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TOS pathophysiology and clinical features

F. M. Crotti, A. Carai, M. Carai, N. Grimoldi, E. Sgaramella, W. Sias, and **F. Tiberio**

Clinica Neurochirurgica dell'Università di Sassari, Sassari, Italy

Summary

The authors present 280 patients operated on for thoracic outlet syndrome (TOS). In a first group of patients anatomical variants were the striking findings. The underlying factor for TOS development is therefore a well defined structural condition and its pathogenetic mechanism is known to be a nerve fibre compression.

In a second group there was no specific salient finding but a postural deviation. The unique pathological features were adhesions of the brachial plexus to the scalenus muscle. Consequently its pathogenetic mechanism is generally recognized as nerve fibre distraction.

In all patients neurological, vascular and myofascial pain symptoms were observed before the operation. Neurological and vascular pain disappeared after surgery, while the myofascial pain remained.

The authors believe that especially in the second, larger group of patients enhancement of the pain-immobility-fibrosis loop is the central pathogenetic factor on which surgical therapy is successful, and that myofascial hemisyndrome – probably arising from a longstanding postural deviation – is not a TOS dependent symptom. In TOS, therefore, there is a pain loop that cannot be resolved by surgical therapy alone.

The connection between myofascial pain syndrome and TOS might explain the many controversial opinions regarding frequency, results and surgical possibilities of this lesion.

Keywords: Thoracic outlet syndrome; myofascial pain; brachial plexus entrapment; trigger point; surgery; posture.

Introduction

The authors present 280 patients operated on for thoracic outlet syndrome (TOS) at the Neurosurgical Department of the University of Milan from 1982 to 1990 and from 1995 to 2000, at the Neurosurgical Department of San Gerardo Hospital of Monza from 1990 to 1995, and at the Neurosurgical Department of the University of Sassari from 2000 to 2003.

Patients were divided into two distinct groups. Fifty-two belonged to a first group where the striking findings from the clinical, diagnostic and pathological points of view were elements described as anatomical variants (cervical ribs, transversal mega-apophysis, fibrous bands, scalenus minimus). The underlying factor for development of TOS is therefore a well defined structural condition, and its pathogenetic mechanism is known to be a neural compression.

The second group of 228 patients was characterized by the absence of diagnostic or intraoperative features mentioned above. The salient finding is not a specific one and could also be observed in the first group, i.e. a postural deviation: tilting or side sliding of the pelvic joint and rotation of the spine. In this group, the unique pathological features were adhesions of the brachial plexus to the scalenus muscle, its pathogenetic mechanism is therefore generally recognized as nerve fibre distraction.

In all 280 patients neurogenic, vascular and myofascial pain symptoms were observed before the operation. Neurogenic and vascular pain disappeared after surgery, while the myofascial pain remained.

The authors believe that especially in the second, larger group of patients, enhancement of the pain-immobility-fibrosis loop is the central pathogenetic factor on which surgical therapy is successful, and that myofascial hemisyndrome, probably arising from a longstanding postural deviation, is not TOS provocated but a TOS independent symptom. In TOS there exists therefore another pain loop that cannot be resolved with surgical therapy alone.

The connection between myofascial pain syndrome (MPS) and TOS may explain the controversial opinions about frequency, results and surgical possibilities.

Materials and methods

Two hundred and eighty patients are reported, 220 females and 60 males, between 27 and 78 years old. 184 patients had surgery on the right side, 96 on the left. 248 patients had follow-up, 196 of them over more than one year.

Pain and other symptoms

Pain is the leading factor in the clinical history of these patients. Complaints consist of lumbago, sciatica and tension headaches. It was always possible to recognize one side of the body to be more affected. Finally a unilateral brachial pain arose with sudden onset in 64 cases and gradual onset in 216 cases.

Other constantly reported symptoms were insomnia, unsteadiness of gait, dizziness without vertigo and visceral disorders grouped under the general heading of ''bowel irritation''. The irritative symptoms (paresthesia) and the neurological deficit occurred later.

