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ELECTERODIAGNOSTIC STUDIES ROLE IN DIAGNOSIS AND MANAGEMENT OF THORACIC OUTLET SYNDROME

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ABSTRACT

Thoracic outlet syndrome (TOS) is caused by the compressive effect on brachial plexus or subclavian vessels at the thoracic outlet. The symptoms are usually neurologic, arterial or venous or the combination of them. Occasionally, the patient complains of chest pain simulating angina pectoris, or pain at the shoulder, neck or anywhere else in the domain of brachial plexus.
There is no pathognomonic test for TOS so far and the diagnosis is dependent to history and physical examination only .Electrodiagnostic studies have been used to show the severity of nerve injuryand see the outcome of surgical intervention and there is not a common finding to make the diagnosis upon that finding. In this study we are trying to prove it.
It has been customary to make the diagnosis of TOS if the nerve injury is limited to C8T1 root but we are going to show that this understanding is not correct and we can have injury to any of the cervical plexus roots. Moreover, It is impossible to differentiate the site of injury from roots and trunk by electrodiagnostic studies since we can't place an electrode over the spinal cord
To prove this fact we selected only the patients who were completely satisfied by the operation and their symptoms was relieved according the quick DASH Questionnaire and thereby we were sure that the diagnosis of TOS has been correct. Retrospectively we evaluated the electrodiagnostic findings in these patients.

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Introduction

Thoracic outlet syndrome (TOS) is a condition caused by compression of nerves or blood vessels at the thoracic outlet(1). Pain, paresthesia, and weakness in the hand, arm, and shoulder, plus neck pain and occipital headaches, are the classical symptoms of neurogenic (NTOS).

Other names according to presumed etiologies such as scalenus anticus, costoclavicular, hyperabduction, cervical rib, and first rib syndromes was assigned to this syndrome.(1) The various syndromes are similar and the specific compression mechanism is often difficult to identify.

The symptoms are usually a neurologic ,venous or arterial or a combination of them. Occasionally, the pain is atypical in distribution and severity and is experienced predominantly in the chest wall and parascapular area, simulating angina pectoris.(1)

Diagnosis of the nerve compression can be objectively substantiated by determining the nerve conduction velocity.

The ulnar nerve conduction velocity (UNCV) test, as described by Jebson(4) and by Caldwell and associates,(3) has widened the clinical recognition of this syndrome and improved diagnosis, selection of treatment, and assessment of therapeutic results.

Although in mild cases with no sign of nerve palsy physiotherapy to improve posture and stretch neck muscles is used primarily, in cases without improvement and the sever cases surgery is advised.

Surgical treatment involves resection of the first rib neurolysis of brachial plexus and scalenectomy , usually through the transaxillary approach except for arterial procedures, which may employ the supraclavicular approach.

Method And Material

We have probably one of the largest series of TOS patients. From these patients we chose 70 patients whom underwent surgery (resection of first rib ,with neurolysis with trans axillary approach) and we had the complete data on EMG -NCV and could

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follow postoperative course and were completely satisfied by the operation and their symptoms was relieved according the quick DASH Questionnaire and thereby the diagnosis of TOS was proved by the result.
58 were female and 12 patients were male (83%-17%) median age was 40.5 years old with the range of 15-62 years.
According to the disabilities of the arm shoulder and hand score (quite DASH)the median score preoperative was 63.6 and median post-operative score with the follow up at least 24 months (2-6 years) was 13.6.

Results

The electro diagnostic tests of 6 patients (1.5%) were completely normal.
The lower trunk symptoms which should be more frequent in TOS was purely present in 7.1%.
Fibrillation and fasciculation along the course of ulnar nerve was present in 10% of cases.
The middle trunk abnormal motor NCV was the pure pathology in2.9%. Fibrillation and fasciculation along the course of median nerve (abductor policis berevis) 2.4% and for
Brachioradialis(radial nerve) NCV was abnormal in 1.8% .
The other finding are summarized in tables 1-12

