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Axillar Compression Syndrome: Anatomical and Clinical Study

Mate Majerović, Željko Jelinčić and Goran Augustin

Department of Surgery, Division of Abdominal Surgery, University Hospital Center, Zagreb, Croatia

A B S T R A C T

In order to evaluate the possibility of compression of axillar artery by medial and lateral fascicle of brachial plexus, authors performed 26 axillar dissections on cadavers. Second part included analysis of 24 selective angiograms of axillar artery of patients with diagnosis of TOS. Third part included the use of modified hyperabduction test for determination of vascular bruit as safe test for diagnosis of axillar compression. Macroscopic changes of axillar artery by compression of medial and lateral fascicle of brachial plexus were present in 11.5%. Specific angiographic horizontal spike-shaped stop of contrast behind the surgical neck of humerus was present in 12.5%. Use of modified hyperabduction test revealed vascular bruit 29.5%. Specific relation of axillar artery and medial and lateral fascicle of brachial plexus revealed another possible etiologic factor in hyperabduction syndrome as a part of TOS. Use of modified hyperabduction test revealed subclinical phase of possible syndrome.

***Key words**: Thoracic outlet syndrome (TOS), axillar compression syndrome, arterial compression, brachial plexus, hyperabduction test, scalenotomy*

Introduction

Thoracic outlet syndrome (TOS) is the preferred term for those syndromes that result from compression of the neurovascular structures to the upper extremities. The syndrome is caused by compression of the brachial plexus or sublavian-axillary artery and/or vein in the region between the thoracic outlet and the insertion of the pectoralis minor muscle onto the coracoid process. Many syndromes are included in the term thoracic outlet syndrome, such as the cervical rib syndrome[1], scalenus anterior syndrome, hyperabduction syndrome, costoclavicular syndrome and the first thoracic rib syndrome. Syndromes that cause similar clinical manifestations include scapulocostal syndrome, suprascapular nerve syndrome and syndrome of lateral axillar hiatus[2,3].

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Clinical evaluation is started with provocative tests of arterial compression[4].

Symptoms may arise from neural, vascular, or combined neural and vascular compression, with neural compression causing 90% to 95% of these symptoms[5].

The reason for investigating further cause of TOS was guided by the results of operative therapy of TOS. Using all possible operative modalities and techniques, there was significant percentage of patients with persisting symptoms ranging from 8% to 66%[6,7,8]. There are two possible answers: one is that current operative procedures are not perfected yet, and another that unknown causes of TOS are present that have not been defined yet.

Accidentaly, we found interesting relationship between axillar artery and plexus brachialis in the region where medial and lateral fascicle cross over the axillar artery to form median nerve. Lord et al. in 1971. described the possibility of compression of axillar artery by median nerve[9], but we did not found any objective signs, tests, diagnostic and therapeutic modalities for this type of compression in the medical literature.

Materials and Methods

Research was divided into three parts. First part included anatomical dissection of axillar regions of 26 cadavers randomly chosen, with preservation of all structures in axilla. All cadavers were fresh, dissected in the Department of Pathology KBC Zagreb 3–6 hours after death determination (legaly, cadaver must be in the department where death occured for 2 hours and then it is transported to department of pathology). The dissected cadavers did not have history of vascular compression and other vascular diseases in their medical files. Axillar dissections were unilateral (13 left and 13 right). We focused on relationship between axillar artery and plexus brachialis in the region where medial and lateral fascicle cross over the axillar artery. We analysed neurovascular relationships in functional positions of upper extremity. These positions included: full adduction, 90° abduction and maximal elevation of 180°.

Second part included clinical analysis of patients with diagnosis of TOS and analysis of 24 selective angiograms of axillar artery in patients with suspected TOS. Between 1995 and 1999 we examined 109 patients with symptoms of TOS. Thirty one of them were subsequently operated. All 24 selective angiograms were indicated in subsequently operated patients. Nineteen of these angiograms were taken with upper extremity in maximal elevation of 180°. All operated patients were clinically evaluated postoperatively (Table 1).

Third part included the possibility of existence of physical signs of compression of axillar artery by tensed medial and lateral fascicle that cross axillar artery in maximal elevation. Physical examination was performed on 122 pupils in Nursing School in Zagreb and included extended history taking, inspection, palpation and auscultation of axillar region. Examina-

TABLE 1
RESULTS AFTER SURGICAL PROCEDURES IN 31 OPERATED PATIENTS

Procedure	Excellent	Good	Poor
Scalenotomy	2	5	7
Resection of the first rib with scalenotomy	1	3	5
Transaxillar resection of the first rib	4	3	1

tion was completed with the use of provocative test. Provocative test was hyperabduction test introduced by Wright in 1945[10] that was modified for detecting eventual compression site between axillar artery and its crossing by medial and lateral fascicle.

