The Shoulder: Acquired Disorders

6

J. Herman Kan

Contents

Abstract

The chapter begins with an in-depth discussion of glenohumeral instability and dislocation. Proximal humeral fractures and Little Leaguer's shoulder are described, followed by a concise discussion of glenohumeral internal rotation deficit (GIRD). Fractures of the clavicle and the scapula are then discussed in depth. A section on chondral injuries of the glenohumeral joint follows, and the chapter concludes with a short description of soft tissue injuries around the shoulder.

The shoulder is composed of three bones: the humerus, scapula, and clavicle. The pediatric shoulder is biomechanically balanced by a very mobile but unstable ball and socket glenohumeral joint and a gliding acromioclavicular joint, with both supported by multiple active (shoulder girdle muscles) and passive (ligaments and labrum) soft tissue stabilizers. It is prone to extra-articular fractures due to its open physis and apophysis, the weakest components of the musculoskeletal unit of the skeletally immature patient [1]. The physis and apophysis tend to fail before soft tissues (ligaments and muscles). Injuries to the pediatric shoulder are frequent and increasing due to the popularity of organized youth sports.

J.H. Kan, MD (✉)
Department of Radiology, Texas Children's Hospital, 6701 Fannin Street, Suite 470, Houston, TX 77005, USA

Department of Radiology, Baylor College of Medicine, Houston, TX, USA
e-mail: jhkan@texaschildrens.org

R. Stein-Wexler et al. (eds.), *Pediatric Orthopedic Imaging*,
DOI 10.1007/978-3-642-45381-6_6,

1 Glenohumeral Instability and Dislocation (Box 6.1)

Box 6.1: Anterior Shoulder Dislocation

Hill-Sachs fracture: subchondral fracture along the posterolateral humeral head

Bankart fracture: anteroinferior glenoid fracture

Glenohumeral dislocations usually result from direct injury during contact sports. In children, these injuries are most common in the second decade [2]. Forty percent of patients present before age 22 [3]. The younger the child is at the time of dislocation, the higher the likelihood of recurrent dislocation and glenohumeral shoulder instability. Approximately 65–90 % of teenagers who suffer one shoulder dislocation will have additional dislocations [4]. Children with collagen disorders such as Ehlers-Danlos or Ulrich collagen VI deficiency have increased incidence of shoulder dislocation.

Most dislocations are anterior, and the child presents with the arm abducted and externally rotated [5]. Less commonly, glenohumeral dislocations may be posterior, inferior (luxatio erecta), or superior. Seizures may cause posterior dislocation. An additional consideration—particularly for atraumatic recurrent posterior dislocations—is underlying primary glenoid dysplasia with excessive retroversion, which can predispose to posterior dislocation.

The glenohumeral joint is a ball and socket joint invested by glenohumeral ligaments, capsule, and shoulder girdle muscles. The static stabilizers include the capsule, labrum, and glenohumeral ligaments. The active stabilizers include the rotator cuff and biceps tendon [6]. The glenoid cavity is concave and slightly retroverted (approximately 2–4°) with respect to the body of the scapula (i.e., the articular surface is directed posteriorly with respect to the scapula). The glenoid cavity itself is shallow, and its perimeter is reinforced by the fibrocartilaginous labrum to increase contact and depth of apposition with the humeral head. Although the glenohumeral joint is technically a ball and socket joint, the relatively small glenoid fossa (compared with a relatively large humeral head) allows for greater mobility at the cost of relative instability. The glenohumeral joint is more predisposed to dislocation than are hinge-type joints.

Atraumatic glenohumeral instability is usually multidirectional and may be bilateral [6]. In addition to underlying glenohumeral dysplasia, risk factors include hypermobility syndromes encountered in mixed connective tissue disease such as Marfan disease and Ehlers-Danlos syndrome [7] or activities that lead to repetitive overstretching such as swimming and gymnastics [8]. Traumatic glenohumeral dislocation tends to be unilateral and unidirectional.

Patients may respond well to rehabilitation after one traumatic dislocation, but primary repair is recommended after two or more, due to the high incidence of recurrent dislocation [9]. In one study, the incidence of recurrent dislocation was 19 % in patients treated operatively, compared with 60 % for a similar group treated nonoperatively [10]. Arthroscopic Bankart repair may be performed if the bone of the glenoid is relatively preserved. If greater than 25 % of the inferior glenoid is deficient as a result of a Bankart fracture, a Latarjet procedure may be indicated [11, 12]. In this procedure, a portion of the coracoid is transferred to the area of bone loss along the anteroinferior glenoid, improving glenoid and humeral head congruency. However, the Latarjet procedure has a relatively high incidence of complications, and, if bone loss is very severe, grafting with iliac crest bone graft may be necessary.

Traumatic multidirectional instability is treated with physical rehabilitation. If physical rehabilitation is unsuccessful, anterior capsular shift is recommended [13]. This procedure tightens the anterior and inferior capsule.

Imaging

Suspected glenohumeral dislocation is evaluated with an axillary view along with frontal radiographs in internal and external rotation. The normal glenohumeral joint has a smooth, round scapulohumeral arch, which is formed by the axillary border of the scapula, the inferior portion of the humeral neck, and the proximal humeral shaft (Fig. 6.1). In both anterior and

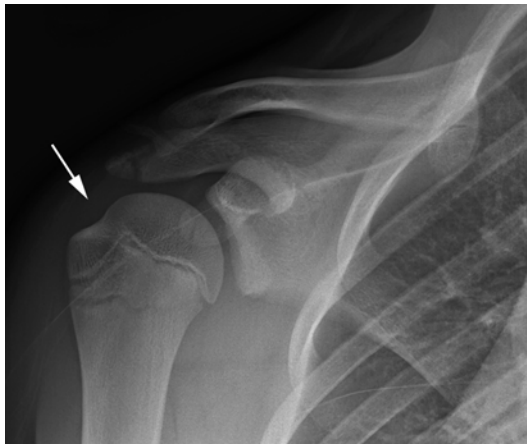

Fig. 6.1 Normal shoulder radiograph with humerus in external rotation in a 13-year-old boy. Note normal smooth scapulohumeral arch. Do not confuse the normal concavity at the interface of the greater tuberosity and humeral head (*arrow*) on this view (or on a poorly obtained frontal internal rotation radiograph) for a Hill-Sachs fracture

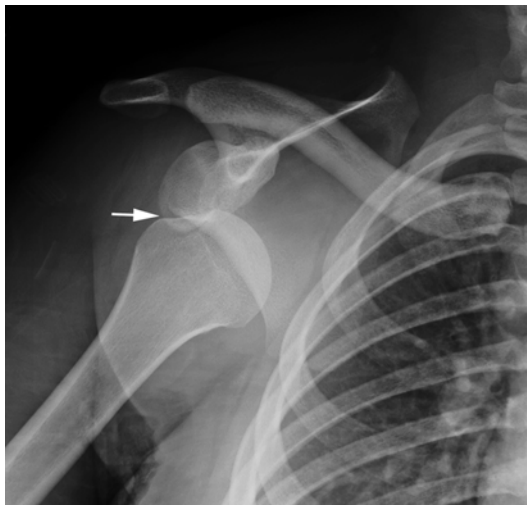

Fig. 6.2 Anterior shoulder dislocation in a 17-year-old girl. The humeral head is displaced inferomedially. Hill-Sachs fracture of the humeral head and bony Bankart fracture of the anteroinferior glenoid rim are also present (*arrow*)

posterior shoulder dislocations, the humeral head moves medially, and the arch is no longer aligned (Fig. 6.2). The extent of medial displacement is subtler with posterior dislocations than anterior dislocations. Axillary, Velpeau, and "Y" views help distinguish posterior from anterior glenohumeral dislocation (Fig. 6.3). The axillary view also evaluates for a glenoid fracture (bony Bankart) and determines if a Hill-Sachs fracture is either engaging (Hill-Sachs fracture depression is locked in place along the inferior glenoid rim) or non-engaging. The Velpeau view provides similar information and is useful for patients who cannot tolerate a traditional axillary view. The "Y" view principally helps determine the anteroposterior relationship but also supplements the frontal views for evaluation of the glenoid (Fig. 6.4). Hill-Sachs and Bankart fractures are presented in greater detail below.

Secondary signs of anterior shoulder dislocation include a Hill-Sachs fracture and osseous Bankart fracture (Figs. 6.5 and 6.6). These fractures result as the posterolateral humeral head and anteroinferior glenoid collide as the humeral head anteriorly dislocates. These fractures are less common in children than in adults and—like shoulder dislocation in general—are more likely in the second decade. The Hill-Sachs fracture occurs along the posterolateral humeral head and is optimally seen on a frontal internal rotation view (Fig. 6.7). The normal concavity at the interface between the humeral head and the greater tuberosity may mimic a Hill-Sachs fracture on a poorly obtained internal rotation AP view. Furthermore, normal glenoid fragmentary ossification may mimic an osseous Bankart fracture. Whereas the former is usually distributed evenly throughout the glenoid rim, the Bankart fracture is more focal, at the anteroinferior glenoid.

Less common glenohumeral dislocations include luxatio erecta (inferior dislocation) and posterior dislocation. Luxatio erecta occurs when the humeral head displaces inferiorly; as a result, the arm is fully abducted and held upward. Secondary signs of posterior shoulder dislocation include a reverse Hill-Sachs fracture and reverse osseous Bankart fracture. A reverse Hill-Sachs fracture occurs along the anteromedial humeral head.

