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SHOULDER

Coracoid impingement: current concepts

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Abstract For many years, coracoid impingement has been a well-recognized cause of anterior shoulder pain. However, a precise diagnosis of coracoid impingement remains difficult in some cases due to the presence of multifactorial pathologies and a paucity of supporting evidence in the literature. This review provides an update on the current anatomical and biomechanical knowledge regarding this pathology, describes the diagnostic process, and discusses the possible treatment options, based on a systematic review of the literature.
Level of evidence V.

Keywords Coracoid impingement · Subcoracoid impingement · Coracohumeral distance · Coracohumeral interval

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Introduction

In 1909, Goldthwait [20] was the first to describe a possible rotator cuff impingement by the coracoid process. During the subsequent century, several anatomical, clinical and biomechanical studies addressed the topic, increasing our understanding of its aetiology. Today, CI is considered a known yet uncommon cause of anterior shoulder pain, resulting from impingement of the subscapularis or biceps tendon between the coracoid process and the lesser tuberosity [8, 11, 17, 39]. More recent studies have reported on a relationship between subcoracoid stenosis, or a narrowed coracohumeral distance (CHD), and anterior shoulder pathologies, and the authors assert subcoracoid stenosis to be relatively common, yet often unrecognized and underreported [31, 35, 42]. Suenaga [45] identified subcoracoid impingement syndromes in 11 of 216 (5.1 %) cases after rotator cuff surgery due to ongoing pain and tenderness over the coracoid process. In 1999, Dumontier noted that 2.8 % or 500 patients with shoulder impingement syndrome had impingement of the rotator interval by the coracoid process [8], while Lo and Burkhart [28] found that 19 % of patients with tears of the supraspinatus, infraspinatus and subscapularis had combined subacromial and coracoid impingement.

In the following sections, predisposing anatomical conditions, current biomechanical data and the current clinical management of CI will be detailed. The goal of this review of the literature was to analyse the existing data on CI and to present the current knowledge about this controversial topic. It was hypothesized there would be only limited evidence in the literature about the diagnosis, treatment outcomes and correlation of coracoid impingement to other shoulder pathologies. Therefore, we performed a systematic literature search in order not to miss important articles.

Materials and methods

Literature review

We performed an electronic search of the United States National Library of Medicine (PubMed) and Embase using the search terms coracoid impingement, subcoracoid impingement, subcoracoid stenosis and coracohumeral distance/space/interval. The most recent search was performed in November 2011. Three independent reviewers (FM, DR, ADW) screened the titles and abstracts for relevance and subsequently read the full text articles to determine inclusion. Since the literature was found to be limited, all original articles, review papers and case reports addressing the topic were included in this study.

Results of literature review

The database searches identified 180 studies, of which 52 were potentially relevant after screening the title and abstract. The 52 articles, related to coracoid impingement included 3 anatomical studies, 8 biomechanical studies, 24 clinical studies, 7 literature reviews and 10 case reports. All reviewers identified the same articles independently. Since there are no prospective randomized trials or comparative studies among these articles, the published data were not found to be strong enough for a high level-of-evidence systematic review and, therefore, a current concepts review was performed.

Anatomy

As long ago as 1909, Goldthwait [20] recognized remarkable differences in the shape and size of individual coracoid processes and believed these variations could explain a possible subcoracoid impingement syndrome. Anetzberger and Putz [1] supported these findings in 1995, after measurement of 343 human scapulae by means of an image analysis system. In 1999, Gumina et al. [21] performed anatomical morphometric studies of the coracoid process and the coracoid–glenoid space in 204 dry scapulae. They described three different types of scapula configurations with Type 1, occurring in 45 % of scapulae, having low values for the coraco-glenoid angle and coracoid overlap, which are known to be associated with a short coracohumeral distance [21]. They theorized Type 1 scapulae, with concomitant severe narrowing of the coraco-glenoid space, would be a predisposing factor for coracohumeral impingement.

