Aseptic Osteonecrosis of the Humeral Head After Anterior Shoulder Dislocation

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Abstract

Anterior shoulder dislocation is a very common trauma and the main complications are well documented. We report a case of aseptic osteonecrosis of the humeral head following an isolated episode of anterior glenohumeral dislocation without fracture that, to our knowledge, has never been reported in the literature.

A 17-year-old male patient sustained an anterior glenohumeral dislocation following a sport accident. It was managed by reduction and immobilization. A radiologically identified aseptic osteonecrosis appeared 6 months later. The instability had been arthroscopically treated. At a follow-up of 4 years, the osteonecrosis has been stabilized leaving a mild arthrosis with stiffness, but without pain.

Key Words

Shoulder dislocation · Trauma · Humeral head osteonecrosis

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Introduction

Anterior shoulder dislocation is very common and constitutes 11% of all shoulder traumas [1]. The main complications are well documented, notably the risk of rotator cuff rupture in older patients and the risk of recurrence in younger patients [2].

We report a case of aseptic osteonecrosis of the humeral head following an isolated episode of anterior glenohumeral dislocation without fracture that to our knowledge has never been reported in the literature.

Patient and Method

A 17-year-old left-handed Caucasian male photography student was admitted to our service following complications from trauma to the right shoulder. The patient (height 165 cm, weight 64 kg) is an amateur athlete (soccer, moto-cross), with no hyperlaxity. In his medical history, only a moderate familial hypercholesterolemia was noted.

Following a football accident (direct postero-anterior shock due to a fall on the shoulder), the patient suffered a right glenohumeral antero-inferior sub-coracoïd dislocation without any initial neurovascular or osseous complications (Figure 1a). Initial reduction using external maneuvers (traction, abduction and internal rotation, one single attempt) was immediately performed in another hospital under general anesthesia, 3 h later (Figure 1b). The shoulder was immobilized by strapping the elbow to the body for 4 weeks prior to a follow-up consultation. After that, physical rehabilitation was prescribed.

At 6 weeks, overall active abduction mobility was measured to be 80° without a painful arc. At 6 weeks, frontal and Lamy's tangential scapular profile X-rays of the shoulder did not reveal any irregularities (Figure 2). At 3 months, overall active abduction mobility was 150°, justifying further rehabilitation.

At 6 months, the pain increased and overall active mobility was no more than 90°. At that time, frontal and Lamy's tangential scapular profile X-rays (Figure 3) revealed a new subchondral bone lesion located at the center of the humeral head, measuring $20 \times 9 \times 10$ mm with a heterogeneous rounded osteolytic center surrounded by a peripheral osteocondensation zone. The humeral head had lost its sphericity in this zone. MRI showed a hyposignal on T1 and T2

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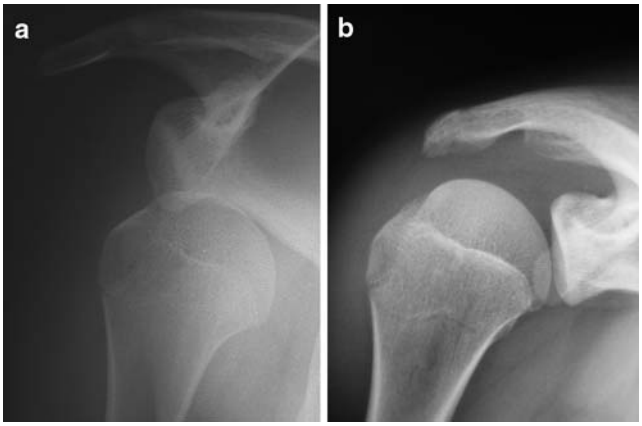

Figure 1. True antero-posterior view of the shoulder. a) Initial antero-inferior shoulder dislocation. b) View immediately after reduction: no particular lesion.

