Scalenotomy for neurogenic thoracic outlet syndrome

Mohamed Hasan, Mustafa Alwalily, Mostafa Elsayed, Mohamed Elgebale, Abdelbaset Saleh, Adel Ragab, Hamdy Behary, Bokhary Mahmoud, and Hatem Elkhouly

Neurosurgery. Faculty of Medicine, Al- Azhar University, Egypt.
neuro_m52@yahoo.com

Abstract: Thoracic outlet syndrome (TOS) must be strongly evaluated in every case of upper limb complain as TOS is often the underlying cause of refractory upper limb conditions facing neurosurgeon or orthopedic surgeon like frozen shoulder, cervical disc (as a double or a multiple crush syndromes) or carpal tunnel syndrome that frequently defy standard treatment protocols and surgeon must select the safest surgical approach for surgically indicated cases. The aim of this study is to evaluate supraclavicular scalenotomy for true neurogenic thoracic outlet syndrome. **Patients and Methods**: twenty patients with neurogenic thoracic outlet syndrome were operated on between 2008 and 2011. Mean age was 38.05±8.85 years (range, 17 to 58 years); female/male ratio was 17/3. The most frequent symptom was paresthesia (75.0%). Seven patients (35%) had bilateral symptoms. All cases (20 cases) were true neurogenic thoracic outlet syndrome (excluding disputed cases). Lower plexus (C8-T1/ulnar nerve) compression was present in 18 patients and upper plexus (C5-C7/median nerve) compression in 2 patients. Preoperative evaluation was done for all patients and includes plain radiography, nerve conduction velocity and MRI cervical, and MRI angiography for selected cases (not all). The indication for surgery was failure of conservative treatment for 6 months. The decision to operate was made if symptoms persisted after conservative therapy in a patient with true neurogenic thoracic outlet syndrome. Cervical rib cases, radiographic occurrence of cervical rib alone is not an indication for surgery unless associated with symptoms. **Results:** there were clinical and electrophysiologic improvement of the preoperative complaint in all cases (Mean ulnar nerve conduction velocity was 58.0±6.07 m/s, range, 43 to 68 m/s) preoperatively and 66.55±5.63 m/s (range, 47 to 70 m/s) postoperatively ($p < 0.05$)) without any recorded complications nor recurrence during 1 year follow up period. **Conclusion:** Surgical decompression for thoracic outlet syndrome by only scalenotomy and release of associated bands(without interruption of osseous elements) is efficient and safe for true neurogenic thoracic outlet syndrome.

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1. Introduction

Thoracic outlet syndrome (TOS) is defined as "upper extremity symptoms due to neurovascular compression in the area of the neck above the first rib" **(1)**. The subclavian vessels and the brachial plexus traverse the cervicoaxillary canal to reach the upper extremity (2). The cervicoaxillary canal is divided by the first rib into two sections: the proximal one, composed of the scalene triangle and the costoclavicular space (the space bounded by the clavicle and the first rib), and the distal one, composed of the axilla. The proximal division is the more critical for neurovascular compression. It is bounded superiorly by the clavicle, inferiorly by the first rib, anteromedially by the costoclavicular ligament, and posterolaterally by the scalenus medius muscle and the long thoracic nerve. The scalenus anticus muscle, which inserts on the scalene tubercle of the first rib, divides the costoclavicular space into two compartments: the anteromedial compartment, which contains the subclavian vein, and the scalene triangle, which is bounded by the scalenus anticus anteriorly, the scalenus medius posteriorly, and the first rib inferiorly and contains the subclavian artery and brachial plexus (3). The anterior scalene muscle (ASM) derives from the anterior tubercles of the transverse processes of the C3-C6 vertebrae, and attaches to the first rib. Functionally, the ASM acts as an accessory muscle of respiration by raising the first rib and slightly bending and rotating the neck **(4).** So that thoracic outlet syndrome (TOS) has three anatomic compartments through which neurovascular structures must traverse to reach the upper extremity: the interscalene triangle, the costoclavicular, and the retropectoralis minor spaces **(5).**

TOS is characterized by symptoms related to compression of the brachial plexus and/or adjacent vascular structures. Compression may be due to a variety of anatomical abnormalities, including a narrow scalene hiatus, fibrous bands between scalene muscles, cervical ribs and a confined costoclavicular space. Also, compression by the coracoid process or the pectoralis minor muscle tendon is believed to sometimes cause these symptoms. There are three basic forms of TOS as TOS complaints can be of arterial (approximately 5%), venous (51%) or neurogenic (95%) origin [6]. A subclassification of NTOS (neurogenic thoracic outlet syndrome) includes “true” NTOS (also known as the “classic” form with objective findings), which accounts for only 1% of neurogenic cases, and “nonspecific” NTOS (also known as the “common” form, with chronic pain symptoms suggestive of brachial plexus compromise but without objective findings). The latter accounts for 99% of neurogenic cases [7]. Clinical tests and electrodiagnostic studies are helpful to evaluate patients with suspected NTOS. Anterior scalene block may serve as a reliable diagnostic test by temporarily blocking or paralyzing the muscle in spasm and reducing symptoms of TOS [8].

