochlea, capitellum, and lateral epicondyle fuse before physeal closure [2,3]. All of the epiphyses of the elbow are intra-articular, whereas the medial and lateral apophyses are essentially extra-articular. The exception to this is a synovial covering over the anterior aspect of the medial epicondyle that may lead to an effusion with a fracture of this structure. The overall contribution of the distal humeral physes to final limb length is approximately 20% [3]. Age at fusion of these ossification centers is variable (and typically delayed in males compared with females by 1 to 2 years) but occurs approximately as follows: the capitellum, trochlea and olecranon close at 14 years of age; the medial epicondyle at 15 years of age; and the radial head and lateral epicondyle close at approximately 16 years of age.

In addition to the importance of understanding the developmental skeletal anatomy of the elbow joint, orthopedic surgeons treating elbow injuries in juvenile and adolescent athletes are better enabled to understand the pathophysiology of, and provide optimal treatment for, specific conditions with an understanding of the extensive vascular network supplying the developing elbow. The majority of the intraosseous blood supply is provided by the posterior perforating vessels of the recurrent system. Haraldsson described two nutrient vessel types in the lateral condyle of the developing elbow. Each type extends into the lateral aspect of the trochlea, with one entering proximal to the articular cartilage and the other entering posterolaterally at the origin of the capsule [4]. Although these two vessels communicate with each other, they do not do so with the metaphyseal vasculature. Vascular embarrassment of this system theoretically places the lateral aspect of the developing elbow at increased risk for osteonecrosis or similar pathology.

The medial aspect of the trochlea has a separate dual blood supply that is vulnerable to injury with fractures. The lateral vessel of this dual supply enters from the posterior aspect of the humerus and crosses the physis, and the more medial vessel enters via the nonarticular, medial aspect of the trochlea. This dual blood supply of the medial aspect of the trochlea may be responsible for the normal trochlear ossification center's occasionally fragmented appearance on plain roentgenograms of the immature elbow.

Medial epicondyle avulsion fractures

Medial epicondyle avulsions are the most common fractures encountered in the juvenile or adolescent throwing athlete [5]. These are acute injuries that characteristically occur with a single throw, pitch, or other powerful tensile force applied to the medial elbow of adolescent athletes [1–3,5–10]. The mechanism of injury is medial traction resulting from acute valgus stress, coupled with violent flexor-pronator mass muscle contraction, which results in acute failure of the medial epicondylar apophysis. The athlete typically presents with the acute onset of medial elbow pain after an especially hard pitch or throw, and is unable to continue playing. The injury may be accompanied by a crack or pop, and usually occurs in the late cocking or early acceleration phases of throwing. Although uncommon, prodromal symptoms of chronic medial elbow pain may precede the injury, and acute ulnar nerve paresthesias may develop at the time of injury.

On physical examination, patients who have medial epicondyle avulsion fractures have focal tenderness to palpation of the medial epicondyle, frequently accompanied by edema and occasionally by ecchymosis. Pain typically limits elbow extension beyond 15°, and may prevent reliable assessment of stability to valgus stress. Coexistent ulnar collateral ligament rupture is unlikely in the setting of a medial epicondyle avulsion fracture, and valgus instability is usually due to the epicondylar avulsion alone. Although acute rupture of the ulnar collateral ligament must be considered, the weak point in the adolescent elbow's medial bone-tendon interface is the physis of the medial epicondyle [11], and as a result, acute ruptures are more commonly seen in skeletally mature individuals. A high index of suspicion for a spontaneously reduced elbow dislocation is important, and careful assessment of the athlete's range of motion and stability is critical, as is the performance of a thorough neurovascular examination with particular attention to ulnar nerve function.

Radiographic evaluation of the suspected medial epicondyle avulsion fracture includes anteroposterior and lateral plain roentgenograms. These typically reveal a minimally displaced avulsion fracture, with variable physeal widening or epicondylar rotation. Findings may be subtle and, in such situations, the diagnosis may be facilitated by a comparison view of the contralateral elbow, a gravity stress test radiograph as described by Woods and Tullos [12], or a manual stress radiograph (Fig. 1). Displacement of the avulsed fragment is due to the pull of the flexor-pronator muscle mass, and although rare, the fragment may displace into

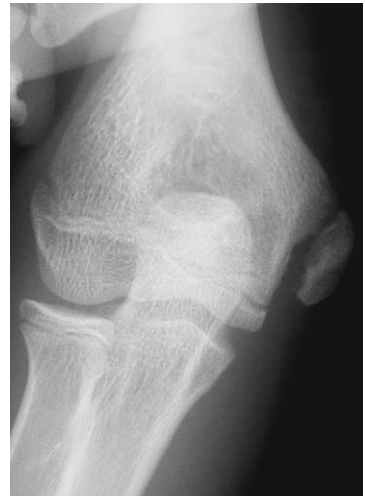


Fig. 1. Manual stress AP radiograph demonstrating a Type I medial epicondyle avulsion fracture in an 11-year-old male.

the elbow joint, particularly when the medial epicondylar avulsion is sustained with an elbow dislocation [13–17]. Assessment of the patient's range of motion is valuable in diagnosing an incarcerated fragment within the joint. Fat pad signs are commonly thought to be unreliable in the radiographic diagnosis, because the degree of hemarthrosis or elbow joint effusion may be limited [18,19]. Although the fracture occurs through the apophyseal plate, the medial epicondylar apophysis does not contribute to the longitudinal growth and alignment of the distal humerus and elbow joint [19,20].

Woods and Tullos [12] presented a three-part classification scheme for medial epicondyle fractures based on patient age and fragment size. Type I injuries occur in children age 14 and under with a fracture fragment typically involving the entire apophysis (see Fig. 1). Displacement and rotation of the fragment are not uncommon. Type II injuries occur in patients 15 and older, and consist of a large fragment that may potentially involve the anterior band of the ulnar collateral ligament. Type III injuries occur in patients 15 and older, and consist of a smaller fragment than Types I or II. It is commonly felt that Type III injuries are more common than Type II, because after age 14 the physis is more likely to be ossified, and a smaller fragment avulses from the medial epicondyle as opposed to the entire apophysis.

Treatment of medial epicondyle avulsion fractures is a subject of some controversy in the literature. Nondisplaced fractures and stress fractures are typically treated conservatively, with a short course of immobilization for comfort followed by activity restriction and physical therapy emphasizing range of motion [21]. Fractures with an incarcerated fragment within the elbow joint are treated operatively if reduction through manipulation [22–24] is unsuccessful. Complete ulnar nerve dysfunction most often requires ulnar nerve exploration

with concomitant open reduction and internal fixation of the fracture. Incomplete ulnar nerve injuries with mild parasthesias or paresis typically resolve over time when treated conservatively [25–27].