Biomechanics

Eight biomechanical studies are available related to coracoid impingement. In 1994, Burns and Whipple [6] examined shoulder impingement against the anterior tip of the acromion, the coracohumeral ligament or the coracoid process in a cadaver study. They reported on a possible biceps tendon impingement against the coracoacromial ligament (CAL) in elevation and external rotation, but also against the coracoid process in elevation and internal rotation. In 2004, Radas and Pieper [41] published results of an examination of 124 cadaver shoulders and showed that the coracohumeral distance significantly decreased with increasing internal rotation. This is not surprising, as the lesser tuberosity diverges from the coracoid process with external rotation and approaches with internal rotation. Since contact between the two bones seemed to be made at a very early stage (25°–60° of internal rotation), the authors assumed a general disposition towards coracoid impingement in normal populations. The decreasing coracohumeral distance during internal rotation was confirmed in a cadaver study on 30 shoulders by Ferreira Neto et al. [10]. These authors also reported a significantly smaller coracohumeral distance among the female sex. In a recently published study, Hughes et al. [22] used pressure transducers placed at the coracoid process, the CAL and the acromion in 9 cadaveric shoulders. The highest pressures were observed in abduction/internal rotation at the coracoid process, in flexion/internal rotation at the CAL (both involving the rotator interval) and in abduction/internal rotation at the CAL (involving the supraspinatus tendon). Visual observation revealed a compression of the infraspinatus and the supraspinatus tendon against posterior/anterior acromion in extension/external rotation and compression of the subscapularis tendon against the coracoid process in flexion/internal and external rotation. These studies demonstrate the possible presence of an anterior impingement syndrome with soft tissue compression against the coracoid process, showing increasing contact pressures for certain positions of the arm. In 2010, Yamamoto et al. [52] also measured contact pressures beneath the coracoacromial arch and bending pressures of the CAL in seven normal cadaveric shoulders during flexion, extension, abduction, horizontal abduction, internal and external rotation. They found contact pressures with the CAL and acromion significantly increased as the arm was moved to greater than 90° flexion, 80° abduction and 50° horizontal abduction and almost constant for internal and external rotation. They also showed bending deformation of the CAL being greater during flexion, extension and horizontal abduction than during internal and external rotation. To summarize, several studies have shown a relationship between specific arm positions and contact pressures under the CAL and the coracoid process [22, 48, 50–52]. Therefore, it seems that both structures might contribute to CI.

Aetiology

The causes for CI can be classified as idiopathic, iatrogenic or traumatic. Idiopathic causes include anatomical variations of the coracoid process [17], calcification within or ossification of the subscapularis tendon [3, 12, 40] and ganglion cysts [26, 46]. The iatrogenic causes, described by Gerber et al. [17], include surgical procedures like coracoid transfer, posterior glenoid neck osteotomy or acromionectomy. CI might also occur after humeral head or neck fractures, fractures of the coracoid process, the glenoid or the scapula neck and posterior sternoclavicular dislocations [17, 53]. A narrowing of the coracohumeral space due to an idiopathic or posttraumatic anterior instability is also conceivable and reported in the literature [38, 41].

Diagnosis

A diagnosis of CI can be challenging. Therefore, it is important for clinicians to be familiar with the diagnostic process involving a detailed medical history of the patient, a thorough clinical examination and appropriate imaging.

Medical history and clinical examination

A thorough history should include previous shoulder pathologies, the duration of discomfort and also the point in time and the activity when it occurred first. Furthermore, the physician should also ask about possible arm positions, motions or actions that provoke the pain. As CI has been reported to be commonly the result of chronic overuse and repetitive micro-trauma [44], the patient's profession and sports activities should also be considered. Since Gerber et al. [17] redefined CI in 1985, the symptoms have been mainly described as dull anterior shoulder pain brought about or aggravated by forward flexion and internal rotation. Abduction and internal rotation may also be described to be painful. The clinical examination includes a complete evaluation of both shoulders. Particular attention should be paid to positive rotator cuff or biceps tendon tests and signs for instability, as these pathologies are reported to be associated with a narrowed coracohumeral distance [16, 23, 36, 38, 42]. Furthermore, obvious tenderness around the coracoid process or the lesser tuberosity can be established through palpation. The coracoid impingement test is performed by passive internal rotation with the arm in cross-body adduction and forward elevation [7]. A lidocaine injection in the subcoracoid region may also be of utility in establishing a diagnosis [11, 13, 17].

