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Abstract

Thoracic outlet syndrome is a condition related to the compression of neurovascular structures within the thoracic outlet, primarily the C8 and T1 nerve roots and/or the subclavian artery/vein. This chapter examines the epidemiology, pathoanatomy, assessment, diagnosis, and treatment options for thoracic outlet syndrome. Data from the pediatric literature is limited, and therefore information from the adult literature will be incorporated.

Introduction

Thoracic outlet syndrome (TOS) is a broad term referring to an array of signs and symptoms that are caused by the compression of neurovascular structures within the constricted space of the thoracic outlet, the area just above the first rib and behind the clavicle (Sanders et al. 2007). There are several neurovascular structures that may be compressed or entrapped in this region, and specific terms have been utilized to describe the principal structure(s) affected (neurogenic, venous, and arterial). Furthermore, neurogenic TOS is subdivided into “true” or electrodiagnostic positive and “disputed” or electrodiagnostic negative. The incidence of true neurogenic TOS is estimated at 1:1,000,000, with disputed neurogenic TOS accounting for approximately 95 % of all TOS cases and 99 % of cases with neurologic symptoms (Atasoy 1996; Roos 1990b). Venous TOS accounts for approximately 3 % of cases, while arterial TOS comprises only 1 % of all cases (Sanders et al. 2007). Thoracic outlet syndrome is diagnosed 3.5–4 times as often in females than in males and occurs most commonly in adults of working age. The true incidence of TOS in the general population is controversial and has been reported to range from 0.3 % to 2 % (Atasoy 1996; Roos 1990a, b).

Pathoanatomy and Applied Anatomy

The thoracic outlet is an anatomic corridor bounded by the bones of the spinal column, first rib, clavicle, and sternum. Within this space,

there are three distinct partitions in which compression of the neurovascular structures may occur: the interscalene triangle, the costoclavicular triangle, and the subcoracoid or pectoralis minor space.

The interscalene triangle is the most commonly involved site in thoracic outlet syndrome and the most common location of brachial plexus compression. This triangle is bordered by the anterior scalene muscle anteriorly, the middle scalene muscle posteriorly, and the superior border of the first rib inferiorly. The anterior scalene muscle originates from the anterior tubercles of the transverse processes of the C3–C6 vertebrae and inserts on the inner superior surface of the first rib. The middle scalene muscle originates from the posterior tubercles of the transverse processes of the C2–C7 vertebrae and inserts broadly onto the posterior aspect of the first rib. The trunks of the brachial plexus along with the subclavian artery pass between the anterior and middle scalene muscles, while the subclavian vein passes anterior to the anterior scalene muscle (Pang and Wessel 1988). Authors have reported that several patients with TOS possess an increased amount of connective tissue in the scalene muscles. Therefore, scalene muscle imbalance and developmental abnormalities have been proposed as potential predisposing factors leading to thoracic outlet syndrome (Sanders 1996).

Several anatomic variants in interscalene triangle can predispose individuals to pathology, including the distance between the anterior and middle scalene muscles at the base of the triangle. The reported range of scalene distance between the anterior and middle scalene muscles is 0–2.2 cm, with an average of 1.1 cm (Atasoy 1996; Makhoul and Machleder 1992). Scalene muscle “intercostalization,” or crossing of insertions, has been observed in up to 15 % of cadaver dissections. This yields a V-shaped anomaly at the base of the triangle that can compress the neural and vascular structures. An alternative U-shaped deformity between the anterior and middle scalene muscles has also been reported, forming a sling effect that places pressure on the structures from below (Atasoy 1996). Additionally, the scalenus minimus, originating from C6 to C7

and inserting on the deep fascia between the subclavian artery and lower trunk of the brachial plexus, can produce a wedge effect on the lower trunk (Atasoy 1996). This muscle may also enclose the subclavian artery and pull it against pleural bands, leading to changes in arterial flow.