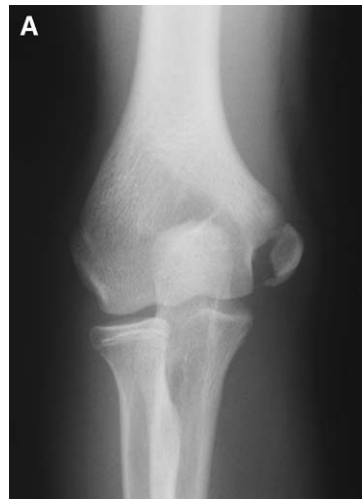
Treatment of displaced medial epicondyle fractures is controversial; numerous authors disagree on the definition of acceptable displacement for conservative management and the long-term results in athletes who have higher demands for elbow function [19,28–32]. Patients who have less than 3 to 5 mm of displacement most often develop an asymptomatic fibrous union that is typically well tolerated [33]. This has led many authors to recommend a nonoperative approach in patients who have less than 3 to 5 mm of displacement, and an absence of associated valgus instability and ulnar nerve dysfunction [14,16,34–37]. Josefsson and Danielsson [33] presented 56 patients treated nonoperatively for widely displaced medial epicondyle fractures. With an average 35-year follow-up, the authors reported good-to-excellent function and range of motion, despite radiographic evidence of fibrous nonunion in 31 of the patients, with no difference between the bony healed and pseudoarthrotic groups. This group, however, did not include or identify a subgroup of throwing athletes, and the authors did note a trend toward an increase in the presence of mild ulnar nerve symptoms in the pseudoarthrotic group [33]. If conservative management is indicated, the authors of this article advocate immobilization at 90° flexion in a long-arm posterior splint with the forearm in moderate pronation for up to 2 to 3 weeks [3,19,38]. A hinged elbow orthosis is applied as soon as the patient's symptoms allow, and is used for up to 6 weeks to achieve optimal range of motion with active-assist range-of-motion exercises. When the fracture site is nontender to palpation, flexor-pronator strengthening is initiated. Once the player is completely asymptomatic and there is radiographic evidence of union, the overhead throwing athlete may begin a strict return-to-throwing program that emphasizes proper mechanics and a gradual return to full participation.

Despite the often adequate results obtained with conservative management of medial epicondyle fractures, a substantial number of authors advocate operative management of fractures with less than 3 to 5 mm of displacement, and most commonly greater than 2 mm of displacement, or with significant fragment rotation, valgus instability, incarceration of the fragment in the joint, or ulnar nerve dysfunction [3,12,39–41]. Ireland and Andrews [31] advocate accepting no displacement with these injuries, and warn against the late sequellae of degenerative radiocapitellar changes with which they may be associated. These considerations become particularly important when dealing with athletes participating in overhead throwing activities or with high functional demands from an injured dominant extremity.

Operative management of medial epicondyle fractures consists of open reduction and internal fixation with one to two cannulated cancellous screws. Rotational instability is best addressed with the latter, if possible. Valgus stability is reassessed intraoperatively following fracture fixation, and if persistent, the surgeon must consider exploration and possible primary repair of the ulnar collateral ligament (Fig. 2) [3,19,42]. Fragments of insufficient size for fixation are optimally

treated with excision [3,12] and primary repair of the ulnar collateral ligament, with or without suture repair of the flexor-pronator muscle mass. If acute ulnar nerve dysfunction is present, exploration and decompression at the cubital tunnel is recommended, but anterior transposition is usually unnecessary.

Postoperative management following open reduction and internal fixation includes a functional hinged elbow orthosis for approximately 6 weeks. If fixation is stable, immediate early range-of-motion exercises are initiated, with the addition of strengthening exercises as comfort allows. When the patient is asymptomatic and clear evidence of radiographic union is present, a progressive



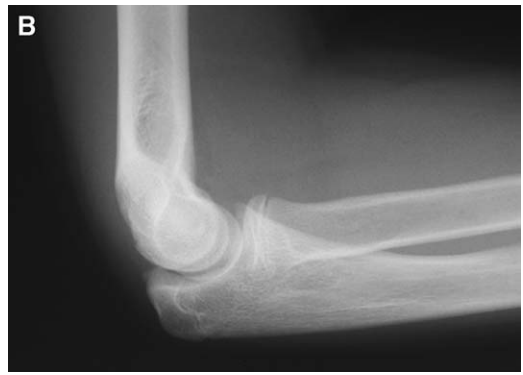
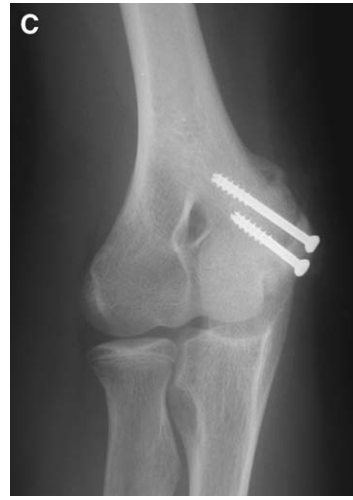


Fig. 2. (*A*) AP radiograph of a displaced Type II medial epicondyle fracture in a 13-year-old male who had persistent valgus instability after open reduction and internal fixation, and was found to have a midsubstance tear of his medial ulnar collateral ligament treated with primary repair. (*B*) Lateral radiograph of displaced medial epicondyle fracture. (*C*) AP radiograph 6 weeks after open reduction and internal fixation with two partially threaded cannulated screws. (*D*) Lateral radiograph 6 weeks after open reduction and internal fixation.



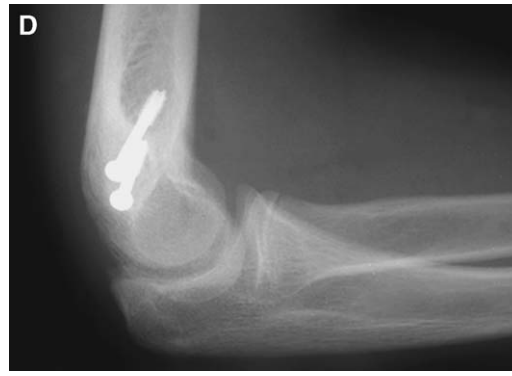


Fig. 2 (*continued*).

return to athletics is allowed. In cases with concomitant ulnar collateral ligament repair, the rehabilitation protocol is modified accordingly.

Medial epicondylar apophysitis

Medial epicondylar apophysitis results from repetitive tensile stress on the medial epicondyle, caused by the flexor-pronator muscle mass and the medial collateral ligament. These valgus stresses result in repetitive microtrauma and ultimately lead to stress fracture failure of the medial epicondylar apophysis [1,38]. Medial epicondylar apophysitis frequently presents with an insidious onset of progressively worsening medial elbow pain that occurs with throwing activities. The athlete characteristically presents with a triad of symptoms that includes pain localized to the medial epicondyle, loss of throwing velocity or

distance, and diminished throwing effectiveness [1,2,43,44]. Pain is exacerbated with throwing, and is most prominent during the late cocking and early acceleration phases, when valgus stress on the elbow is maximal.

Physical examination commonly reveals point tenderness at the medial epicondyle, and a flexion contracture may be present. Swelling is an inconsistent finding, and although there may be pain elicited with valgus stress, frank instability is not present. Radiographic findings are subtle and not uniform, because some patients have normal plain roentgenograms. Most often, a subtle widening of the apophysis is present and is more readily identified with a comparison view of the contralateral elbow (Fig. 3). Less commonly, epicondylar fragmentation or hypertrophy may be present [9,13,45,46]. In a 1965 study by Adams of 80 California Little League pitchers 9 to 14 years of age, 49% demonstrated medial epicondylar fragmentation [9]. A subsequent study by Torg et al of recreational players 9 to 18 years of age in Philadelphia presented a 4% incidence of fragmentation, and attributed the lower incidence to the less intensely competitive nature of the league studied [46].