The management of TOS can be both non-operative and operative. Non-operative management includes modification of behavior by avoiding provocative activities and arm positions, in addition to individually tailored physical therapy programs that strengthen the muscles of the pectoral girdle and help to restore normal posture [9]. Surgical options include first rib resection through a transaxillary approach [10] and decompression with neurolysis of the involved regions of the brachial plexus, especially the C7, C8, and T1 nerve roots through a supraclavicular approach [11] which is called supraclavicular neuroplasty. The first rib with the compressive elements may also be removed using the supraclavicular approach which enables careful evaluation of the brachial plexus [12].

In this study, supraclavicular decompression of anterior scalene and associated bands for treatment of true neurogenic thoracic outlet syndrome has proved to be effective in long term relief of symptoms, and thus, it is a reasonable surgical treatment option.

2. Patients and methods

The present study included twenty patients with neurogenic thoracic outlet syndrome. They were selected from Al-Azhar University hospitals between 2008 and 2011.

Clinical evaluation included a detailed history and physical examination. At the initial consultation, the patient's symptoms were recorded and a complete clinical examination of the neck, shoulder, and upper extremity was performed.

The following procedures were used routinely for the diagnosis of thoracic outlet syndrome:

1. Clinical evaluation: inspection, palpation, auscultation in the supraclavicular fossa with the arm in the neutral position and up into the abducted and externally rotated position, and muscle strength test,

2. Provocative clinical tests: three classic maneuvers: Adson or Scalene Test. This maneuver tightens the anterior and middle scalene muscles Costoclavicular Test (military position),This maneuver narrows the costoclavicular space by approximating the clavicle to the first rib and thus tends to compress the neurovascular bundle and Hyperabduction Test the components of the neurovascular bundle are pulled around the pectoralis minor tendon, the coracoid process, and the head of the humerus.

3. Electrophysiologic tests: somatosensory evoked potential recording across the brachial plexus,

4. Anatomic studies: plain radiograph and magnetic resonance imaging, and

5. Provocative stimulation and anesthetic blocks of the anterior scalene muscle.

The indication for surgery in this study was only primary true neurogenic thoracic outlet syndrome with exclusion of disputed cases of neurogenic thoracic outlet syndrome and cases of TOS secondary to pancost tumor, subclavian aneurysms and others.

Surgical technique

After adequate general endotracheal anesthesia with the patient in the supine position, the supraclavicular and infraclavicular areas were prepared and draped in a standard fashion. A big roll was placed under the cervical and thoracic spine, and the head was turned in the opposite direction. An S-shaped incision (or linear) was made along the posterior border of the sternocleidomastoid muscle and along the proximal third of the clavicle. The brachial plexus was exposed in the usual fashion from lateral to medial using the subclavian vein, subclavian artery and the scalenus anterior muscle in between as a landmark with meticulous dissection of the phrenic nerve along the scalenus anterior muscle

Neurolysis of the brachial plexus was performed using the operative microscope. Vessel loops were placed around the nerve roots, and special care was taken to remove any bands or other soft tissue that might be compressing the roots or lower trunk. The extent of microneurolysis was based on the patient's symptoms, the preoperative assessment, and the intraoperative findings. Each root, trunk, and cord and division of the supraclavicular plexus were inspected under high magnification for signs of fibrosis, palpated for softness or hardness, and accordingly decompressed from the entrapping hypertrophic epineural sheath. The microneurolysis commenced with simple epineurotomy and progressed, as needed, to interfascicular scar excision. The goal was to restore “softness” in the plexus elements and to reach normal-looking neural tissue both proximally and distally.

Follow-up was obtained by standard visits with the clinician for all patients. The surgical results were gathered and analyzed in 6-month time increments, with an additional time point at 2 months after surgery.

3. Results

In the present study, females represent 85% of all studied cases and males represent 15%; age ranged from 17 to 58 years with a mean of 38.05±8.85 years. The most common presenting symptom was paresthesia reported in 75%, followed by lack of power in 55% and finally pain at rest in 50%; symptoms were unilateral in 65% and bilateral in 35%; plexus compression was in upper plexus in 10.0% and lower plexus in 90.0% (Table 1).

