Natural History

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Eraclio Siuni

The anatomopathological aspects of the glenohumeral arthropathy associated with the massive rupture of the rotator cuff were already described in the nineteenth century.

R. W. Smith, a professor of surgery in the University of Dublin, in the original communications section published in the *Quarterly Journal of Medical Science* in Dublin in February 1853 [1, 2], described the case of a man of about 60 years of age where a chronic rheumatic arthritis had been established in the shoulder joint.

He wrote "Upon the right side, the head of the humerus, placed much farther back than natural, and elevated so as to be in contact with the under surface of the acromion process"…."it was found that no articular surface existed in the normal situation of the glenoid cavity, but upon the external aspect of the neck of the scapula, there had been formed a glenoid-shaped, concave surface, for the reception of the head of the humerus"…."The head of the humerus had lost completely the globular form which it possesses in the normal state; it was flattened from within outwards."

Adams, a Regius Professor of surgery at the University of Dublin, explained in his *A Treatise on Rheumatic Gout, or Chronic Rheumatic Arthritis of All the Joints* published in 1873 [3] a localized form of chronic rheumatoid arthritis involving the shoulder, characterized by biceps tendon rupture and rotator cuff tear, erosion of the upper portion of the humeral head, and erosion of the undersurface of the acromion process and of the distal third of the clavicle.

Codman, in his text *The Shoulder* published in 1934 [4], described the case of a 51-year-old woman suffering from a traumatic rotator cuff tear resulting from a fall. At the time of the intervention, 6 years after the traumatic event, he found a major defect in the rotator cuff associated with an atrophy of the surrounding muscle, a severe glenohumeral arthropathy, intra-articular loose bodies, a chronic synovitis, and effusion of the bursa or joint. He attributed the pathological changes to the mechanical forces acting on the shoulder joint as a result of the functional insufficiency of a chronically neglected large rotator cuff tear.

In the 1950s and 1960s of the last century, several authors, Galmiche and Deshayes [5] in 1958, Shephard [6] and Snook [7] in 1963, Burman [8] and Banna [9] in 1964, and Bauduin and Famaey [10] in 1969, have published numerous cases of elderly patients with supraspinatus tendon tears and shoulder arthropathy associated with recurrent spontaneous hemorrhage into the subdeltoid bursa and glenohumeral joint.

DeSeze [11] in 1968 exposed "l'épaule sénile hémorragique" characterized by a chronic rupture of the rotator cuff, associated with recurrent hemorrhage of the shoulder and severe

E. Siuni (✉)
Responsible "Shoulder Surgery Unit", SCOMR
Marino Hospital, Cagliari Ats Sardinia, Italy

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arthropathy. Also referring to previous reports in the literature, he then studied this clinical entity in three elderly women who did not have a history of trauma.

Lamboley [12] in 1977 presented a report of nine cases of elderly women who suffered from recurrent painful swelling on the shoulder and identified an association between rotator cuff tear and glenohumeral arthropathy and arthritis of the knee joint.

Charles S. Neer in 1977 [13] then introduced, for the first time, the term "cuff tear arthropathy" to describe degenerative changes in the glenohumeral joint associated with a chronic cuff tear, including erosion of the articular surfaces, restricted shoulder motion, osteopenia, and collapse of the humeral head.

Halverson et al. in 1981 [14–16] coined the term "Milwaukee shoulder" to describe the condition of elderly women who had chronic rotator cuff tear with recurrent bilateral shoulder effusions and radiographic destructive changes of the glenohumeral joints. They found out that basic calcium phosphate (BCP) crystals, such as hydroxyapatite, accumulate in elevated levels, in the synovial tissue and fluid of shoulders with rotator cuff deficiency and arthropathy.

They then suggested the so-called inflammatory-mediated theory to explain the arthropathy associated with the rotator cuff tear and hypothesized that the basic calcium phosphate crystals such as hydroxyapatite initiate a cascade of events. The resulting phagocytosis of these crystals by macrophages would induce the release of proteolytic enzymes such as collagenases and proteases that would cause degradation of cartilage matrix components and periarticular and articular structures [17–19].

Tissue damage involves an additional release of crystals in the synovial fluid by triggering a vicious circle and resulting in an accelerated degeneration of the rotator cuff and biceps tendon, leading to glenohumeral joint destruction [20, 21].