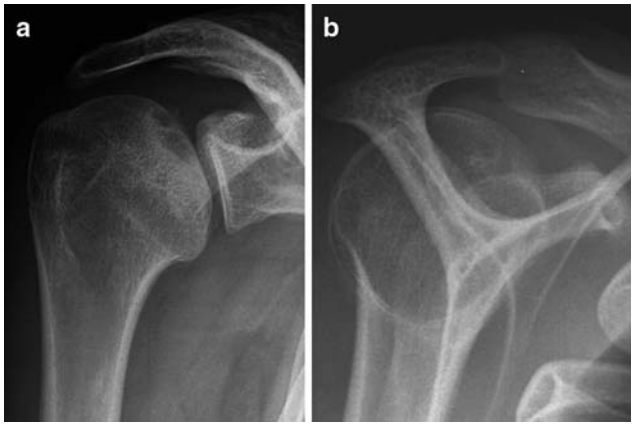

Figure 3. Radiographic control at 6 months: head deformation and osteolysis. a) True antero-posterior view of the shoulder. b) Y view of the shoulder.

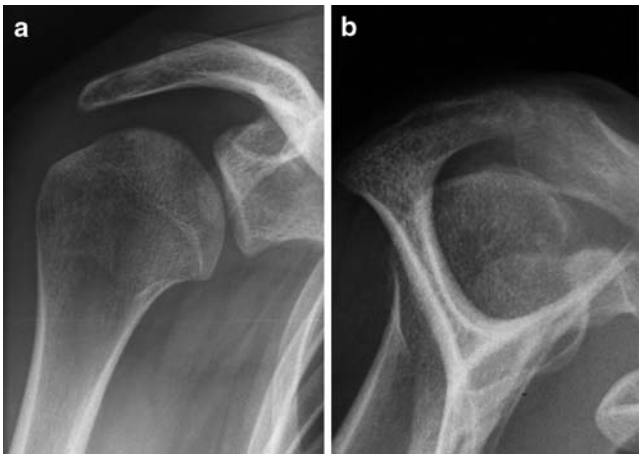

Figure 2. Radiographic control at 6 weeks, considered initially as normal. a) True antero-posterior view of the shoulder. b) Y view of the shoulder.

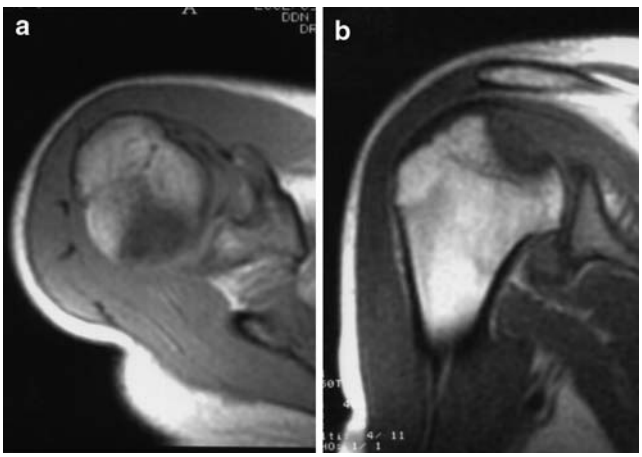

Figure 4. MRI at 6 months. a) Transversal plane (T1 sequence, TR/TE = 360/12; flip angle 90°; matrix 160 H/160 V, slide thickness 5 mm). b) Coronal plane (T1 sequence, TR/TE = 400/20; flip angle 90°; matrix 192 H/192 V, slide thickness 5 mm).

sequences that corresponded with the humeral head lesion (Figure 4). An arthroCT scan showed a hypodense lesion at the superior pole of the humeral head, surrounded by a hyperdense border, opposite an indentation without cartilaginous continuity loss (Figure 5). In addition, there was a subscapularis muscle detachment pocket and a minimal fracture at the edge of glenoïd cavity's antero-inferior ridge; the shoulder cuff was continent.

Complementary rheumatology workup did not uncover any underlying disease, or any plausible systemic cause for osteonecrosis.

At 10 months, mobility had improved with symmetrical overall active abduction and anteversion at 170°; insufficient external rotation (elbow to body) persisted (10° vs. 60° on the uninjured side) and during 90° abduction, external rotation was 70° versus 90° on the healthy side. Internal rotation was symmetrical. With once-weekly analgesics, pain diminished. However, significant apprehension persisted, impairing the patient's activities.

