Thoracic outlet syndrome: an overview

Thoracic outlet syndrome is a non-specific label that encompasses the various presentations resulting from compression of neurovascular structures at the thoracic outlet. This overview focuses on its underlying aetiology and management.

The term thoracic outlet syndrome was coined by Peet et al (1956) to encompass the various presentations caused by compression of one or more of the neurovascular structures (brachial plexus, subclavian vein and artery) exiting the thorax via its superior aperture (anatomically this is referred to as the thoracic inlet).

Thoracic outlet syndrome has in the past and still continues to gather much controversy regarding its diagnosis and recognition as a clinical entity and the best method of treatment (Mackinnon and Novak, 2002).

Mechanisms

Compression of these structures occurs either as a result of anatomical factors, trauma or repetitive activities. Anatomically, as the brachial plexus trunks and subclavian vessels exit via the thoracic inlet, there are three narrow passageways where compression may occur (Mackinnon and Novak, 2002). The most important of these is the interscalene triangle (space between scalenus anterior and medius and the first rib), followed by the costoclavicular triangle (space between the clavicle, first rib and upper border of the scapula) and subcoracoid space (beneath the coracoid process and pectoralis minor). In addition to these fixed anatomical narrowings, certain manoeuvres as well as the presence of anomalous structures (such as fibrous bands and cervical ribs) can further reduce this space. In addition, muscular instability caused by chronically abnormal posture and movements have been implicated in contributing to or propagating the compression.

Trauma (such as a whiplash injury) and vigorous or repetitive movements can directly damage the neurovascular bundle at the thoracic inlet and are particularly associated with compression of the blood vessels.

Classification

Broadly, thoracic outlet syndrome can be classified into two groups: neurogenic (involving the brachial plexus) and vascular (involving the subclavian vessels) (Colli et al, 2006). Vascular cases should then be differentiated into arterial or venous thoracic outlet syndrome.

The importance of this classification is that the different types have distinctive clinical and pathological features. Neurogenic thoracic outlet syndrome is the most common (more than 90%) but also the most variable in terms of the spectrum of disease. Early damage seen on a histological level may not even be detectable by any objective means, merely presenting with intermittent symptoms (Mackinnon and Dellon, 1988). In addition, when there is further compression more distally symptoms may occur even with minimal compression at the thoracic inlet – this is referred to as 'multiple (or double) crush syndrome' (Upton and McComas, 1973; Mackinnon and Dellon, 1988). Compression of the subclavian artery is a rare phenomenon and in young patients should lead one to suspect the presence of anomalous ribs or fibrous bands (Schneider et al, 1999). The complications of sustained compression include stenosis, aneurysm formation, ulceration and of course distal ischaemia, which is often how patients first present, having tolerated the primary symptoms fairly well (Mackinnon and Novak, 2002). However, this accounts for about 1% of the total thoracic outlet syndrome.

In venous compression, a presumed inflammatory reaction of the endothelium can lead to thrombosis of the subclavian vein (Paget–Schroetter syndrome or effort thrombosis), although subsequent pulmonary embolism is rare (Schneider et al, 1999; Mackinnon and Novak, 2002). The symptoms are identical to the symptoms of deep venous thrombosis of the leg, with arm swelling, discoloration and oedema.

Diagnosis

The most reliable diagnostic aid is the clinical evaluation, beginning with a careful history.

Presentation

Thoracic outlet syndrome mainly affects younger adults, neurogenic thoracic outlet syndrome being around three times more common in females than males. In an older patient, one should first consider other differential diagnoses (such as degenerative disease of the spine) as a cause of peripheral neurological symptoms (Urschel and Razzuk, 1998).

By far the most common presentation is neurological. Of this group approximately 95% present with pain or paraesthesia, mostly (90%) in the distribution of C8–T1

Mr Umar Sadat *is Clinical Research Associate in Vascular Imaging and Fellow in Vascular Surgery,* **Dr Ruwan A Weerakkody** *is Foundation Year 1 Doctor in General Surgery and* **Mr Kevin Varty** *is Consultant Vascular Surgeon in the Regional Cambridge Vascular Unit, Addenbrooke's Hospital, Cambridge University Hospitals NHS Foundation Trust, Cambridge CB2 0QQ*

Correspondence to: Mr U Sadat

or solely in the distribution of the ulnar nerve (Greep et al, 1979). Involvement of the upper cords of the brachial plexus (C5,6,7) is associated with pain or paraesthesia involving the thumb to middle finger and sometimes the neck, ear, upper chest, back and outer arm (radial nerve distribution) (Greep et al, 1979; Urschel and Razzuk, 1998). Deep chest pain (so-called pseudoangina) may also occur. Fewer than 10% of cases present with motor symptoms such as weakness or loss of dexterity (Urschel and Razzuk, 1998).

Rarely, patients may present with wasting of the intrinsic muscles of the hand (the so-called Gilliatt–Sumner hand); in this case the diagnosis of thoracic outlet syndrome is clear cut and specifically greater atrophy of the thenar muscles (T1) compared with the intrinsic hand muscles (C8) is seen (Upton and McComas, 1973). *Table 1* summarizes the symptoms according to frequency.