Congenital cervical fibrous bands have also been described as etiologies for thoracic outlet syndrome. In cases of incomplete cervical rib development, a fibrous band or “anlagen” connects the bony tip of the cervical rib to the first rib or deep fascia. These bands have been reported in 0.5–0.6 % of the population and may be bilateral in almost 80 % of affected individuals (Urschel et al. 1973). In studies by Roos and Poitevin, 12 different locations of fibrous bands causing nerve compression were reported (Poitevin 1988; Roos 1966).

The costoclavicular triangle is composed of the area between the first rib and the clavicle. This space is bounded by the clavicle, subclavius muscle, and costocoracoid ligament (thickened portion of the clavipectoral fascia extending from the first rib to the coracoid process) anteriorly, the first rib posteromedially, and the superior border of the scapula posterolaterally. The brachial plexus, subclavian artery, and subclavian vein all pass through the costoclavicular space. The subclavian vein is most susceptible to compression especially in the presence of a hypertrophied subclavius tendon insertion, as can be seen in Paget-Schroetter syndrome (axillary-subclavian vein thrombosis associated with strenuous and repetitive activity of the upper extremities) (Drakos and Gausche-Hill 2013).

The boundaries of the subcoracoid (pectoralis minor) space include the pectoralis minor muscle anteriorly and the chest wall posteriorly. Although not technically part of the thoracic outlet, compression of the neurovascular bundle can occur due to stretching of the neurovascular structures around the coracoid. This pulley effect occurs with arm positioned in abduction and concomitant external rotation of the scapula. Arm abduction also applies tension to the pectoralis muscle tendon, further compromising the space.

Assessment of Thoracic Outlet Syndrome

Signs and Symptoms

The clinical manifestations of thoracic outlet syndrome vary considerably based on the principal structure(s) affected (neurogenic, venous, and arterial), although some overlap exists. The vast majority of patients with TOS have the electrodiagnostic negative disputed neurogenic form. Therefore, the principal diagnostic modality is the physical examination findings including provocative maneuvers. Chronic pain involving the shoulder girdle, neck, and upper back with concomitant paresthesias of the upper extremity is the most common symptom reported, occurring in up to 95 % of patients with TOS (Urschel et al. 1973). These paresthesias typically affect the medial arm, forearm, and ulnar two digits (C8 and T1 nerve root distribution), although paresthesias involving the median nerve distribution or entire hand have been reported. The chronic pain is generally described as “dull” or “throbbing.”

Patients may report symptoms occurring during overhead lifting, prolonged typing, driving, and speaking on the telephone. Women in particular tend to have difficulty handling their hair and may have to flex their neck forward in order to limit arm motion (Fig. 1). In addition, there is soft evidence that some women with macromastia may experience neurologic TOS secondary to compression of the lower trunk against the first rib and the tilting forward of the coracoid process (Iwuagwu et al. 2005). Pain at the base of the neck is another common complaint, as are nocturnal symptoms (Roos 1990; Sanders et al. 2007). Patients with disputed TOS may also complain of vascular-type symptoms, including a subjective feeling of swelling and coolness of the hand.

Symptoms of venous TOS typically arise in patients who repeatedly exert their upper extremity with the arms above shoulder level. Fatigue of the affected forearm may occur within minutes of use. Swelling accompanied by pain and/or cyanosis is a common manifestation of venous TOS and may

lead to paresthesias in the digits (Fig. 2a). Collateral veins that are visible in the skin around the affected shoulder, neck, and chest wall may develop due to venous compression (Marine et al. 2013; Fig. 2b). Additionally, due to the



Fig. 1 A 14-year-old female with pain and right thoracic outlet syndrome attributed to cervical rib. The patient has decreased right arm abduction secondary to pain (Courtesy of Shriners Hospital for Children, Philadelphia)

diameter of the veins being decreased and flow being interrupted (stasis), spontaneous upper extremity venous thrombosis (Paget-Schroetter syndrome) may develop (Urschel and Razzuk 1991).

