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REVIEW ARTICLE

Neurogenic Thoracic Outlet Syndrome Reviewed

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Abstract

Neurogenic Thoracic Outlet Syndrome (NTOS) is a rare, but controversial syndrome in relation to its diagnosis, treatment modality, and approaches in case of surgical treatment. In the English literature, there are sparse studies dealing with these aspects.

We conducted a PubMed 2000-2017 literature review and found a total of 3953 cases reported with NTOS. The clinical characteristics, etiology, electrophysiological and radiological work-up and treatment options were reviewed and reported.

It seems that, as far as surgical indication criteria are concerned, there is a consensus for NTOS in its motor deficit stage and its techniques are generally well established.

This review showed that differential diagnosis, radiological, and electrophysiological criteria for correct diagnosis of NTOS are not controversial. However, surgical indications and types of approaches and techniques reflect the surgeon's affiliation with specialties dealing with NTOS (vascular, plastic, hand, orthopedic or neurosurgeons), and the surgeon's experience with this specific and rare syndrome.

Keywords: Neurogenic Thoracic Outlet Syndrome, Muscle spasms, nerve compression

Abbreviations: TOS Thoracic outlet syndrome, NTOS Neurogenic Thoracic outlet syndrome, FRR First Rib Resection, CRR Cervical Rib Resection, S Scalenotomy

1. Definition, Introduction, and classification

Neurogenic Thoracic Outlet Syndrome (NTOS) is an entrapment syndrome that refers to brachial plexus compression at the thoracic outlet level, namely an opening bordered laterally by the first rib, medially by the vertebral column, and anteriorly by the clavicle manubrium complex, which is defined by early anatomists more precisely as thoracic inlet.[1] NTOS is the second most commonly published entrapment syndrome in the literature after carpal tunnel syndrome.[2]

NTOS can coexist with arterial and venous TOS, which are syndromes characterized by compression of the brachial artery or vein respectively, but which remains a separate entity. NTOS is an object of the neurological surgery practice due to symptomatology of brachial plexus involvement. NTOS and Vascular TOS symptoms often overlap, and these patients frequently come to the attention of both vascular and neurological surgeons.[3] Although literature is rich in reports on TOS case series, an established classification of this group of syndromes, clinical criteria, and agreement on how cases should be presented in the literature has been until recently unclear. According to a recent report from the Society of Vascular Surgery, NTOS is confirmed when three of the following four criteria are present: (i) signs and symptoms of pathology occurring at the thoracic outlet (pain and/or tenderness), (ii) signs and symptoms of nerve compression (distal neurologic changes, often worse with arms overhead or dangling), (iii) absence of other pathology potentially explaining the symptoms, and (iv) a positive response to a properly performed scalene muscle test injection.[4]

When neurological subjective complaints prevail the clinical situation and when objective evidence on

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impairment in nerve conduction is not found, neurological TOS is referred as unspecific or disputed TOS. A lack of objective signs makes these patients difficult to evaluate. Patients that fall into the disputed TOS category may not respond well to surgery.[5] Unfortunately, disputed TOS often causes significant impairment of patient quality of life. [6] In our opinion, subjective complaints in disputed TOS reflect nerve suffering despite missing objective findings. Technical limits to detect nerve dysfunction in this stage could hypothetically explain the lack of documentation in the literature of nerve dysfunction in this group of patients.

2. Etiology of NTOS

Thoracic outlet syndrome is caused by the entrapment of the neurovascular bundle passing through the thoracic outlet. It is widely accepted that TOS is basically a problem of "space" for the neurovascular bundle passing through several narrow anatomical paths from the neck to the axilla. The brachial plexus as well as subclavian vessels are vulnerable to compression as they cross three distinct areas in the cervico-axillary canal: the interscalene triangle, the costoclavicular triangle, and the subcoracoid space.[7] Repeated compression and minor trauma to the bundle are caused when anatomical variation, anthropomorphical, and physiological conditions such as congenital abnormalities with superimposed traumatic injury, muscle spasm, and tissue fibrosis are met. [1, 7] The severity of bundle compression determines the clinical context. [1, 7-12]

Main anatomical variation associated with NTOS is the presence of cervical rib. About 10% of the patients with cervical ribs who are symptomatic (i.e. show signs of TOS) usually also show neurogenic symptoms.[9, 13] Only 50% of the patients in this group refer improvement after surgical cervical rib removal , indicating that presence of cervical rib may be a risk factor rather than the main cause of cervical bundle suffering.