Computed tomography (CT) helps determine the type of surgical stabilization necessary to repair the glenohumeral joint. Sagittal and coronal reformatted images along with three-dimensional (3D) maximum intensity projections (MIPs) are especially helpful for determining the amount of bone loss at the anteroinferior glenoid. The normal glenoid is pear-shaped. It

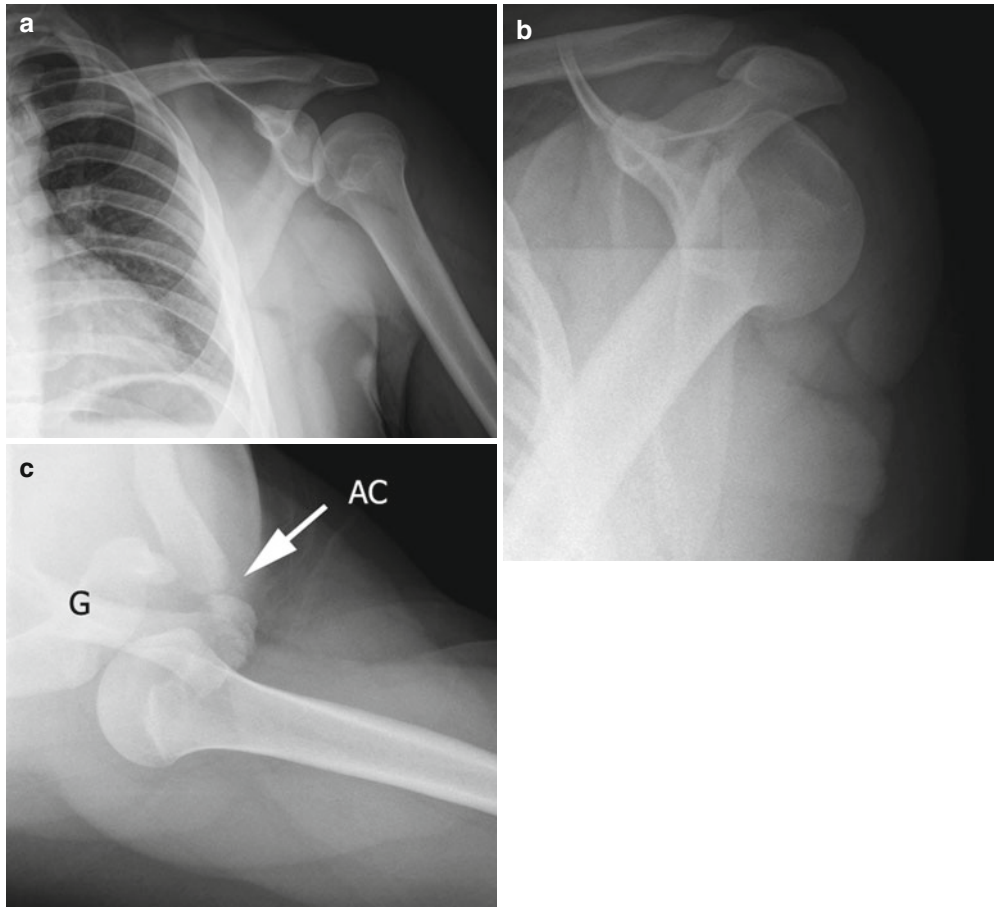

Fig. 6.3 Posterior shoulder dislocation in a 15-year-old girl. (**a**) There is subtle disruption of the scapulohumeral arch with medial humeral head displacement. The "Y" (**b**) and axillary (**c**) views confirm posterior shoulder dislocation. *AC* acromioclavicular joint. *G* glenoid

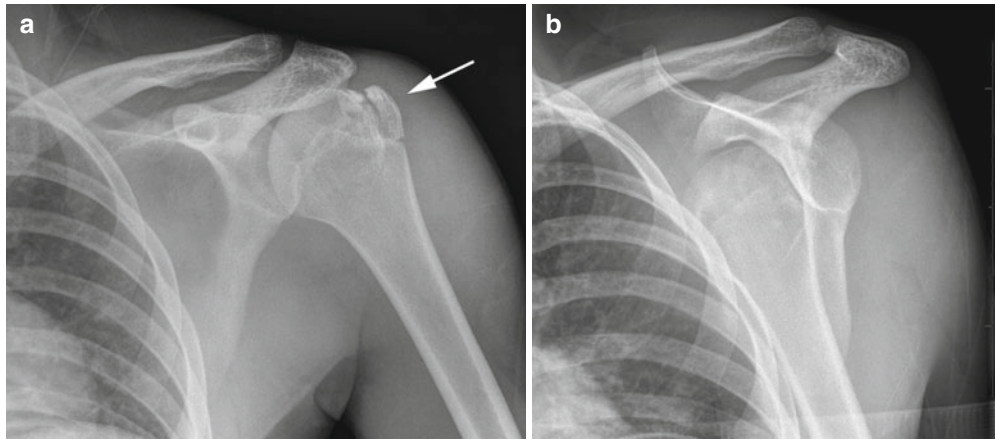

Fig. 6.4 Greater tuberosity fracture in an 11-year-old boy. (**a**) On the frontal view, the fracture fragment (*arrow*) is slightly separated, and glenohumeral alignment appears normal. (**b**) The "Y" view shows the humeral head is actually dislocated

may become an "inverted pear" (large end up) if a Bankart fracture causes bone loss along the anteroinferior quadrant (Fig. 6.8). If more than 25 % of the glenoid is deficient, surgical fixation with a Latarjet procedure or iliac crest grafting may be indicated [11, 12].

Magnetic resonance arthrography (MRA) delineates the status of the static stabilizers of the glenohumeral joint and the various soft tissue Bankart injuries [14]. Although CT arthrography (CTA) and MRA may have comparable accuracy for the evaluation of Bankart injuries [15], MRA avoids radiation and can also evaluate marrow and soft tissue edema, as well as delineate subtypes of Bankart injuries. In the acute setting, posttraumatic glenohumeral joint fluid may have an arthrographic effect, making intra-articular gadolinium injection unnecessary.

Anteroinferior glenoid and labroligamentous injuries are classified into two subtypes: osseous Bankart and soft tissue Bankart lesions. The true soft tissue Bankart lesion is an anteroinferior labral tear with a torn scapular periosteum (Fig. 6.9), but there are many subtypes for soft tissue injuries (Fig. 6.10). It is more important

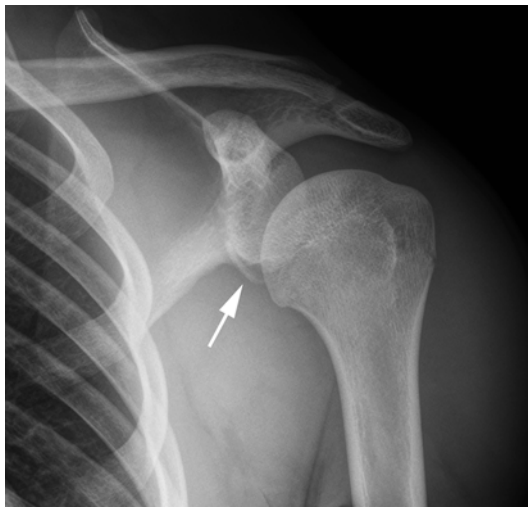

Fig. 6.5 Anterior shoulder dislocation in a 17-year-old boy. There is a bony Bankart fracture (*arrow*)

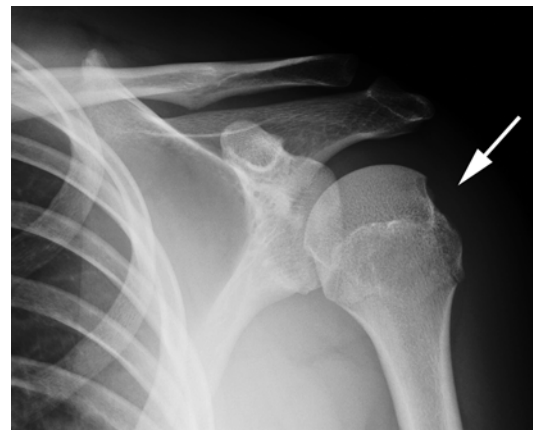

Fig. 6.7 Sequela of anterior shoulder dislocation in a 15-year-old boy. There is a large Hill-Sachs fracture (*arrow*) on this frontal view with internal rotation