Discussion

As the electro diagnostic reports are performed according the most frequent pathology compatible with the findings, TOS was only reported in 22 patients (in 31%) of our cases.
The mainstay diagnostic consideration for thoracic outlet syndrome includes the history and physical examination and although EMG-NCV findings are not necessary to confirm the diagnosis and cannot reliably rule out this diagnosis. These test are performed to show the extent of nerve damage before the surgery and save the surgeon from legal issues concerning the intraoperative nerve damage.
The subclavian vessels and brachial plexus traverse the cervicoaxillary canal to reach the upper extremity. The outer border of the first rib divides this canal into a proximal division triangle. This proximal division is composed of the scalene triangle and the space bounded by the clavicle and the first rib (the costoclavicular space. (1)
The proximal division is the critical space in which the neurovascular compression happens. This space is bounded superiorly by the clavicle and the subclavious muscle; inferiorly by the first rib; anteromedially by the border of the sternum, the clavi pectoral fascia, and the costoclavicular ligament; and posterolaterally by the scalenus medius muscle and the long thoracic nerve. (1)
The scalenus anticus, inserting on the scalene tubercle of the first rib, divides the costoclavicular space into two compartments: an anterior compartment, containing the subclavian vein, and a posterior compartment, containing the subclavian artery and brachial plexus. (1)
Many factors can cause compression of the neurovascular bundle at the thoracic outlet. The basic factor, which was pointed out by Rosati and Lord (5) is deranged anatomy, to which congenital, traumatic, and atherosclerotic factors may contribute. Bony abnormalities are present in approximately 30% of patients, either as a cervical rib, a bifid first rib, and fusion of first and second ribs, clavicular deformities, or previous thoracoplasty.
Pathologic changes in the configuration of the cervicoaxillary canal alter the normal functional dynamics and serve as the basis of the clinical maneuvers used in the diagnosis of thoracic outlet syndrome.(1)
The mainstay diagnostic consideration for thoracic outlet syndrome includes the history and physical examination. Radiographs of the chest and cervical spine, EMG-NCV, cervical myelography, magnetic resonance imaging (MRI), computed tomography (CT), coronary angiography, venography, or arteriography are not necessary to confirm the diagnosis.(1)
The symptoms and signs of thoracic outlet syndrome depend on whether the nerves, blood vessels, or both are compressed at the thoracic outlet.
Signs and symptoms of nerve compression occur most frequently; arm pain and paresthesias are present in about 95% of patients and motor weakness in < 10%. Pain and paresthesias are segmented in 75% of cases, with the ulnar nerve distribution involvement more prominent. Pain is usually insidious in onset and commonly involves the neck, shoulder, arm, and hand.(1)
In some patients the pain is atypical, involving the anterior chest wall or the parascapular area; it is then termed pseudoangina because it simulates angina pectoris.(1)
Urschel and associates (7) reported that these patients have normal coronary arteriograms and decreased ulnar nerve conduction velocities, strongly suggesting the diagnosis of thoracic outlet syndrome.
This atypical manifestation of the thoracic outlet syndrome is due to the neuroanatomy, innervation, and pain pathways of the arm, chest wall, and heart.
To confirm the diagnosis we can use the compression tests from which Adson or Scalene Test is the most helpful one.
The Adson test, described by Adson in 1951(2) tightens the anterior and middle scalene muscles, thus decreasing the interscalene space and magnifying any preexisting compression of the subclavian artery and brachial plexus. The patient is instructed to take and hold a deep breath, extend his or her neck fully, and turn his or her face toward the side. Obliteration or diminution in the radial pulse suggests compression.
There is no single diagnostic test that can confirm the presence of a NTOS.(1) Variable presenting symptoms, complicated by multiple anatomical anomalies, present a diagnostic dilemma to the physician. Diagnosis is usually confirmed by a combination of a proper history and physical examination and electrophysiological studies are only performed to rule out other pathologies and to have a confirmation of the extent of disease before the operation.
Electrodiagnostic studies fall under two categories: electromyography (EMG) and nerve conduction studies (NCS). EMG studies have been found useful in the diagnosis of neurogenic TOS, but may not be sensitive enough in patients with a milder form of the disease.[13,14] Motor NCS can test the brachial plexus motor component at the root or cord level. Any decrease in amplitude of response is suggestive of axonal loss. Delayed conduction may implicate a demyelinating disease.[14]
Motor conduction velocities of the ulnar, median, radial, and musculocutaneous nerves can be reliably measured, as described by Jebson. Caldwell and associates(3) improved and adapted to clinical use the technique of measuring UNCV in evaluating patients with thoracic outlet compression. Conduction velocities over proximal and distal segments of the ulnar and median

nerves are determined by recording the action potentials generated in the hypothenar or first dorsal interosseous muscles. The points of stimulation are the supraclavicular fossa, mid-upper arm, area below the elbow, and wrist.
Urschel and colleagues reported that the normal UNCV values are 85 m/s across the thoracic outlet, 55 m/ s around the elbow, 59 m/s in the forearm, and 2.5 to 3.5 m/s at the wrist.
In patients with thoracic outlet syndrome, the average UNCV value was reduced to 53 m/s across the outlet (range of 32-65 m/s). (6-10)
Somatosensory-evoked potential studies have been utilized to diagnose neurogenic TOS,[17,18,] but may be nonspecific.[15,19]
Electromyography (EMG) and nerve conduction velocity (NCV) tests are normal in the large majority of patients with clinical signs of NTOS. When positive, the changes are usually nonspecific. In a small number of patients, usually those with cervical ribs and hand atrophy, electrodiagnostic studies reveal typical changes of ulnar neuropathy.

TABLE 1

		Frequency	Percent
Gender	Female	58	82.90%
	Male	12	17.10%

TABLE 2

Age	
mean±sd	median(min to max)
39.17±11.26	40.5(15to62)

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