Muscle spasms are another physiological condition highly associated with NTOS syndrome. Cervical muscle spasms, particularly those involving the anterior scalene muscle, transmits traction on the brachial plexus at the thoracic outlet. Muscle and nerve edema follows brachial plexus traction causing worsening of the space-content conflict in the thoracic outlet space. Inflammation, fibrotic changes, and scar development may follow. Finally, normal nerve conduction may be hampered from scar development and fibrotic changes. [1, 11, 12] Because the brachial plexus and subclavian artery trace the same trajectories, neurologic symptoms or arterial signs overlap. In addition, given that the brachial plexus also innervates vessels of the arm, vasomotor disturbances may also reflect brachial plexus compression pathology. Arterial TOS is not considered present unless symptomatic ischemia with compression or actual physical injury is proven.

3. Clinical signs and symptoms, differential diagnosis

Like in all other entrapment syndromes, symptoms of NTOS usually begin with a first irritating stage that may develop toward a second lesion stage.[7] Sensory-motor disturbance, and vascular disturbance are part of the constellation of symptoms in patients suffering from NTOS. The most common symptoms, including pain in the neck, shoulder, arm, and hand, are typically unilateral but can also occur bilaterally.[1] Women are referred to be three to four times more likely to develop NTOS.[8]

Clinical situations may be dominated by upper plexus or lower plexus symptoms. In upper plexus compression (median nerve), symptoms secondary to C5, C6 and C7 nerve roots involvement are present. Sensory changes occur in the first three fingers and muscle weakness or pain occurs in the anterior chest, triceps, deltoids, as well as at the parascapular muscle areas and down the outer arm from the extensor muscles to the forearm. Pain in the neck, mandible, and ear with occipital headaches have also been reported. Dizziness, vertigo, and blurred vision may be present. [8, 14] The most frequently observed symptoms are pain and paresthesia present in approximately 95% of patients. In 75% of this group symptoms are segmental, and in 90% of these cases the ulnar nerve distribution is affected or involved. Motor weakness is present in less than 10% of cases. [9]

Lower plexus irritation involves C8 and T1 nerve root compression with sensory changes occurring mainly in the fourth and fifth finger often present. Pain or muscle weakness is referred at rhomboid and scapular muscles to the posterior axilla, down the ulnar distribution to the forearm and involving the elbow, to flexors of the wrist and intrinsic muscles of the hand. Objectively, slowing of median nerve conduction indicates upper plexus compression whereas slowing of ulnar nerve conduction indicates lower plexus compression.[8]

In one of the largest studies of NTOS, symptoms of lower plexus compression were most common (68%), followed by symptoms of both upper and lower plexus compression (20%) and symptoms of upper plexus compression alone (12%).[8]

A classic finding in patients with true Neurogenic TOS is the Gilliatt-Sumner hand. First described by Gillatt *et al.* in 1970, Gilliatt-Sumner hand is clinically characterized by the most dramatic degree of atrophy observed in the abductor pollicis brevis, with a lesser involvement of the interossei and hypothenar muscles. Thenar, hypothenar, and interossei weakness and/or atrophy, ulnar and medial ante-brachial cutaneous hypoesthesia can be present in the affected arm, whereas sensibility in the median nerve region can be normally preserved. Sensory loss is restricted to the ulnar aspect of the hand and forearm, as predicted primarily by the known compression of the lower trunk. [7, 15, 16]

The diagnosis is based on patient history, physical exam, and objective findings. A history of trauma is often referred in anamnesis. Physical examination reveals the above described clinical signs. Maneuvers that either narrow the thoracic outlet (lifting the arms overhead) or stretch the brachial plexus (dangling; often driving or walking/running) exacerbate the symptoms and are key to examination.

In addition to ENMG findings, more recently MR neurogram and tractography may be useful to confirm NTOS diagnosis and identify the location of nerve compression, allowing tailored surgical decompression [5, 17] Trauma, mass lesions, vascular lesions, brachial plexitis, and cervical spinal cord or foraminal stenosis share similar diagnostic features and are to be considered in the differential diagnosis.[18] Particularly, in patients with nerve compression symptoms older than 60, causes such as degenerative or traumatic cervical spine, cardiac, or pulmonary pathologies are to be suspected and excluded before a final diagnosis of TOS is made.[8]

4. Electrophysiological findings

In this entrapment neuropathy, the T1 fibres tend to be preferentally affected. The characteristic features of True N-TOS are T1>C8 nerve fiber involvement. A comprehensive electrophysiological examination of the lower plexus with contralateral comparison studies is imperative to diagnose this disorder accurately.[19]

Proximal focal conduction abnormalities, such as conduction block or decreased conduction velocity in the lower plexus, are described in the literature. However, it is difficult to apply them in practice as their measurements appear to be unreliable and difficult to reproduce.