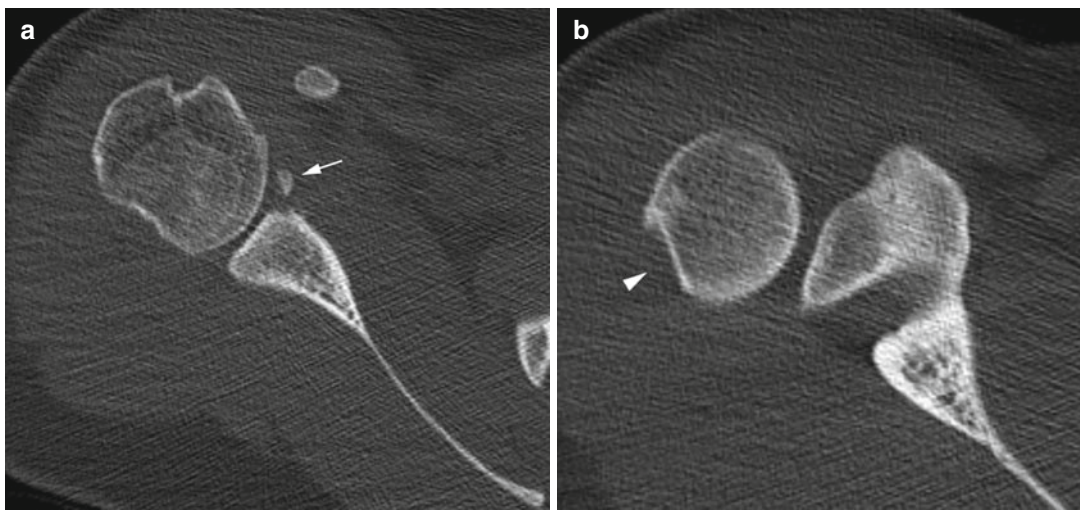

Fig. 6.6 Fractures in a 16-year-old girl with prior anterior shoulder dislocation. Axial CT images show (**a**) mildly displaced bony Bankart (*arrow*) and (**b**) depressed Hill-Sachs (*arrowhead*) fractures

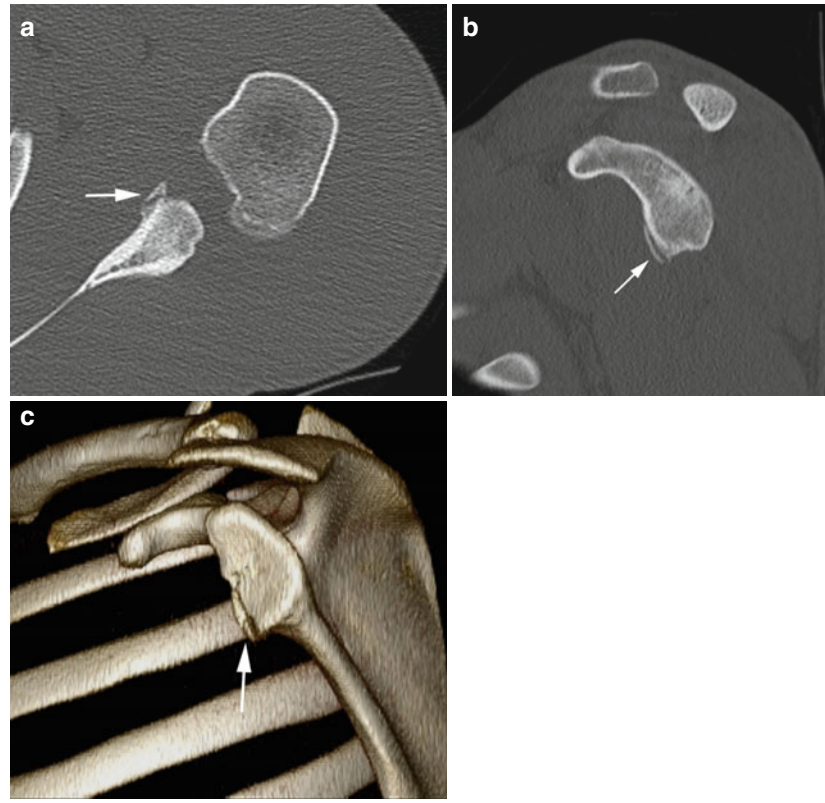

Fig. 6.8 A 16-year-old girl with osseous Bankart (*arrows*) demonstrated on axial (**a**), sagittal (**b**), and 3D (**c**) reconstructions. 3D-CT best delineates the inverted pear appearance of the glenoid due to anteroinferior deficiency after Bankart fracture (Courtesy of Tal Laor, Cincinnati Children's Hospital)

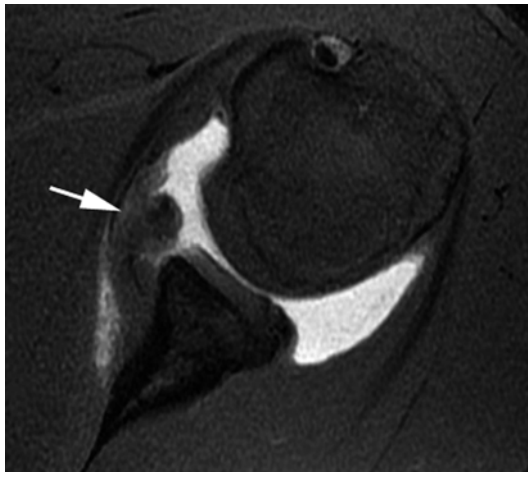

Fig. 6.9 Soft tissue Bankart lesion in a 16-year-old boy. Axial T1-weighted (*T1-W*) fat-suppressed (*FS*) image from MR arthrography (*MRA*) shows intra-articular contrast material insinuating between the glenoid and the displaced, torn, mass-like labral tissue (*arrow*)

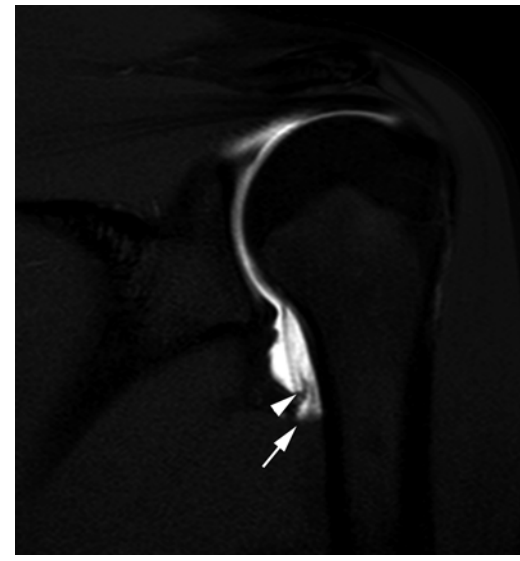

Fig. 6.10 Humeral avulsion of the glenohumeral ligament injury (*HAGL*) in a 16-year-old girl. Coronal T1-W FS MRA shows extra-articular contrast extension (*arrow*) and retracted inferior glenohumeral ligament (*arrowhead*)

Table 6.1 Soft tissue Bankart variations

Soft tissue Bankart variations	Description
Bankart lesion (Fig. 6.9)	Anteroinferior labral tear with a torn scapular periosteum
Perthes	Non-displaced Bankart with intact scapular periosteum
Anterior labroligamentous periosteal sleeve avulsion (ALPSA)	Bankart lesion with medial displacement with intact scapular periosteum
Glenolabral articular disruption (GLAD) (Fig. 6.11)	Bankart with associated glenoid chondral defect
Humeral avulsion glenohumeral ligament (HAGL) (Fig. 6.10)	Avulsion of the anterior inferior glenohumeral ligament at its humeral insertion
Bony avulsion glenohumeral ligament (BAGHL)	HAGL but with a humeral bone avulsion injury
Floating anterior inferior glenohumeral ligament (AIGHL)	Combined Bankart and HAGL lesion

Adapted from Ref. [16]

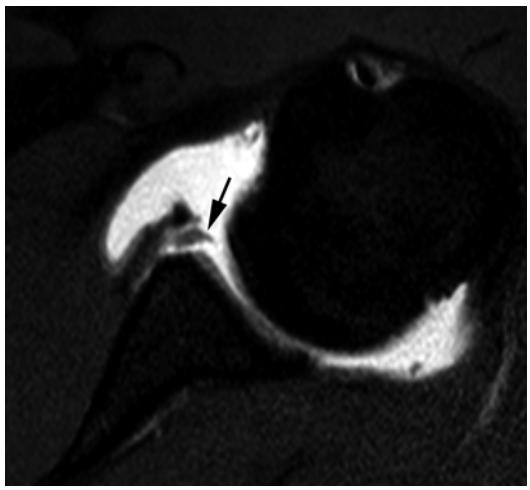

Fig. 6.11 Glenolabral articular disruption (*GLAD*) subtype of soft tissue Bankart lesion in a 17-year-old boy. Axial T1-W FS MRA shows a component of cartilage has been stripped (*arrow*)

to accurately describe findings than to label the exact subtype (Table 6.1). The status of the glenoid cartilage (Fig. 6.11), inferior glenohumeral ligament, labrum, and glenoid bone must all be closely evaluated.

Superior labral anterior-posterior (SLAP) injuries are sometimes associated with glenohumeral instability and at least in adults occur in approximately 20 % of patients with glenohumeral instability [17]. However, treatment is not always necessary.

There are no specific imaging findings for atraumatic multidirectional shoulder instability other than a patulous glenohumeral capsule, which is best seen along the axillary recess after distention during arthrography. Therefore, the diagnosis is generally made on gestalt interpretation in conjunction with clinical findings [8]. Associated findings include articular and labral injuries resulting from recurrent dislocations.

2 Proximal Humeral Fractures

Proximal humeral fractures comprise 0.5 % of all fractures in children and between 4 and 7 % of all epiphyseal fractures [18]. The mechanism and force of injury resemble those for shoulder dislocations, but the force decompresses along the proximal humerus. The unbalanced biomechanical pull of muscle insertions proximal and distal to the site of fracture leads to angular and displacement deformities.

