39 Case Example 2: Combined Massive Rotator Cuff Tear and Recurrent Shoulder Instability

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39.1 Introduction

Recurrent shoulder instability which results from a massive rotator cuff tear is uncommon [1]. Massive rotator cuff tear is defined as a complete detachment of at least two tendons [2]. The term shoulder instability is used to refer to the inability to maintain the humeral head in the glenoid fossa. Glenohumeral joint is the most mobile joint in our body. Its significant range of motion is achieved due to the lack of bony constraints which sets the stage for pathologic instability. This balance between shoulder mobility and stability is related to a complex combination of dynamic and static stabilizers around the shoulder joint.

Static stabilizers are as follows:

1. Vacuum effect provided by intracapsular negative pressure, suction effect of the glenoid labrum against humeral head, and adhesion-cohesion effect between two wet surfaces.
2. Bonny geometry: Normally the glenoid is anteverted in relation to the humerus which prevents posterior instability. A loss of that physiological version may affect the stability as we see in glenoid dysplasia.
3. Glenoid labrum increases the glenoid surface, serves as attachment for the glenohumeral ligaments and the long head of biceps, and prevents translation of the humeral head.
4. Glenohumeral capsule works as a socket. Together with glenohumeral ligaments it stabilizes the humeral head in all directions.

Dynamic stabilizers are as follows:

1. Proprioception: Glenohumeral capsule with its receptors sends information to periscapular muscles. In case of stretching of the capsule, the periscapular muscles contract, which prevents dislocation.
2. Rotator cuff (RC) creates concavity compression mechanism which maintains the center of rotation and stabilizes the shoulder at middle range of motion (ROM) when the ligamentous structures are lax and at terminal ROM through muscle activity that limits motion and decreases strain on the glenohumeral ligaments [3].

Disruption of the balance between dynamic and static stabilizers due to their loss of integrity may lead to instability.

Case Example

A 63-year-old female patient presented to our outpatient department was complaining of a right

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N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_39

shoulder pain lasting for a year, which has been worsened after a fall directly on the shoulder 6 days prior. The injury was managed initially by another institution. On the day of the injury she was examined in the emergency department where fracture of the injured shoulder was excluded on plain radiographs. On examination in our hospital she complained of inability to use her right upper limb in everyday activities. She complained of shoulder instability. She had up to five shoulder dislocations per day after the injury. The dislocated shoulder was relocated by herself. Before the injury she had a feeling of instability but no dislocations. Pain was present in every movement of the affected arm. She complained of a shoulder pain at night, which frequently woke her up. Before the recent injury she performed one cycle of physiotherapy (hydrogymnastics, laser therapy, exercises for the shoulder, diadynamic therapy) prescribed by her physiatrist, which was unsuccessful. Four months prior she had an ultrasound examination of her right shoulder, which showed a complete rupture of the supraspinatus tendon with subacromial bursitis. During the examination of her shoulder an atrophy of the supraspinatus muscle region was present. Active anteflexion and abduction was up to 90° but very painful, active retroflexion up to 20°. Jobe test, belly-press test, infraspinatus test, anterior apprehension test, and sulcus sign test were positive. Function of the axillary nerve was intact; no motor or sensory deficits were identified during neurological examination. The neck was well movable and painless. X-ray of the right shoulder was performed (Fig. 39.1).

Computed tomography arthrography (CTA) of her right shoulder showed medium-sized Hill-Sachs lesion, Bankart lesion with continuation along the anterior labrum into a minor superior labrum anterior to posterior (SLAP) lesion, complete rupture of supraspinatus (SSP) tendon with retraction about 1.5 cm laterally from the acromioclavicular joint, partial rupture of infraspinatus (ISP) tendon, moderate atrophy of the SSP