Somatosensory evoked potentials might be abnormal in patients with objective signs of TOS but, as demonstrated by Veilleux, this does not affect the diagnosis of NTOS. Studies on nerve conduction have shown that lower brachial plexus lesions have a distinctive pattern of axonal loss with low CMAP (Compound Muscle Action Potential) amplitudes in both median and ulnar nerves, but preferentially affect the median innervated thenar muscles. F wave prolongation may be found but it should be interpreted with caution and considered in combination with other abnormal parameters that point to the lower plexus. [20, 21] Sensory nerve conduction studies also reveal a distinctive pattern that suggests lower trunk involvement. In this case, despite the decreased median CMAP amplitude, the median SNAP (Sensory Nerve Action Potential) is normal. This occurs due to sensory median fibers being unable to travel through the lower trunk. In most cases the ulnar SNAP is low but not absent.

Needle EMG abnormalities are found in median rather than ulnar innervated C8-T1 muscles, and less so in radial innervated C8 muscles. Medial antebrachial cutaneous nerve sensory action potential is an important parameter to consider in the electrophysiological diagnosis of lower brachial plexus lesions. It has been demonstrated that in different clinical settings, this nerve is preferrentially damaged early in the lower trunk lesions. Seror reported that low or comparatively low MABCN SNAP amplitude could be the only abnormal feature suggesting a mild lower brachial plexus lesion, meaning this abnormality could identify patients before important axonal loss occurs in motor fibres.[22] Radiography, MRI, Angiography and Echography can further help evaluate structural anomalies known to influence the syndrome. [5]

5. Management

Typical clinical and electrophysiological changes present in patients with true NTOS respond well to surgical treatment. However, patients with disputed/nonspecific NTOS show undefined electrophysiological changes which respond unfavorably to surgical treatment.[23]

Evidence suggests that delaying surgical decompression may lead to irreversible nerve injury and limit potential symptomatic relief and thus emphasizing the need for clear diagnostic criteria to promptly confirm the diagnosis of NTOS rather than waiting for prolonged failure of conservative measures.[24]

Different surgical approaches have been developed with time. Supraclavicular scalenectomy and partial scalenectomy, with neurolysis of the brachial plexus when indicated, combined with resection of a cervical rib when present, were the most common procedures in the early 1950's.[8] The posterior approach for resection of the first rib was used ten years later. As the mortality rate was high with the above- mentioned techniques, trans-axillary approach became the technique of choice in years 60's-70's due to lower mortality rates and no requirement for muscle division.

A supraclavicular or infraclavicualar approach was mainly reserved for vascular lesions.[8, 25] According to Ross *et al*., the anterior approach has given an extremely satisfactory immediate post operatory result. A trans axillary approach is recommended for resection of the first rib, reserving the posterior approach for second procedures in patients with recurrent symptoms.[25] Studies also show that first rib resection alone can be enough to improve upper plexus symptoms.[8]

When there is an indication for surgery, an earlier treatment increases the probability for a better outcome. [24] As for all entrapment syndromes, physical therapy is fundamental for the proper management in cases that do or do not have indications for surgery. Disputed NTOS is best managed with a trial of conservative therapy before surgical treatment options are considered. Cases that are resistant to conservative treatment may require surgical intervention. In True neurogenic and vascular TOS, surgical indication is widely accepted. Post-operative rehabilitation

is recommended after surgical decompression to address factors that could lead to a recurrence of the patient's symptoms.[26] Botulinum toxin and lidocaine injection to scalene or pectoralis muscles may improve symptoms in patients with a strong component of muscle spasm in their pathology. However, they cannot replace an indication for surgery when present and are often applied as adjunctive therapies in operated patients. [9, 10, 16]

Discussion

According to Tender *et al.,* 2004, "True NTOS is the only universally acknowledged type of NTOS and is defined as a demonstrable, chronic C8, T1, and lower trunk brachial plexopathy, usually caused by congenital bony and soft tissue anomalies".[15, 16] Cases with neurogenic symptoms are reported to represent about 95-98% of cases in literature,

Table 1. Shows a summary of the literature on the treatment, reported as published PubMed NTOS case series in the last 20 years.