An understanding of the timing and appearance of the ossification centers of the proximal humerus helps in interpreting radiographs in the setting of trauma and elbow ossification centers also helps determine the clinical significance of different fracture patterns. There are three ossification centers at the proximal humerus. The humeral head epiphysis begins to ossify at or shortly after birth, and the greater tuberosity apophysis begins to ossify at age 3 years. The lesser tuberosity begins to ossify at age 5 years, and the humeral head, greater tuberosity, and lesser tuberosity fuse at age 6 [19]. The proximal humeral physis remains open until age 14–17 in girls and 16–18 in boys [20]. Damage to this physis can result in significant growth disturbance, as it is responsible for 80 % of the longitudinal growth of the humerus. However, as a general rule, proximal humeral fractures are more likely

to remodel in younger children than in older children due to the open physis.

Non-displaced or minimally displaced proximal humeral fractures are treated nonoperatively. Severely angulated or displaced fractures may require operative fixation in children older than 10–13 years of age [18, 21]. In children under 11 years of age, most if not all fractures will remodel without long-term sequelae and can be treated nonoperatively [19]. Surgical reduction is indicated in open proximal humeral fractures or if there is evidence of neurovascular compromise.

Imaging

Three-view shoulder (frontal internal and external rotation along with axillary) or two-view humerus radiographs adequately assess most proximal humeral fractures. It is important to determine the amount of displacement, as this predicts prognosis and determines appropriate treatment. The Neer classification offers a convenient way to categorize proximal humeral fractures (Table 6.2), although it is not generally used with children [19]. Angulation should also be addressed (Fig. 6.12), although the need to report the precise degree of fracture angulation varies with institution and orthopedic surgeon preference [18]. In older children, fractures are frequently complete (Fig. 6.12), while in younger children, buckle fractures are common (Fig. 6.13). The fracture line orientation, physeal involvement (humeral head, greater tuberosity, lesser tuberosity) (Fig. 6.14), and presence of

Table 6.2 Neer classification of proximal humeral fractures

Grade	Displacement
I	<5 mm
II	<1/3 shaft width
III	2/3 shaft width
IV	>2/3 shaft width

Adapted from Ref. [19]

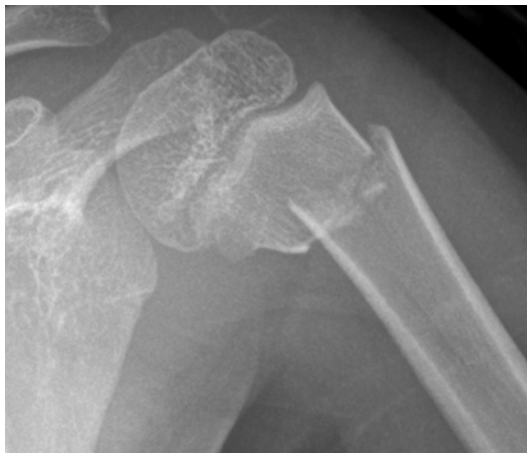

Fig. 6.12 Neer type I displaced and mildly comminuted fracture of the surgical neck of the humerus in a 5-year-old girl. There is less than 5 mm of lateral displacement of the distal fragment

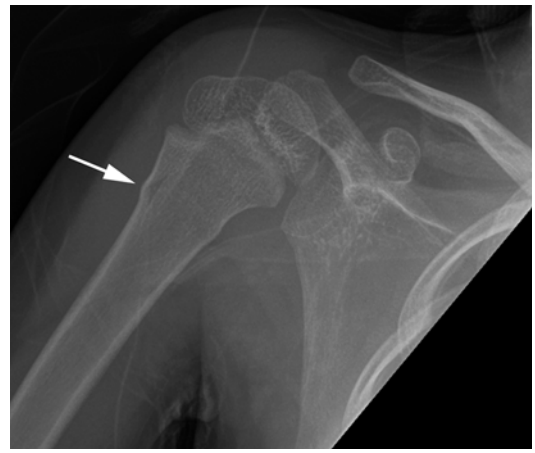

Fig. 6.13 Non-displaced buckle fracture (*arrow*) of the surgical neck of the humerus in a 3-year-old girl

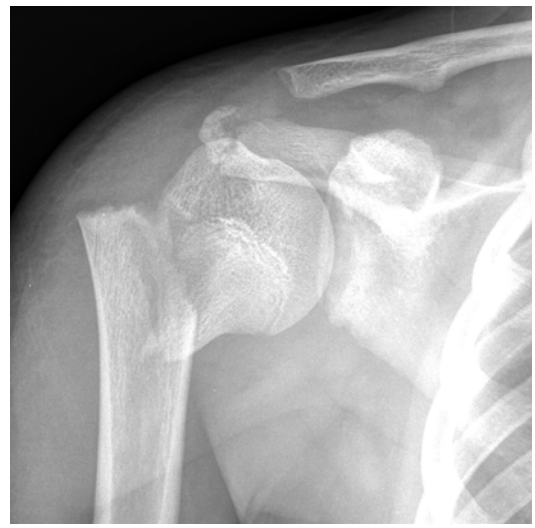

Fig. 6.14 Neer grade 4 proximal humeral fracture in a 13-year-old boy. The fracture extends from the lateral physis to the greater tuberosity and is displaced by more than two-third of the width of the shaft

comminution should be discussed, as with any fracture.

Radiography is almost always sufficient for imaging proximal humeral fractures. Occasionally, for complicated fractures, magnetic resonance imaging (MRI) may help identify tendon, periosteum, or muscle interposition at the fracture site [22], which could impede proper healing.

3 Little Leaguer's Shoulder (Box 6.2)

Box 6.2: Little Leaguer's Shoulder (Young Throwing Athlete)

Radiographs:

Proximal metaphyseal sclerosis, lateral metaphyseal fragmentation

Wide physis (resembles acute Salter-Harris I fracture)

MRI:

T1: Physeal widening

Fluid sensitive: metaphyseal edema, periosteal reaction, and juxtacortical soft tissue edema

Less common: epiphyseal edema, juxtaphyseal cysts, and periosteal edema

Little Leaguer's shoulder, seen in athletes who throw overhead, is an overuse injury of the proximal humeral physis and is most likely a chronic non-displaced Salter-Harris I fracture. The injury most commonly occurs during growth spurts in boys between 11 and 14 years of age [23]. Rotational torqueing generated during the acceleration phase of an overhead throw probably causes the injury.

The glenohumeral joint in childhood is quite lax. However, in early adolescence, a dramatic change in range of motion occurs, with decreased internal and external rotation, along with increased shoulder elevation. The decrease in rotational motion may contribute to excessive stress at the physis with throwing and thus predispose children to this injury [24].

A single acute event generally does not cause the injury; symptoms develop gradually and correlate with pitch count. Symptoms include pain during throwing, with palpation of the proximal humerus, and during resisted external rotation of the humerus [25].

The treatment for Little Leaguer's shoulder is rest from overhead throwing [23]. Surgery is not indicated. If the child returns to throwing too early, continued physeal damage may lead to displacement and eventual early physeal closure. Prevention of this injury is one of the reasons for pitch count regulations for Little League players.

Imaging

Little Leaguer's shoulder is usually diagnosed clinically. Radiographs can confirm the diagnosis or suggest alternative etiologies for symptoms. Findings include proximal metaphyseal sclerosis, lateral metaphyseal fragmentation, physeal widening, and demineralization (Fig. 6.15) [25, 26]. Note that this appearance is indistinguishable from an acute non-displaced Salter-Harris I fracture. The term Little Leaguer's shoulder should not be used when there is a displaced Salter-Harris I proximal humeral fracture (Fig. 6.16).

Distinguishing Little Leaguer's shoulder from normal variations of physeal widening is particularly challenging because the injury occurs during adolescent growth spurts when normal physiologic widening is present. Radiographs of the contralateral asymptomatic shoulder may be obtained to determine if the physeal widening is physiologic or due to chronic physeal injury (Fig. 6.17).

MRI demonstrates physeal widening, and there may be edema—usually in the metaphysis [27]. Less common findings include epiphyseal edema, juxtaphyseal cysts, and periosteal edema [28, 29]. T1-weighted (T1-W) sequences best evaluate physeal widening. Fluid-sensitive sequences help assess marrow edema, periosteal reaction, and juxtacortical soft tissue edema (Fig. 6.15). In the presence of physeal widening and/or juxtaphyseal cysts (but no edema), MRI may suggest chronic injury. However, if there are cysts and physeal widening along with juxtaphyseal and juxtacortical marrow edema, the process is likely acute superimposed on chronic.