Dieppe et al. in 1984 [22] introduced the term "apatite-associated destructive arthritis," and in 1985 [23], after noticing that the basic calcium phosphate crystals were found in osteoarthritis, neuropathic joints, and joint tissue of healthy elderly patients, they argued that BCP crystals were actually the product of the wear and destruction of the articular surfaces and not the triggering cause. In 1988 [24] they later proposed the term "idiopathic destructive arthritis" to describe the arthropathy of the shoulder associated with rotator cuff tear.

In contrast, Neer et al. hypothesized in 1983 [25] that a massive rotator cuff tear was the inciting event in the development of rotator cuff tear arthropathy and that both mechanical and nutritional factors contributed to the subsequent progression of the arthropathy.

The scapulohumeral cingulum muscles, the rotator cuff, and the deltoid muscle act synergistically to maintain the balance of the shoulder joint on both the coronal and the transverse plane.

The mechanical theory suggests that the glenohumeral arthropathy was due to the deficient rotator cuff because without the superior stabilizing effect of the supraspinatus tendon, the humeral head for the action of the deltoid muscle tends to subluxate superiorly (Fig. 7.1a, b). The deterioration of the articular cartilage is a direct result of abnormal physical stresses imparted to the humeral head, leading to erosion of the upper portion of the humeral head (Fig. 7.2a, b, c), the superior glenoid fossa, and undersurface of the acromion (Fig. 7.3a, b) and erosion of the acromioclavicular joint and the coracoid (Fig. 7.4a, b).

The rotator cuff acts in the dynamic stability of the shoulder with balance between the subscapularis anteriorly and infraspinatus and teres minor posteriorly. A massive rotator cuff tear extends posteriorly with infraspinatus, and teres minor involvement entails an imbalance of this "force couples" (Burkhart 1992) resulting in further wear on the articular surfaces and acromion [26].

The concept of "concavity-compression" was coined by Hurov in 2009 [27], to explain the role of the rotator cuff in the dynamic stabilization of the shoulder by centering the convex humeral head on the concave glenoid fossa in all directions of movement.

A massive rotator cuff tear and rupture or dislocation of the long head of the biceps leads to unbalanced force coupling and loss of the con-

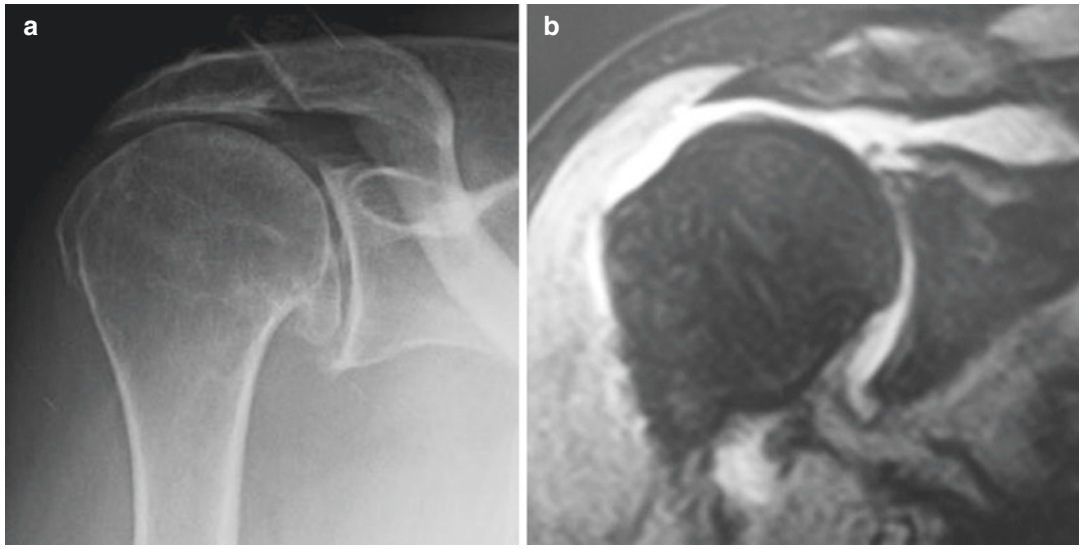

Fig. 7.1 (**a**, **b**) The humeral head, for the action of the deltoid muscle, tends to subluxate superiorly