Imaging

In patients with coracoid impingement, standard radiographs may reveal anatomical variations, such as a far laterally projecting coracoid process in the anteroposterior view or a chevron-shaped coracoid process in the supraspinatus outlet view [27, 37]. However, MRI or CT examinations appear to be more precise in establishing the diagnosis [32] as demonstrated by several studies in the literature addressing coracoid impingement and the corresponding MRI and CT findings. In most cases, axial sequences are used to measure the CHD, defined as shortest distance between the humeral head and the coracoid process [2, 4, 14, 17–19, 32, 34].

Gerber et al. [18] studied 47 healthy shoulders by computed tomography (CT) in adduction and an additional 20 in forward flexion and internal rotation. The CHD was 8.7 mm for the adducted arm and 6.8 mm for the arm in flexion and internal rotation. Coracoid impingement appeared particularly likely during forward flexion and of a shoulder with a coracoid tip close to the scapular neck or projecting far laterally. Bonutti et al. [4] and Friedman et al. [14] described an abnormal CHD to be less than 11 mm on MRIs in patient with shoulder pain. Giaroli et al. [19] were the only group who found that a CHD of 10.5–11.5 mm was significantly related to surgically confirmed subcoracoid impingement but was poorly predictive and, therefore, concluded that diagnosis of subcoracoid impingement could not be made on MRI findings alone.

Other MRI studies have suggested a correlation between coracoid impingement, a narrowed coracohumeral distance, and rotator cuff or biceps pathologies. Nove-Josserand et al. [35] reported a CHD of 9 mm in patients with supraspinatus tear and in patients with isolated subscapularis tears. However, patients with large rotator cuff tears involving the subscapularis and infraspinatus averaged a coracohumeral distance of 7.7 mm with 30 % having a value of less than 6 mm. Similar results were shown for patients with fatty degeneration of the subscapularis and infraspinatus [36]. Richards et al. [42] also reported on the narrowed CHD in patients with tears of the subscapularis. They found an average coracohumeral distance of 10 mm in patients without rotator cuff pathology and a decreased distance of 5 mm in patients with subscapularis tears.

Ultrasonography of the coracohumeral interval can also be used for diagnosing coracoid impingement. Tracy et al. [47] found the CHD to be narrowed in patients with clinically diagnosed coracoid impingement ($n = 8$) compared to healthy volunteers ($n = 19$).

The main radiological criterion for assessment of possible CI in all these studies was measurement of the CHD. In addition to the coracohumeral distance, the coracoid index, defined as the lateral projection of the coracoid

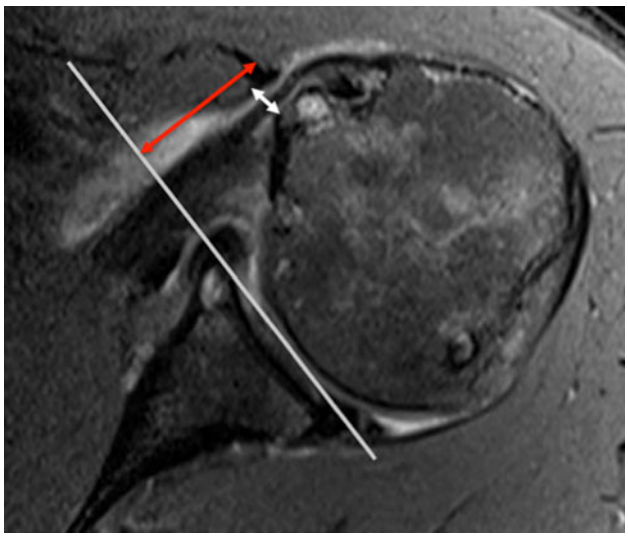

Fig. 1 MRI of left shoulder of patient with coracoid impingement. The narrowed CHD is marked with a *white arrow*; the *red arrow* indicates the coracoid index. A concomitant lesion of the subscapularis tendon and a cyst within the lesser tuberosity can also be detected

