

CASE REPORT

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Co-existence of os acromiale with suprascapular osseous bridge: a case report and review of the literature

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We report on a very rare case of co-existence of os acromiale with suprascapular osseous bridge in a dry scapula. The frequency of os acromiale alone ranges from 1.3 to 15%, while the frequency of suprascapular osseous bridge varies between 0.036% and 12.5%. We review the relative literature and emphasize the fact that such knowledge is important for a physician in order to avoid misdiagnosis of an acromion fracture and lytic lesion of the scapula. (Folia Morphol 2009; 68, 2: 109–112)

Key words: os acromiale, suprascapular osseous bridge, scapula, co-existence

INTRODUCTION

Os acromiale (OA) refers to the unfused epiphysis of the acromion along with separate ossicles or a fused elongated acromion, which can occur unilaterally or bilaterally. For some authors it is a rare anatomical abnormality [4, 13] and for others a common one [14]. Usually there are three separate ossification centres for the acromion process: meta-acromion, meso-acromion, and pre-acromion. The osseous union of centres occurs between 22 and 25 years. Consequently, diagnosis should be made after the age of 25. OA classification is determined by an ossification centre that failed to fuse [8, 20].

The junction between OA and acromion can be a discrete synovial joint, a synchondrosis, or a syndesmosis. The frequency of OA ranges from 1.3% [13] to 15% [14] in anatomical and radiographic studies. This anatomical variation can be either asymptomatic or painful by impingement mechanism. Symptomatic cases can be relieved by conservative therapy or by a wide variety of surgical techniques including acromioplasty, bone grafting, acromion osteosynthesis, resection of OA, and others. Suprascapular osseous bridge (SOB) is the outcome of superior transverse scapular ligament (STSL) ossification. STSL has no specific clinical function in humans and is the only piece where a bony bridge can appear [7]. If the ligament is ossified then the area underneath the ligament is limited and this may result in suprascapular nerve (SN) entrapment. Other reasons for SN entrapment include direct trauma, fracture of the scapula, ganglion cysts, lipomas, tumours, occupational overuse, and traction injuries. This is due to narrow morphology and the sharp edges of the suprascapular notch [16].

The aim of our study is to present a very rare case of co-existence of OA and SOB, which to our best of knowledge has not been reported in the literature.

CASE REPORT

In the macerated right scapula of a male cadaver of Caucasian race aged 68 years selected from the osteological collection of the Anatomy Department, OA was noticed along with SOB. The mea-

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Figure 1. A right dry scapula with co-existence of os acromiale (small arrow) and suprascapular osseous bridge (large arrow).

surements were made with the assistance of a digital sliding calliper with a sensitivity of 0.001 mm.

The case represents a fusion of OA and acromion with partial synostosis and partial synchondrosis. We considered the distal portion of the scapular spine as meta-acromion with a length of 12.27 mm and width of 29.41 mm. The maximum length of the acromion along with the unfused OA was 25.93 mm, maximum width was 28.49 mm, and maximum height was 18.44 mm. The mean proportional length was 0.68 mm. The proportional length was compared with the total length of the acromion.

The SOB had a length of 8.89 mm and width of 5.51 mm. The formed foramen was ovoid in shape with a maximum vertical diameter of 9.81 mm (Fig. 1). From case history, the cadaver did not appear to have any pathological conditions such as fracture, tumour, or shoulder pathology during his life.

The research was approved by the Ethical Committee of the Aristotle University of Thessaloniki.

DISCUSSION

The first anatomical report regarding OA was made in 1833 [6]; the first detailed description was made by Grüber in 1863 [9]. In that first detailed approach to this anatomical variation, in 3 out of 100 cadavers OA was identified with a distinct synovial joint. OA ossification centres can be recorded in patients as young as 18 years and they usually unite from posterior to anterior [17]. This leads to a failure of pre-acromial and meso-acromial centres to unite with the spine and the meta-acromial centre [13, 14].

Three types of OA are registered: type A, represents a failure of fusion between meso-acromion and metaacromion. In type B, there is failure of fusion between pre-acromion and meta-acromion, and type C

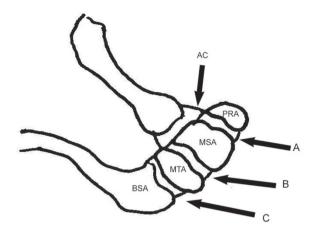


Figure 2. The three types of os acromiale are demonstrated. A, B, and C are indicating the spaces between PRA-MSA, MSA-MTA, and MTA-BSA, correspondingly; PRA — pre-acromion, MSA — meso-acromion, MTA — meta-acromion, BSA — basi-acromion, AC — acromicolavicular joint.

represents a failure of fusion between meta-acromion and basi-acromion (Fig. 2). Their frequency, according to Warner et al. [23], is 73.33%, 6.66%, and 20% for types A, B, and C, respectively, while Park et al. [17] recorded values as follows, 80%, 10%, and 10% for types A, B, and C, respectively. In very rare cases meta-acromion, meso-acromion, and preacromion are completely separated in a variation named as double OA. Furthermore, in another rare variation, three interruptions between ossification centres can be recognized, this refers to OA basale. Other variations in delayed ossification of metaacromion, meso-acromion, and pre-acromion are OA posterior and accessorium [8].

