

DEGENERATION OF THE GLENO-HUMERAL JOINT

An Anatomical Study

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One hundred and fifty-one shoulder dissections were performed on 76 cadavera, 41 men and 35 women, with an average age of 68 (range 18–92). Before the age of 60 no degenerative changes were encountered but after 60, degeneration and full thickness ruptures of the rotator cuff, cartilage degeneration and degeneration and ruptures of the long biceps tendon appeared in an increasing frequency with age. A highly significant relationship between cuff degeneration and cartilage degeneration was found. Measurement of the thickness of normal joint cartilage of the caput humeri did not show any changes with time. Gleno-humeral degeneration was encountered bilaterally in 82 per cent and was more frequent in women, and there is little evidence that occupation is of major importance for the development of shoulder joint degeneration.

Key words: cartilage degeneration; osteoarthritis; rotator cuff degeneration; shoulder rotator

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Various factors have been suggested to be important for the development of degenerative gleno-humeral joint disease. Heine (1926), De Palma (1950) and Jurmain (1980) reported increasing frequency of gleno-humeral osteoarthritis with age. Kellgren & Lawrence (1952) and Lawrence (1955) found that the prevalence of osteoarthritis in men was to some extent influenced by occupation. Neer (1961) reported degenerative changes and avascular necrosis of the humeral head as late complications following trauma and certain abnormalities such as bone infarction in sickle cell anaemia and Caisson's disease.

The purpose of the present investigation was to estimate the prevalence of gleno-humeral joint degeneration in different age-groups and to

evaluate the relationship between cartilage degeneration of the gleno-humeral joint and degeneration of the rotator cuff.

MATERIAL AND METHODS

One-hundred and fifty-one shoulder dissections were performed on 76 cadavera, 41 men and 35 women, at the Departments of Pathology and Forensic Medicine at the Malmö General Hospital. The average age of the deceased was 68, ranging from 18 to 92 (Figure 1). The most frequent causes of death were malignant and cardio-vascular diseases. Six cadavera were dissected in the Department of Forensic Medicine. In this group of younger subjects, suicide and sudden cardiovascular death were the predominant causes of death.

Information about shoulder distress in life-time was extracted from the case stories. Since most of the departed were old and had been retired for years the information about their former occupations frequently was incomplete. One woman, age 78, had a recent fracture of the surgical neck of the right shoulder and in

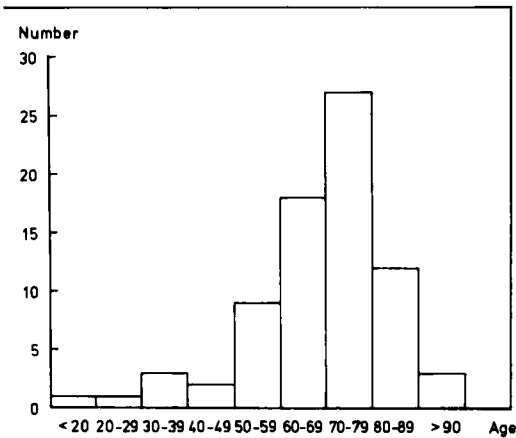


Figure 1. Age distribution.

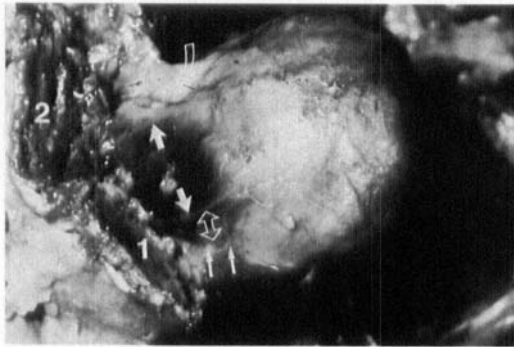


Figure 2a. The normal rotator cuff of the right shoulder of a man aged 70 after the acromion (1) and the clavicle (2) have been excised. The supraspinatus tendon (thick, white arrows), the infraspinatus tendon (open arrows) and the teres minor tendon (double headed arrows) are exposed. The open, curved arrow indicates the rotator interval.