Treatment of medial epicondylar apophysitis focuses on the elimination of repetitive valgus stress, and therefore requires the cessation of all throwing activities. The authors of this article recommend a minimum of 6 weeks of restriction from throwing. Initial treatment with ice and nonsteroidal anti-inflammatory drugs (NSAIDs) may provide symptomatic relief. Severe cases may require an initial short course of elbow immobilization (7–14 days). If the athlete presents with a flexion contracture on examination, physical therapy should be initiated as soon as tolerable, to emphasize range of motion, muscle stretching, and strengthening. Return to throwing or athletics is only initiated after complete resolution of

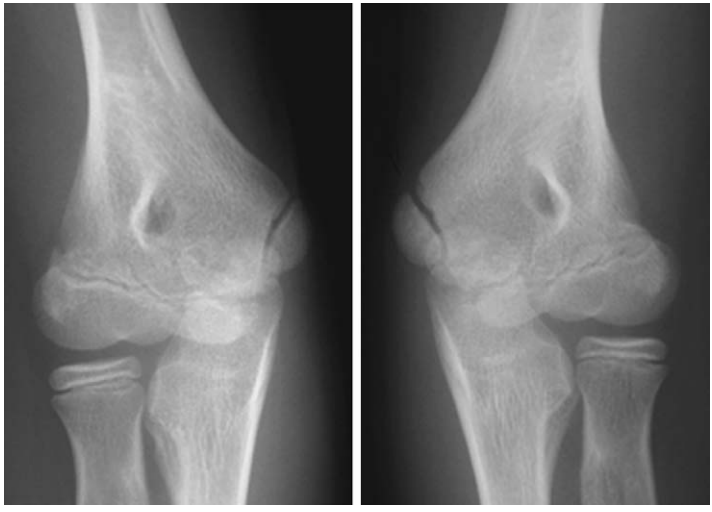


Fig. 3. Comparison AP radiographs demonstrating left-elbow medial epicondyle apophysitis with subtle widening in the throwing arm of a 10-year-old male.

symptoms and the absence of tenderness on physical examination. Demonstration of radiographic healing is not essential before returning to athletics. It is imperative for the overhead throwing athlete to undertake a gradual return, with a strict throwing program that emphasizes proper mechanics. Athletes who recover from medial epicondylar apophysitis may have a recurrence of symptoms; however, this condition typically responds well to conservative, nonoperative management and resolves without a functional deficit.

Olecranon apophyseal injury

The acceleration phase of throwing in the overhead athlete subjects the olecranon to repetitive forceful contraction of the triceps, which may result in olecranon apophyseal injury [19,31,38,47]. These repetitive forceful triceps contractions required for powerful overhead throwing are thought to place a distraction force across the epiphysis, and Gore et al hypothesized that the etiology of olecranon apophyseal injury is a traction apophysitis similar to that which occurs at the medial epicondyle [48]. Other authors have similarly proposed a connection to Osgood-Schlatter disease (in which stress on the tibial tubercle in association with rapid growth and musculotendinous imbalance results in apophysitis), and separated olecranon secondary ossification centers with persistence into adulthood (Fig. 4) has been described in the literature [31,49,50]. Patients may present with complaints of acute (more common) or chronic pain at the posterior aspect of the elbow, frequently accompanied by swelling and decreased range of motion. Physical examination findings include olecranon tenderness to palpation and pain with resisted elbow extension.

Radiographs characteristically demonstrate widening or fragmentation of the olecranon physis, as well as sclerosis in comparison to the contralateral elbow [51]. Torg and Moyer in 1977 presented a case of nonunion through a stress fracture of the olecranon epiphyseal plate, and postulated that the repetitious stress of pitching prevented physeal closure in addition to causing a stress fracture through the epiphyseal plate [52]. Pavlov et al [51]and Torg and Moyer [52] proposed that once the epiphysis is distracted, continued stressful activity may prevent normal closure and result in olecranon epiphyseal nonunion. Pavlov et al [51] presented a report of two cases with radiographic and histologic confirmation of nonunion of the olecranon epiphysis in adolescent baseball pitchers. Whether a stress fracture or simple traction apophysitis is present, the diagnosis of olecranon apophyseal injury is not excluded by normal radiographs, and a high index of suspicion is required in patients with the appropriate clinical findings. Technetium bone scan has been suggested as an adjunct in confirming nondisplaced fractures or subtle stress fractures [53].

Treatment of olecranon apophyseal injury is determined by the degree of fragment displacement, chronicity of the injury, and the athlete's symptoms. Initially, treatment consists of activity modification, NSAIDs therapy, ice, and physical therapy if limited range of motion is present. An adequate response to

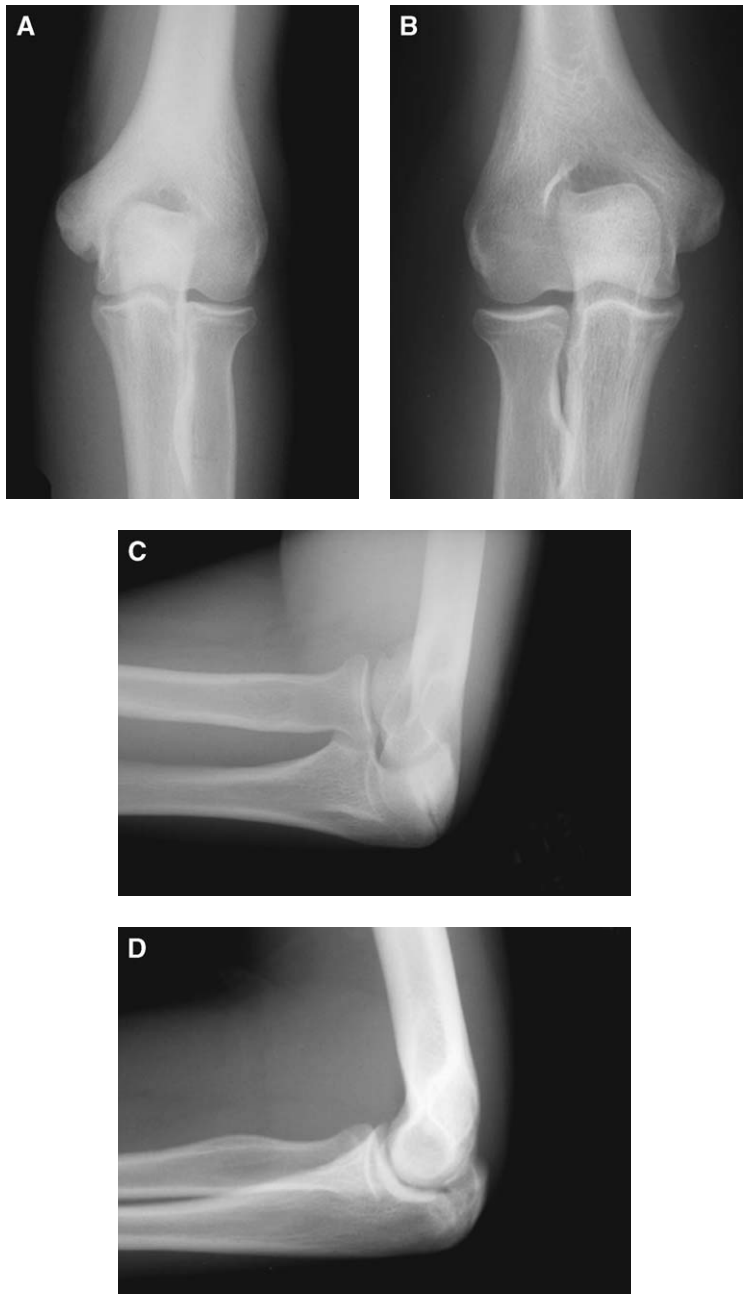


Fig. 4. (*A*) AP radiograph demonstrating persistent olecranon apophysis in the throwing arm of a 16-year-old male. (*B*) Contralateral AP comparison radiograph. (*C*) Radiocapitellar radiograph of persistent olecranon apophysis. (*D*) Lateral radiograph of persistent olecranon apophysis. (*E*) Contralateral lateral comparison radiograph.