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Table (1): Demographic characteristics and clinical presentations of studied cases

	Values
Sex (M/F) (no, %)	5 (15%)/15 (85%)
Age (mean ± SD): range	38.05±8.85: 17- 58
Symptoms	
Paresthesia	15 (75%)
Lack of power	11 (55.0%)
Pain at rest	10 (50.0%)
Laterality	
Unilateral	13 (65.0%)
Bilateral	7 (35.0%)
Plexus compression	
Lower	18 (90.0%)
Upper	2 (10.0%)

Significant decrease of VAS postoperative compared to preoperative (paired samples t test)

Table (2): Operative indications and outcome in studied cases

	Values
Operative indications	
Persistence of symptoms after conservative treatment	16 (80.0%)
Decreased ulnar conduction (< 60m/s)	4 (20.0%)
Ulnar conduction (mean ± SD): range	
Preoperative	58.00±6.07: 43-62
Postoperative	66.55±5.63: 47-70*
Visual analogue scale (mean±SD): range	
Preoperative	5.70±2.15: 3-9
Postoperative	2.10±2.22: 0-7#
Success	
Upper plexus compression	14 (77.8%)
Lower plexus compression	1 (50.0%)
Total	15 (75.0%)

* Significant increase of ulnar conduction postoperative compared to preoperative (paired samples t test)

In the present work, operative indications were persistence of symptoms after conservative treatment in 80% of cases and decreased ulnar conduction < 60m/s in 20%. Ulnar conduction in preoperative period ranged from 43 to 62 with a mean of 58.0±6.07 while postoperative, it ranged from 47 to 70 with a mean of 66.55±5.63 m/s and there was significant increase of ulnar conduction in postoperative time when compared to preoperative value. In addition, visual analogue scale in preoperative time ranged from 3-9 with a mean of 5.70±2.15 while in postoperative time, it ranged from 0-7 with a mean of 2.10±2.22 and there was significant decrease in postoperative time when compared to preoperative values. The success rate was 75% in all studied cases; in upper plexus compression, the success rate was 77.8% and in lower plexus compression, it was 50% (Table 2)

4. Discussion

Thoracic outlet syndrome (TOS) refers to compression of the neurovascular structures at the superior aperture of the thorax [13]. So it is a misnomer as it is thoracic inlet not thoracic outlet.

The number of surgically indicated cases in this study was limited (20 cases) in comparison to other studies like **Urschel and Razzuk**[14] at 1998 (Thousands of cases), as they extend the study to include all clinical types of TOS over 50 years, while at this study the included cases were limited to true neurogenic thoracic outlet syndrome which represent 1% of neurogenic TOS, while disputed neurogenic thoracic outlet syndrome, which represent 99% of neurogenic TOS, and vascular TOS were excluded.

Regarding the gender and age:, females represent 85% of all studied cases and males represent 15%; age ranged from 17 to 58 years with a mean of 38.05±8.85 years. While, **Bhattacharya et al.** [15] reported that, females: males represent 62:8 with a mean age of 37 years.

Several reasons for the preponderance of female patients have been postulated, including relative underdevelopment of the muscles inserting around the shoulder girdle or relatively lower origin of the brachial plexus with frequent contributions from the second thoracic nerve root [21].

Regarding symptoms, the most common presenting symptom was paresthesia reported in 75%, followed by lack of power in 55% and finally pain at rest in 50%; symptoms were unilateral in 65% and bilateral in 35%; plexus compression was in upper plexus in 10.0% and lower plexus in 90.0%. These results are comparable to those reported by **Balci et al.** [16] who reported that, the most frequent symptom was paresthesia in the arms and hands (72.3%). Twenty-six (53%) patients complained that their symptoms were aggravated when lifting objects above the head or when placing the arms in an exaggerated hyperabduction position. C8-T1/ulnar nerve (lower plexus) compression was present in 36 patients, and C5-C7/median nerve (upper plexus) compression was present in 6. Therefore, the lower plexus in 85.7% and the upper plexus in 14.3% of patients were compressed in neurologic cases. In addition, **Bhattacharya et al.**[15] who reported that, 71% of the cases presented with neurological symptoms, females/male represent 62/8 and mean age was 37 years. Furthermore, it is in agreement with **Terzis and Kokkalis** [17] who reported that; mean age was 36.9±11.5 (range, 21–55 years), about two thirds of the patients presented with neurologic symptoms (weakness was found in 88% of the cases, and decrease of sensation was revealed in 68% of cases) and positive electrophysiological signs (65% of the cases).