beyond the glenoid joint line in axial CT or MR images, is theorized to have an influence on developing CI. Dines et al. [7] described the mean value in healthy shoulders to be 8.2 mm. Besides, cysts within the lesser tuberosity are reported to correlate with a narrowed CHD and supraspinatus/subscapularis abnormalities [49]. Figure 1 shows the technique for measurement of the CHD and the coracoid index in a patient with coracoid impingement.

Treatment

Once diagnosis of coracoid impingement is established, the first line of treatment should be conservative, including activity modifications with avoidance of provocative positions, rotator cuff and scapula-stabilizing musculature strengthening [16, 17, 43, 44].

Surgical treatment

If non-operative measures fail, surgical treatment might be necessary in cases of persistent complaints. Operative management should be considered and discussed with the patient, because both open and arthroscopic coracoplasty have been demonstrated to provide good and reliable clinical results in terms of pain relief and functional improvement [8, 15, 17, 24, 28, 31, 45].

Indications

Indications for surgical intervention include ongoing pain due to secondary causes of coracoid impingement, including calcifications or ossification of the subscapularis tendon [3, 40], ganglion cysts [26, 46], prior surgeries such as coracoid transfer, posterior glenoid neck osteotomy or acromionectomy [17], and previous fractures of the humeral head or neck, fractures of the coracoid process, the glenoid or the scapula neck and posterior sternoclavicular dislocation [17, 53]. In addition, primary intractable anterior shoulder pain in patients with clinically and radiologically suspected CI may be an indication for surgical treatment [8, 17, 31].

Surgical techniques

Several arthroscopic and open surgical techniques have been described in the literature in order to perform a decompression of the coracohumeral space. Dines et al. [7] described an open technique, using a standard deltopectoral approach, with dissection of the conjoined tendon from the coracoid process, resection (10–15 mm) of the coracoid tip and re-attachment of the conjoined tendon to the remaining base of the coracoid process. Another option to perform open coracoplasty is to divide the conjoined tendon for 2 cm and continuing the incision for 2 cm into the coracohumeral ligament. After removal of the lateral portion of the coracoid process, the conjoined tendon is repaired side to side [27, 37]. Also, the coracobrachialis attachment can be elevated close to its origin to allow visualization and osteotomy of the posterior, lateral and inferior portions of the coracoid [8, 45]. Gerber et al. [17] suggest that isolated coracoid impingement is rare and advocate additional resection of the coracoacromial ligament and acromioplasty.

The arthroscopic technique can be performed via a transarticular or extraarticular approach from the subacromial space [16, 24, 28]. In cases of persistent pain and suspected but unclear diagnosis of CI, diagnostic arthroscopy can provide clarification. It enables the surgeon to directly examine the subcoracoid space, looking for signs of impingement such as inflammation of the surrounding capsular tissue, tendinitis or even tearing at the insertion of the subscapularis tendon. Furthermore, the coracohumeral distance can be visualized and measured directly in different arm positions, and bone or soft tissue impingement can be evaluated. Moreover, concomitant intra-articular pathologies that might be responsible for ongoing shoulder pain and could be missed during open surgery can be detected and addressed.

In 2007, Kleist et al. [25] investigated the effectiveness and safety of arthroscopic coracoplasty. They performed an arthroscopic coracoplasty in 5 cadaveric specimens, evaluating the coracoid index and CHD in pre- and postoperative CT scans. After gross dissection, they summarized that arthroscopic coracoplasty could effectively improve coracoid index and CHD and that neurovascular structures

were at a safe distance from the dissection site. The results of an anatomical study by Lo et al. [30] agree with these findings, showing the safety of arthroscopic coracoplasty when avoiding dissection medially to the coracoid process.