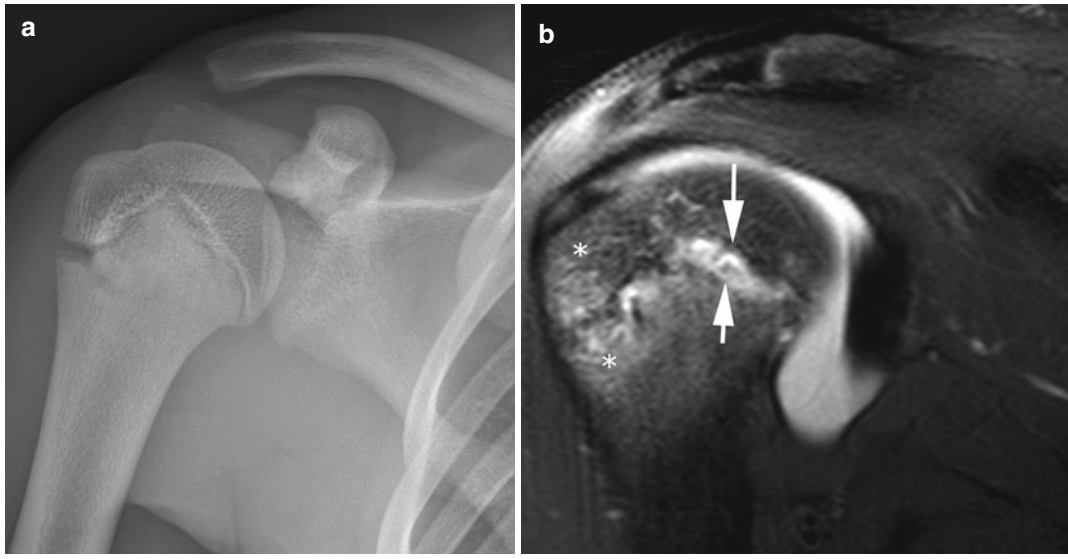

Fig. 6.15 Little leaguer's shoulder in a 14-year-old boy. (**a**) Widening of the proximal humeral physis. (**b**) Coronal T2-W FS MRA shows juxtaphyseal marrow edema (*) and physeal widening particularly along the medial physis (*between arrows*)

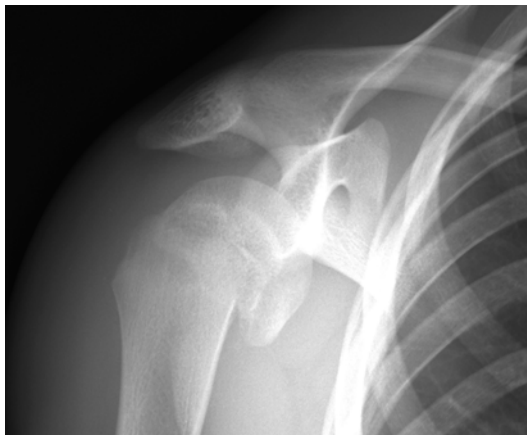

Fig. 6.16 Displaced Salter-Harris I fracture of the proximal humerus with glenohumeral dislocation in a 15-year-old boy

Differential Diagnosis

Little Leaguer's shoulder is indistinguishable from acute non-displaced Salter-Harris I fractures of the proximal humerus, and diagnosis is predicated on clinical history. The injury may also be difficult to distinguish from normal physiologic physeal widening, but imaging the asymptomatic shoulder should clarify this.

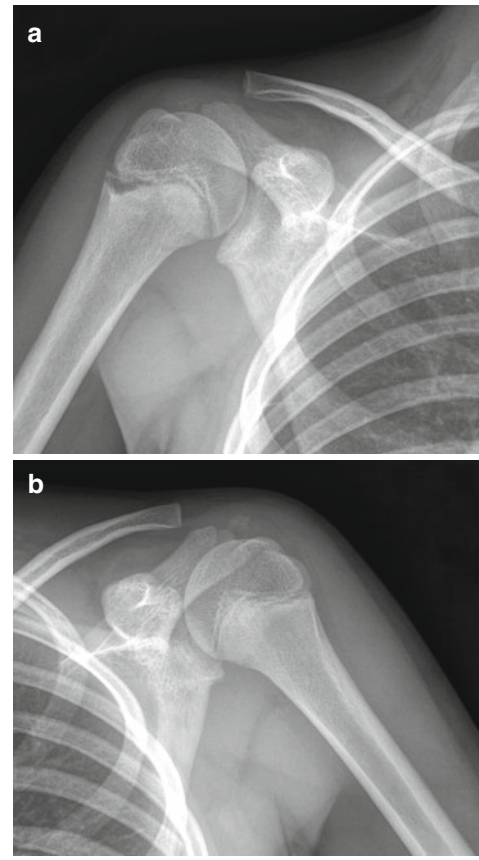

Fig. 6.17 Little leaguer's shoulder in a 13-year-old boy. (**a**) On the right, the proximal humeral physis is too wide. (**b**) Normal left shoulder (for comparison)

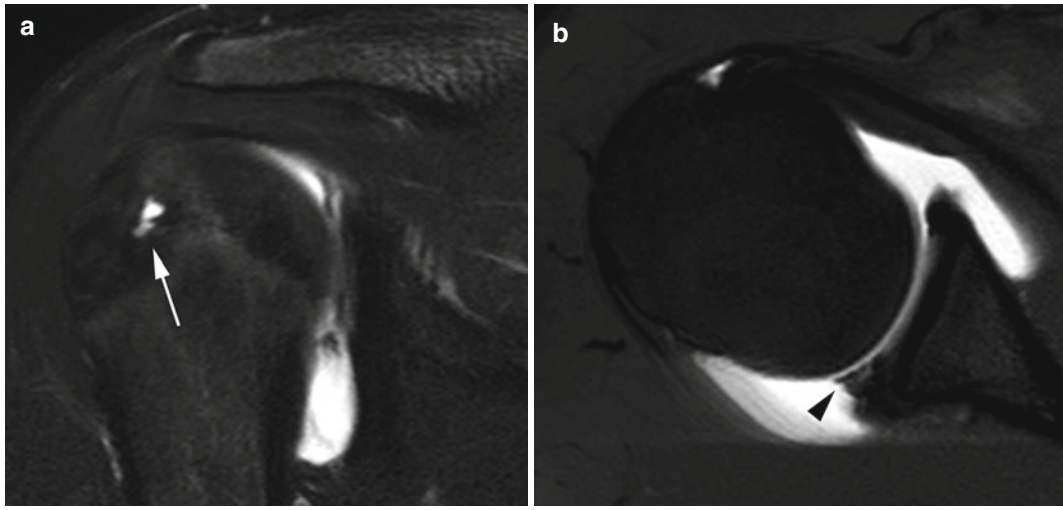

Fig. 6.18 Glenohumeral internal reduction deficit (*GIRD*) in a 17-year-old pitcher. (**a**) Coronal T2-weighted (*T2-W*) FS MRA shows posterolateral humeral head subchondral cysts (*arrow*). (**b**) Axial T1-W FS image shows a degenerative tear of the posterior labrum (*arrowhead*)

4 Glenohumeral Internal Rotation Deficit (GIRD)

Glenohumeral internal rotation deficit (GIRD) occurs in athletes who throw overhead, most commonly affecting baseball players but occasionally swimmers and tennis players. Repetitive overhead rotational motion causes anterior capsular laxity and posterior fibrosis of the glenohumeral joint, leading to reduced internal rotation [30]. Over time, the humeral head axis shifts posteriorly. Clinical GIRD is defined as loss of 25° of internal rotation in the affected shoulder.

If there are no associated labral or rotator cuff tears, treatment includes physical therapy to decrease muscle imbalance and capsular stiffness. If this is unsuccessful, posterior capsular release can be performed [30].

Imaging

Radiographs are often normal in the setting of GIRD. MRA optimally evaluates for GIRD, demonstrating undersurface tears of the posterior aspect of the supraspinatus, tears of the infraspinatus, posterosuperior labral tears, posterolateral humeral head cysts, and posterior capsular fibrosis (Fig. 6.18) [31, 32]. Posterolateral humeral head cysts are usually located at the site of attachment of the infraspinatus tendon and the posterior fibers of the supraspinatus tendon [31]. These cysts resolve after treatment.

Differential Diagnosis

Posterolateral humeral head cysts should not be confused with changes related to Hill-Sachs fractures from anterior glenohumeral dislocation. Unlike Hill-Sachs fractures, posterolateral humeral head cysts usually do not distort the spherical shape of the humeral head. These cysts should also not be confused with subchondral cysts related to osteoarthritis or with neoplastic entities such as giant cell tumor or chondroblastoma.

5 Clavicular Fractures (Box 6.3)

Box 6.3: Clavicular Fractures

Proximal and distal fractures are Salter-Harris equivalents

Distal fractures likely to be displaced if the fracture line is lateral to the coracoclavicular ligament

Floating shoulder is unstable: midclavicular fracture combined with glenoid neck fracture

Acromioclavicular or sternoclavicular dislocations are extremely rare in children/adolescents

Fractures of the clavicle may result from a direct blow while playing contact sports or from an indirect force transmitted across the shoulder, for instance, falling on an outstretched arm [33]. Contact sport injury is the most common cause of clavicular fracture in older children and adolescents. About one-third of clavicular fractures occur in preschool children who fall from a crib or bed. Only 4 % of all clavicular fractures result from birth trauma [34].

Several major ligaments and muscles attach to the clavicle; therefore, muscle spasm and/or unbalanced ligamentous retraction predisposes clavicular fractures to displacement and angulation. The coracoclavicular ligament, acromioclavicular capsule, and deltoid and trapezius muscles attach to the distal clavicle, and the acromioclavicular capsule, sternocleidomastoid muscle, and pectoralis major muscle attach proximally. The clavicle is located just above the subclavian artery and vein, and the brachial plexus is also nearby. Displaced fractures therefore carry significant risk of neurovascular injury.