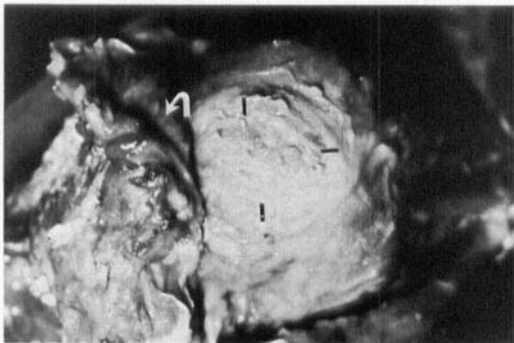


Figure 2b. The right shoulder of a man aged 72. After the subacromial bursa has been reflected over the acromion (white arrow), an area (20 × 25 mm) with severe penetrating degeneration in the supraspinatus tendon is exposed (black rods).

this case only the left was dissected, in the remainder of the cadavera both shoulders were scrutinized.

After excision of the acromion the rotator cuff was inspected *in situ* (Figure 2a) and types, size and localisation of the degenerative changes were recorded (Figure 2b). Areas with thinning, fraying or fibrous degradation of the cuff were classified as degenerated as were of course full thickness ruptures which were also recorded separately.

The gleno-humeral joint was then opened with an incision transversing the muscle-tendon junction of the rotator cuff and paralleling the borders of the glenoid cavity. After the cuff and capsule had been reflected the inner aspect of the joint could be scrutinized. Areas of internal non-penetrating cuff degeneration were now recorded. The condition and position of the long biceps tendon were registered and, after dislocation of the joint, degenerative changes of the articular cartilage of the caput humeri and the glenoid cavity recorded. Surface changes of the cartilage with thinning and fraying were registered as superficial degeneration. Severe degeneration was characterized by deep fraying of the cartilage and, in the most advanced cases, areas of full thickness cartilage deterioration denuding the subchondral bone.

In a separate series, 19 heads of the humerus without macroscopic degenerative changes and from normally appearing joints of 11 cadavera, six women and five men, with an average age of 68 (range 43–86) were excised. The heads were bisected in the frontal plane and the thickness of the articular cartilage measured with a ruler at three sites (Figure 3), site a) being the mid-point of the caput joint surface, site c) 5 mm from the lower cartilage border and site b) on the mid-point between a) and c).

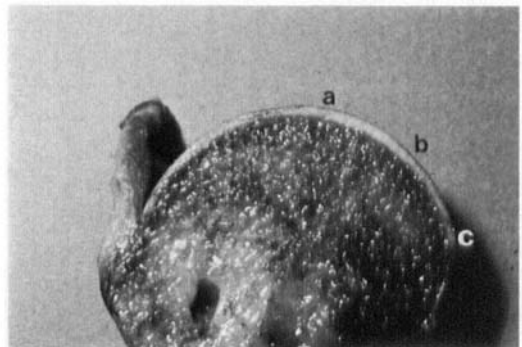


Figure 3. The bisected right caput humeri of a woman aged 78. The distal part of a normal supraspinatus tendon and its insertion is seen at the left. The sites of measuring the cartilage thickness are indicated. The measurements in this caput was: site a) 2 mm, site b) 2 mm, and site c) 1.5 mm.

RESULTS

Macroscopic degeneration of the rotator cuff was registered in 25 shoulders and full thickness cuff ruptures in 23 shoulders. The degeneration or rupture was bilateral in 42 shoulders (87 per cent). In 12 of the 25 shoulders with cuff degeneration the changes were localized to the inner

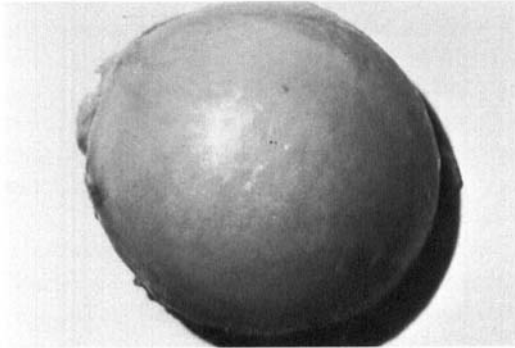


Figure 4a. The right caput humeri of a woman aged 80. Normal cartilage.