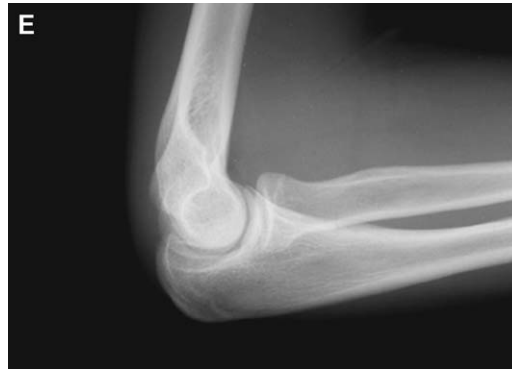


Fig. 4. (*continued*).

this form of conservative management is normally seen within approximately 4 to 6 weeks; however, as described above, physeal stress fractures and partial triceps tendon avulsions have been described in the setting of chronic olecranon apophysitis, and persistence of the olecranon apophysis into adulthood may occur, all of which may require surgical management (see Fig. 4) [3,31,50,52,54–57]. Surgical treatment is indicated for patients who have persistent symptoms and radiographically documented failure of apophyseal closure after 3 to 6 months of conservative treatment. The authors of this article prefer surgical fixation with a single cancellous screw (Fig. 5). Postoperatively, patients are immobilized for approximately 10 days, and subsequently placed in a physical therapy program of active elbow flexion and passive extension. No forceful, active extension is permitted for 6 weeks.

Ulnar collateral ligament injury

Ulnar collateral ligament injuries of the elbow are uncommon in the juvenile and adolescent athlete [31]. Chronic attritional tears in this cohort are exceptionally uncommon, and when an ulnar collateral ligament rupture does occur, it is typically the result of an acute failure [58,59]. Patients therefore complain of pain and the acute onset of inability to continue participating in their sport (most commonly overhead throwing; eg, pitching). Physical examination typically reveals a flexion contracture; point tenderness to palpation medially, usually distal to the medial epicondyle; and pain and instability with Jobe's valgus stress test, the moving valgus stress test, or the milking maneuver. Radiographs are imperative to evaluate for a possible medial epicondyle avulsion fracture. Valgus stress antero-posterior (AP) radiographs, as described previously, with gravity or manual manipulation, can be helpful in the assessment of instability (Hughes and Paletta, submitted for publication, 2002) [40]. Although the exact amount of medial opening with an ulnar collateral ligament tear is a subject of controversy, a relative increase of 2 mm or more in comparison with the contralateral elbow is

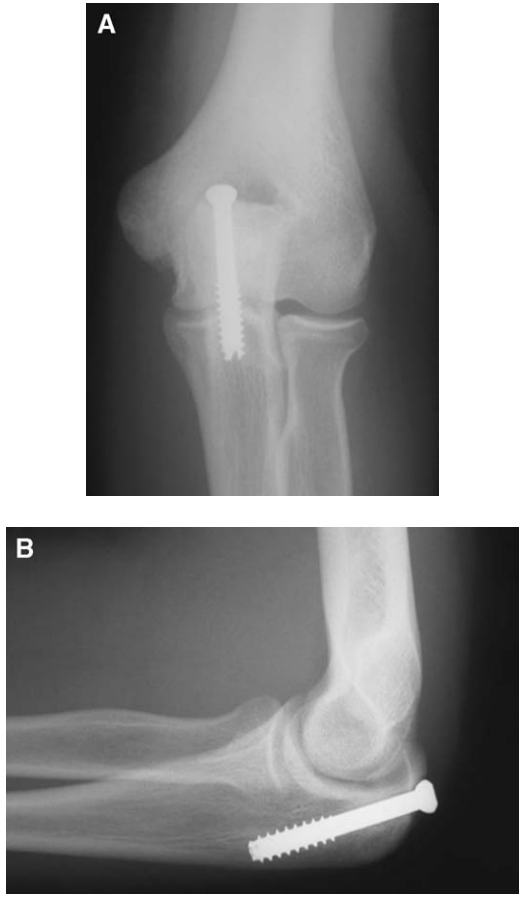


Fig. 5. (*A*) AP and (*B*) lateral radiographs 12 weeks after open reduction and internal fixation with one partially threaded cannulated screw.

considered pathologic. MR arthrography with gadolinium contrast can be of great assistance in diagnosing and documenting the presence of an ulnar collateral ligament tear.

Treatment of an ulnar collateral ligament tear in the juvenile or adolescent athlete initially consists of a short period of immobilization for pain control, NSAIDs, and ice. Upon recovery from the initial pain of the injury, physical therapy is initiated, with an emphasis on regaining optimal motion and maintaining strength. The authors of this article typically use a hinged elbow brace to protect the patient from valgus stress. Approximately 6 weeks after the initial injury, stability of the elbow is reassessed with physical examination and stress radiographs. In the young athlete who has a complete ulnar collateral ligament tear and instability and who wishes to return to participation in a sport that places valgus stress across the elbow, surgical intervention is recommended. Operative

management is also considered in patients who have a documented partial tear and who lack instability, but have persistent medial elbow pain with activity for 3 months or greater of rest and rehabilitation. Operative management may consist of direct repair of the ulnar collateral ligament in this cohort in the rare setting of an acute avulsion injury [31]. If there is any question regarding the potential stability of a direct repair, then reconstruction with the use of autograft tendon (preferably palmaris longus) should be performed according to the same principles that guide ulnar collateral ligament reconstruction in adults. When performing a medial ulnar collateral ligament reconstruction in patients with open physes, premature closure of the medial epicondylar apophysis is a potential issue; however, this typically is not clinically relevant, because premature closure will not affect the longitudinal growth of the distal humerus.

Panner's disease

First described in 1927 [60,61], osteochondrosis of the capitellum was identified in the context of its similarity to Legg-Calvé-Perthes disease of the hip on plain roentgenograms. Panner's disease is defined as a focal lesion of the capitellar subchondral bone and its overlying articular cartilage, characterized by a disorder of the capitellar ossification center, which begins as degeneration or necrosis and is followed by recalcification or regeneration of the ossific nucleus [3,4,38,59,62–65]. Panner's disease is the most common cause of lateral elbow pain in young children, and is most commonly seen in patients less than 10 years of age. The etiology has not been determined; however, a proposed mechanism involves alteration in the vascularity of the developing capitellum [38,66].

Although Panner's disease is similar to osteochondritis of the capitellum, the age of presentation and prognosis of each is different. Panner's disease characteristically occurs in children younger than 10 years old, and its natural history involves a benign, self-limited process, with eventual restoration of the normal capitellar size, contour, and appearance in the overwhelming majority of patients. True collapse of the subchondral bone and persistent deformity are rare. Osteochondritis dissecans (OCD), in comparison, is a common cause of lateral elbow pain in children and adolescents that characteristically presents between 11 and 16 years of age, and is more frequently seen in athletes who sustain repetitive trauma through valgus stress and lateral compression across the elbow (eg, baseball players and gymnasts) [64,67]. A comprehensive discussion of OCD follows below. The clinical distinction between Panner's disease and OCD is of great importance, in that the natural histories significantly affect treatment options.