Modified hyperabduction test is performed with patient sitting straight with head looking straight forward and upper extremity fully adducted. In this position auscultation is performed in supraclavicular and infraclavicular region. Auscultation is repeated with patient taking deep breath. Now patient performes 90° abduction and auscultations in the same positions are repeated. With this manoeuvre more proximal compression is excluded if exists. Next step is patients maximal elevation of the upper extremity in supination. In this position, as in previous, simultaneous palpation of radial artery with auscultation of anterior and upper border of axilla is performed. The same examination can be performed through whole process of hand elevation to its maximal elevation. Decrease of palpabile radial pulse or its absence and detection of localization and intensity of vascular bruit, is reliable sign of the height (localization) and the degree of compression of axillary artery (Fig. 1).

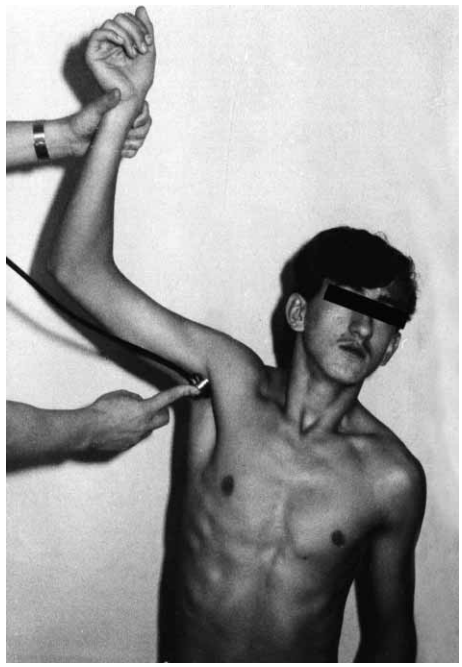

Fig. 1. Modified hyperabduction test for detection of localization of vascular bruit in axillar compression syndrome.

Results

Dissection of axillar regions of 26 cadavers, randomly chosen, with special attention on region of crossing of axillar artery by medial and lateral fascicle revealed interesting biomechanical relations. Nervous fibers of brachial plexus glide over the axillar artery in the range of full adduction to 90° abduction of upper extremity, and finally, in the maximal elevation, axillar artery is compressed between these nervous fibers. Anatomical relations were similar, only difference was level of connection of medial and lateral fascicle into median nerve, which differed in 1–2 cm.

In 3 of 26 cases (11.5%) of dissected cadaveric axillar arteries, we found advanced atherosclerotic-stenotic changes on crossing of medial and lateral fascicle over axillar artery. Another interesting moment is that in these three cases with atherosclerotic-stenotic changes of axillar artery, signs of generalized atherosclerosis were not present. In all three cases trophical changes of digits were present (Fig. 2), and in two of this three cases we found poststenotic dilatation of axillar artery (Fig. 3).

Second part of investigation included clinical analysis of 24 selective arteriograms of axillar artery. Stop of contrast was evident in 9 cases (37.5%) in maximal elevation of upper extremity. Anot-

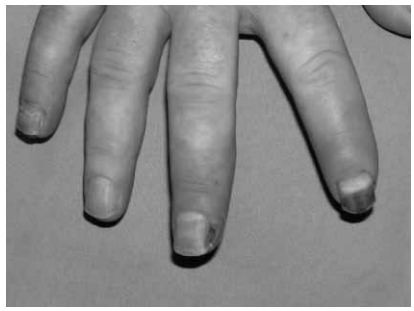

Fig. 2. Trophical changes of digits observed in three cases with atherosclerotic-stenotic changes of axillar artery, without signs of generalized atherosclerosis.

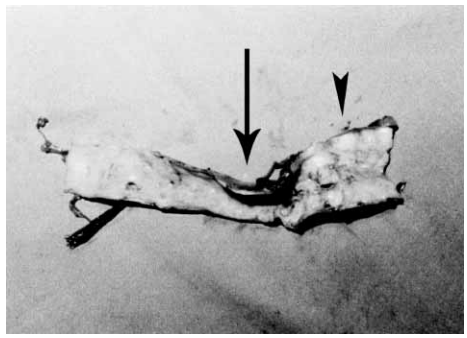

Fig. 3. Poststenotic dilatation of axillar artery. Arrow points to the place where medial and lateral fascicle unite and cross over axillar artery. Poststenotic dilatation evident on the right side of resected segment (arrowhead).