Compression of the arteries within the thoracic outlet is virtually always associated with a cervical or anomalous rib. Arterial TOS is extremely rare, and symptoms develop spontaneously, commonly in young patients with little or no risk factors for atherosclerosis. Clinical manifestations of arterial TOS are typically caused by arterial thromboembolism and include symptoms of hand and finger ischemia, including pain, pallor, paresthesias, and coldness. Arterial thromboembolisms most often develop from mural thrombus of the subclavian artery. Rarely, a thrombus from the subclavian artery can embolize in a retrograde fashion causing a stroke (Desai and Robbs 1995; Lee and Hines 2007).

Distal nerve compression in association with TOS has been suggested as a cause of double-crush syndrome. Carpal tunnel syndrome has been reported in 21–45 % of patients with TOS, and cubital tunnel syndrome has been seen in up to 10 % of cases (Leffert 1992; Lishman and Russell 1961; MacKinnon 1992; Putters et al. 1992). The double-crush theory remains controversial. Opponents of the double-crush theory propose that the wide range of variable symptoms is due to nerve compression at a

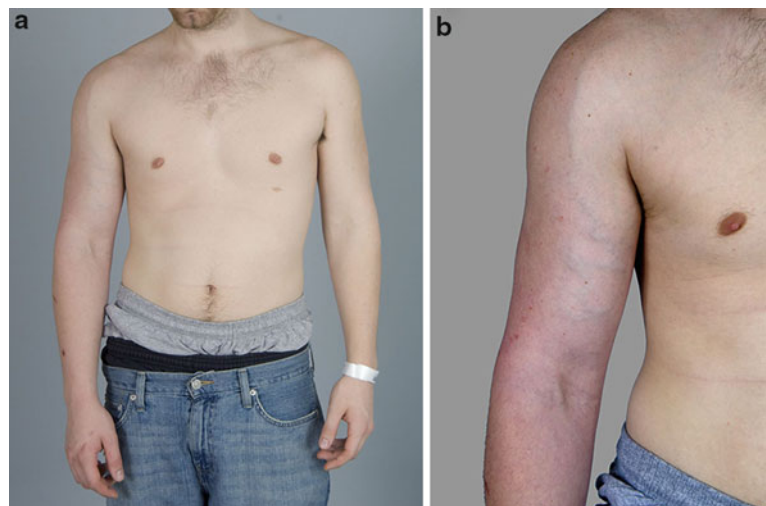


Fig. 2 An 18-year-old male who presented with complaints of pain in the right arm after working as short-order cook. (a) Swollen right arm with venous congestion present due to venous thoracic outlet syndrome. (b) Presence of distended veins (Courtesy of Shriners Hospital for Children, Philadelphia)

single site, based on symptom relief achieved following surgery at one location (Carroll and Hurst 1982).

Due to the chronic, unremitting nature and clouded differential diagnosis of TOS, patients may experience continued psychological stress. Numerous tests and physical examinations may be performed, furthering the psychological strain on patients. Depression, anxiety, anger, and frustration can become more visible as symptoms persist without a definitive diagnosis (Jamieson and Merskey 1985; Luoma and Nelems 1991). Therefore, psychological evaluation and treatment should be available for any patient who exhibits signs of psychological stress.

Physical Examination

Although the history of patients with TOS is often nonspecific, physical examination is diagnostic in up to 97 % of patients (Rayan 1998). Physical examination should begin with observation of the patient's habitus and assessment of any visible asymmetry. Slouching, shoulder droop, and scapular asymmetry are clues that may lead to a diagnosis of TOS but could also represent alternative pathologies. The skin over the chest wall, neck, and shoulder girdle should be examined for the presence of venous engorgement or collateral sprouting. Muscle tone and bulk should be evaluated via palpation with and without resistance, especially in the neck, upper back, shoulder, arm, forearm, and hand. A sensory examination should be performed utilizing two-point discrimination and Semmes-Weinstein monofilaments, as well grip and pinch measurements. Blood pressure measurements in both arms, as well as auscultation of the subclavian arteries, should be performed to assess for thromboembolism or aneurysm. In a study by Braun and colleagues, pulse oximetry measurements in patients with symptomatic TOS were compared with measurements taken in a control group prior to and after a provocative maneuver. Oxygen saturation dropped to 86 % in the symptomatic TOS group while only dropping to 94 % in the control group. Therefore, it was concluded that pulse oximetry

may be a quick and inexpensive tool to aide in the diagnosis of thoracic outlet syndrome (Braun et al. 2012).