Physeal and metaphyseal fractures are more likely to occur than are acromioclavicular or sternoclavicular capsular rupture, as these capsules are more resilient to biomechanical forces than are the adjacent physis and metaphysis. Pediatric clavicle fractures near the acromioclavicular or sternoclavicular joints are Salter-Harris equivalent fractures (the lateral clavicular physis fuses to the body of the clavicle at 19 years, and the medial physis fuses at 25 years) [35]. A so-called acromioclavicular pseudodislocation, or coracoclavicular periosteal sleeve fracture, may occur because the coracoclavicular ligament is more resilient than is the distal metaphysis [36].

Most clavicular fractures are treated nonoperatively, as even those with significant angulation or displacement remodel over time to a normal shape. Malalignment and nonunion are very rare in children. Midclavicular fractures are immobilized, and young children can usually return to their usual activities (including sports) within 6 weeks, although adolescents may need up to 10 weeks [33]. Particularly in younger patients, distal clavicle fractures near the acromioclavicular joint do not require surgery. However, patients older than 8 years with significantly displaced distal clavicular fractures may have a better cosmetic outcome if treated operatively [37].

Operative reduction is indicated if the fracture is open or severely displaced or if there is neurovascular injury. The latter complication is rare in distal and midclavicular fractures. However, medial clavicular fractures that are displaced posteriorly near the sternoclavicular joint may require surgical intervention due to mediastinal impingement [35]. Nonunion also mandates surgical reduction.

Imaging

Both frontal and angled frontal radiographs allow initial assessment of clavicular fractures. It is important to recognize that the mid-diaphysis of the normal clavicle may appear sharply angulated, especially on the angled view.

Midclavicular fractures are generally incomplete buckle fractures in younger children, whereas these fractures in adolescents tend to be complete. They may be displaced, angulated, or comminuted. If the major fracture fragments are near the coracoclavicular ligament and sternocleidomastoid muscle, angulation and displacement of the fracture fragments are common and may increase until healing supervenes (Fig. 6.19).

Proximal and distal clavicular fractures (Fig. 6.20) are usually Salter-Harris equivalent fractures, and extension to the physes of the associated ossification centers must be assessed. Since the medial and lateral apophyseal ossifica-

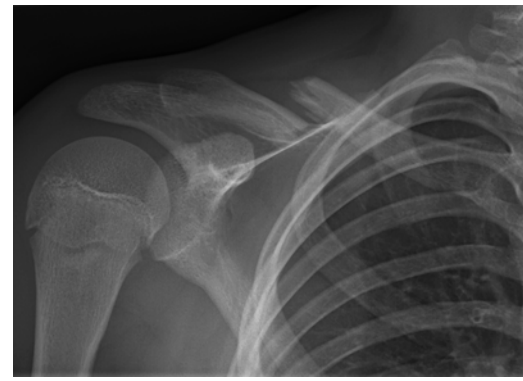

Fig. 6.19 Displaced clavicular fracture in a 12-year-old boy. The distal fracture is displaced inferiorly due to an intact coracoclavicular ligament. The proximal fracture fragment is displaced superiorly due to unbalanced force of the sternocleidomastoid muscle

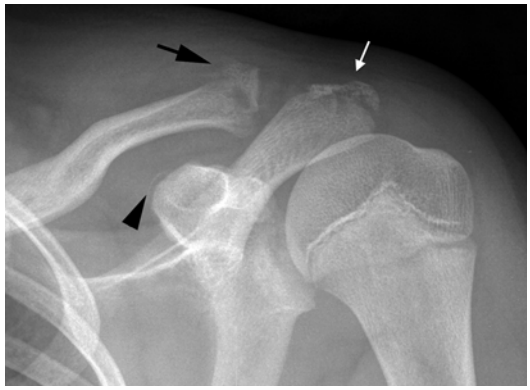

Fig. 6.20 Distal clavicular fracture in a 12 year-old boy. The clavicle fracture (*black arrow*) is a Salter-Harris II equivalent fracture since it extends to the distal clavicular physis. Normal fragmentary appearance of the acromial ossification center (*white arrow*) and apophysis of the coracoid at the level of the coracoclavicualr ligament (*black arrowhead*) should not be construed as fractures

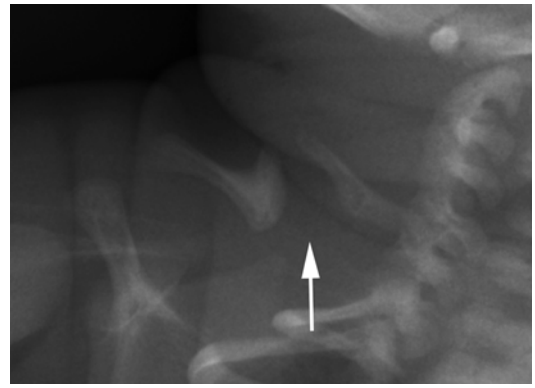

Fig. 6.21 Right clavicular pseudarthrosis (*arrow*) in a 6-day-old boy

tion centers do not appear until late adolescence [35], any fracture line extending to the medial or lateral clavicular end should be considered a Salter-Harris fracture.

Proximal clavicular fractures are easily missed on standard clavicular views due to the overlapping structures but are readily diagnosed by CT. CT is especially important for posteriorly displaced clavicular fractures since these may impinge upon the mediastinum.

The Neer and Rockwood classification system helps categorize distal clavicular fractures [38, 39]. Type 1 fractures are distal to the coracoclavicular ligaments, and therefore displacement is uncommon. Type 2 fractures are more medial, between the coracoclavicular ligaments. These fractures tend to be displaced because the distal component retains its alignment with the acromioclavicular joint, whereas the proximal component is aligned with the coracoid. Nonunion is more common with type 2 fractures. There may be coracoid avulsion injuries as well.

The floating shoulder, defined as a midclavicular fracture associated with a glenoid neck fracture, is considered unstable [40]. It is therefore important that clavicle radiograph series include the ipsilateral glenohumeral joint so that this fracture will be properly identified.

If acromioclavicular or coracoclavicular injury is suspected, it is essential to evaluate both the normal and the symptomatic shoulder with wide-view radiographs with and without weights. Views of the normal shoulder are important because normative adult measurements of the acromioclavicular and coracoclavicular distances on radiographs cannot be used in children, since the true ends of the acromion and coracoid are cartilaginous until late adolescence and even into early adulthood. For instance, the secondary ossification center of the acromion does not develop until between 15 and 18 years of age, and it does not fully fuse until age 22–25 [41]. The coracoid process has multiple ossification centers: the first appears by 1 year of age, the second at 10, and the third during puberty (Fig. 6.20). All three fuse by age 15 [42].

Differential Diagnosis

Midclavicular fractures should be distinguished from congenital pseudarthrosis of the clavicle (Fig. 6.21) (see Chap. 5), which almost always affects the right clavicle (except in cases of situs inversus). This results from failure of the medial and lateral clavicular mesenchymal primary centers to coalesce [43]. The ends of clavicular pseudarthrosis tend to be rounded and well corticated.

6 Scapular Fractures

Scapular fractures usually result from direct trauma related to either a motor vehicle accident in older patients or child abuse in infants and toddlers [44, 45]. Child abuse should always be suspected in infants and toddlers with scapular or

acromial fractures (Fig. 6.22) [41, 45]. The scapula is also prone to avulsion fractures due to its numerous muscle attachments.

Direct trauma usually causes fractures of the lateral scapular body (Fig. 6.23), although occasionally greenstick fractures occur [46]. Athletes (especially those who throw a great deal) may develop scapular stress fractures along the lateral border due to repetitive tug injury at the teres minor origin [47].

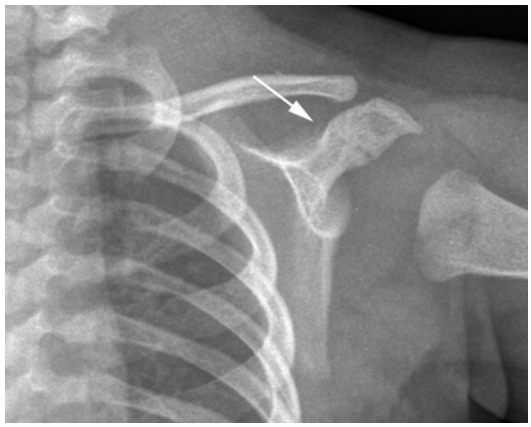

Fig. 6.22 Healing non-displaced acromion fracture (*arrow*) in a 5-month-old boy, a victim of non-accidental trauma

Avulsion fractures can occur at the many different sites of muscle attachment. Winging of the scapula—most often a result of injury to the long thoracic nerve—can result from avulsion fracture at the insertion of the serratus anterior muscle along the inferior angle of the scapula [48]. Additional sites for scapular avulsion fracture include the infraglenoid tubercle (the attachment site for the triceps muscle), acromion (the deltoid muscle), coracoid (the coracoclavicular ligament), and coracoid tip (the short head biceps and coracobrachialis muscles), among others.

Extra-articular scapular fractures are usually treated nonoperatively if there is less than 2 cm of displacement [49]. Nonoperative management of scapular fractures usually results in healing without complications because the adjacent muscle bulk helps stabilize the fracture fragments. Surgical reduction is recommended when fractures involve the glenoid fossa and are displaced more than 5 mm [50].