In addition to the raw symptoms (which may be accounted for by many aetiologies), there are certain typical precipitating factors. Overhead activity of the arm or hand, or repetitive activity in an elevated position (e.g. applying make-up in women) are well-recognized exacerbating mechanisms (Upton and McComas, 1973). Furthermore, although symptoms are often insidious in nature, a clear precipitating cause such as whiplash injury may also be given.

When there is vascular involvement, the symptoms are more clear cut. Compression of the subclavian artery is typically seen in young adults and associated with vigorous arm activity. Compressive symptoms include pain or claudication, coldness and Raynaud's phenomenon. Note that pain in arterial thoracic outlet syndrome may be particularly severe owing to involvement of sympathetic fibres which travel closely apposed to the artery (and may also be associated with causalgia or sympathetic maintained pain syndrome; Urschel and Razzuk, 1998). Arterial thrombosis (complete occlusion) will lead to persistent cyanosis and pallor and will present in the acute emergency setting.

Venous involvement in the form of Paget–Schroetter syndrome (effort thrombosis) is very uncommon (Mackinnon and Novak, 2002). Typically, it is seen in young men with a history of strenuous work. In addition to the usual compressive elements, cold, trauma and the usual thrombogenic factors increase the propensity for this syndrome to develop (Adams and DeWeese, 1971; Urschel and Razzuk, 1991, 1998). Such a patient will typically present with oedema and/or cyanosis of the upper extremity with distended superficial veins of the shoulder and chest.

Table 1. Presentations of neurogenic thoracic outlet syndrome

Common	Paraesthesia or pain	C8/T1: medial forearm or hand
		C5,6,7: median nerve territory
	Weakness of arm or hand	
	Headache	
Less common	Facial numbness or pain	
	Anterior chest wall pain (pseudoangina)	
	Colour alteration (sympathetic fibres)	
Rare	Wasting of small muscles of hand (thenar > intrinsic)	

Examination

In neurogenic thoracic outlet syndrome, where the diagnosis may not be entirely clear at first, further evaluation requires objective corroborating evidence of nerve compression specifically at the thoracic inlet as well as methods to exclude other possible diagnoses.

The mainstay of the physical evaluation is provocative tests which aim to reproduce the symptoms by inducing artificial compression at the thoracic inlet. Originally, a number of provocative manoeuvres were designed to assess vascular integrity in thoracic outlet syndrome (*Table 2*) but they have not been reliable in evaluating nerve compression (Sanders and Haug, 1991). Reproduction of symptoms in an arm-elevated position is thought to be a useful diagnostic indicator of thoracic outlet syndrome (in particular, the modified Roos manoeuvre is thought to be more accurate as this excludes concomitant provocation at distal sites) (Sanders and Haug, 1991; Novak et al, 1993). Patients with thoracic outlet syndrome will also exhibit tenderness over the scalene muscles (Mackinnon and Novak, 2002).

It is also particularly important to exclude distal compression since a majority of patients with thoracic outlet

Table 2. Provocative manoeuvres in thoracic outlet syndrome

Test	Description	Positive result
Adson's	Head turned towards affected side, arm by side	Obliteration radial pulse
Halstead	Shoulders backward and downward	Obliteration radial pulse
Wright's hyperabduction	Shoulder hyperabducted (180°), elbow flexed	Obliteration radial pulse
Roos	Shoulder abduction (90°), external rotation, arm flexion (90°) and opening and closing hand	Reproduction of symptoms
Modified Roos	Arm extended to avoid provocation at multiple sites	Reproduction of symptoms

syndrome exhibit signs of co-existent carpal tunnel or cubital tunnel syndrome (Novak et al, 1993). This can be done by the usual specific provocation tests at possible sites of distal compression. To exclude proximal impingement, examination of the cervical spine (including the Spurling test) can be performed.

On inspection, abnormal postures (particularly the head-forward position) that predispose to thoracic outlet syndrome by causing altered length and strength of muscles in the region of the neck may be seen (Janda, 1993; Kendall et al, 1993; Sahrmann, 2002). Evaluation of the shoulder and scapular muscles will often reveal instability or weakness of the scapular stabilizers (trapezius and serratus anterior), often associated with rotator cuff tendonitis (Mackinnon and Dellon, 1988). Finally, directed pain evaluation questionnaires are used as a means of identifying psychological and other causes of pain (Mackinnon and Novak, 2002).

Investigation

For neurogenic thoracic outlet syndrome, further tests are used as corroborative evidence or to investigate underlying aetiology. X-rays of the chest and cervical spine may reveal the presence of cervical ribs (6–11% incidence in thoracic outlet syndrome patients; Sanders and Haug, 1991), prominent C7 transverse processes, degenerative changes at the cervical spine or even a Pancoast tumour. Computed tomography scanning has also been used for more detailed evaluation (Gebarski et al, 1982; Bilbey et al, 1989).