The hallmark of the physical examination for thoracic outlet syndrome is the performance of provocative tests. Positive results are documented if a maneuver either reproduces the patient's symptoms or diminishes/obliterates the radial pulse. Several historically utilized maneuvers such as Adson's, Wright's, and Halstead's tests are less commonly performed due to their lack of sensitivity and specificity for TOS (Gillard et al. 2001).

The Roos' test (90° abduction external rotation test, stickup test, elevated arm stress test [EAST]) involves the patient holding the affected arm in an abducted and externally rotated position while repeatedly pumping the hand open and closed for 3 min. Symptoms and rapid fatigue are indicative of a positive test. The test may be poorly tolerated in patients with positional nerve compression. This maneuver is often considered the most sensitive test for thoracic outlet syndrome (Roos 1990).

The upper limb tension test involves the patient elevating both arms out to the sides with elbows straight and head facing forward. The patient's wrists are then extended, and the head is tilted away from the affected side. When this test is positive, the patient reports increasing discomfort or paresthesias due to the increasing tension on the brachial plexus (Sanders et al. 2007).

Imaging and Other Diagnostic Studies

Several imaging modalities may be utilized in the workup of thoracic outlet syndrome. Initial radiographs of the cervical spine and chest should be obtained at the time of first evaluation to assess for the presence of an elongated C7 transverse process or cervical rib on the affected side (Wilbourn 1999; Fig. 3). Fibrous bands associated with abnormal cervical ribs are radiolucent and therefore are better visualized on MRI (Panegyres et al. 1993).

Ultrasonography is often utilized in patients with vascular pathology due to its low cost and ease of use. Color duplex ultrasonography is highly sensitive and specific when diagnosing stenosis, occlusion, and/or flow abnormalities

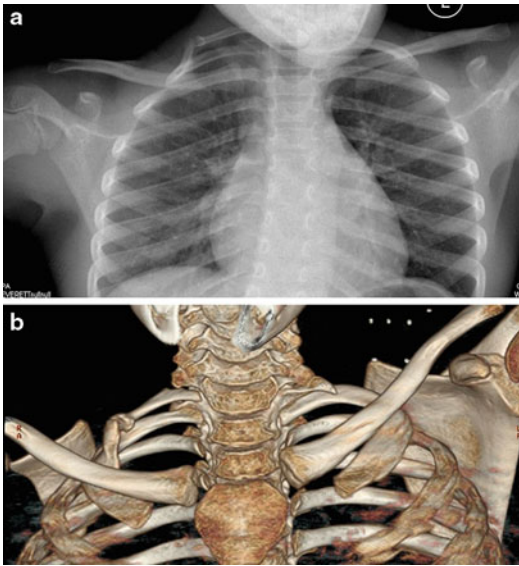


Fig. 3 (a) Chest radiograph demonstrates cervical rib on the right side with intervening synchondrosis. (b) CT scan better delineates the cervical rib and synchondrosis (Courtesy of Shriners Hospital for Children, Philadelphia)

(Demondion et al. 2006; Wadhvani et al. 2001). Pulse volume recordings can be used to evaluate arterial compression and can be followed up by MR angiography or arteriography (Kleinert and Gupta 1993).

Angiography provides the best visualization of the arterial anatomy and is considered the gold standard for the diagnosis of arterial TOS. MR angiography is noninvasive, but conventional angiography allows for simultaneous therapeutic thrombolysis in an acute scenario (Ohkawa et al. 1992; Sharafuddin et al. 2002). The bony anatomy should be carefully assessed, as bone anomalies are the most common cause of arterial compression.