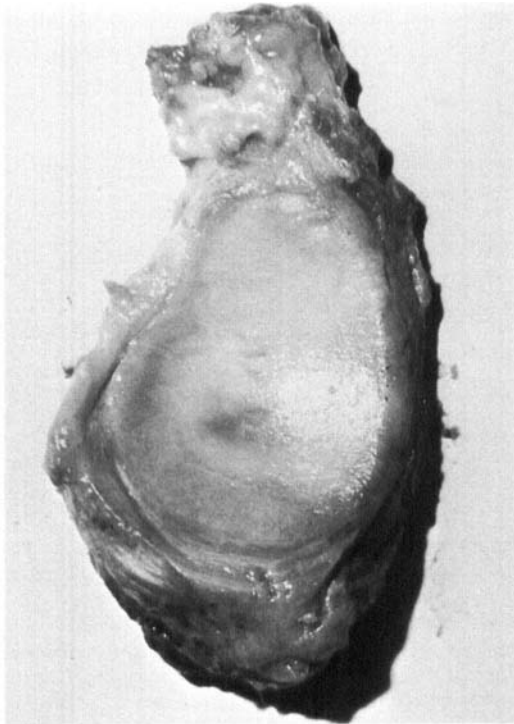


Figure 4b. The glenoid cavity of the same shoulder. Normal cartilage.

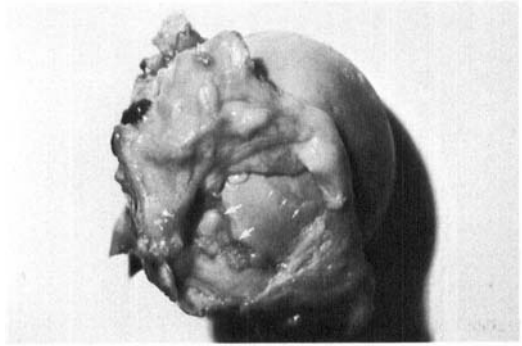


Figure 5. The right caput humeri and the rotator cuff of a man aged 84. A full thickness rupture is seen in the supraspinatus tendon, measuring 20 × 30 mm. An area of superficial cartilage degeneration is seen in the right superior quadrant (smaller arrows). Osteophytes and deep cartilage degeneration are exposed near the tuberculum majus (larger arrows).



Figure 6a. The left shoulder of a woman of 67 with an isolated rupture of 5 × 15 mm in the infraspinatus tendon, close to its superior border. The thick arrows indicate the edge of the acromion.

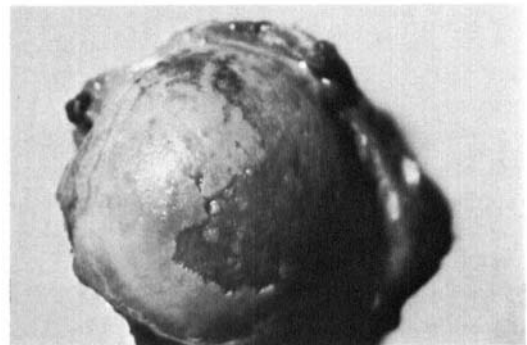


Figure 6b. The caput humeri of the same shoulder. Severe cartilage degeneration and a large, irregularly formed area without cartilage.

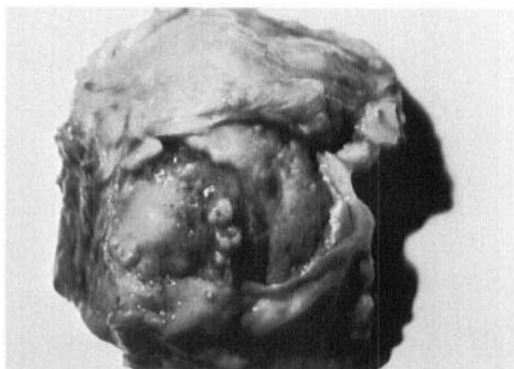


Figure 7a. The right caput humeri and the rotator cuff of a woman aged 70 with a full thickness rupture of the subscapularis tendon, measuring 20 × 30 mm. Large osteophytes at the tuberculum minus.

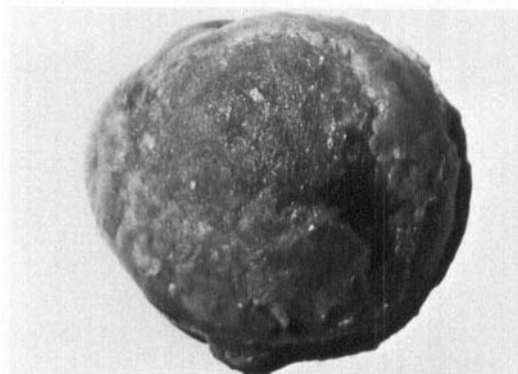


Figure 7b. The caput humeri of the same shoulder. Severe cartilage degeneration with fraying and a large central area without cartilage.

surface of the supraspinatus tendon, close to the tendon insertion. Most of the full thickness ruptures were in the supraspinatus tendon (Figure 5) but one shoulder had an isolated rupture, sized 5 × 15 mm, of the infraspinatus tendon (Figure 6a and b) and three shoulders had full thickness ruptures of the subscapularis tendon (Figure 7a and b).