In our experience, an operative indication is confirmed after the third dislocation. In this case, the unexplained pain of the shoulder pushed us to propose an earlier stabilization. A Bankart procedure was proposed in relation to the CT exam views. Given the significant apprehension, the impossibility of resuming normal sports activities, the detachment pocket visu-

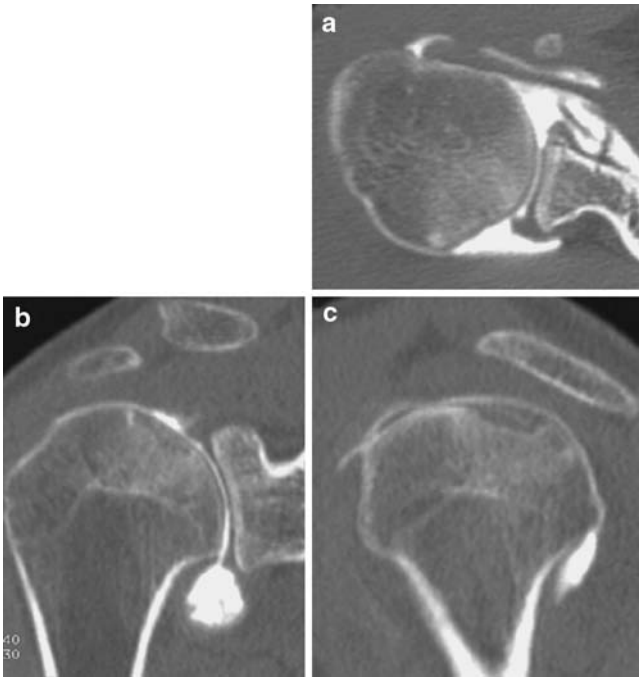

Figure 5. ArthroCT scan at 6 months, intact cartilage (140 kV, 130 mA, slide thickness 1 mm). a) Transversal plane b) Coronal plane c) Parasagittal plane.

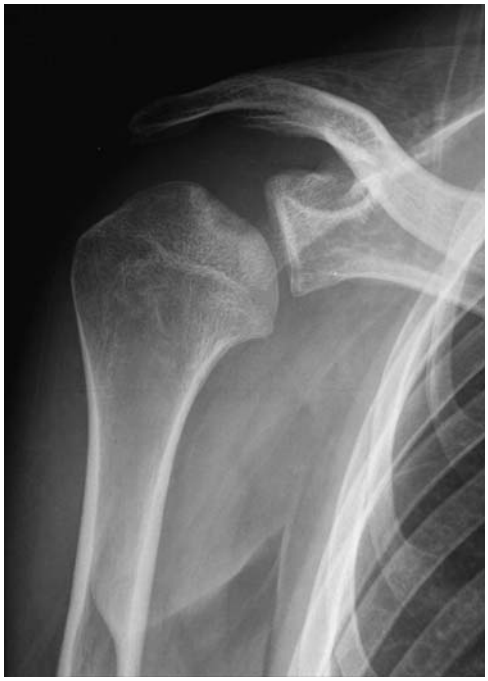

Figure 6. True antero-posterior view of the shoulder at 10 months, lesion stabilized.

alized by the arthroCT scan and the persistent patient demand, a Bankart type capsuloplasty repair under arthroscopy was performed at 10 months following the initial trauma. At this stage, X-rays showed a stabilization of the bone lesion (Figure 6). The procedure was proposed after 6 months' follow-up. Due to personal convenience of the patient, the surgery was only performed at 10 months' follow-up.

This intervention was performed in a beach chair position, with a posterior optical access route and an anterior instrument access route, interior toward exterior.