The incidence of OA varies greatly between various literature from 1% to 15% [4, 13], but is widely accepted as a common condition. Some researchers claim that the greater the distance of the acromioclavicular joint from the anterior edge of the acromion, the higher the eventuality of an OA [10].

Studies reveal that OA is less common in women than in men, and less common in white than black race [21]. Another study suggests that there is a genetic correlation for the appearance of OA. In addition, heavy manual labour during childhood can lead to the same condition [2]. OA more often appears unilaterally than bilaterally. Other studies suggest bilateral involvement in half of the cases [21]. Liberson [13] refers a 62% bilateral incidence in his study.

The classic approach for radiological diagnosis of OA requires lateral and anteroposterial axillary views, a contralateral view comparison can also help. Diagnostic imaging criteria includes osteophytic lipping at the acromial gap margins in 90% of cases [18], or in 66.67% of cases according to Edelson et al. [7]. Axial CT is helpful for diagnosis [22], but according to Granieri et al. [8] computed tomography and magnetic resonance imaging have not significantly increased the diagnostic information. OA is usually an incidental asymptomatic finding. Pain can be derived from impingement syndrome, rotator cuff tears, or from motion of unfused acromion [23]. If symptoms emerge, then this may be the result of a small unstable fraction pull from the deltoid muscle towards the rotator cuff, such that the supraspinatus outlet decreases in size or by abnormal motion at the non-union site [1]. Another pain release mechanism involves the anterior part of the deltoid muscle that may force OA to impinge towards the rotator cuff tendons during contraction [23]. Patients complain about limited range of motion at the shoulder joint and a sensation of clicking during movement. Furthermore, pain can emerge during overhead activity such as in various sports or occupations. Night pain is also common [20].

Differential diagnosis includes other painful shoulder syndromes, acromion and clavicle fracture. In addition, physicians and radiologists must be aware of this in order to distinguish fused OA from fibrocartilaginous union [7]. Fused OA can sometimes be difficult to identify on radiographs from unfused OA [21]. OA always has rounded-regular edges at the interacromial joint, whereas a bony fracture usually has sharp edges [8].

There is no need for treatment in asymptomatic os acromiale cases. First approach management includes conservative treatment with anti-inflammatory drugs and manual therapy as treatment of choice [20]. A variety of surgical techniques have been proposed for symptomatic cases with or without rotator cuff tears, including excision of fragments smaller than 4 cm, fusion with external fixation of large fragments, bone grafting, and anterior arthroscopic acromioplasty.

Anatomical studies support the fact that SOB incidence was as low as 0.036% [11]. Other studies claim incidence ranges from 3.7 to 12.5% [3, 7, 19]. Suprascapular notch in humans varies from complete absence to a small round opening that can be bridged. Partial bridges of bone can be found in 8.1% of cases [7]. In some rare cases, the ossification takes place in an accessory band of STSL. These bands employ a significant role in bony bridge

direction, which can be transverse, within the suprascapular notch or below the suprascapular notch. The type of U-shaped suprascapular notch, which shows partial ossification of the medial part of the STSL, is of particular interest [16]. Hrdlicka [11] supports the theory that bony bridge incidence increases with age; this may be related to enthesopathic changes. Insertion sites of STSL are subject to tensile and compressive forces due to its fibrocartilaginous origin. The same study suggests that, regardless of a weak muscle, omohyoid contributes to insertional angle changes and movements accentuated by the shortness of the ligament^{*}[15]. Bayramoglu et al. [3] reported that the inferior belly of the omohyoid muscle was attached to the medial part of STSL in all cases in his study.

The presence of SOB may predispose to SN entrapment syndrome [3]. It is possible to misdiagnose suprascapular foramen as a lytic lesion of the superior part of the scapula. Diagnosis for symptomatic SOB includes atrophy of the supraspinatus and infraspinatus muscles [12]. Shoulder discomfort and pain from SN entrapment is usually located to the posterior and lateral aspects of the shoulders and is characterized by its deep and diffuse nature [3, 5]. Another mechanism that can cause SN entrapment syndrome and pain concerns shoulder abduction, which can lead to traction of SN and squeezing [23]. Treatment can be conservative or surgical, including resection of SOB in order to decompress SN. The latter seems to relieve palsy from nerve compression [5].

The significance of our case yields that for the first time in the literature an OA is associated with a bony bridge of the suprascapular notch in a dry scapula. Such knowledge is important for physicians in order to avoid misdiagnosis of an acromion fracture and lytic lesion of the scapula. Moreover, patients with such a combination of anatomical variants may have undiagnosed symptoms of shoulder subacromial impingement and suprascapular neuropathy.

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Written consent was obtained from the cadaver's next of kin for the publication of this article.

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