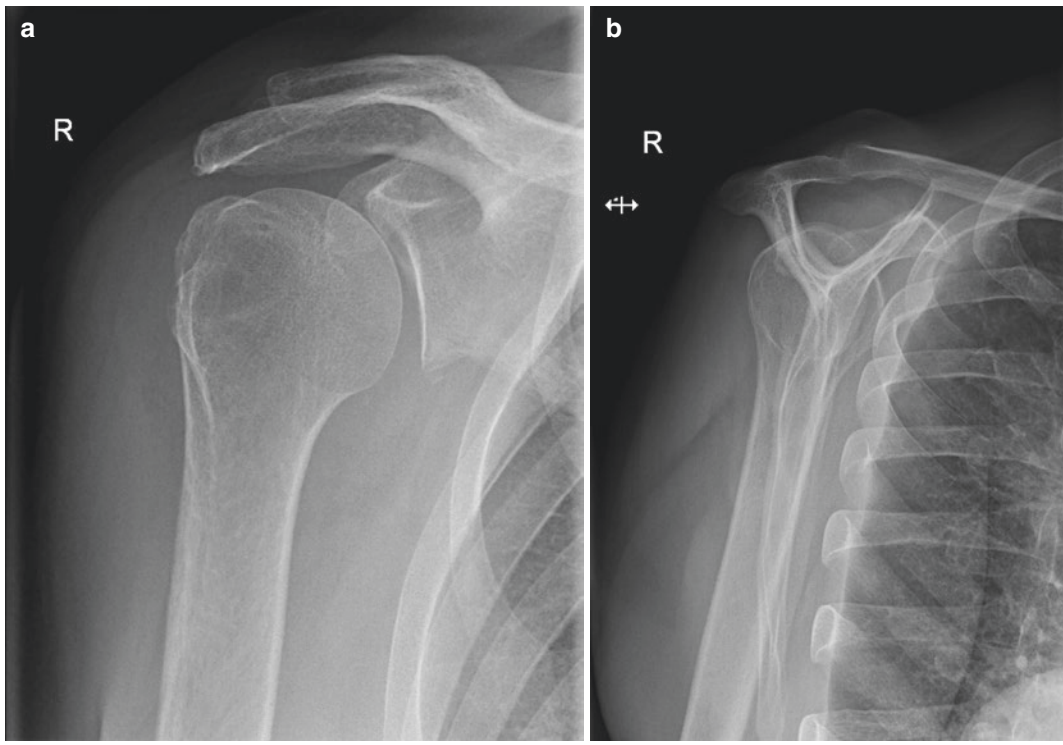

Fig. 39.1 X-ray of the right shoulder with AP view (**a**) and Y view (**b**): A slight cranial translation of the humeral head without glenohumeral arthrosis is seen