Arthroscopy showed a capsulolabral detachment with an Anterior Labroligamentous Periosteal Sleeve Avulsion (ALPSA lesion), a chondropathy aspect in the posterosuperior quadrant of the humeral head, without cartilaginous continuity loss; the cartilage of the glenoïd and the deep layers of the cuff were uninjured. Capsuloplasty was performed by reinserting the capsule at the inferior margin of the glenoïd cavity with three bioresorbable anchors (polylactic acid), 3.5 mm in diameter (PANALOK®, Mitek worldwide Ethicon, Inc., Johnson & Johnson Company, MA, USA) at 3, 4 and 6 o'clock. There were neither perioperative, nor immediate postoperative complications. The shoulder was immobilized, elbow to body, for 4 weeks and then rehabilitated. The patient resumed his sports activities 3 months after surgery.

Results

Three years and 10 months after surgery, the patient has resumed his normal professional activities. He quit football and moto-cross for reasons unrelated to his shoulder, but he plays squash. He experienced neither recurrent instability, nor dislocation. He has intermittent pain, only after very significant efforts, without impairment during the activity, not requiring analgesics. Clinical examination showed neither amyotrophy, nor sensory deficit of the shoulder; abduction was 170° versus 180°, anteversion 165° versus 180°, external rotation at 90° abduction was 20° versus 50°, internal rotation was symmetrical at D6. The apprehension test was positive, without apprehension during everyday activities. The Jobe relocation test was painful without loss of muscle strength. The palm-up test, the lift-off test and the belly press test were normal. There were no sign of subacromial conflict. The patient did not show any hyperlaxity.

Forty-six months after injury, radiological control (Figure 7) showed homogenous bone trabecula with a persistent spherical defect of the humeral head and

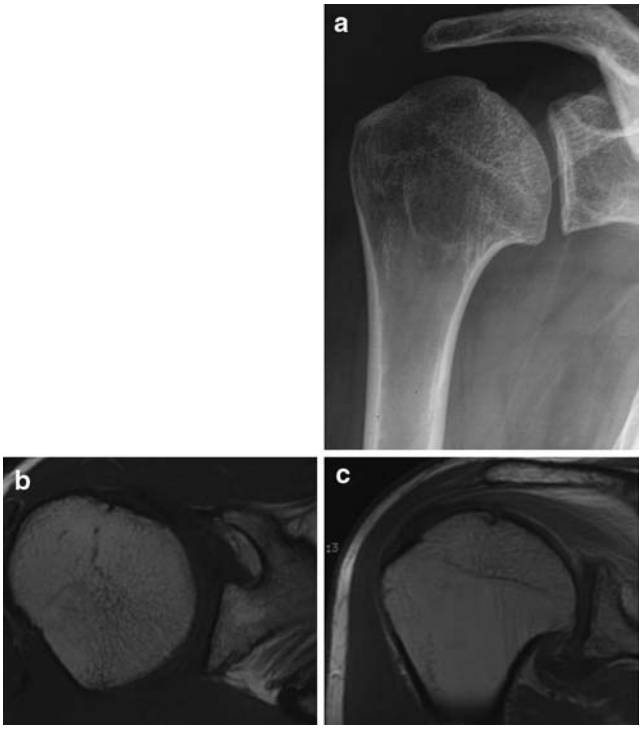

Figure 7. Follow-up at 46 months. a) True antero-posterior view of the shoulder. b) MRI transversal plane (T1 sequence, TR/TE = 500/13.2; flip angle 90°; matrix 480 H/320 V, slide thickness 3 mm). c) MRI coronal plane (T1 sequence, TR/TE = 480/9.8; flip angle 90°; matrix 320 H/256 V, slide thickness 4 mm).

mild arthrosis (Samilson and Prieto grade 1) [3]. MRI showed a homogenous signal without any anomalies of the humeral head.

Discussion

Aseptic osteonecrosis of the humeral head is the second most frequent osteonecroses site, after the femoral head. The causes are well known, both non-traumatic [4] – corticotherapy, alcoholism, sickle-cell disease, barotrauma, hypercholesterolemia, Gaucher's disease, certain systemic inflammatory diseases – and traumatic causes, especially complex cephalotuberosity fractures with three or four fragments [5, 6].

In this report, we considered our patient as having stage III aseptic osteonecrosis of the humeral head according to Ficat's classification [7] transposed to the humeral head by Cruess [8]. Differential diagnoses to avascular osteonecrosis included osteochondral fracture and intraosseous contusion.