The joint cartilage of the caput humeri and the glenoid cavity was macroscopically normal in 117 shoulders (77 per cent) (Figure 4a and b). The cartilage degeneration was bilateral in 28 out of 34 shoulders (82 per cent). The degeneration was recorded as superficial in 10 shoulders (Figure 5)

and severe in 24, 12 of which had full thickness degeneration denuding the subchondral bone in irregularly shaped areas (Figures 6b and 7b).

The degenerative changes were often more pronounced in the glenoid cavity. Osteophytes were found most frequently in the vicinity of the tubercles and the sulcus and sometimes along the borders of the glenoid cavity (Figures 5 and 7a).

One woman, age 90, had a total cuff rupture in her right shoulder; there was also severe cartilage destruction with total absence of cartilage in the glenoid cavity and about 50 per cent absence of cartilage of the caput humeri. The caput had migrated superiorly and reshaped the inferior surface of the acromion forming a shallow joint cavity-like structure not dissimilar in appearance from the glenoid cavity (Figure 8).

The long biceps tendon had degenerative changes with fraying and flattening in 12 shoulders and had ruptured in 6 shoulders. The tendons had dislocated forward in 4 joints. Degeneration or rupture of the long biceps tendon was, with one exception, always associated with degenerative disease of the rotator cuff. One man, age 74, had been operated on in his left shoulder 27 years ago because of recurrent dislocation. His

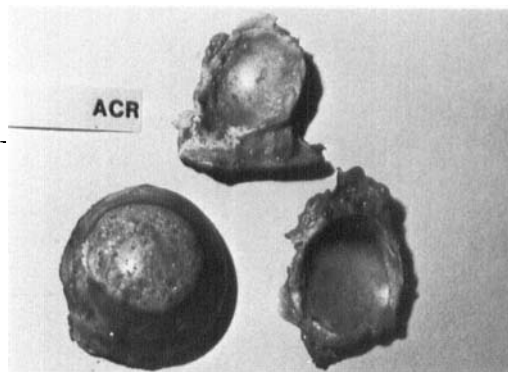


Figure 8. The caput humeri, the glenoid cavity (to the right) and the inferior aspect of the acromion (at the top) of a woman aged 91, who had a full thickness cuff rupture including the supra and infraspinatus tendons and most of the teres minor and the subscapularis tendons. There is a total absence of cartilage in the glenoid cavity and in the central area of the caput, where the subchondral bone is denuded and shiny. The caput had migrated superiorly and created a neoarthrosis at the inferior surface of the acromion.

Table 1. Distribution of degenerative gleno-humeral joint lesions in the different age groups

Age groups	<60	60–69	70–79	80–89	90	Men	Women	Total
No. of shoulders in the age group	32	36	53	24	6			
Cartilage degeneration	0	4 (11%)	16 (30%)	10 (41%)	4 (60%)	14	20	34
Cuff degeneration	0	6 (17%)	9 (17%)	8 (33%)	2 (33%)	16	9	25
Full thickness cuff rupture	0	4 (11%)	11 (21%)	4 (17%)	4 (60%)	11	12	23
Degeneration of the long biceps tendon	0	0	6 (11%)	1 (4%)	5 (83%)	4	8	12
Rupture of the long biceps tendon	0	2 (6%)	4 (8%)	0	0	1	5	6

rotator cuff was normal but there was superficial cartilage degeneration of the caput humeri and the long biceps tendon was severely degenerated.

The prevalence of degenerative lesions in relation to age is demonstrated in Table 1. The coincidence of degeneration of the rotator cuff and degeneration of the joint cartilage is presented in Table 2. Degenerative cuff disease, including

Table 2. Coincidence of degenerative changes in the rotator cuff and the gleno-humeral joint cartilage in 151 shoulders

	Cartilage	
	No degeneration	Degeneration
Cuff		
No degeneration	95	8
Degeneration	4	7
Rupture	4	19

ruptures, was present in 26 out of 34 shoulders with cartilage degeneration and in 22 out of 117 shoulders with normal cartilage. This difference is highly significant ($P < 0.001$, chi-square test). Forty-three of the 76 deceased had macroscopically normal shoulders.