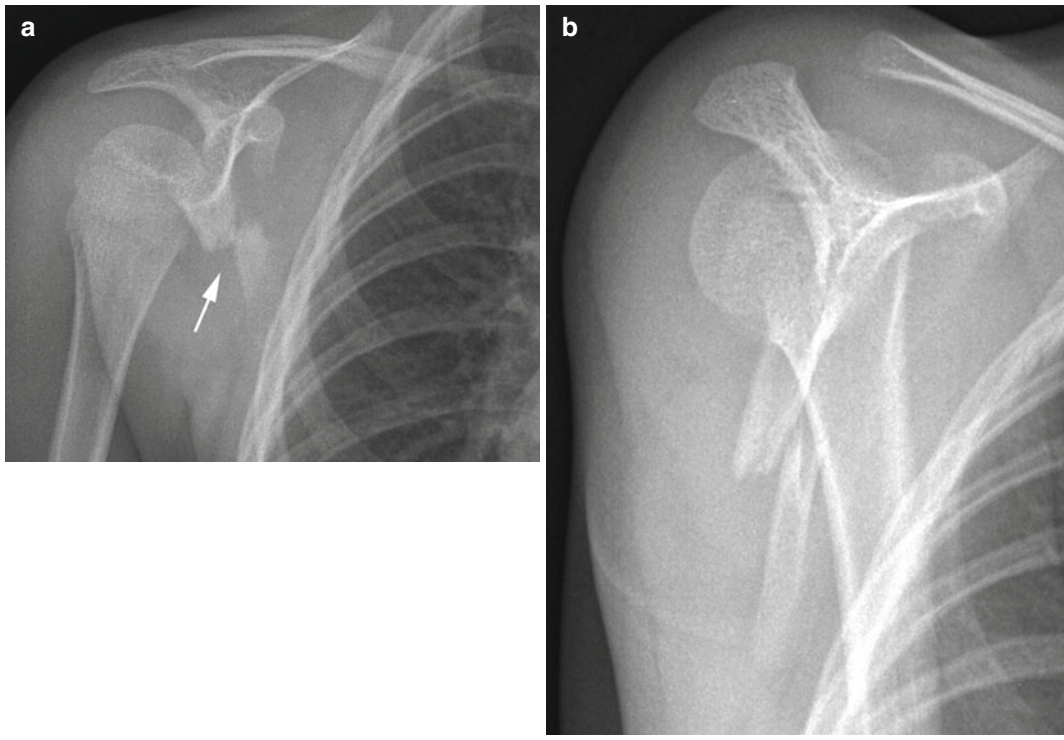

Fig. 6.23 Lateral scapular body fracture in a 4-year-old girl. Frontal view (**a**) shows medial displacement (*arrow*), and "Y" view (**b**) shows anterior displacement of the distal fragment

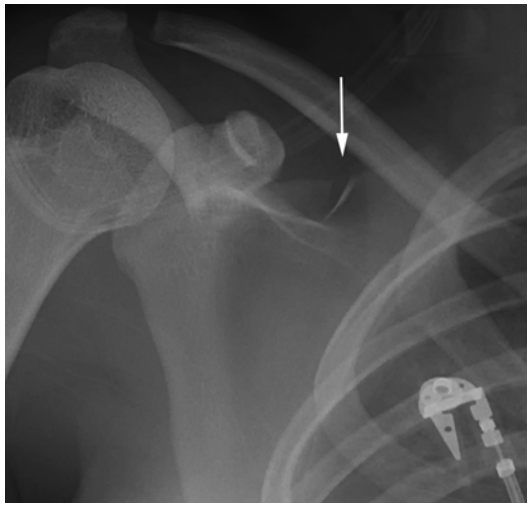

Fig. 6.24 Extra-articular superior scapular fracture (*arrow*) in a 13-year-old girl, a result of direct impaction injury

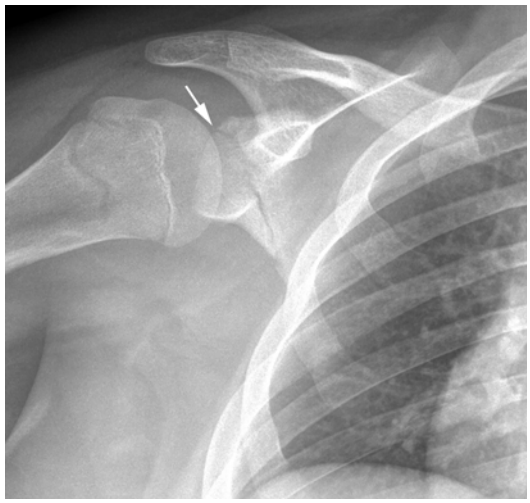

Fig. 6.25 Fractured scapular body and glenoid neck in a 10-year-old boy. There is intra-articular extension (*arrow*)

Imaging

If a fracture is suspected, the initial work-up includes shoulder and/or scapular radiographs (Fig. 6.24). It is important to address the degree of displacement, relationship of the scapular fracture to major muscle attachments, and involvement of the glenoid fossa (Figs. 6.24 and 6.25), since these factors affect management. If the fracture extends to the glenoid fossa, CT may help define the articular margin and determine displacement. However, the shoulder has often already been imaged in the course of a chest CT, as patients who suffer scapular fractures have usually sustained extensive additional trauma.

Differential Diagnosis

Scapular fractures should be differentiated from normal secondary ossification centers. These appear throughout the scapula during childhood and adolescence, as discussed above. They tend to have well-defined cortical margins and are rounded or ovoid, whereas true fracture lines have sharp non-corticated margins.

7 Chondral Injuries of the Glenohumeral Joint

Osteochondritis dissecans (OCD) most commonly occurs at the knee, usually during the second decade. Involvement of the glenohumeral joint is rare, but when OCD does affect the shoulder, it usually involves the anterosuperior humeral head [51]. Acute and/or repetitive microtrauma—both quite common in athletes—causes humeral head and glenoid OCD [52, 53]. Presenting symptoms include stiffness, locking, and clicking.

Stable OCD lesions are usually treated nonoperatively. If loose bodies or other features of instability are present, debridement, drilling, or allograft placement may be indicated [54]. As with OCD lesions elsewhere, severity of symptoms and the extent of physeal closure dictate the need for surgery. If the physis is still open, spontaneous healing is more likely. If an acute chondral or osteochondral fracture is unstable (discussed below), or if the patient experiences locking, debridement or possibly stabilization is generally advised.

Imaging

The appearance of glenohumeral OCD is similar to OCD elsewhere. If lesions are small, radiographs are usually normal. Larger lesions may demonstrate a subchondral lytic defect with or without underlying depression [51]. MRA best assesses these lesions, although they can usually be identified with routine shoulder MRI.

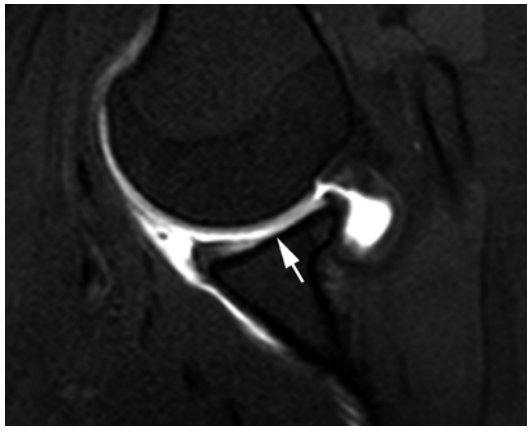

Fig. 6.26 The normal central bare area of the glenoid in a 16-year-old girl. Abduction external rotation (*ABER*) T1-W FS MRA shows typical smooth, tapering central bare area of the glenoid (*arrow*)

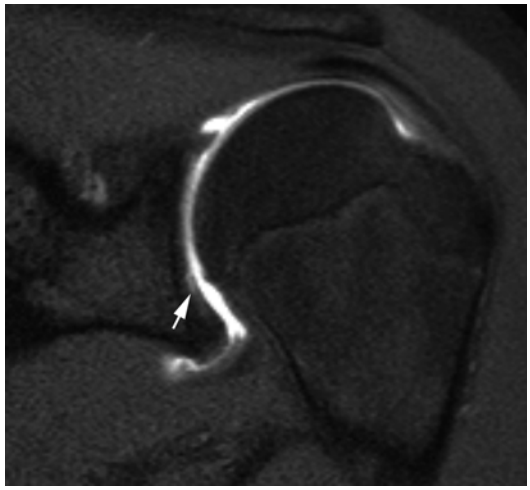

Fig. 6.27 Non-displaced delaminating chondral injury of the glenoid in a 16-year-old boy. Coronal T1-W FS MRA shows contrast insinuating (*arrow*) between the deep and superficial cartilage layers

Repeated insult, partial healing response, and remodeling result in MRA features that include overlying cartilage loss, cartilage fissuring, gadolinium insinuation between the cartilage fragment and parent bone, subchondral cysts, and loose body formation. Associated subchondral marrow edema and joint effusions may be seen. Any of these findings suggest the fragment is unstable.

Differential Diagnosis

Chondral injuries should be differentiated from the normal bare spot of the glenoid fossa [55]. On MRI, the normal glenoid bare spot is usually centrally located and has smoothly tapering margins without underlying cortical abnormality (Fig. 6.26). Alternative considerations for humeral head OCD include epiphyseal osteonecrosis, chondroblastoma, epiphyseal Brodie abscess related to osteomyelitis, and sequelae of prior shoulder dislocation with a depressed Hill-Sachs fracture. OCD injuries should be differentiated from acute cartilage and osteochondral fractures, which have more sharply demarcated margins (Fig. 6.27).