Children presenting with Panner's disease typically complain of a vague, dull, and aching pain of the lateral elbow that is exacerbated with activity and relieved by rest. It is frequently accompanied by a loss of elbow motion and subjective stiffness. Physical examination may reveal lateral tenderness to palpation at the radio-capitellar joint, with a 10° to 20° flexion contracture. Crepitus is not uncommon, but edema or effusion is rare. Plain radiographs typically reveal diffuse

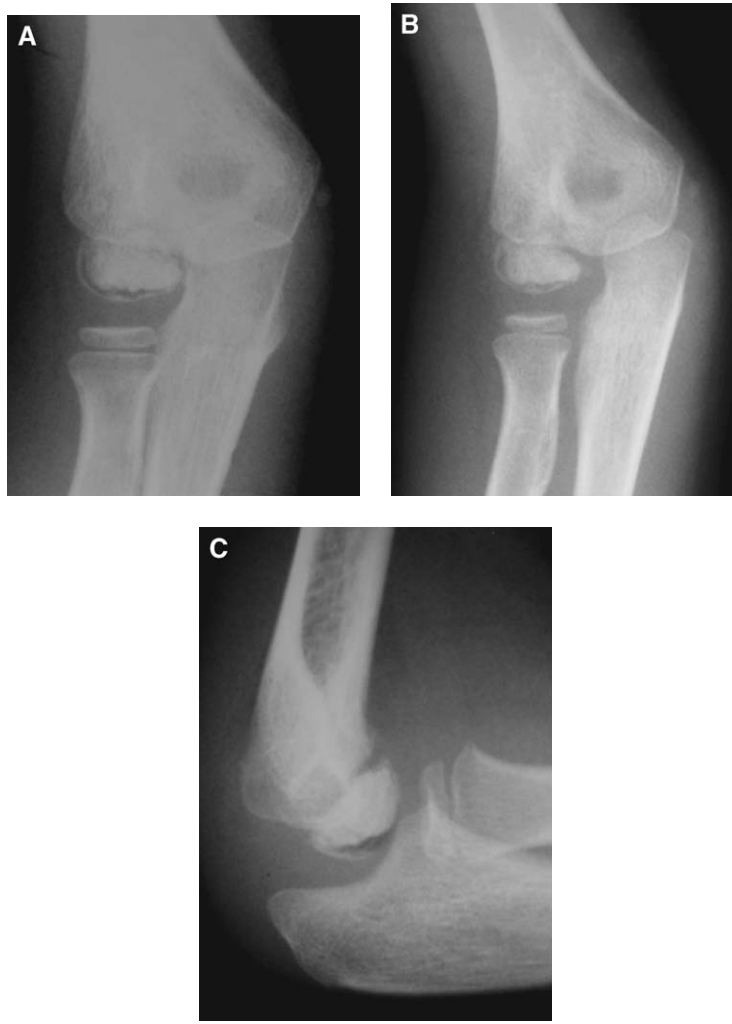


Fig. 6. (*A*) AP, (*B*) modified AP, and (*C*) lateral radiographs demonstrating Panner's disease in a 9 year-old male.

involvement of the central aspect of the anterior capitellum, with an irregular appearance and a variable area of rarefaction and fragmentation; however, the entire ossific center of the capitellum may be involved (Fig. 6).

The natural history of Panner's disease is self-limited and normally results in resolution, with restoration of the normal appearance, contour, size, and subchondral architecture [60,65,68–70]. Uncommonly, late deformity and collapse can occur. Arthroscopic treatment of Panner's disease has been described [63]; however, the consensus in the literature supports the conservative management of this condition, with activity modification, rest, avoidance of valgus stress, ice,

NSAIDs, emphasis on maintenance of range of motion, and temporary posterior splinting in patients who have severe symptoms. A prolonged period of healing up to 3 years is characteristic, and excellent long-term radiographic and clinical results can be expected.

Osteochondritis dissecans

First described in the knee by Konig in 1889, OCD was initially presented as a condition with an appearance suggestive of a subchondral inflammatory process causing dissection of a fragment of overlying articular cartilage [61,71];. however, the accepted term is a misnomer, because the condition has never been shown to involve a true inflammatory process [72], and the term osteochondritis technically refers to inflammation of bone and cartilage. Nevertheless, OCD is a condition involving focal injury to subchondral bone that results in loss of structural support for the overlying articular cartilage. As a result, degeneration and fragmentation of the articular cartilage and underlying bone occur, often with the formation of loose bodies. The condition has been described in the capitellum, trochlea, wrist, femoral head and condyles, distal tibia, patella, and talus [73–75].

As described above, OCD is a common cause of lateral elbow pain in children and adolescents that characteristically occurs between 11 and 16 years of age, and is more frequently seen in athletes who sustain repetitive trauma through valgus stress and lateral compression across the elbow (eg, throwing athletes and gymnasts) [9,40,64,66,76–78]. In throwers, the radiocapitellar joint is subjected to compressive forces during repetitive valgus loading [39], and the gymnast's elbow is subjected to repetitive compressile and shear forces when the upper-extremity functions as a weight-bearing joint that receives up to 60% of the force from compressive axial loads [79–81]. These activities are thought to affect the tenuous blood supply to the capitellum of the developing elbow.

The precise etiology of capitellar OCD remains unclear; however, most authors believe it is due to a combination of repetitive microtrauma in the setting of a tenuous blood supply in the developing elbow [3,57,74,82]. Haraldsson [4] provided support for the ischemic theory of OCD in demonstrating that the rapidly expanding capitellar epiphysis in the developing elbow receives its blood supply from one or two isolated transchondroepihyseal vessels that enter the epiphysis posteriorly, and therefore function as end-arteries passing through the cartilaginous epiphysis to the capitellum. In addition, the metaphyseal vascular anastomoses do not make significant contributions to the capitellum until approximately 19 years of age. Histopathologically, OCD is consistent with an ischemic etiology, because findings typically reveal subchondral osteonecrosis. A genetic predisposition to the development of OCD has been proposed in the literature [83–85]; however, convincing scientific evidence of OCD as a heritable condition does not currently exist.

The traumatic theory of OCD, as described above, is based on the high prevalence of the condition in the dominant upper extremities of athletes who

sustain repetitive valgus loading and compressile forces across the radiocapitellar joint. The repetitive microtrauma caused by these forces has been proposed to weaken the capitellar subchondral bone and result in fatigue fracture [77]. Should failure of osseous repair mechanisms occur in this setting, an avascular portion of bone may then undergo resorption, with further weakening of the subchondral architecture. This coincides with the characteristic rarefaction typically seen at the periphery of the lesion. The altered subchondral architecture can no longer support the overlying articular cartilage, and renders it vulnerable to shear stresses that may lead to fragmentation. Further support for the traumatic theory of OCD has been provided by Schenk and associates in their demonstration of the significant differences in the cartilage topography and biomechanical properties of the radial head and capitellum, which create a mismatch between the stiffer central aspect of the radial head and the lateral aspect of the capitellum and may contribute to the pathogenesis of capitellar OCD [82]. In addition to these data, variable enlargement and irregularity of the radial head may occasionally develop in patients who have OCD, and have been reported to occur with variable frequency [75,86–88].