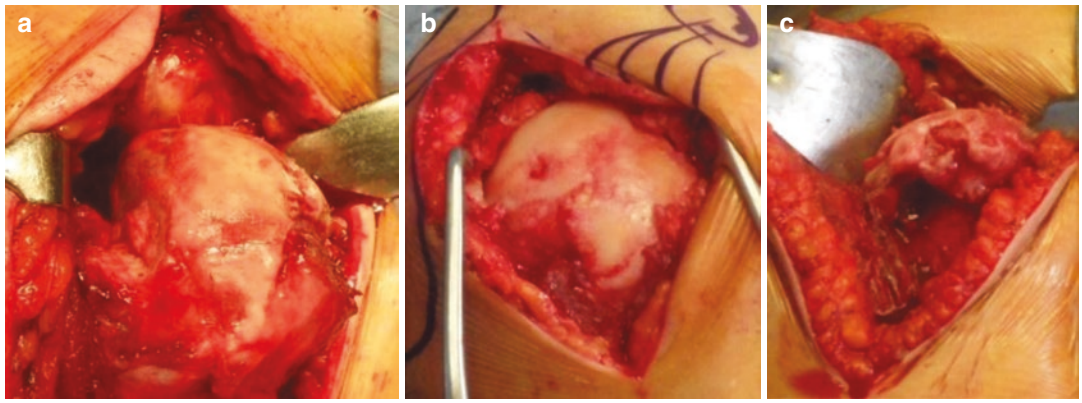

Fig. 7.2 (**a–c**) The deterioration of the articular cartilage is a direct result of abnormal physical stresses imparted to the humeral head, leading to erosion of the upper portion of the humeral head

cavity-compression mechanism which ultimately result in altered glenohumeral joint biomechanics [28–30]. In a study on eight cadaver shoulders performed by Ho et al. in 2011 [31], it was noted that biomechanical alterations in the shoulder develop after a full-thickness supraspinatus tear and at least 50% of the infraspinatus.

Neer, in 1983, also described the nutritional theory that, in association with alteration of the shoulder joint kinematic due to the massive full-thickness rotator cuff tear, leads to the development of the cuff tear arthropathy. The defect of the rotator cuff results in the loss of normal negative pressure within the joint space and the consequent spread of the synovial fluid in the surrounding tissues with loss of regular nutrition of articular cartilaginous surfaces. The defect of the rotator cuff also involves the reduction of joint movement and function, resulting in biochemical alteration of the synovial fluid and glycosaminoglycan content of cartilage, osteoporosis, cartilage atrophy and subchondral collapse (Fig. 7.5a, b), and the development of arthropathy cuff tear.

Collins and Harryman in 1997 [32] have suggested the combination of mechanical, nutritional, and biological concepts. The repeated

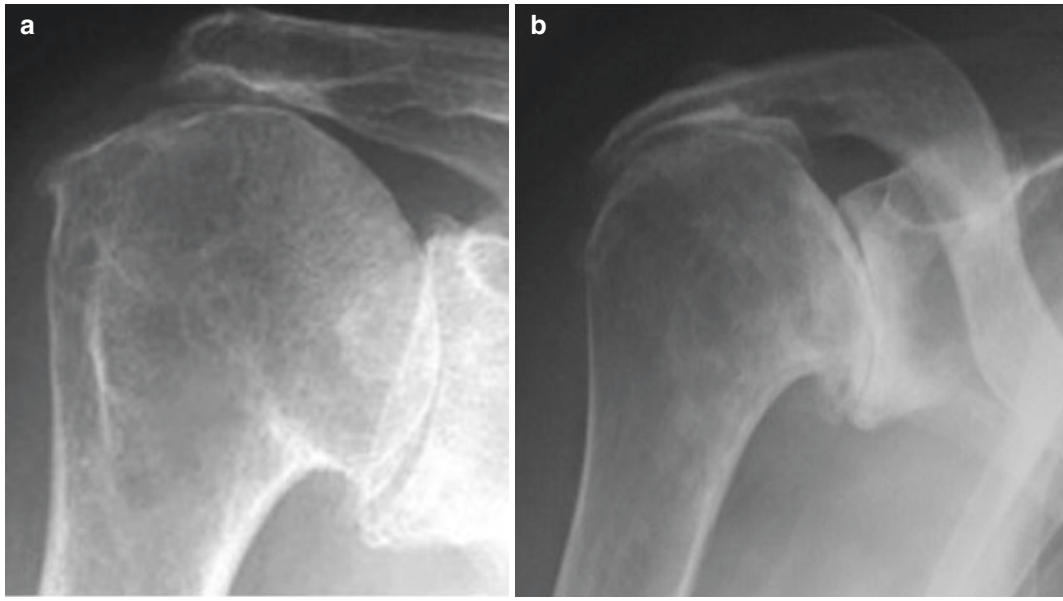

Fig. 7.3 (**a**, **b**) Erosion of the superior glenoid fossa and undersurface of the acromion