8 Soft Tissue Injuries About the Shoulder

Pediatric rotator cuff tears are extremely rare and occur in athletes suffering either acute injury or repetitive microtrauma [56]. Rotator cuff tears may also be seen in the setting of glenohumeral instability [17]. The scarcity of this injury has two causes: one, children have not lived long enough for repetitive microtrauma and overuse to compromise the rotator cuff, and, two, because the physis—not the tendon or myotendinous junction—is the weakest component of the musculoskeletal unit, biomechanical insult occurs at the physis before the rotator cuff is compromised.

References

1. Ecklund K. Magnetic resonance imaging of pediatric musculoskeletal trauma. Top Magn Reson Imaging. 2002;13(4):203–17.
2. Rowe CR. Prognosis in dislocations of the shoulder. J Bone Joint Surg Am. 1956;38-A(5):957–77.
3. Emery KH. Imaging of sports injuries of the upper extremity in children. Clin Sports Med. 2006;25(3):543–68, viii.
4. Davis KW. Imaging pediatric sports injuries: upper extremity. Radiol Clin North Am. 2010;48(6):1199–211.
5. Boone JL, Arciero RA. First-time anterior shoulder dislocations: has the standard changed? Br J Sports Med. 2010;44(5):355–60.

6. Omoumi P, et al. Glenohumeral joint instability. J Magn Reson Imaging. 2011;33(1):2–16.
7. Grahame R, Hakim AJ. Hypermobility. Curr Opin Rheumatol. 2008;20(1):106–10.
8. Macmahon PJ, Palmer WE. Magnetic resonance imaging in glenohumeral instability. Magn Reson Imaging Clin N Am. 2012;20(2):295–312, xi.
9. Jones KJ, et al. Functional outcomes of early arthroscopic Bankart repair in adolescents aged 11–18 years. J Pediatr Orthop. 2007;27(2):209–13.
10. Kirkley A, et al. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder: long-term evaluation. Arthroscopy. 2005;21(1):55–63.
11. Chuang TY, Adams CR, Burkhart SS. Use of preoperative three-dimensional computed tomography to quantify glenoid bone loss in shoulder instability. Arthroscopy. 2008;24(4):376–82.
12. Burkhart SS, De Beer JF. Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill-Sachs lesion. Arthroscopy. 2000;16(7):677–94.
13. Paxinos A, et al. Advances in the management of traumatic anterior and atraumatic multidirectional shoulder instability. Sports Med. 2001;31(11):819–28.
14. Waldt S, et al. Anterior shoulder instability: accuracy of MR arthrography in the classification of anteroinferior labroligamentous injuries. Radiology. 2005;237(2):578–83.
15. Oh JH, et al. Effectiveness of multidetector computed tomography arthrography for the diagnosis of shoulder pathology: comparison with magnetic resonance imaging with arthroscopic correlation. J Shoulder Elbow Surg. 2010;19(1):14–20.
16. Beltran J, et al. Glenohumeral instability: evaluation with MR arthrography. Radiographics. 1997;17(3):657–73.
17. Yiannakopoulos CK, Mataragas E, Antonogiannakis E. A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. Arthroscopy. 2007;23(9):985–90.
18. Pahlavan S, et al. Proximal humerus fractures in the pediatric population: a systematic review. J Child Orthop. 2011;5(3):187–94.
19. Neer 2nd CS, Horwitz BS. Fractures of the proximal humeral epiphysial plate. Clin Orthop Relat Res. 1965;41:24–31.
20. Bishop JY, Flatow EL. Pediatric shoulder trauma. Clin Orthop Relat Res. 2005;432:41–8.
21. Beringer DC, et al. Severely displaced proximal humeral epiphyseal fractures: a follow-up study. J Pediatr Orthop. 1998;18(1):31–7.
22. Lucas JC, Mehlman CT, Laor T. The location of the biceps tendon in completely displaced proximal humerus fractures in children: a report of four cases with magnetic resonance imaging and cadaveric correlation. J Pediatr Orthop. 2004;24(3):249–53.
23. Osbahr DC, Kim HJ, Dugas JR. Little league shoulder. Curr Opin Pediatr. 2010;22(1):35–40.
24. Meister K, et al. Rotational motion changes in the glenohumeral joint of the adolescent/Little League baseball player. Am J Sports Med. 2005;33(5):693–8.
25. Carson Jr WG, Gasser SI. Little Leaguer's shoulder. A report of 23 cases. Am J Sports Med. 1998;26(4):575–80.
26. Fleming JL, et al. Little Leaguer's shoulder. Skelet Radiol. 2004;33(6):352–4.
27. Obembe OO, et al. Little Leaguer's shoulder (proximal humeral epiphysiolysis): MRI findings in four boys. Pediatr Radiol. 2007;37(9):885–9.
28. Song JC, Lazarus ML, Song AP. MRI findings in Little Leaguer's shoulder. Skelet Radiol. 2006;35(2):107–9.
29. Hatem SF, Recht MP, Profitt B. MRI of Little Leaguer's shoulder. Skelet Radiol. 2006;35(2):103–6.
30. Kibler WB, Sciascia A, Thomas SJ. Glenohumeral internal rotation deficit: pathogenesis and response to acute throwing. Sports Med Arthrosc. 2012;20(1):34–8.
31. Giaroli EL, Major NM, Higgins LD. MRI of internal impingement of the shoulder. AJR Am J Roentgenol. 2005;185(4):925–9.
32. Tehranzadeh AD, Fronek J, Resnick D. Posterior capsular fibrosis in professional baseball pitchers: case series of MR arthrographic findings in six patients with glenohumeral internal rotational deficit. Clin Imaging. 2007;31(5):343–8.
33. Kocher MS, Waters PM, Micheli LJ. Upper extremity injuries in the paediatric athlete. Sports Med. 2000;30(2):117–35.
34. Soto F, et al. Presentation, evaluation, and treatment of clavicle fractures in preschool children presenting to an emergency department. Pediatr Emerg Care. 2009;25(11):744–7.
35. Kubiak R, Slongo T. Operative treatment of clavicle fractures in children: a review of 21 years. J Pediatr Orthop. 2002;22(6):736–9.
36. Black GB, McPherson JA, Reed MH. Traumatic pseudodislocation of the acromioclavicular joint in children. A fifteen year review. Am J Sports Med. 1991;19(6):644–6.
37. Nenopoulos SP, et al. Outcome of distal clavicular fracture separations and dislocations in immature skeleton. Injury. 2011;42(4):376–80.
38. Neer CS. Fractures of the distal third of the clavicle. Clin Orthop Relat Res. 1968;58:43.
39. Rockwood CA. Fractures of the outer clavicle in children and adults. J Bone Joint Surg Br. 1982;64B:642 (abstract).
40. Owens BD, Goss TP. The floating shoulder. J Bone Joint Surg Br. 2006;88(11):1419–24.
41. Currarino G, Prescott P. Fractures of the acromion in young children and a description of a variant in acromial ossification which may mimic a fracture. Pediatr Radiol. 1994;24(4):251–5.
42. Kim SJ, et al. Bilateral unfused coracoid process: report of a case. J Korean Med Sci. 1998;13(5):563–5.
43. Currarino G, Herring JA. Congenital pseudarthrosis of the clavicle. Pediatr Radiol. 2009;39(12):1343–9.

44. Wilber MC, Evans EB. Fractures of the scapula. An analysis of forty cases and a review of the literature. J Bone Joint Surg Am. 1977;59(3):358–62.
45. Kogutt MS, Swischuk LE, Fagan CJ. Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Ther Nucl Med. 1974;121(1):143–9.
46. Bowen TR, Miller F. Greenstick fracture of the scapula: a cause of scapular winging. J Orthop Trauma. 2006;20(2):147–9.
47. Herickhoff PK, et al. Scapular stress fracture in a professional baseball player: a case report and review of the literature. Am J Sports Med. 2007;35(7):1193–6.
48. Heyse-Moore GH, Stoker DJ. Avulsion fractures of the scapula. Skelet Radiol. 1982;9(1):27–32.
49. Jones CB, Sietsema DL. Analysis of operative versus nonoperative treatment of displaced scapular fractures. Clin Orthop Relat Res. 2011;469(12):3379–89.
50. Lantry JM, Roberts CS, Giannoudis PV. Operative treatment of scapular fractures: a systematic review. Injury. 2008;39(3):271–83.
51. Hamada S, et al. Osteochondritis dissecans of the humeral head. Arthroscopy. 1992;8(1):132–7.
52. Mahirogullari M, et al. Osteochondritis dissecans of the humeral head. Joint Bone Spine. 2008;75(2):226–8.
53. Chu PJ, et al. Osteochondritis dissecans of the glenoid: a rare injury secondary to repetitive microtrauma. J Trauma. 2009;67(3):E62–4.
54. Johnson DL, Warner JJ. Osteochondritis dissecans of the humeral head: treatment with a matched osteochondral allograft. J Shoulder Elb Surg. 1997;6(2):160–3.
55. Kim HK, Emery KH, Salisbury SR. Bare spot of the glenoid fossa in children: incidence and MRI features. Pediatr Radiol. 2010;40(7):1190–6.
56. Tarkin IS, et al. Rotator cuff tears in adolescent athletes. Am J Sports Med. 2005;33(4):596–601.