The clinical presentation of capitellar OCD is typically characterized by the insidious onset of poorly localized, progressive lateral elbow pain in the dominant arm of an adolescent athlete who participates in the aforementioned activities that place repetitive stress on the radiocapitellar joint. It is important to note, however, that prodromal pain is not always present. Typically, pain is exacerbated with activity and relieved by rest. In advanced cases in which a fragment has become unstable or loose body formation has occurred, mechanical symptoms of elbow locking, clicking, or catching may be present and may provide important data regarding treatment options. Physical examination often reveals tenderness at the anterolateral aspect of the elbow, with or without swelling and crepitus. In the early stages of OCD, no motion loss may be appreciated [89]; however, loss of extension is the most common limitation, and decreased forearm rotation is occasionally present [2]. Provocative testing includes the active radiocapitellar compression test, which consists of forearm pronation and supination with the elbow in full extension in an attempt to reproduce symptoms [90].

Diagnostic evaluation begins with plain radiographs, which typically demonstrate the classic radiolucency or rarefaction of the capitellum in addition to irregularity or flattening of the articular surface. The lesion frequently appears as a focal rim of sclerotic bone surrounding a radiolucent crater, with rarefaction located in the anterolateral aspect of the capitellum (Fig. 7). Depending on the chronicity of the lesion, diagnosis may be facilitated by obtaining AP radiographs with the elbow in 45° of flexion, as described by Poehling [67] and Takahara et al [89]. Despite technique, plain radiographs may not reveal lesions in the earlier stages of the condition, may be nondiagnostic in up to half of cases, and have been suggested to be inadequate in evaluating the chondral component of OCD lesions [3,91]. In advanced cases, articular surface collapse, loose bodies, subchondral cysts, radial head enlargement, and osteophyte formation may be seen (Figs. 8, 9). The traditional classification adapted from Minami's description

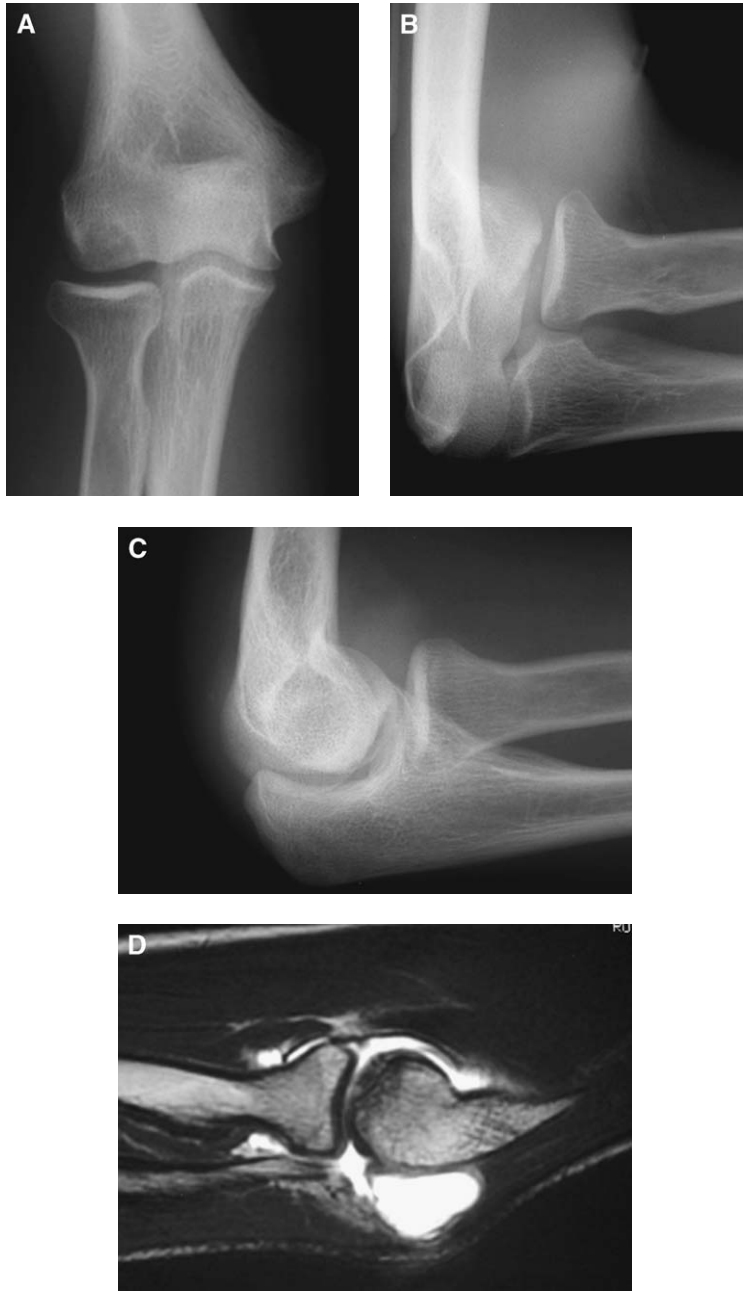


Fig. 7. (*A*) AP, (*B*) radiocapitellar, and (*C*) lateral radiographs demonstrating a Type IB osteochondritis dissecans lesion in a 15-year-old male pitcher. (*D*) T2-weighted, gadolinium-enhanced MR arthrogram of patient in Fig. 7A, demonstrating intact articular surface with disruption of the subchondral architecture and subchondral edema.

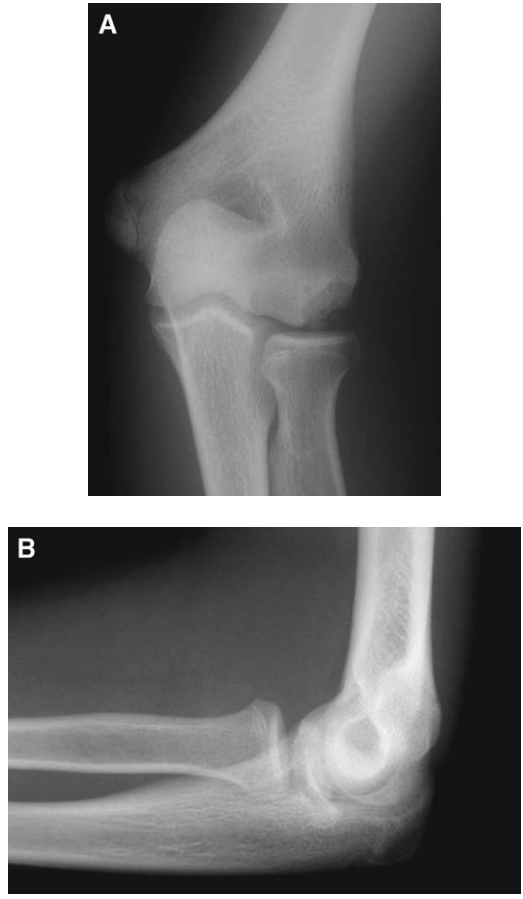


Fig. 8. (*A*) AP and (*B*) lateral radiographs demonstrating a Type III OCD lesion with fragmentation and loose body formation in a 21-year-old male.

involves three grades based on an AP view of the elbow as follows: Grade I, translucent shadow in central or lateral capitellum; Grade II, clear zone or split line between the subchondral bone and the lesion; and Grade III, loose bodies identified [64,92,93].