Perioperative considerations performing arthroscopic coracoplasty

The procedure can be performed both under general or interscalene block anaesthesia. Maintenance of a mean arterial pressure of 70–90 mm Hg or a systolic pressure near 100 mm Hg allows maximal visualization and minimizes bleeding. A thorough examination of both shoulders under anaesthesia is performed on every patient after induction of anaesthesia but before positioning. Range of motion and signs of instability are documented. This is important, as, for example, in case of anterior instability coracoplasty alone might not be sufficient [38].

Diagnostic arthroscopy and management of concomitant lesions

Diagnostic arthroscopy is performed through a standard posterior portal. The possible bone or soft tissue impingement between the coracoid process and the humeral head is examined in different arm positions, especially in abduction/flexion and internal rotation. Furthermore, signs of inflammation of the surrounding capsule tissue, tendinitis or lesions of the biceps tendon, the biceps pulley, the rotator interval or the rotator cuff are carefully searched for, since these are potentially related to CI [29, 31, 36, 42].

In addition, signs of anterior shoulder instability such as a capsulolabral lesion or an elongated capsule–ligament complex must be detected in order to choose the correct treatment. In the presence of anterior shoulder instability, stabilization should be performed first, as this might solve the anterior impingement pathology [15, 38]. Tears of the long head biceps tendon and/or its pulley system need to be addressed by biceps tenodesis or tenotomy in order to avoid the risk of persistent pain [9]. In case of a subscapularis tendon tear, accompanied by coracoid impingement, it is recommended performing a coracoplasty first. By doing so, one can avoid mechanical compression between the coracoid and subscapularis repair. Furthermore, one creates more space within the anterior shoulder compartment, which makes the surgery technically easier for the subsequent subscapularis repair [5].

Arthroscopic coracoplasty

For performing an arthroscopic coracoplasty, an anterolateral portal is established approximately 1.5 cm lateral to the anterolateral tip of the acromion. The key to proper portal placement is first verifying the intra-articular position and the expected work angles with a spinal needle.

In order to expose the coracoid, the joint capsule between the superior glenohumeral ligament (SGHL) and the middle glenohumeral ligament (MGHL) is opened with a shaver or radiofrequency device, preserving the medial sling of the biceps sheath and the MGHL and SGHL (Fig. 2). The coracoacromial ligament serves as landmark, safely leading to the lateral coracoid process. Further landmarks are the conjoined tendon inferiorly and the base of the coracoid medially. By dissecting on the lateral side of the coracoid, the neurovascular structures remain safe. When reaching the lateral aspect of the coracoid, the radiofrequency device can be used to remove the soft tissue from the tip and posterior aspect of the coracoid. Next, a 4-mm burr is used to remove approximately 5 mm of the posterolateral tip of the coracoid process (Fig. 3). Subsequent dynamic examination should confirm sufficient resection. The goal should be to medialize and anteriorize the coracoid, while preserving the major tendinous and ligamentous attachments and preventing an iatrogenic fracture (Fig. 4).

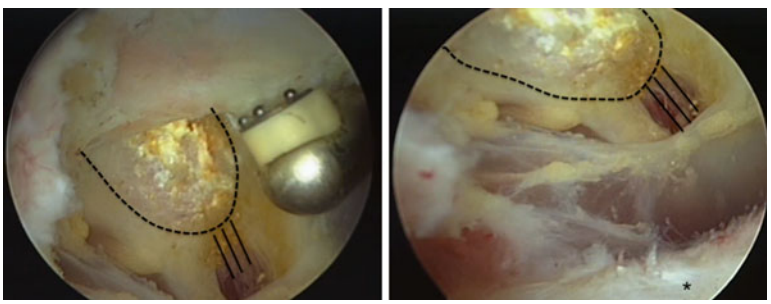

Fig. 2 Right shoulder with patient in modified beach chair position, viewing from posterior (Same applies to Figs. 3, 4). *Left side* preparation of coracoid process (*dotted black line*) with electrocautery through the open joint capsule. *Right side* nice exposition of the coracoid process, awaiting coracoplasty. The partially torn subscapularis tendon is marked with (*asterisk*) and the conjoined tendon can also be seen (*black lines* indicate its course)