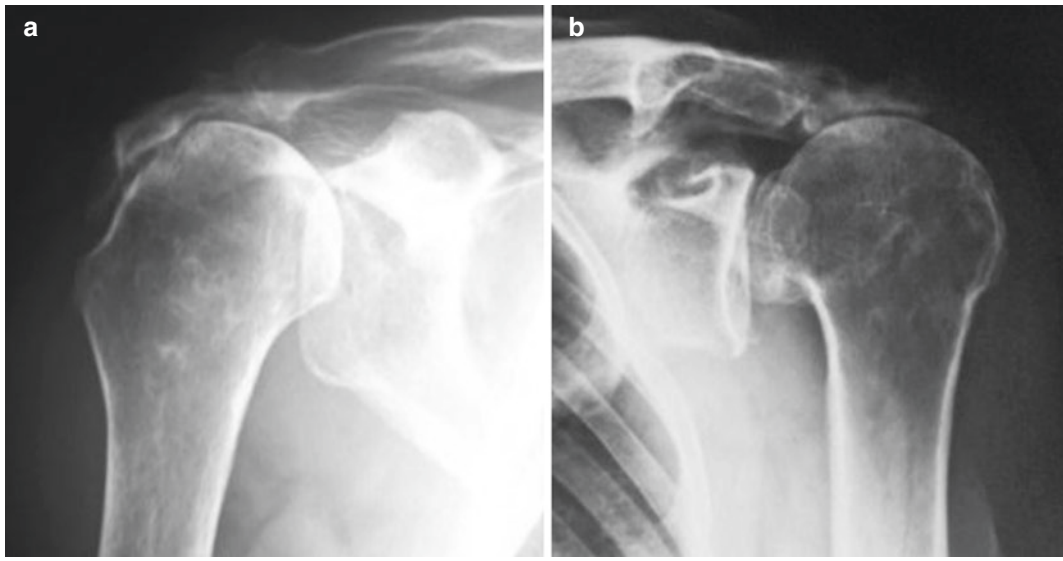

Fig. 7.4 (**a**, **b**) Erosion of the acromion and acromioclavicular joint

microtrauma created by the humeral head resting on the acromion leads to a fragmentation of the cartilage and particulate debris. Something that not only induces an enzymatic response, always associated with the pain and loss of movement, but also leads to further damage to the articular cartilage surface.

Nutrition theory has been questioned by some studies showing an increase in cartilage matrix metalloproteinase and both the correlation between the cytokine concentration and catabolic enzymes and the deterioration of articular cartilage after a massive rotator cuff tear [33–35].

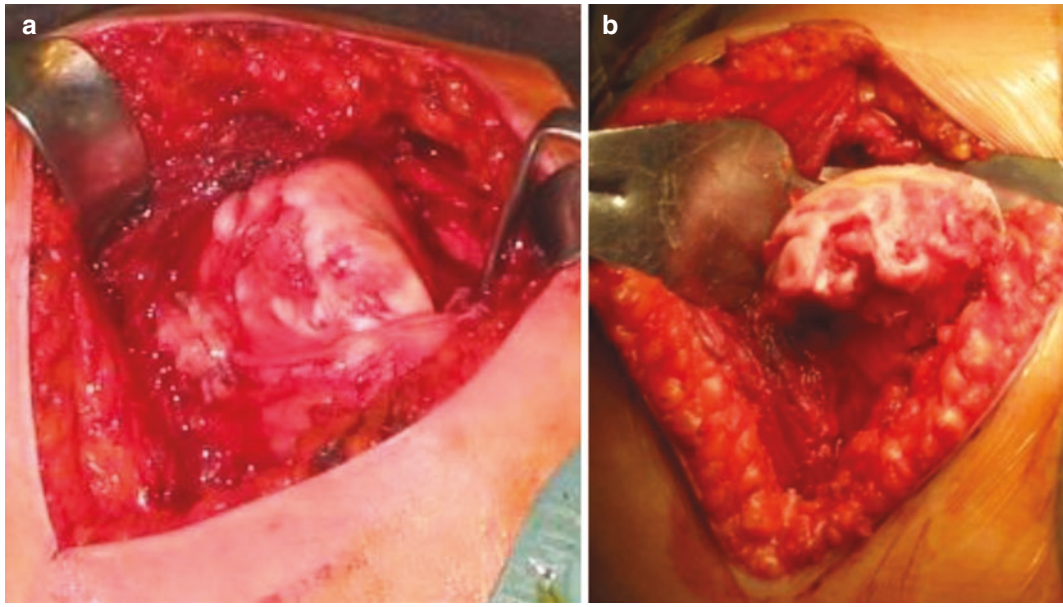

Fig. 7.5 (**a**, **b**) Cartilage atrophy and subchondral collapse of the humeral head