In cases of suspected venous TOS, venography is the gold standard. The patient may be asked to perform a provocative maneuver while imaging takes place, as this may be diagnostic for venous compression from TOS. As with angiography, thrombolysis can also be achieved during venography if necessary. Additionally, collateral vein sprouting may be visible on venography (Sharafuddin et al. 2002).

Electrodiagnostic evaluation is recommended in patients with suspected neurogenic TOS as part of the initial workup. In patients with

symptomatic neurogenic TOS, electromyographic (EMG) changes showing chronic denervation of the intrinsic hand muscles are the initial abnormalities observed, while nerve conduction velocity changes are not seen until late in the disease (Marcaud and Métral 2000; Passero et al. 1994).

Classification Scheme

A scheme was introduced by Roos that classified TOS based on the affected segment(s) of the brachial plexus. These categories include upper trunk compression, lower trunk compression, and combined compression. Eighty-five to ninety percent of his patients exhibited symptoms of lower trunk and/or combined compression (Roos 1982).

Wilbourn categorized thoracic outlet syndrome into two basic types, each with two subtypes (Wilbourn 1999). Vascular TOS is subdivided into arterial and venous, while neurogenic TOS is subdivided into true, or electrically positive, and disputed, or electrically negative.

Outcome Tools

There are no validated outcome measures to specifically assess thoracic outlet syndrome outcomes. Several quality of life scores, including the Cervical Brachial Symptom Questionnaire (CBSQ), the Brief Pain Inventory (BPI), and the Short-Form 12 (SF-12), have been utilized to evaluate long-term TOS outcomes following operative intervention. Additionally, functional outcomes following surgical repair of TOS have been measured utilizing the Disability of Arm, Shoulder, and Hand (DASH) score (Rochlin et al. 2012, 2013).

Nonoperative Treatment

Nonoperative management is universally agreed upon as the first-line treatment of thoracic outlet syndrome. The goal is to alleviate the compression of the thoracic outlet as well as avoidance of exacerbating factors (Leffert 1992; Novak et al. 1993; Table 1).

Table 1

Thoracic outlet syndrome	
Nonoperative management	
Indications	Contraindications
All suspected cases of TOS	The presence of a mass
	Acute arterial or venous thrombosis

Techniques

Supervised postural training and exercises directed at strengthening the parascapular muscles and widening of the thoracic outlet are the strategy for patients with poor posture. Additional exercises aimed at strengthening the muscles of the shoulder girdle, stretching the scalene muscles, and relaxing the first rib can be performed. These exercises should attempt to expand the thoracic outlet so that the brachial plexus can pass unobstructed (Atasoy 1996; Pratt 1986).

For women with macromastia, better breast support may improve symptoms. Reduction mammoplasty may be warranted in severe cases, although this treatment remains controversial (Iwuagwu et al. 2005; Kaye 1972; Leffert 1992). Decreasing overhead activity and reducing downward force on the shoulder girdle through ergonomic modification can also be helpful. Symptomatic improvement is achieved in the majority of TOS patients treated without surgery. Only 10–30 % of TOS patients will progress to becoming surgical candidates (Oates and Daley 1996; Sanders et al. 1979; Selke and Kelly 1988; Thompson and Petrinc 1997; Thompson et al. 1997).

Operative Treatment

Indications/Contraindications

Indications for surgical treatment include intractable pain, neurologic deficits, vascular compromise, the presence of a mass or abnormal anatomy, or failure of nonoperative treatment (Oates and Daley 1996; Sanders et al. 1979;

Selke and Kelly 1988; Thompson and Petrinc 1997; Thompson et al. 1997).