Further diagnostic imaging of OCD lesions primarily consists of MRI, although ultrasonography [88] and bone scintigraphy are less commonly used [77]. MRI is especially valuable in assessing the cartilage overlying the OCD lesion [88,90,94] as well as in diagnosing OCD in it early stages. Takahara et al [89] presented data suggesting that early OCD lesions may be diagnosed with the demonstration of a low signal change on T1-weighted images of the superficial capitellum, despite no evidence of changes on T2-weighted images. Intervening fluid between a fragment and the capitellum on T2-weighted images is indicative of detachment. Controversy exists over the utility of contrast-enhanced MR

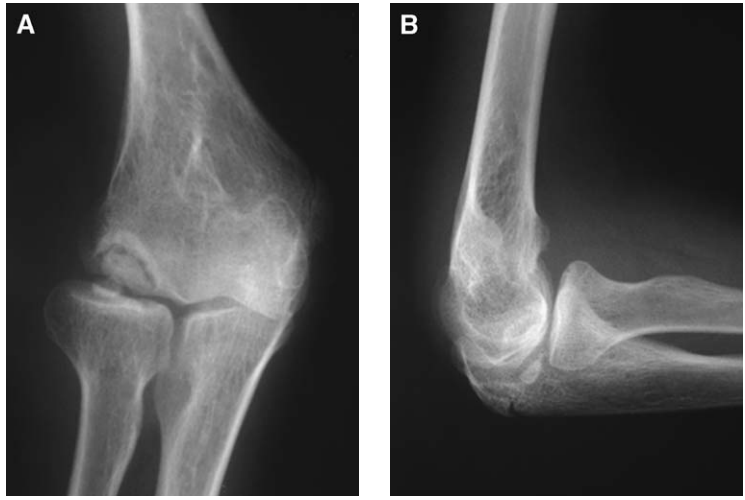


Fig. 9. (*A*) AP and (*B*) lateral radiographs demonstrating advanced OCD (Type IV) with fragmentation, arthrosis, osteophyte formation, and radial head enlargement in a 24-year-old male.

arthrography [95,96]; however, this technique can potentially provide additional information regarding the status of the articular cartilage (see Fig. 7D) and identification of loose bodies. Limitations of MRI for evaluation of OCD lesions include a possible decreased ability to reliably assess radial head involvement [90]. In addition, with cases of OCD in which clear indications for arthroscopy exist, MRI has been shown to be of questionable added benefit and may be unnecessary [90].

The natural history of capitellar OCD is difficult to predict, and no reliable criteria exist for predicting which lesions will collapse with subsequent joint incongruity and which will go on to heal without further sequellae. If healing does take place, this usually occurs by the time of physeal closure. As described previously, should healing not take place, repetitive microtrauma and shear stresses to the articular surface of a lesion that has lost its supportive subchondral architecture may result in further subchondral collapse and deformation with joint incongruity, as well as articular cartilage injury, fragmentation, and loose body formation [1,3,75,77]. In advanced cases, degenerative changes accompanied by a decreased range of motion are likely to develop. As a result, staging and appropriate treatment of OCD lesions in the adolescent athlete is not only important for a potential return to same level of competition, but imperative for optimal long-term elbow function with activities of daily living.

A universally accepted classification system for OCD lesions does not exist; however, attempts have been made to describe and stratify lesions based on a combination of clinical examination data, diagnostic imaging, and arthroscopy findings [69,89]. Baumgarten et al [69] presented an arthroscopic classification adapted from Ferkel et al's arthroscopic classification of talar OCD lesions [97].

Based on 17 elbows in 16 patients, lesions were stratified into five types, with treatment recommendations suggested accordingly (Table 1).

Another useful classification system presented by Petrie and Bradley [65] expands upon the traditional system and combines clinical, diagnostic imaging, and arthroscopic data to categorize lesions into five types, based on the status of the articular cartilage and stability of the underlying subchondral bone [3,59,89]. In this system, early lesions (Type I) are subtyped to differentiate between those with subtle findings on diagnostic imaging (eg, normal plain radiographs and low-signal changes on T1-weighted images with normal T2-weighted images) and those with more classical findings. This subdivision is based upon the work of Takahara et al [89], which demonstrated a subset of nascent lesions that may have a better prognosis with conservative management following early diagnosis. Therefore, Type IA lesions represent OCD in its earliest stages, and diagnostic imaging of this group is remarkable only for low-signal change on T1-weighted images or subtle changes identified on ultrasound. The articular cartilage is intact and no significant loss of subchondral bone stability has developed. Type IB lesions are intact lesions, at significant risk for becoming unstable, that display the more typical radiographic findings of OCD on diagnostic imaging, including capitellar flattening, rarefaction, and sclerosis (Fig. 10), as well as increased signal on T2-weighted MR imaging. The articular cartilage is intact; however, the architecture of the underlying subchondral bone is unstable, placing the lesion at significant risk for progression. The use of intra-articular contrast for MRI of these lesions may dramatically enhance assessment of the overlying articular cartilage and degree of instability.

The initial treatment of Type I lesions with a viable, stable fragment consists of conservative management with rest, activity modification, consideration of immobilization (less than 2–3 weeks, depending on symptoms), ice, NSAIDs, and early active-assist range of motion. Serial radiographs are obtained at 10 to 12 week intervals to monitor healing, and activity modification is strongly recommended until the radiographic appearance of revascularization [67] and healing [1,3,64,98,99]. Radiographic findings of OCD may persist for several years [61,88,93], and as a result, after conservative management, the most critical

Table 1
Arthroscopic classification and treatment recommendations for talar OCD lesions

Type	Articular cartilage status	Treatment recommendations
I	Smooth, soft, ballotable	Observation or drilling
II	Fissuring or fibrillation	Resection of degenerative cartilage
III	Exposed bone with fixed osteochondral fragment	Fragment excision
IV	Loose, nondisplaced fragment	Fragment excision
V	Displaced fragment with loose body formation	Loose body removal, followed by sclerotic bone debridement to bleeding bone surface and synovial or osteophyte debridement

Data from Baumgarten TE, Andrews JR, Satterwhite YE. The arthroscopic classification and treatment of osteochondritis dissecans of the capitellum. Am J Sports Med 1998;26(4):520–3.

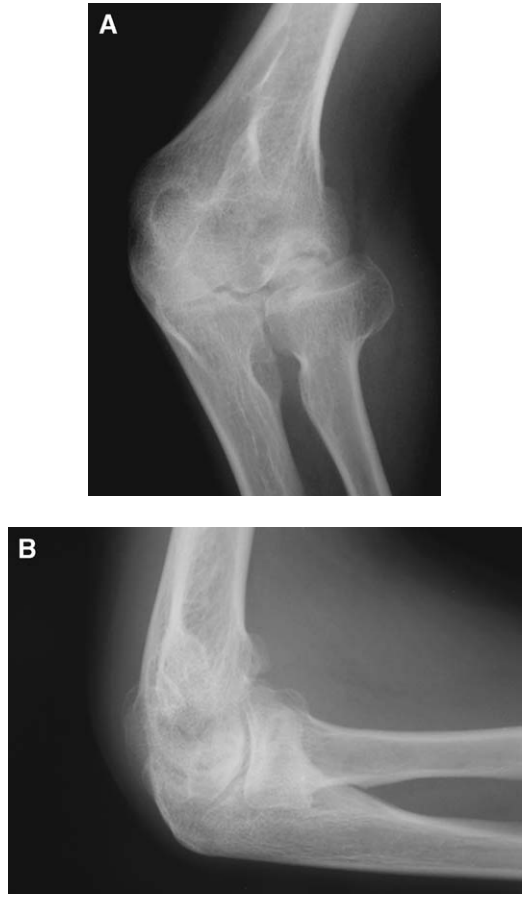


Fig. 10. (*A*) AP and (*B*) lateral radiographs demonstrating a Type IB OCD lesion in a 15-year-old male pitcher.

issue regarding an athlete's ability to return to sports is symptom resolution. Unfortunately the results of conservative treatment of OCD, in comparison with Panner's disease, are not uniformly successful [59,87,93,100,101]. Takahara et al [94,101] presented the results of nonoperative management of early OCD lesions with an average follow-up of 5.2 years, and reported that over half of these patients had pain with activities, and fewer than half of the lesions demonstrated radiographic improvement. Careful consideration and physician–patient/family counseling is imperative regarding the clinical scenario, the athlete's motivation to return to competition, and the levels at which he or she may expect to be able or unable to compete. Surgical indications for operative management of Type I lesions include radiographic evidence of lesion progression and failure of symptom resolution despite a 6-month trial of a conservative, nonoperative regimen. Arthroscopic examination, débridement as needed, and drilling or

microfracture of the OCD lesion (with or without in-situ pinning) constitute the preferred surgical treatment.