Kramer et al. in 2013 [36] performed a histological study on the shoulder joint cartilage of two rat groups, one with an induced rotator cuff tear and another with suprascapular nerve injury and intact rotator cuff. The results obtained over time have shown that cartilage deterioration was similar in the two groups. It must have been thought that perhaps the wear of the cartilage was due to the loss of balance of the mechanical forces acting on the glenohumeral joint.

Literature data [37, 38] indicates that rotator cuff injuries increase with age progression and that the percentage evolves from an average of 10.7% between the ages of 50 and 59 to 36.6% in the age from 80 to 89 years, with no significant differences between male and female subject [39]. Already Tempelhof et al. [40] in an ultrasound study published in 1999 highlighted the prevalence of 51% of full-thickness rotator cuff tears in asymptomatic patients over the age of 80 years.

However, a recent study published in 2016 [41] on the ultrasound examination of 486 volunteers showed contrasting results, with a prevalence of 11.1% of full-thickness rotator cuff tears in asymptomatic patients over the age of 70 years.

From the literature we gather that a rotator cuff tear, not subjected to surgical repair, though asymptomatic, can evolve toward a massive tear [42–45].

In a recent study published in 2017 [46], 69 patients were evaluated, of whom 45 with partial-thickness tears (PTT) and 24 with full-thickness tears (FTT), undergoing acromioplasty without tendon repair. It has been observed in a 22-year long-term evaluation that 74% of patients with FTT had developed X-ray cuff tear arthropathy >2° according to the Hamada classification and 87% had increased the tear size with ultrasonic examination. These authors stated that patients with full-thickness cuff tear and undergoing acromioplasty without cuff repair have, after 22 years, a high frequency of tear progression and cuff tear arthropathy. They concluded that only full thickness was a significant variable.

Several authors [29, 46, 47] also analyzed risk factors such as advanced age, sex, cuff tendon and biceps tendon status, trauma, high shoulder activity and manual labor, and their possible influence on the onset of cuff tear arthropathy for the progression of the tear rotator cuff. Nevertheless, other risk factors, such as hypercholesterolemia [48], hypertension [49, 50], and smoking [51, 52], only studied to evaluate the evolutionary rotator cuff tear on a degenerative

basis could also be cited for possible influences on the arthropathy cuff tear.

Neer et al. [25] reported that the arthropathy cuff tear would only develop in 4% of patients suffering from a complete tear of the rotator cuff.

It can be therefore stated that the progressive worsening of rotator cuff tear toward arthropathy is quite difficult to predict.

In a recent study [53] of 138 subjects, evaluated with 8-year radiographic and ultrasound examination, 24% of whom were control patients and 28% had partial-thickness and 49% full-thickness rotator cuff tears, it was showed that the magnitude of radiographic progression in Hamada grade is not influenced by the tear severity or enlargement. The size of rotator cuff damage alone does not seem to be associated with the development of typical alterations of the cuff tear arthropathy. Other individual, biological, or genetic factors may interfere with the natural progression to this severe and disabling pathology of the shoulder [37].

Cuff tear arthropathy is more common in elderly women [19, 29, 54–56] and dominant shoulder [55].

To explain the reason as to why arthropathy cuff tear is more common in female patients, Gumina et al. in 2017 [56] hypothesized both an autoimmune theory and a theory related to joint hyperlaxity, as the two pathological conditions are more frequent in women. In the first hypothesis, estrogens may interfere with the autoimmune mechanism seen that estrogen receptors are also present in the immune system complex. In the second theory, static stabilizers may be less effective and therefore conditions for an unstable shoulder in patients with massive rotator cuff tear. Unfortunately the available data did not allow the authors to make any valid conclusions.