Regarding the diagnosis of true TOS, it is complementary by triad of 1. Clinical examination which is the main stone including anterior scalene muscle block, 2. Nerve conduction velocity assessment, 3. Anatomical diagnostic studies like MRI cervical spine, MRI angiography and others.

Christo et al., [8] stated that: Anterior scalene block may serve as a reliable diagnostic test by temporarily blocking or paralyzing the muscle in spasm and reducing symptoms of TOS.

Scalene muscle blocks are especially useful to confirm a diagnosis of NTOS**,** with a good response being highly correlated with a good response to surgery [1, 18]**,** while **Harold and Harry** [19] stated that The primary objective test for thoracic outlet peripheral nerve compression is the nerve conduction velocity (NCV), but other deny that as unfortunately, the results of conventional EMG / NC studies are usually negative or nonspecific in patients with neurogenic TOS. This is probably due to two causes; the first is the extremely proximal location of brachial plexus nerve root compression, where it may be technically difficult to obtain accurate NC readings. The second is the nerve compression in neurogenic TOS is typically intermittent, only rarely causing the type of permanent changes in motor nerve function that are most easily detected by EMG / NC studies, for that the diagnosis of TOS is complementary by triad of clinical examination which is the main stone and scalene muscle block test., nerve conduction velocity assessment, and anatomical diagnostic studies like MRI cervical spine, MRI angiography and others.

We introduce a new diagnostic scale for diagnosis of TOS, in which the true neurogenic thoracic outlet syndrome can be postulated as 6 points, 3 points for clinical examination (1 for provocative tests and 2 for scalene block test), 1 point for nerve conduction velocity, and 2 points for anatomic studies. It is necessary to be at least 5 points to diagnose neurogenic TOS.

Regarding the cause of TOS; Controversies exist concerning its causes [12]**,** but the postulation of intermittent spastic scalene muscles with compression of brachial plexus and subclavian artery at the scalene hiatus is the primary cause of TOS. This postulation is supported by the following 1. Lack of subclavian vein compression symptoms in cases of TOS as it is outside the scalene hiatus. 2. Hypertrophy of the anterior scalene muscle was seen in 12.5% of the dissections as observed by **Marius et al** [20] 3. Lack of constant objective findings by nerve conduction velocity studies as the muscular compression is intermittent**.** This postulation is the substrate of this study for anterior scalenotomy alone without resection of the first rib. So that scalenotomy (of anterior plus or minus middle scalene) and anterior decompression of any offending compressing soft tissue bands without interrupting bony element (as in cases of carpal tunnel, the surgeon deal with the compressing soft tissue without interrupting carpal bones). Also supraclavicular approach is better for good visualization of Brachial plexus and phrenic nerve. For upper plexus (median nerve) compression, **Stoney *et al.*** [21] wrote that transaxillary rib resection alone was not enough and that it should be combined with the supraclavicular approach to achieve best results. So that the surgical outcome of all cases was satisfactory without any recorded surgical complications, while **Urschel and Razzuk** [22] reported that, of 1508 procedures on patients with symptoms of ulnar nerve compression (lower plexus), 76% achieved an excellent result, 20% a good result, 4% a fair result, and fewer than 1% a poor result

With first rib resection and scalenotomy in upper plexus TOS, 77% reported good or excellent results, 92% had long-term relief, and 2.2% had recurrent symptoms requiring reoperation [23].

In this study, there is no recorded surgical complications, while in transaxillary approach with rib resection [18] there was many recorded complications like (1) recurrence resulted from a rib remnant left by the initial surgeon that regenerated fibrocartilage and new bone, producing a high incidence of recurrence. (2) Bleeding requiring a second procedure occurred after only 4 of 7407 procedures. Significant infection requiring drainage occurred after nine procedures. (3) Venous bleeding after surgery that required thoracotomy and repair. Venous injuries usually “suck” air. (4) Significant nerve injuries of the brachial plexus with residual signs occurred in four patients due to prolonged stretching, inappropriate retraction, or direct surgical injury. **Dale's** review [23] of morbidity rates in 881 patients revealed significant bleeding in 11 (1.4%) and nerve injury of the brachial plexus in 13 (1.5%), the phrenic in 39 (4.9%), the long thoracic in 3 (0.1 %), and the recurrent laryngeal in 6 (0.2%). In another 168 patients reported [21], the phrenic nerve was injured in 6 (4%) and the long thoracic and recurrent laryngeal nerve in 1 (0.5%); Horner's syndrome occurred in 9 (6%). So that supraclavicular decompression of anterior scalene and associated bands for treatment of true neurogenic thoracic outlet syndrome has proved to be effective, easy, safe and reasonable surgical treatment option for true neurogenic thoracic outlet syndrome .

