Thoracic Outlet Syndrome: Historical Aspects of Anatomy and Physiology

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Abstract—Throughout the years complexes of symptoms of the upper extremity, caused by compression, were to be explained. Pain, paresthesia, edema, ischemia and motor dysfunction of the arm was and still is a diagnostic problem. From the start many separated syndromes have been developed in order to explain the patient's problems which at the end came they all together to a nosological entity, the thoracic outlet compression syndrome (TOS). This paper reviews the evolution of the syndromes throughout the years and the historical landmarks that formed this multifactorial problem as we understand it today.

Index Terms—Thoracic outlet syndrome, neurogenic thoracic outlet syndrome, cervical rib, scalenus muscle, scalenotomy, compression syndromes.

I. INTRODUCTION

The thoracic outlet syndrome is an obscure cause of shoulder pain which is often overlooked. Diagnosis of the TOS is difficult, particularly in patients without osseous abnormalities on plain radiographs and with wide variation in symptoms. The purpose of this article is to review the literature on the anatomical, diagnostic, and therapeutic considerations involved with TOS form a historical aspect.

II. CERVICAL RIB SYNDROME

Cervical rib was first described, by Galen and Vesalius, and since their time many and prolific have been the writers who have observed this anomaly and have recorded their observations in the literature mostly due to vascular obstruction. At 1818 a woman with arm ischemia was evaluated by Sir Astley Cooper [1] who identified a connection between cervical ribs and hand ischemia when he described the cause of her symptoms as a result of “a projection of the lower cervical vertebrae towards the clavicle and consequent pressure on the subclavian artery.” So the Cervical rib syndrome, a compression syndrome of the vascular bundles was born. The first official diagnosis was made by Willshire [2] in 1860 and by Gruber in 1869 [3]. In 1861, at St. Bartholomew’s Hospital in London recorded an early attempt to remove the first rib, an exostosis of the transverse process of the seventh cervical vertebra to treat a weak, painful ischemic hand by Coote [4] and in 1865, Paget first described axillary vein thrombosis due to the presence of a cervical rib [5].

As we see the initial emphasis was on cervical ribs as a source of anatomic compression, especially of the subclavian vessels. In the years to follow the neurological symptoms were added to the equation. At 1905 the syndrome included not only the vascular bundles but also the brachial plexus compression symptoms and Murphy gave the first definition: 1) pressure on the trunks of the brachial plexus with pain, paresthesia, hyposthesia, or anesthesia in the peripheral area of distribution of the involved sensory fibers, and paresis or paralysis of the muscles supplied by the involved motor fibers, 2) pressure on the subclavian artery with brachial ischemia and possible aneurysmal formation, thrombosis and gangrene, and 3) development of a tumor in the supraclavicular triangle [6]. The study of a nosological entity with such interest pointed out patients with similar symptoms in the absence of a bony anomaly. That was recognized by Murphy in 1910, who described a case which presented many of the manifestations of the cervical rib syndrome but with no cervical rib present [7]. He outlined the significance of the muscle system of the area and particularly the scaleneus anterior muscle. The same conclusion had at 1910 and Jones who also stated that in some individuals the eighth cervical and first thoracic segments contribute an unusually large proportion of the fibers to the brachial plexus and in these cases the first thoracic rib may traumatize the lower trunk of the plexus [8]. It is clear by then that there is no need of a cervical rib to ignite TOS symptoms which have started to be recognized and analyzed.

At 1912 Todd [10] - [13] disagrees with Murphy [15] and previous statements that the subclavian artery could be compressed by the scalenus anterior muscle or the first rib. In cases of cervical rib the nervous symptoms are caused by an exaggerated form of the normal relation existing between the brachial plexus and the first rib. He believed that the vascular symptoms in cases of cervical rib syndrome are not mechanical in origin but trophic in character and are caused by paralysis of the sympathetic fibers passing to the vessels. He also stated that the vascular symptoms depends on normal dynamic factors like: action of the scalenes in inspiration, descent of the diaphragm in inspiration, descent of the heart in inspiration.

The theories of both Jones and Todd were supported by Stopford who highlighted another anatomical structure of the area, the clavicle [16]. It was reiterated that the medial portion of the clavicle may be elevated or depressed depending upon the tonicity and development of the rectus
abdominis muscles and that the position of the lateral portion may be similarly changed depending upon the strength and tone of the trapezius muscle. He concluded that these two factors acting on the clavicle are of importance in the production of compression of the lower trunk of the brachial plexus by the first rib and also explain the greater incidence of this syndrome in females [10],[16].

III. SCALENUS ANTICUS MUSCLE SYNDROM

Until now we had a solid bone – cervical rib, clavicle etc. that surely causing problems but TOS can occur without the presence of any bony anomaly. This is the point that other theories are gaining ground in order to fill in the gap in pathophysiology. The scalenus anticus syndrome was born and involved especially with the work of Adson and Coffey in 1927 [17]. They disagreed with Todd's statement that the contracting scalenus anticus muscle could not compress the subclavian artery. They advocated that the cervical rib not be removed for the relief of symptoms but that only the scalenus anticus muscle be severed. Adson described the “Adson test” which we still use today in clinical evaluation of patients, and it was considering then a pathognomonic sign of scalene anticus syndrome.