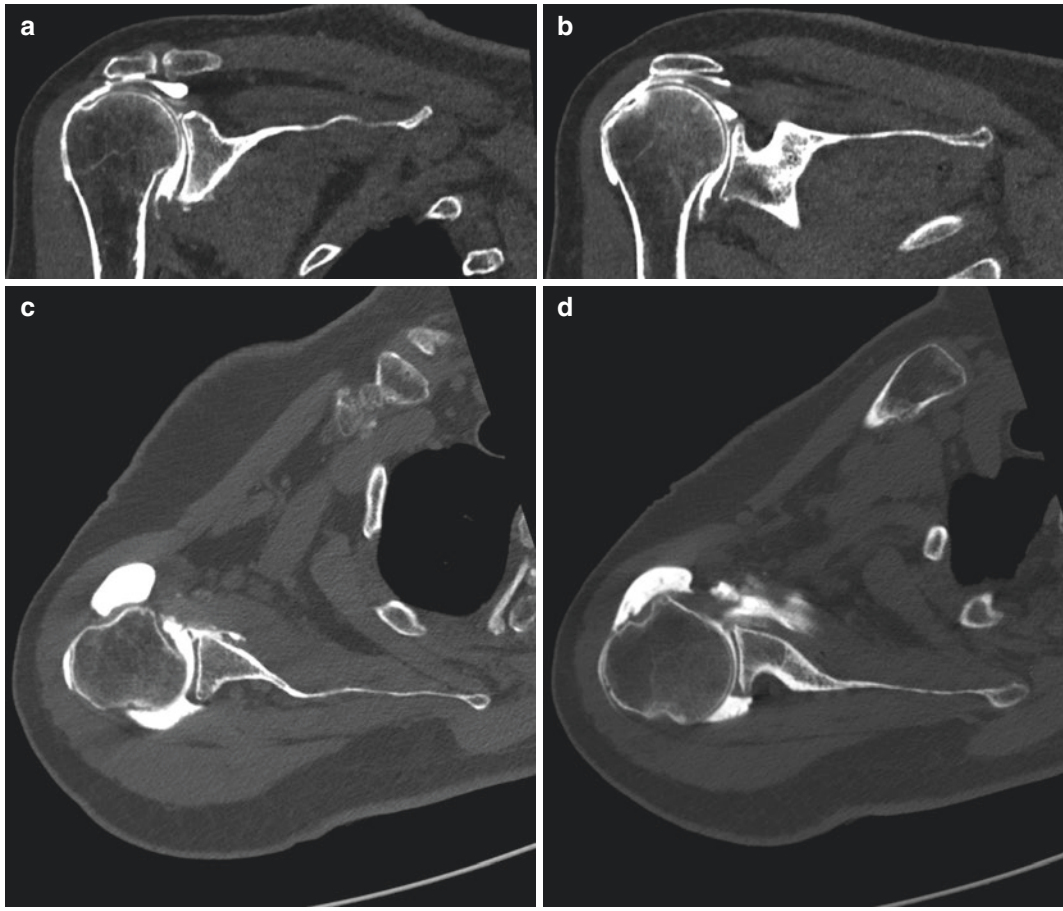

Fig. 39.2 CTA of the right shoulder: Coronal reconstruction shows a minor SLAP lesion (**a**) and a SSP lesion with cranial translation (**b**). On transverse reconstruction a Bankart lesion (**c**) and a Hill-Sachs lesion (**d**) were seen

and ISP muscle, and absence of glenohumeral joint arthrosis (Fig. 39.2).

Due to the evidence of gross instability both an arthroscopic stabilization and RC reconstruction of the injured shoulder was advised. We decided not to implant a reverse total shoulder prosthesis due to the absence of glenohumeral joint arthrosis. Arthroscopy of the shoulder confirmed the radiologically identified pathological lesions and additional finding was a rupture of the upper part of the subscapularis tendon, medially subluxated long head of the biceps tendon, and not a partial but a complete rupture of ISP tendon. During the surgery we performed an arthroscopic Bankart repair, remplissage and reconstruction of the ISP to the footprint, reconstruction of subscapularis, SSP, and long head of the biceps tenotomy. After the surgery the patient started with rehabilitation. Six months postoperatively the patient was satisfied with the result. She had no feeling of instability, no pain in her shoulder with a very good range of motion (Fig. 39.3).

39.2 Discussion

The prevalence of traumatic anterior shoulder dislocations in the elderly population has been increasing due to prolonged life expectancy [1, 4]. Shoulder dislocations in the elderly have a different pathological spectrum with specific surgical implications compared to the younger population. Especially the RC tears have been the

Fig. 39.3 Patient and her ROM 6 months postoperatively

predominant pathological lesion that induce shoulder instability in the elderly population [1]. Robinson et al. have shown that a massive RC tear is one of the most important risk factors for early re-dislocation within a week after a first-time anterior traumatic dislocation [5].