Sensory testing is used to measure changes in vibration or pressure thresholds and two-point discrimination. A change in sensory thresholds following provocative testing is a sensitive sign of early nerve damage that would not otherwise be detectable. On the other hand, a derangement in two-point discrimination suggests more extensive damage and usually signifies multiple crush syndrome.

The use of electrophysiological tests as diagnostic tests is controversial (Wilbourn and Lederman, 1984; Wilbourn, 1999). However, some advocate using electrophysiological markers as further corroborative evidence: specifically it has been suggested that a reduction in ulnar or median nerve conduction velocity to less than 85 m/s across the thoracic inlet serves as good corroborative evidence for thoracic outlet syndrome (Jebsen, 1967; Caldwell et al, 1971; Urschel et al, 1971; Urschel, 1993; Urschel and Razzuk, 1998). Seror (2004) have reported that the nerve conduction velocity of the sensory medial antebrachial cutaneous nerve is abnormal in clinical neurogenic thoracic outlet syndrome patients in whom all other electrodiagnostic studies were normal.

In vascular thoracic outlet syndrome, clinical examination will often be sufficient for the diagnosis. However, duplex ultrasound scanning or angiography may be indicated in certain situations.

Management

Emergency management (vascular thoracic outlet syndrome)

The management of thrombosis of the subclavian vein is prompt thrombolysis, followed by surgical decompression (ideally by first rib resection). However, in late presentations anticoagulation is warfarin, usually for 6 months. Patients presenting with thrombosis or distal embolization from a stenosed subclavian artery in thoracic outlet syndrome should be treated with first rib resection, thrombectomy or embolectomy, and dorsal sympathectomy (if indicated) (Urschel and Razzuk, 1998).

Elective management

Conservative management

The treatment of thoracic outlet syndrome has been the most important point of discussion since Peet et al (1956) suggested conservative treatment. They prepared a specific exercise programme, which included moist heat, massage, stretching of pectoralis, strengthening of levator scapulae and postural correction exercises. Since then different researchers have suggested physiotherapy as the first line of management. Physiotherapy aims to open up the space between the first rib and clavicle, and improve posture and muscular stability (Caldwell et al, 1971; Novak and Mackinnon, 1996).

Whatever the method used, all studies demonstrate some degree of efficacy for conservative treatment with good or very good results in 76–100% at short-term follow up (within a month) and 59–88% at medium to long-term follow up (after at least 1 year) (Vanti et al, 2007).

If conservative approaches do not succeed and having excluded other aetiologies, surgery may be undertaken.

Surgical options

Surgery usually involves excision of the first rib or accessory rib with or without scalenectomy. The approaches are either transaxillary or supraclavicular.

Supraclavicular decompression permits not only an option for thoracic outlet decompression and cervical rib or first rib excision but also allows scalenectomy (anterior and middle) and brachial plexus neurolysis to be performed (Hempel et al, 1996). Hempel and colleagues have reported one of the largest case series with 770 supraclavicular first rib resections and scalenectomies performed for thoracic outlet syndrome. They found that an excellent response was achieved in 455 (59%) cases, good result in 206 (27%) cases, fair outcome in 95 (13%) cases and a poor result in only 13 (1%) cases. There was a single occurrence of lymphatic leakage and no brachial plexus injuries developed. Only two cases developed postoperative causalgia which required sympathectomy. There were no vascular or permanent nerve injuries. McCarthy and colleagues (1999) have also

reported satisfactory results with this approach concluding that supraclavicular scalenectomy and cervical rib excision with selective first rib excision is a safe and effective procedure for most patients with thoracic outlet syndrome.

On the other hand, the supraclavicular approach alone does not give adequate exposure for complete removal of first rib. The transaxillary approach overcomes this problem because ideally the posterior stump of the first rib should be left so short that it lies posterior to the T1 cervical root clearly seen at the operation and should not extend more than 1 cm anterolaterally when seen by chest X-ray (Roos, 1971; Barkhordarian, 2007). In most series, transaxillary first rib resection provides better relief of symptoms than supraclavicular neuroplasty of the brachial plexus and two thirds of patients return to normal activity (Fulford et al, 2001).

The studies comparing surgical and conservative management provide quite controversial results with surgery favouring patients in traumatic cases and conservative management favouring non-trauma cases.

However, as concluded by Vanti et al (2007), almost all authors report good correlation of favourable outcomes with an appropriate exercise regimen either with or without surgery.

Conclusions

The diagnosis and treatment of thoracic outlet syndrome is quite complex but usually rewarding. Therefore symptom complexes should be identified and managed appropriately, ideally by a multidisciplinary team. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Thoracic outlet syndrome is a complex multifactorial condition.
- There are multiple diagnostic steps and multiple treatment options which can be non-operative (e.g. physiotherapy) or surgical (e.g. cervical rib excision).
- Patients should be advised that surgical decompression may not completely alleviate all symptoms.