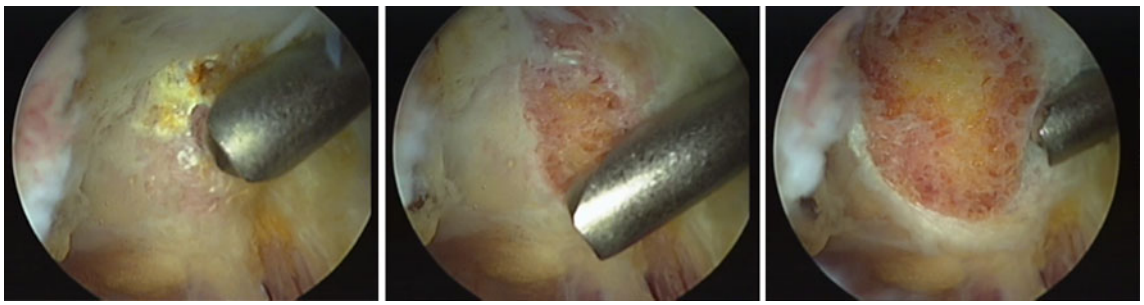

Fig. 3 Successive coracoplasty, performed from lateral to medial through the anterolateral portal, using a 4-mm burr to resect the posterior aspect of the coracoid tip (*Left*, *middle*). Finally, the lateral edge of the coracoid tip is resected, in order to medialize the coracoid process (*Right*)

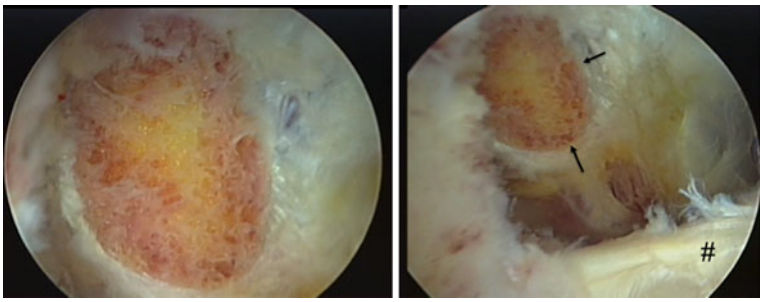

Fig. 4 Completed coracoplasty. The *black arrows* show the direction of resection in order to anteriorize and medialize the coracoid process and create more space for the subscapularis tendon (*hash*)

Rehabilitation and outcomes

The postoperative rehabilitation depends on the additional procedures performed. After isolated coracoplasty, active range of motion is allowed as early as possible. We recommend avoiding impingement positions for the first 2 weeks after surgery. Regarding the outcome after surgical treatment of coracoid impingement, the literature indicates good and reliable results in terms of pain relief and improvement of shoulder function for open and arthroscopic coracoplasty [8, 15, 17, 24, 28, 31, 45].

Discussion

The most important finding of the present study was that our knowledge about coracoid impingement, especially regarding clinical aspects such as diagnosis, clinical examination, imaging, treatment options and expectable outcomes, is not well supported by rigorous scientific studies at this time.

Regarding *anatomy* and possible predispositions for coracoid impingement, we know the scapula and the coraco-glenoid space demonstrate different configurations which influence the coracohumeral distance [21]. In addition, the coracohumeral distance decreases with increasing internal rotation of the humeral head [10, 41]. However, these cadaveric studies can only suggest a possible influence of scapula configuration and rotation angles of the humeral head on coracoid impingement. Two recent studies demonstrated increased pressures at the coracoid process and/or the coracoacromial ligament for certain shoulder motions [22, 51, 52]. However, it is unclear whether the described contact pressures have to be assumed as pathologic, because measurement of the coracohumeral distance was lacking. Therefore, it remains unclear whether a normal population or specimens with narrowed coracohumeral space were observed. Studies comparing contact pressures in specimens with normal and narrowed CHD do not yet exist in the literature.