The measurements of the caput humeri cartilage thickness at three different points are demonstrated in Table 3 for three age groups. There were no significant changes in cartilage thickness with age. In three cases only, it was known that the deceased had suffered from shoulder distress in his life-time, in one of these no changes were found, in two there was degeneration of the joint cartilage and the rotator cuff.

DISCUSSION

Age is an important contributory factor in degenerative joint disease and relevant also for the shoulder structures according to several authors

Table 3. Cartilage thickness (mm) of the caput humeri at three measuring sites in three age groups ($\bar{A} \pm S.D.$) (mm)

		Measuring site		
		a)	b)	c)
Age group				
<65	n=8	1.94±0.18	1.88±0.23	1.19±0.37
65–75	n=5	2.0 ±0	2.0 ±0	1.2 ±0.27
>75	n=6	2.0 ±0.31	1.83±0.25	1.0 ±0
Total	n=19	1.98±0.25	1.90±0.24	1.13±0.32

who have performed dissection studies (Heine 1926, De Palma 1950, Olsson 1953, Jurmain 1980). The statement is valid also for the present investigation of 151 shoulder joints. All 32 shoulders from individuals younger than 60 were free from macroscopic degeneration of the rotator cuff, joint cartilages or long biceps tendon, whereas after the age of 60 degenerative changes of these structures became increasingly frequent with advancing age.

The data of the present study also suggest, however, that the joint cartilages of an otherwise normal gleno-humeral joint do not deteriorate with time. No difference in the thickness of the articular cartilage of caput humeri was found between the age groups. This is in accordance with the results of Petersson & Redlund-Johnell (1983), who measured the joint space of normal gleno-humeral joint radiographs and found no change with age except in women, in whom the joint space was even slightly increased.

It has been a matter of debate whether strenuous work is of importance for the development of degenerative gleno-humeral disease (Kellgren & Lawrence 1952, Lawrence 1955). In the present study most of the deceased were old and had retired from work several years before departure, and it was not possible, in most instances, to analyse their former occupation. The results of the study suggest, however, that occupation is of minor importance since there was a higher frequency of both full thickness cuff ruptures and of cartilage degeneration in women. Another striking feature of the present investigation indicating a limited importance of occupation, strenuous work and handedness on the pathogenesis of gleno-humeral degeneration was the finding of cartilage degeneration being bilateral in 82 per cent and rotator cuff degeneration, including full thickness ruptures, being bilateral in 87 per cent. Seventy-six per cent of the shoulders with cartilage degeneration were also afflicted with rotator cuff degeneration or full thickness rupture. Whether the rotator cuff degeneration and the cartilage degeneration are two expressions of a common degenerative gleno-humeral joint disease, a degenerative syndrome, or one lesion causes the other is not possible to conclude from the present study. However, in all age groups

with degenerative lesions, the prevalence of rotator cuff degeneration, including ruptures, exceeding that of cartilage degeneration, may indicate that cuff degeneration precedes cartilage degeneration as well as degeneration of the long biceps tendon. The patho-mechanism might be two-fold. Firstly, penetrating cuff degeneration or full thickness ruptures might cause leakage of synovia from the gleno-humeral joint, resulting in subnutrition of the articular cartilages. Secondly, full thickness ruptures cause mechanical imbalance with a tendency of superior migration of the caput humeri, resulting in a displaced articulation and increased wear of the cartilage. An obvious drawback of dissection studies such as this is the lack of information about symptoms in life-time. Olsson (1953) dissected 106 shoulders from persons whom he had been able to examine clinically before death when they were admitted to a hospital for chronic illness. He found no correlation between recorded signs of degeneration and shoulder pain at the time of death or in the history. De Palma (1950) in a similar study found that degenerative lesions of the shoulder joint, however severe, need not be associated with loss of function or pain. In the present investigation only three out of 76 deceased had a known history of shoulder distress before death and two of those had degenerative changes in their shoulders. As for the rest of the deceased with degenerative changes in their shoulders, one can only assume, on basis of the reports mentioned earlier, that many, in spite of severe combined degeneration of the rotator cuff and articular cartilages, had suffered only slight discomfort from their shoulders during their last years of life.

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