TABLE 2
LEVEL OF STOP OF CONTRAST ON 24 SELECTIVE AXILLAR ARTERIOGRAMS

Level	Number of patients	%
Did not occured	15	62.5
Scalenus triangle	2	8.3
Costoclavicular triangle	5*	20.8
Humeroscapular space	1	4.2
Surgical neck of humerus	3*	12.5

*There was compression on two levels on same angiogram in two patients and their results are in both costoclavicular triangle and behind surgical neck of humerus

TABLE 3
VASCULAR BRUIT IN 122 HEALTHY FEMALE VOLUNTEERS IN ANTERIOR AND UPPER BORDER OF AXILLA DURING MODIFIED HYPERABDUCTION TEST

Axilla	Positive test	%
Left	17	13.9
Right	19	15.6
Total	36	29.5

her crucial element is that stop of contrast is observed on different levels of subclavian-axillar arterial segment (Table 2). In three angiograms which showed stop of contrast behind surgical neck of humerus (12.5%), we found horizontal spike-shaped stop of contrast. Position and shape correspond to anatomical site of crossing of medial and lateral fascicle over axillar artery. On additional selective arteriograms in 90° abduction of upper extremity stop of contrast was not present. All three arteriograms of spike-shaped stop of contrast around surgical neck of humerus are seen on figure 4 (Fig. 4).

Third part. Use of modified hyperabduction test in 122 healthy female volunteers showed results and distribution summarized in table 3. Positive sign was vascular bruit in anterior and upper border of axilla where anatomical site of crossing of medial and lateral fascicle over axillar artery is located. The sign was positive in 36 of 122 cases (29.5%) (Table 3).

Discussion

Anatomical structures in axilla can compress either artery, vein, or nerves in some pathologic conditions because of interstructural incongruence[11–13].

The subject of this study are anatomical relations between structures of axilla, namely nerves and axillar artery as a cause of development of compression syn-

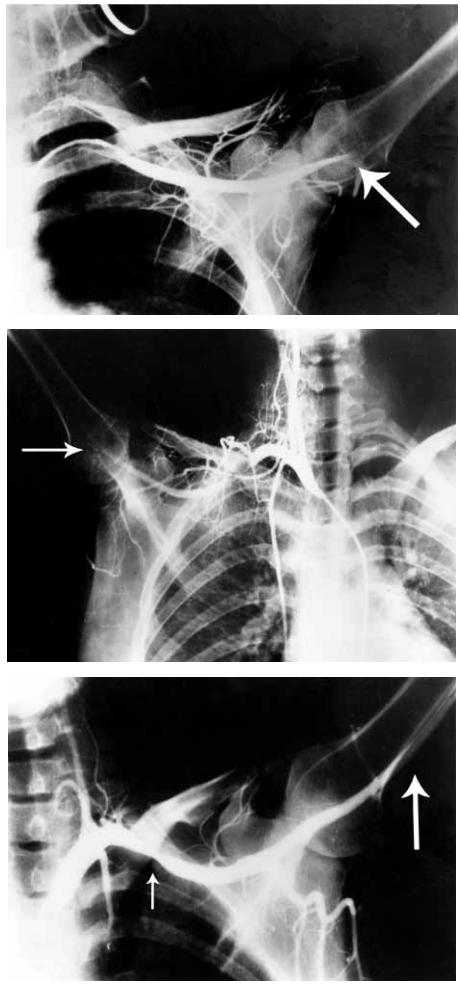

Fig. 4. Spike-shaped stop of contrast (arrows) around surgical neck in anatomical position where medial and lateral fascicle unite in 'V' shaped form to form median nerve when arm is in maximal elevation (note narrowing of contrast in costoclaviclar space in figure 3C pointed with smaller arrow).

drome. In normal state, when arm is in full adduction, medial and lateral fascicle surround axillar artery and on the anterior surface of the artery they connect in the shape of letter "V" to form median nerve. Such anatomical relations of axillar artery and neural bundle predispose to arterial compression in specific positions of upper extremity.

When the upper extremity is in maximal elevation, neurovascular bundle becomes tensed over the first rib, muscles of that region and ligamentous structures. Geometricaly, axillar artery travels longer linear distance in cranial direction than nervous fibers because of two reasons: first, nervous fibers are positioned above the axillar artery, and second, origin of nervous fibers of plexus brachialis (C5-Th1) is localized above the origin of subclavian artery. "V" formation of lateral and medial fascicle above the axillar artery serve as fixation point of axillar artery thus further decreasing its mobility (Fig. 5).

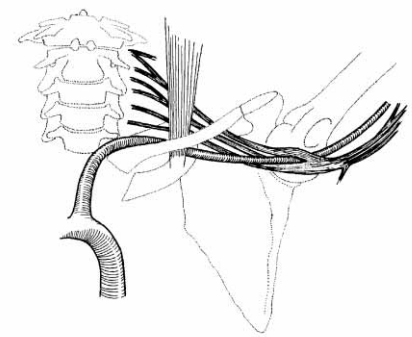

Fig. 5. Anatomical relations between axillar artery and medial and lateral fascicle of brachial plexus (see text for details).