In recent years, numerous studies have been conducted to investigate the etiopathogenesis, but several aspects that would better define patients with massive rotator cuff tears that may face an arthropathy still remain unknown.

Being able to identify and fully understand the risk factors related to rotator cuff disease, but above all to progression to the cuff tear arthropathy, would be crucial to implement a prevention strategy aimed at avoiding this severe disabling disease (Fig. 7.6a–d).

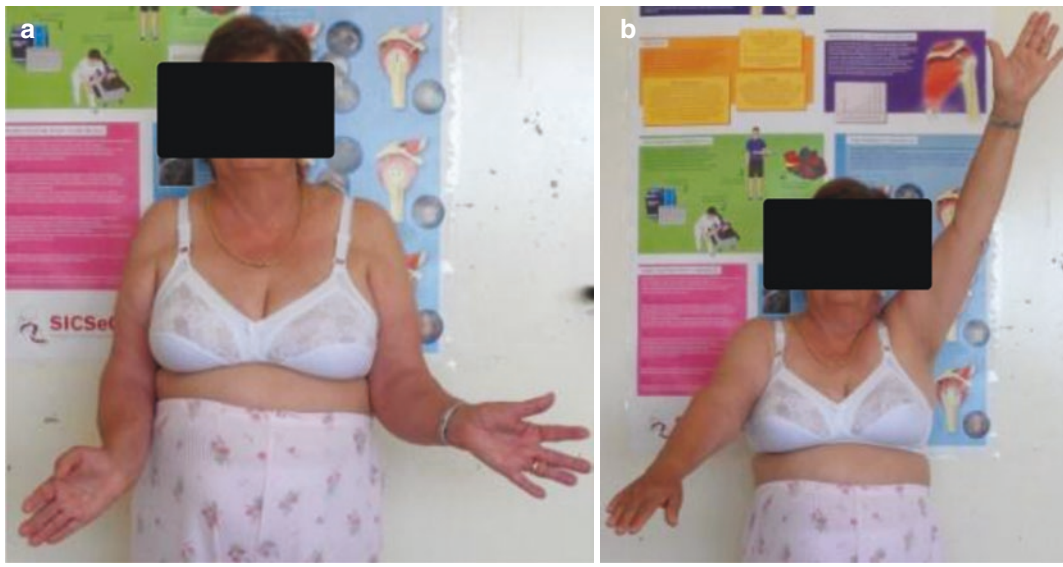

Fig. 7.6 (**a–d**) Active preoperative range of movement in woman patient with a cuff tear arthropathy: severe disabling disease

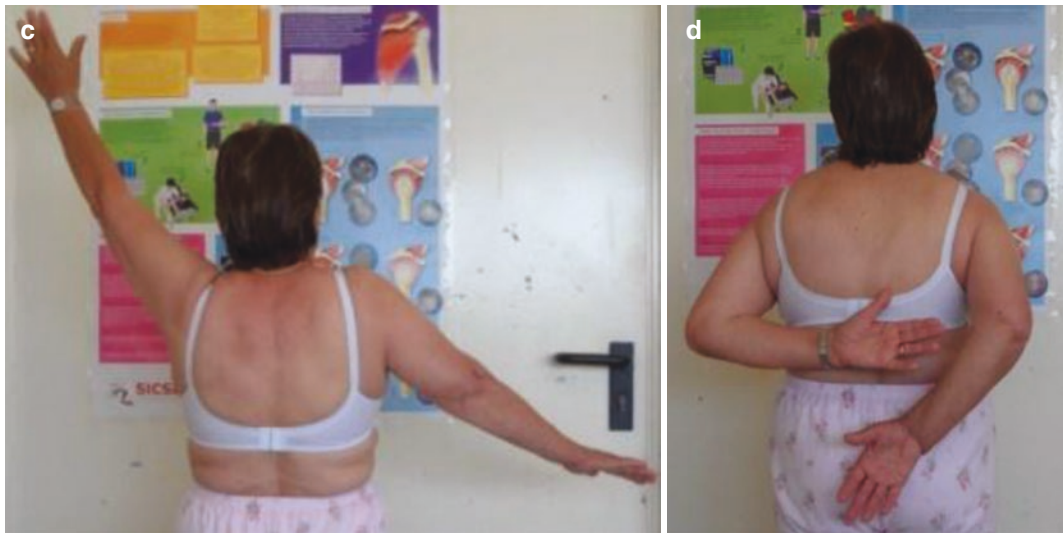

Fig. 7.6 (continued)

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