Gerber et al. [17] stated the *diagnosis* of coracoid impingement should be made clinically. Since then, the clinical examination for diagnosis of coracoid impingement has been well described in several articles [7, 8, 17, 45]. However, the clinical examination is subjective by nature, and to date, a scientific examination of the validity of clinical tests is missing in the literature. The quality of clinical diagnosis might also depend on the experience and skills of the examining physician. The same applies to the validity and accuracy of diagnostic injections [7, 17, 33].

Standard *radiographs* might reveal anatomical variations, such as a far laterally projecting coracoid process in anteroposterior view or a chevron-shaped coracoid process in the supraspinatus outlet view [27, 37]. However, a study showing a reliable correlation between radiographic findings and coracoid impingement is lacking in the literature.

The value of *ultrasonograhy* for diagnosis of coracoid impingement is reported in one single study in the literature, showing a narrowed CHD in patients with clinically diagnosed coracoid impingement ($n = 8$) [47]. However,

reliability and validity of this procedure need to be proven in further studies.

MRI examination was found to be only 5.3 % sensitive yet 97 % specific for coracoid impingement (EBM level III) and, therefore, diagnosis of coracoid impingement cannot be established on imaging studies alone [19]. However, there are several clinical MRI-based studies, showing correlations between a narrowed CHD and anterior shoulder pathologies (EBM levels II, III, IV) [31, 35, 36, 42]. Nevertheless, these kinds of studies can only describe existing correlations but cannot answer the question whether a narrowed CHD is cause or effect.

Diagnostic arthroscopy is reserved as the final option of the diagnostic process. However, a proper way of intraoperative measurement of the coracohumeral distance has not yet been described in the literature. Therefore, an intraoperative diagnosis may be made through examining the shoulder for secondary signs of CI.

Regarding *treatment and outcomes* of coracoid impingement, most authors recommend a conservative treatment as a first option followed by surgical intervention for intractable cases. However, no well-designed prospective study in the literature shows specific outcomes after conservative treatment of coracoid impingement and, therefore, there is no evidence in the literature for its success. The literature reports good and reliable results for open and arthroscopic treatment of coracoid impingement [8, 15, 17, 24, 28, 31, 45]. However, coracoid impingement is a rare finding, and available studies report only small case series [8, 15, 24, 28, 45] (EBM level IV) or patients treated with concurrent procedures in addition to the coracoplasty [15, 17, 31, 45]. Furthermore, possible concomitant injuries missed during open surgery or addressed arthroscopically might have crucial influence on the postoperative outcome. A prospective randomized study, comparing the outcomes after open versus arthroscopic coracoplasty, does not exist in the literature.

The main limitation of this study is that the scientific data about CI are weak, and therefore, despite the systematic nature of the review, the level of evidence of the study itself has to be assigned as level V. Much of the information on the topic is based on clinical experience and expert opinion. Hopefully, this will encourage researchers to perform further studies on this controversial topic. The paper does nevertheless provide an update on the latest knowledge about CI and hopefully it can help shoulder surgeons who face clinical decisions about CI in daily practice.

Conclusion

The current systematic review supports the hypothesis that our evidence-based knowledge about coracoid impingement is limited. Valid clinical studies are difficult to perform due to its infrequent isolated occurrence and coexisting pathologies. However, further biomechanical, radiological and clinical investigations will be necessary to answer the remaining questions about the validity of our diagnostic process, the treatment options and associated outcomes. Nevertheless, while there is evidence in the literature that a narrowed coracohumeral distance is closely related to anterior shoulder pathologies, we need to elucidate whether this is cause or effect.

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