Ochsner, Gage and DeBakey reported a series of cases without cervical rib in which they believed abnormal compression to the neurovascular structures passing to the arm was produced by the scalenus anticus and medius muscles with increased irritation to the plexus [18]. In these cases they found fibrositis of the offending muscle. They believed that the fibrositis led to spasm of the muscle elevating the first rib, which resulted in compression and stretching of the large nerve trunks. It was postulated a vicious circle was established which must be broken for relief of the discomfort and could be done so most practically by anterior scalenotomy. They stated that the brachial plexus and subclavian artery are pinched between the scalenus anticus and medius muscles during the phase of muscular spasm. Naffziger and Grant in 1938 supporting the previous theories of etiology and they presented the term - scalenus syndrome [19].

IV. CERVICOBRACHIAL SYNDROME

In 1940, in response to the Naffziger terminology Aynesworth says that there are a number of pathologic conditions besides the compression of the scalenus anterior muscle which may produce identical symptoms, the Naffziger terminology, therefore, is too limited [21]. He described 20 patients with “cervicobrachial syndrome” which was meant to be an all-inclusive term to include all causes of neurovascular compression in the supraclavicular region. At the same time knew correlations were made between causes and symptoms. Semmes and Murphey [22] discussed the simulation of scalenus anticus syndrome by a herniated sixth intervertebral disk and Jelsma, added trauma, who believed that was a precipitating factor in 29 of 115 cases of scalenus anticus syndrome in his report [20].

V. COSTOCLAVICULAR CMPRESSION SYNDROM

In 1943 Falconer and Weddell introduced the costoclavicular compression syndrome [23]. They reported 4 cases in which they demonstrated for the first time that postural changes without an anatomic defect could precipitate neurovascular symptoms in the upper extremity as a result of narrowing of the space between the clavicle and first rib by a backward and downward thrust of the shoulders. Telford and Moddershead in 1947 discussed the costoclavicular syndrome and suggested that obliteration of the radial pulse was due to positional changes that were distal to the clavicle and not related to costoclavicular syndrome [24]. These investigators described the sling that is created by the anterior and middle scalene muscle fusion and the next year described fibrous bands that run in the middle scalene muscle, bands from the cervical rib to the first rib, and the scalene minimus muscle as a frequent contribution to brachial plexus compression.

VI. THORACIC OUTLET SYNDROM

Until now is relative clear that the symptoms of the upper limb can be caused from many things and not by only one. The understanding of the shoulder girdle syndromes has evolved and the complexity of the causes needed to be arranged. At 1953 Lord attempts to gather all the passing evidence and to propose a guideline for treatment [25]. He says that symptoms and signs depend on which of the three important structures are involved, namely, the subclavian artery, the subclavian vein, and the brachial plexus. The mechanism of pressure and torsion may differ from patient to patient, and a careful analysis is necessary to evaluate whether the difficulty is due to scalenus anticus pressure, with or without a cervical rib, to the narrowing of the costoclavicular space, or, finally, to the hyperabduction syndrome. It was time for someone to look at the group of symptoms as an entirety with a spherical view and to assemble an acceptable syndrome. This was done in 1956 by Peet who described for the first time the term thoracic outlet syndrome [26]. He included all of the compression syndromes in one, giving a name in the anatomic region were compression is happening. This is the term that is used still today.

Since then a turn was made by researchers and writers to investigate the surgical anatomy of the thoracic outlet (or inlet by some writers [27]) anatomical area and to optimize the best treatment either this is conservative treatment or surgical. Big series of operations was done and until the present days the study of the TOS continues.

VII. OPERATIVE APPROACHES AND TECHNIQUES

From the beginning surgical therapy for TOS is based on decompression of the compressed structure. The evolution of the surgical treatment was always according to the trend of the medical diagnosis. Initially, surgical approaches to TOS were directed primarily at subclavian aneurysms that were associated with anomalous ribs. By 1916, Halstead described a collection of 716 cases of cervical rib with at least 27 subclavian aneurysms [28]. Later the understanding of the role of the scalenus muscles, conjunction with sectioning the
The thoracic outlet syndrome (TOS) is a condition that involves compression of the brachial plexus, subclavian artery, and subclavian vein as they pass through the thoracic outlet. The symptoms can be caused by anatomical anomalies in the thoracic outlet, including scalene muscle hypertrophy, cervical rib, or first rib anomalies. Various surgical procedures have been developed to relieve symptoms by decompressing the thoracic outlet, including scalenectomy, transaxillary and anterolateral thoracotomy, and Costotransversectomy. The choice of treatment depends on the individual patient's anatomy, symptoms, and the presence of other medical conditions. The end results of surgical treatment show that a substantial number of patients achieve marked improvement in symptoms, and the surgical outcomes are often long-lasting. However, some patients may still experience recurrence of symptoms.

REFERENCES