References

1. Sanders RJ, Hammond SL, Rao NM. Thoracic outlet syndrome: a review. Neurologist, 2008; 14: 365–373
2. Ravitch MM, Steichen FM. Atlas of General Thoracic Surgery. Philadelphia: WB Saunders; 1988.
3. Ranney D. Thoracic outlet: an anatomical redefinition that makes clinical sense. Clin Anat. 1996;9(1):50–52.
4. Atasoy E. Thoracic outlet syndrome: Anatomy. Hand Clin 2004; 20(1):7–14,.
5. Demondion X, Herbinet P, Van Sint Jan S, Boutry N, Chantelot C, Cotten A. Imaging assessment of thoracic outlet syndrome. Radiographics 2006; 26: 1735–1750.
6. Thompson RW, Bartoli MA. Neurogenic thoracic outlet syndrome. In: Rutherford vascular surgery, 6th ed. Elsevier Saunders. Place of Publication: Philadelphia, PA, USA, 2005.
7. Atasoy, E., Thoracic outlet compression syndrome. Orthop Clin North Am, 1996. 27(2): p. 265–303.
8. Christo PJ, Christo DK, Carinci AJ, Freischlag JA. Single CT-Guided Chemodenervation of the Anterior Scalene Muscle with Botulinum Toxin for Neurogenic Thoracic Outlet Syndrome. Pain Medicine 2010; 11: 504–511.
9. Vanti C, Natalini L, Romeo A, Tosarelli D, Pillastrini P. Conservative treatment of thoracic outlet syndrome. A review of the literature. Eura Medicophys. 2007; 43(1):55–70.
10. Barkhordarian S. First rib resection in thoracic outlet syndrome. J Hand Surg Am. 2007; 32(4):565–570. doi: 10.1016/j.jhsa.2007.01.018.
11. Sheth RN, Campbell JN. Surgical treatment of thoracic outlet syndrome: a randomized trial comparing two operations. J Neurosurg Spine. 2005; 3(5):355–363.
12. Degeorges R, Reynaud C, Becquemin JP. Thoracic outlet syndrome surgery: long-term functional results. Ann Vasc Surg. 2004; 18(5):558–565.
13. Huang JH, Zager EL. Thoracic outlet syndrome. Neurosurgery. 2004; 55(4):897–902.
14. Urschel HC, Razzuk MA. Neurovascular compression in the thoracic outlet. Changing management over 50 years. Ann Surg 1998; 228:609–17.
15. Bhattacharya V, Hansrani M, Wyatt MG, Lambert D. Outcome following Surgery for Thoracic Outlet Syndrome. Eur J Vasc Endovasc Surg 2003; 26, 170-175.
16. Balci AE, Balci TA, Eren S, Nesimi M. Surgical treatment of thoracic outlet syndrome: effect and results of surgery. Ann Thorac Surg 2003;75:1091-1096
17. Terzis JK, Kokkalis ZT. Supraclavicular approach for thoracic outlet syndrome.Hand (NY). 2010 Sep; 5(3):326-37.
18. Braun RM, Sahadevan DC, Feinstein J. Confirmatory needle placement technique for scalene muscle block in the diagnosis of thoracic outlet syndrome. Tech Hand Up Extrem Surg 2006; 10: 173–176.
19. Harold C. Urschel, JR., and Harry Kourlis, JR. Thoracic outlet syndrome: a 50-year experience at Baylor University Medical Center.. Proc (Bayl Univ Med Cent). 2007 April; 20(2): 125–135.
20. Marius Fodor, Lucian Fodor, Constantin Ciuce.Vascular Surgery Unit, First Surgical Clinic, Emergency District Hospital, University of Medicine and Pharmacy, Cluj Napoca, Romania.Annals of vascular surgery (impact factor: 1.04). 08/2011; 25(7):961-8.).
21. Stoney WS, Addlestone RB, Alford WC, Jr, Burrus GR, Frist RA, Thomas CS., Jr The incidence of venous thrombosis following long-term transvenous pacing. Ann Thorac Surg. 1976; 22(2):166–170.
22. Urschel HC, Razzuk MA. Upper plexus thoracic outlet syndrome: optimal therapy. Ann Thorac Surg 1997; 63:935–9.
23. Dale WA. Thoracic outlet compression syndrome: critique in 1982. Arch Surg. 1982; 117(11):1437–1445.

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