Surgical Objective

The goal of operative management is decompression of the neurovascular structures within the thoracic outlet. A variety of procedures and approaches have been proposed, and there is no consensus on which procedure is the gold standard. Procedures include the following in isolation or combination: cervical rib resection, first rib resection, scalenotomy, scalenectomy, anomalous fascial band excision, claviclectomy, and pectoralis minor release.

Preoperative Planning

See Table 2.

Positioning

The position of the patient will vary according to the surgical approach. The patient may be supine, lateral decubitus, or in the beach chair position. The arm is prepped and draped within the sterile field. All bony prominences are meticulously padded.

Surgical Approach

There is currently no universally accepted approach for operative treatment of thoracic outlet syndrome (Oates and Daley 1996). Several approaches have been described including a supraclavicular, transclavicular, subclavicular, transaxillary, posterior, and a combined approach.

First rib resection, with or without anterior scalenectomy, is most commonly performed for TOS (Leffert 1992; Oates and Daley 1996; Roos and Owens 1966, 1982). The rare upper plexus TOS can be managed via isolated scalenectomy, where 80–90 % of the anterior scalene muscle and 40–50 % of the middle scalene muscle are excised

Table 2

Thoracic outlet syndrome
Preoperative planning
OR table: standard OR table
Position/positioning aids: variable depending upon the approach
Fluoroscopy location: unnecessary
Equipment: Spurling Kerrison rongeur, chest tube
Tourniquet (sterile/nonsterile): none
Draping: entire arm and hemithorax to contralateral sternoclavicular joint

(Alnot 1995; Sanders et al. 1979; Thompson and Petrinc 1997). The supraclavicular approach is commonly utilized for scalenectomy and upper plexus exploration but may limit the exposure of the first rib (Thompson et al. 1997).

Removal of the first thoracic rib can alleviate several potential sites of compression as the first rib acts as a fulcrum for T1, a site of attachment for the scalene muscles, and also borders the costoclavicular space. The transaxillary approach provides the greatest access to the first rib and is cosmetically more appealing but sacrifices the possibility of scalenectomy and upper plexus exploration (Alnot 1995; Karamustafaoglu et al. 2011; Thompson and Petrinc 1997). The transaxillary approach can be utilized for removal of cervical ribs, and the pectoralis minor tendon can be divided if subcoracoid compression is suspected. A supraclavicular and transaxillary combined approach may be useful in recurrent or complicated cases (Qvarfordt et al. 1984).

Technique

First rib resection utilizing the supraclavicular approach is performed via a transverse incision just above the clavicle. The incision extends from the sternocleidomastoid to the anterior edge of the trapezius. Cutaneous nerves are mobilized and the platysma incised. The external jugular vein is ligated and the underlying omohyoid identified and retracted. The omohyoid is the “door” to the supraclavicular plexus. The fat pad is mobilized to reveal the underlying plexus. The anterior and middle scalene muscles are identified. The phrenic nerve is visualized lying on the anterior surface of the anterior scalene.

The plexus is isolated from superior to inferior beginning from the C5 and C6 nerve roots (upper trunk). The C7 nerve roots lie slightly inferior and posterior to the upper trunk. Further inferior the lower trunk and subclavian artery are isolated. Deep to the lower trunk, the first rib is visualized. The entire plexus, especially the lower trunk, is gently mobilized away from the first rib. Any fibrous bands are resected. The anterior and middle scalene muscles are released at their insertions into the first rib.

The first rib is carefully separated from the underlying pleura. The rib is exposed along the entire base of the thoracic outlet triangle. Once the rib is isolated, the anterior and posterior aspects are cut with a bone cutter or Spurling Kerrison rongeur. The resected piece is then removed from the surgical field. Any remaining sharp edges are removed, and bone wax is applied to the cut ends. Wound closure is straightforward. The fat pad is brought over the brachial plexus. The subcutaneous tissue and skin are closed with absorbable suture. The arm is placed in a sling, and range of motion is started in a week (Fig. 4; Table 3).