As described earlier one of the main dynamic stabilizers is represented by the RC through concavity compression mechanism. Pouliart et al. have shown in a cadaveric study that smaller capsuloligamentous lesions were needed to lead to dislocation in the presence of RC deficient model [6]. Rowe found similar rates of shoulder dislocations among patients younger or older than 45 years of age [7]. Gumina and Postacchini have reported an incidence of shoulder instability in patients above 60 years of age to be up to 20% of acute anterior dislocations [8]. One of the major differences between shoulder instability in younger and elderly patients is the low recurrence rate in the elderly compared to the high recurrence rate in young population. The percentage of recurrent shoulder dislocations in patients bellow the age of 20 is reported to be between 68 and 95%, whereas in patients older than 60 years between 11 and 31% [9–11].

Shoulder dislocation may result in a RC tear, both in younger sportsmen or older patients with age-related degenerative tendinopathy. Shoulder dislocations in elderly are more commonly associated with neurovascular injuries, fractures, and RC tears. In younger patients, it is thought that anterior dislocation often causes more damage to the anterior capsulolabral support structures.

Whereas in elderly, it has been considered that dislocation results more in a disruption of the posterior support structures such as the posterosuperior RC, while it rarely affects the anterior glenoid labrum and/or capsular structures [12]. Craig popularized the posterior mechanism of instability in a reported series of three patients older than 60 years who had suffered a RC tear combined with recurrent instability after an anterior dislocation. He proposed that recurrent anterior instability may be a result of posterior RC failure [13].

The prevalence of RC tears after an anterior shoulder dislocation is estimated to be between 7 and 32% and is more common with advancing age. The percentage of elderly patients with this comorbidity is 34–100% [1]. This age-related pathophysiology of anterior shoulder dislocation is attributed to deterioration of the structure and mechanical properties of RC tendons [14]. A cadaveric study by Lehman et al. has shown an age-related increase in full-thickness RC tears. In cadavers aged under 60 years the incidence of RC tears was 6% whereas in those above 60 years of age the incidence rose to 30% [15]. Gombera et al. postulated in their systematic review that pain and weakness persisting for up to 3 weeks after an anterior dislocation should set a high suspicion for a RC tear and further investigations should be taken [16].

Once the patient is diagnosed with a combination of shoulder instability and RC lesion, treatment options must be considered. If a patient has a balanced RC lesion, minimal pain and absence of recurrent instability conservative treatment may be considered [17]. However, in patients who continue to suffer from persistent pain and shoulder dysfunction due to recurrent dislocations, surgery may result in less pain and improved function [16].

There is no clear consensus as to whether either the RC tear or the anterior capsulolabral complex injury or both should be treated surgically if they occur simultaneously in a patient with recurrent anterior dislocation of the shoulder. Itoi and Tabata reported a satisfactory outcome in 8 out of 11 (73%) patients when only the RC tear was repaired while the Bankart lesion was left unrepaired [18]. On the other hand, Gumina and Postacchini argued that both RC tear and Bankart lesion must be repaired to achieve satisfactory result [8]. Shin et al. suggested that since massive RC tears alone can induce shoulder joint instability, their repair is sufficient to stabilize the joint, while the Bankart lesion is left unrepaired [19]. In this cases anterior capsulolabral repair does not affect the stability of the joint [1]. However, when the size of RC tear was small to medium, Bankart repair should be performed as well, since small RC tear alone is insufficient to induce instability of the shoulder, which in this case appears to be in a large part caused by the anterior capsulolabral lesion [19].

39.3 Summary

Rotator cuff plays an important role in preventing glenohumeral instability as it stabilizes and centers the humeral head in the glenoid fossa through concavity compression mechanism. Capsulolabral lesion is a very common finding in a dislocated shoulder but a single dislocation event may also disrupt a rotator cuff, which is already weakened from overuse injury in younger athletes and from age-related degeneration in elderly. We must be careful in assessing a patient with persisting pain and weakness up to 3 weeks after an anterior shoulder dislocation and consider further investigations to exclude a rotator cuff injury. Surgical repair of either the rotator cuff, the capsulolabral structure, or both has been proposed in patients with recurrent anterior shoulder dislocation. Currently, there are no definitive answers on which surgical treatments bring the best clinical results.

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