As for an osteochondral fracture, it is true that many authors [9, 10] have reported an important risk of osteochondral lesions in adolescents. According to Flachsmann [11], the cause of these lesions is linked to a significantly more fragile bone–cartilage interface in adolescents than in children or adults. This fragility is linked to sudden modifications of the mechanical properties of cartilage (supple) and subchondral bone (rigid) associated with an immature bone-cartilage bonding system (few interdigitations).

We did not retain the diagnostis of humeral head notching nor osteochondral fracture since no such radiographic images existed, either upon dislocation, or upon control at 45 days. Furthermore, imagery and arthroscopy found continuous cartilage, thus excluding an osteochondral fracture. Finally, the images progressively worsen in parallel with the clinical observations.

An intraosseous contusion was ruled out since at 6 months, a hyposignal in sequences T1 and T2 was observed upon MRI, in contrast to bone contusions where a hypersignal exists in sequence T2 [12]. In general, bone contusions are not visible on standard X-rays and the humeral head remains spherical. In this patient, clinical and biological examinations eliminated any known medical cause and radiography showed no controlateral lesions.

We believe that this osteonecrosis is a direct consequence of the dislocation episode.

When using the description of anterior shoulder dislocation without associated fracture and its complications, no similar cases were found in the literature.

A case of aseptic osteonecrosis following dislocation associated with a non displaced fracture of the greater tuberosity [13] and a case of aseptic osteonecrosis after capsular reconstruction with installment of an anchor on the humeral epiphysis due to chronic instability [14] have been published in the literature. According to the authors, these two cases are linked to a lesion of the intraosseus branch of the anterior circumflex artery: the arcuate artery.

Many authors [15–18] have studied the vascularization of the proximal humeral epiphysis. Its arterial vascularization stems from two branches of the axillary artery: the anterior and posterior circumflex arteries.

In cadaver studies, it has been shown that the anterior circumflex artery predominates, and in healthy patients, vascularizes the supero-anterior portion of the head, the lesser tuberosity and the delto-pectoïdal groove. Despite a larger diameter, the posterior circumflex artery vascularizes a lesser surface: the postero-inferior portion of the humeral head and the posterior portion of the greater tuberosity. For all authors, this systematization is subject to numerous intra- and inter-individual anatomical variations. Auxiliary anastomoses with vessels from the rotator cuff, the thoraco-acromial artery, suprascapular artery and

arteria profonda brachii have been observed but have never been studied because of the very fine caliber of these vessels. Intra-osseous distribution is principally comprised of two anastomotic networks located on both sides of the epiphysal plaque linked by numerous perforating vessels. These networks are fed by the arcuate artery – intra-osseous branch of the anterior circumflex artery – and by the numerous intra-osseous branches stemming from the posterior circonflex artery.

From a purely quantitative standpoint, Lane [19] showed that the number of subchondral blood vessels is maximal at the age of 20 and decreases with age. These anatomical observations may explain the absence of osteonecrosis upon dislocation, thus making this clinical case an exception.

We eliminated all sub-periosteal vascular and intraosseous lesions for anatomical reasons. In fact, the mechanism itself (anterior dislocation) cannot cause a direct lesion to the anterior circonflex bundle.

Given the position of the necrotic lesion within the humerus-scapula contact zone in a dislocated position, our pathophysiological hypothesis is a lesion by subchondral bone shearing that triggered intra-osseous edema, which would decrease local capillary perfusion pressures thus causing osteonecrosis.

Because of the benefit/risk ratio to the patient, this hypothesis could not be confirmed since this would implicate performing highly invasive arteriographic or histological examinations.

The lesion stabilized at 10 months post-trauma with recovery at approximately 4 years; however, there were complications in the form of articular stiffness.

Conclusion

In addition to the well-recognized complications of anterior shoulder dislocation, the absence of rapid clinical improvement, especially in young patients, a diagnosis of humeral head osteonecrosis should be evoked. It is best visualized by MRI within 4–6 months following trauma. Unless necrosis is widespread, no specific intervention is necessary.

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