In the process of arm abduction nervous fibers of brachial plexus glide over the artery, and finally, in the maximal elevation artery is compressed between these nervous fibers, because, simply geometricaly, axillar artery travels longer linear distance in cranial direction than nervous fibers. "V" formation of medial and lateral and fascicle serve as fixation point. Another element which contribute

to this phenomenon is that arteries are softer and more elastic that nerves. Probable pathophysiological processes on the site of compression are the result of chronic irritation and include reparative processes: fibrous changes of arterial wall and activation of sympathetic nervous system, with resulting atherosclerotic-stenotic changes on crossing of medial and lateral fascicle over axillar artery. Final changes include hemodynamic disturbances with development of poststenotic dilatation of axillar artery, mural thrombi and consecutive microembolisation of digital arteries with subsequent trophical changes of digits. It was not possible to find out if these cadavers had any clinical symptoms of upper extremity, but trophical changes of digits of involved upper extremity made that asumption almost certain.

Searching through literature there are studies that define some structures and conditions as a cause of compression. Wright[10] described compression of axillar artery under the insertion of minor pectoral muscle to coracoid process, and Lord[9] described mutual compression of neural bundle and axillar artery but without specific localization and objective signs of this entity.

In 36 of 122 healthy female volunteers, presence of vascular bruit in anterior and upper part of axilla indicate that there is large number of potential candidates for the development of this syndrome which may be termed fascicular compression syndrome of axillar artery. Gergoudis and Barnes[14] concluded that significant arterial obstruction resulted from one or more of provocative tests in healthy persons in 60% unilaterally and 33% bilaterally. Rare symptomatology of this, relativley frequent condition in "healthy" population with predominant neurologic simptoms is explained with the hypothesis that potential candidates subconsciously avoid movements that cause neurologic symptoms with painful sensations[15].

Modified hyperabduction test for determination of mutual compression of axillar artery and medial and lateral fascicle point that there is significant percentage of subclinical types of this syndrome (simptomless volunteers), which, with further progression of the disease, may become manifest, or remain asymptomatic because of subconscious avoidance of provocative positions with subsequent ischaemia.

Conclusion

After anatomical dissections of cadaveric axilla, it was obvious that clinical manifestations and radiologic findings were not the result of anatomic variations, but specific functional relations of axillar artery and medial and lateral fascicle. Positive modified hyperabduction test used for determination of mutual compression of axillar artery and brachial plexus, confirms that these components, both vascular and neural are compressed, either mutualy, or by rigid structures of the roof of axilla.

Vascular bruit in the axilla in maximal elevation of upper extremity with point of maximal loudness in the anterior and upper part of axilla is evident sign of axillar artery compression by neural bundles of brachial plexus.

Now, with possible new etiologic factor in TOS there is much more to be done. If angiographic diagnosis is evident there is a question of therapy of this fascicular compression syndrome of axillar artery. Probably neurolysis or transposition of axillar artery will give some answers to that question but it has to be evaluated in the future.

Abbrevations:

TOS – thoracic outlet syndrome

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M. Majerović

Department of Surgery, Division of Abdominal Surgery, University Hospital Center Zagreb, Kišpatićeva 12, 10000 Zagreb, Croatia
e-mail: mate.majerovic@zg.htnel.hr

AKSILARNI KOMPRESIVNI SINDROM – ANATOMSKA I KLINIČKA STUDIJA

SAŽETAK

Autori su proveli 26 sekcija na kadaverima da bi istražili mogućnost kompresije aksilarne arterije medijalnim i lateralnim snopom brahijalnog pleksusa. Drugi dio rada sastojao se od analize 24 selektivna angiograma aksilarne arterije s dijagnosticiranim sindromom gornje aperture toraksa. Treći dio rada uključivao je modificirani test hiperabdukcije s vaskularnim šumom kao siguran dijagnostički test aksilarne kompresije. Makroskopske promjene aksilarne arterije zbog kompresije bile su prisutne u 11,5%. Posebno oblikovani horizontalno ušiljeni obris kontrasta iza kirurškog vrata humerusa bio je prisutan u 12,5%. Korištenje hiperabdukcijskog testa pokazalo je šum u 29,5%. Veza između aksilarne arterije i brahijalnog pleksusa otkrila je još jedan mogući etiološki čimbenik u hiperabdukcijskom sindromu kao dio sindroma gornje aperture toraksa. Korištenje modificiranog hiperabdukcijskog testa ukazivalo je na subkliničku fazu mogućeg sindroma.