Year	Author	Cases with NTOS	Surgical approach for the primary procedure	Affiliation
1998	Urschel and Razzuk [8]	2210	Trans axillary FRR	Cardiovascular and thoracic surgery
2002	Sanders et al[13]	46	*Supraclavicular approach CRR+S in 22 cases; CRR+FRR 17 cases Anormal FRR 5 cases *Trans axillary FRR	Vascular Surgery
2004	Tender et al[15, 16]	33	Anterior approach: 18 cases Posterior subscapular: 15 cases NO first rib resection in all	Neurosurgery
2005	Altobelli et al[27]	185 patients 254 operative sides	Transaxillary FRR+S	Vascular Surgery
2010	Vegelin et al[28]	11 NTOS 11 Disp TOS	Supraclavicular approach 21/24 FRR	Plastic, Reconstructive and Hand Surgery
2010	Scalli et al. [29]	26	Transaxillary FRR (8) Supraclavicular FRR (10) CRR (6) And / or S	Vascular Surgery
2010	Atasoy [30]	750	TransaxillaryFRR+Transcervical Anterior S	Hand Surgery
2011	Chandra et al. [31]	55	Supraclavicular decompression FRRS+BPNeuorlisis	Vascular Surgery
2012	Rochlin et al. [32]	162	FRRS +S	Vascular Surgery
2013	Caputo et al. [33]	189	Supraclavicular decompression (FRRS+ BP neurolysis with or without pectoralis minor tenotomy	Vascular Surgery
2015	Likes et al. [34]	286	FRR+S	Vascular Surgery

whereas NTOS with objective findings on brachial plexus structure compression (known as True NTOS), considered as the "classic" form, accounts for only 1% of cases.[11, 12] About 99% of neurogenic cases are characterized by clinical features of brachial plexus compression, such as pain and other symptoms, without objective findings.[12]

In a PubMed 2000-2017 literature review, we found a total of 3177 cases reported with NTOS (Table 1) with the biggest series from Urchel and Razzuck, 1998 with 2210 patients. Urchel and Razzuck indicate that the best treatment is surgery, however, these cases do not consist of patients diagnosed singularly with NTOS.

In general, surgery is indicated only in the presence of symptoms that fail to respond to conservative therapy and produce disability.[13] Duration of symptoms before surgical treatment is the main prognostic factor.[35] Postoperative ENG registration often does not support clinical improvement after surgery. Individual therapeutic concepts with special consideration of anatomy and the clinical picture have to be considered for the best treatment options.[36] Surgical therapy yields positive results, particularly if true pathologic compression of the neurovascular structure is present.[31, 37] Among the authors' strictly investigating and following NTOS cases, Marty *et al.,* 2012 referred 43% of cases with complete or marked recovery.[35] The remaining cases showed minimal improvement. Le Forester *et al.,* 2000 referred complete improvement in their series of 10 patients.[38] Recovery, however, is less probable in cases associated with hand atrophy.[36, 39] Clinical improvement is not always followed by improvement in electrical indicators, suggesting that ENMG may not be the tool of choice for follow up.[36]

Conclusion

NTOS is the second most commonly published entrapment syndrome in the literature after carpal tunnel syndrome.[2]

NTOS is characterized by objective clinical findings of nerve suffering. When neurological subjective complaints prevail the clinical situation and when objective evidence on impairment in nerve conduction is not found, neurological TOS is referred as unspecific or disputed TOS. The pathology is due to the entrapment of neurovascular bundle passing through the thoracic outlet. The most frequently observed symptoms are pain and paresthesia present in approximately 95% of patients.[9]

Women are referred to be three to four times more likely to develop NTOS. Most common symptoms, including pain in the neck, shoulder, arm, and hand, are typically unilateral but can also occur bilaterally. Upper plexus syndromes or lower plexus syndromes may dominate the clinic. Gilliatt-Sumner hand is a classical finding. In addition to ENMG findings, more recently MR neurogram and tractography may be useful to confirm NTOS diagnosis and identify the location of nerve compression, allowing tailored surgical decompression [7, 16]

When objective evidence for nerve suffering is present, the indication for surgery is clear and there is no need to wait for physical therapy results. Supraclavicular approach with first rib resection and brachial plexus neurolysis with or without Scalenotomy is the most commonly used approach. Physical therapy and thorough follow up is necessary after surgery. A decision for surgical treatment should be taken with consideration of the overall clinical situation, rate of disability, and patient expectations.

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