Treatment-Specific Outcomes

Outcomes of thoracic outlet syndrome procedures vary considerably based on the operation performed and the definitions of success and failure. Two or more years may be required for recovery and improvement following any procedure. Unfortunately, the initial symptomatic relief provided by these procedures may be negated due to scar formation and recurrent compression.

Isolated scalenotomy has a reported failure rate of 50 %, with recurrence of symptoms being common. Results for scalectomy with or without first rib resection are more encouraging with success rates reported between 68 % and 90 %. Isolated first rib resection has a widely variable success rate of 37–92 %. Studies directly comparing first rib resection and scalectomy have not demonstrated significant differences in patient outcomes.

Following surgical decompression for disputed neurogenic TOS, success rates have been reported



Fig. 4 (a) Supraclavicular approach. Note the brachial plexus tented over the cervical rib. (b) Exposure and resection of the cervical rib. (c) Specimen of resected cervical rib and synchondrosis. (d) Postoperative photograph of the

patient in Fig. 1 following resection of the cervical rib. The patient had resolution of pain and symmetric shoulder motion (Courtesy of Shriners Hospital for Children, Philadelphia)

between 91 % and 93 % for all procedures. However, at 10-year follow-up, those rates decline to 64–71 % (Sanders et al. 1979; Sanders and Pearce 1989; Sanders 1996). Operations performed to treat true neurogenic TOS via release of fibrous bands or cervical rib resection commonly alleviate painful symptoms, but strength may or may not improve dependent upon the extent of axonal and muscle damage (Le Forestier et al. 1998).

Arterial TOS treated with surgical decompression has a reported success rate of 91 % at a mean follow-up of only 5.7 months (Cormier et al. 1989). Outcomes following thrombolysis and subsequent surgical decompression for venous TOS are excellent with a greater than 95 % vein patency rate at 5 years (Lee et al. 2006; Molina et al. 2007; Schneider et al. 2004; Urschel and Razzuk 2000).

Table 3

Thoracic outlet syndrome
Surgical steps
Transverse skin incision just above the clavicle
Incise the platysma and ligate external jugular vein
Identify omohyoid
Visualize phrenic nerve on anterior scalene
Identify upper, middle, and lower trunks
Protect brachial artery
Release anterior and middle scalene insertions into first rib
Incise the periosteum of first rib
Resect first rib at base of thoracic outlet
Smooth edges of bone

Preferred Treatment

Nonoperative management is always the first-line treatment for thoracic outlet syndrome. However, if this fails and an operation is required, the first rib is removed via a supraclavicular approach. Additional etiologies are also addressed at the time of surgery, including scalene release, fibrous band resection, and cervical rib removal. The rib is removed carefully separated from the underlying pleura and removed piecemeal to avoid injury to a pneumothorax.

Surgical Pitfalls and Prevention

See Table 4

Management of Complications

Brachial plexus injury following rib resection for TOS occurs in less than 1 % of cases, while vascular injuries are reported in 1–2 % of cases (Chang et al. 2007). Additional complications include pneumothorax, which requires chest tube placement. The most frustrating complication is the failure to alleviate the patient's symptoms. The reason is often multifactorial and may be due to inadequate decompression, wrong diagnosis, or secondary gain issues (Table 5).

Table 4

Thoracic outlet syndrome	
Potential pitfalls and preventions	
Potential pitfall	Pearls for prevention
Pneumothorax	Remove rib in small pieces with protection of underlying pleura

Table 5

Thoracic outlet syndrome	
Common complication	Management
Pneumothorax	Chest tube insertion
Failure to improve symptoms	Consider alternative diagnoses

Summary and Future Research to Improve Diagnosis, Treatment, and Outcome Assessment

Thoracic outlet syndrome is a complex condition that may have no identifiable pathology. The mainstay of initial management is nonoperative treatment with exercises to improved posture and to widen the thoracic outlet. Patients with persistent symptomatology due to anatomic variations or a mass effect have excellent outcomes with decompression. However, those with no discrete cause have variable success rates with operative intervention, regardless of the approach utilized.

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