Type II lesions are open, unstable lesions characterized by cartilage injury and instability, as well as collapse or disruption of the subchondral bone architecture. These lesions are frequently flap lesions that characteristically present with more advanced radiographic changes (including a well-demarcated fragment surrounded by a sclerotic margin), and some authors have suggested that Type II lesions have an increased propensity for decreased viability [64,95]. Currently, a clear consensus does not exist regarding operative management with fragment excision or open reduction and internal fixation. Most authors advocate excision of displaced fragments, potentially accompanied by drilling or microfracture [64,67,68,75,77,102]. Critical considerations in operative planning include the size and integrity (viability) of the fragment, the subchondral architecture on the fragment and the opposing bony bed, the potential for anatomic restoration of the articular surface, and the method of fixation if attempted. The literature to date includes descriptions of metallic screw, bioabsorbable screw, Kirschner wire, bone peg, and dynamic staple fixation [64,92,103–107]. Some surgeons have also used osteoarticular allograft techniques in treatment of more advanced lesions, but data regarding experience with this method are limited [108,109].

Type III lesions are distinguished from Type II by the presence of loose bodies, which indicate a more advanced, and likely long-standing, lesion. Contrast MR arthrography is the diagnostic imaging study of choice for identification of loose bodies. The cartilaginous bodies may become enlarged via synovial nutrition of the chondrocytes, whereas the host bed is often decreased in size due to fibrous tissue in-growth [89]. Treatment of Type III chronic lesions primarily consists of arthroscopic examination, loose body removal, débridement, and drilling or microfracture of the OCD lesion, because the chronicity of the lesion and loose body formation leave no role for open reduction and internal fixation. Acutely displaced fragments of sufficient size and integrity for stable fixation are often given serious consideration for treatment as such, however.

The diagnosis of a Type IV lesion constitutes concomitant radial head involvement, which, as described previously, accompanies capitellar lesions with variable frequency. Although simple radial head enlargement (less than ~30% of the articular surface) may require no additional formal treatment, the large degenerative ''bipolar'' lesions that may develop with severe degenerative changes at the radiocapitellar joint constitute a serious problem, because radial head replacement and radial head resection in this population are unlikely to have favorable results.

Reports in the literature regarding follow-up of the conservative and surgical management of OCD are difficult to compare and interpret collectively, because there is a lack of uniformity in their methods. The lack of a universally accepted classification system, the limited numbers of patients in most series, and the disparities present in description of age at presentation, symptomatology, lesion size, location, stability, and viability, method of diagnostic imaging used, surgical technique, and length of follow-up make drawing conclusions from the currently

available literature difficult. Nevertheless, familiarity with results of the literature to date is imperative to treating patients who have OCD and to the continued progression of knowledge regarding it. A general consensus exists in the literature regarding the need to ideally limit continual high-stress loading of the radiocapitellar joint in patients treated (even successfully) who have OCD, in order to prevent the deterioration of the frequently obtained short-term favorable results. As a result, most pitchers are counseled to transition to other positions, and gymnasts are advised of the difficulty in returning to continued high-level competitive gymnastics.

In one of the longest follow-up studies available in the elbow OCD literature, Bauer et al [110] presented the results of 31 patients (23 of whom were treated surgically with lesion or loose body excision) who had capitellar OCD followed for an average of 23 years. At follow-up, the most common complaints were decreased range of motion (average 9° flexion loss, 2° extension loss, and 6° pronation/supination loss) and pain with activity. Radiographic evidence of degenerative changes involving the elbow joint was present in 61% and radial head enlargement in 58%.

McManama et al [68] presented data on 14 adolescents who had radiocapitellar OCD lesions treated with excision via a lateral arthrotomy, with average follow-up of 2 years. Lesions were not sized, but 93% had good or excellent results. Jackson et al reported on the roughly three year follow-up of OCD lesions in ten female gymnasts treated primarily with curettage of loose cartilage, drilling, and loose body excision [80]. All of the patients reported symptomatic relief; however, only one patient returned to competition, and did so with discomfort. Average loss of extension at follow-up in this series was 9°, which is consistent with other reports in the literature.

Ruch et al presented the follow-up at an average of 3.2 years after arthroscopic débridement alone for management of elbow OCD in 12 adolescents [111]. The average flexion contracture improved 13° (23° preoperatively to 10° postoperatively). All patients had capitellar remodeling on follow-up radiographs, and approximately 42% had associated radial head enlargement. Ninety-two percent of patients in this series were highly satisfied with minimal symptoms, and of note, five patients (42%) had a triangular lateral capsular avulsion fragment (seen radiographically but not at arthroscopy), which had a statistically significant association with a worse subjective outcome. Baumgarten et al [69] presented the aforementioned arthroscopic classification system of elbow OCD along with average 4-year follow-up (range: 24–75 months) on 17 elbows treated in 16 patients. Lesion size and age were not revealed in this study; however, several interesting observations at follow-up came from this series: average flexion contracture improvement was 14° (19° preoperatively to 5° postoperatively), approximately 24% had pain, seven of nine (78%) throwers and four of five (80%) gymnasts were able to return to sport, and no patient had demonstrable degenerative joint disease.

Takahara et al [94] presented a series of 53 patients, 14 of whom were treated nonoperatively, and 39 of whom were treated surgically, in the first study

to correlate lesion size with outcome. Average follow-up in this study was 12.6 years, and lesion chronicity had no value in outcome prediction; however, poor radiographic outcomes were predicted by early degenerative joint disease and large lesions (greater than 70% of the capitellum with a defect angle of 90°). The future treatment options for OCD are likely to change significantly with continued investigation regarding the diagnosis of fragment viability; the stability and optimal outcomes of metallic, bioabsorbable, and bone fixation methods; and the potential for osteochondral replacement with osteoarticular allograft, recombinant human bone morphogenetic protein-impregnated collagen sponges, and chondrocyte-impregnated collagen bilayer techniques.

Summary

Elbow pathology in skeletally immature athletes continues to increase as younger age groups enter competitive play and with the increased intensity illustrated by single-sport specialization. Advances in arthroscopy and imaging technology have significantly contributed to the diagnosis and treatment of these disorders. While clinical outcomes studies have begun to shed light on the natural history of many of these disorders to clarify operative indications, this is an area in great need of further research. As operatives techniques continue to expand and enhance surgical intervention, these studies will become critical in assessing long-term outcome which is imperative in determing the optimal treatment for this patient population.

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