In order to select the patients for surgery, we wanted to distinguish personal subjective symptoms (pain and paresthesia) and interpersonal objective symptoms (impairment of function, reduced activity, modification of habits, hand muscle atrophy, hypesthesia).

Pain

The pain described by the patients covered the full range of possibilities from throbbing to dull, burning and stabbing. In general patients complained about different types of pain with different patterns. Location of the pain varied greatly: hand, forearm, or hemithorax irradiating also to the controlateral hand, neck and head.

We distinguished three specific pain patterns from different origins:

1) ''Neurogenic pain'': found in all cases, irradiating to radicular dermatomes (in 252 cases C8–T1, in 28 cases C5–C6). This pain appeared in paresthetic areas and occurred like a parossystic spike (shooting pain).

2) ''Vascular pain'': was present in 216 patients (80%), rather varied in intensity, endurance and extension. It spreads over areas remote from the radicular dermatomes. Sometimes it is characterized by psychological enhancement (chest pain, angor). The area overlaps one or more vascular territories and the pain is always accompanied by physical changes like rubor, tumor, calor, or pallor to the fingers, to the whole hand, limb, neck, breast region up to the contralateral hand. Typical is the ''glove'' pattern. Vascular pain has a slow climbing up to a plateau that can persist for many hours. The characteristic attribute is described as ''throbbing''.

3) ''Myofascial pain'': was present in 252 patients (90%) and is described as tension ache or burning. It extends along the fascias and is commonly perceived on biceps, triceps, trapetius, scalenus and pectoralis muscles up to the muscles of the arm. Distribution of spontaneous (myofascial) pain was identical in all patients. It overlaps an equal pattern of tender points on the periosteous attachment or on the belly muscles. Tapping some of these, around the scapula, triggers the referred pain similarly when occurring spontaneously. The area of myofascial pain generally covered the upper quarter of the body, extending to the arm in segmental distribution. In the temporal pattern, the myofascial pain was represented by the baseline (''underground'' pain), sometimes with parossystic outbursts.

Paresthesia

Paresthesia was present in all patients day and night. In 10% of cases (28 patients) paresthesia was distributed on the radial side of the hand/forearm, while in 90% of cases (252 patients) it was found on the ulnar side.

Impairment of performance and change in habits

Conscious and unconscious avoidance of certain movements or postures, e.g. those involving the upper quarter of the body (washing windows, painting ceilings, static load bearing on the shoulder), was a common feature in all patients. It is difficult to set the borderline between avoiding pain dysfunction of movement and neurologic deficit.

Symptoms of lesion

Only in 20 of our patients impairment of performance was clearly due to motor deficit (interosseous muscle atrophy); in the other patients there was no evidence neither clinical nor electrophysiological of denervation, even with an important functional limitation. In 45 patients there was an almost insensate hand.

Clinical features

General clinical evaluation

Based on Viola's typology, our patients could be divided into: asthenic longitype: 28 cases (10%), sthenis normotype: 20 cases (7%), brachitype: 232 cases (83%), picnic (none).

All patients observed presented a postural habit with forward bending of the head-neck complex on the sagittal plane. 252 patients (90%) showed a lateral tilt of the pelvis toward the injured side on the frontal plane, and 28 patients (10%) toward the opposite. All patients presented an enhancement of the supraclavear cavities and a lateral flexion of the head toward the injured side. On the same side, a constellation of tender points marked the bone attachment of peculiar muscles (enthesytis). Above the levator scapulae and the trapezius, trigger points (TPs) for referred pain to shoulder/arm were found. A postural scoliosis C-type was observed in 224 patients (80%), in 140 patients (50%) homolateral, in 84 patients (30%) contralateral to the painful limb. In 56 patients (20%) we observed a postural scoliosis S-type.

In 192 cases (68%) homolateral, in 88 cases (32%) contralateral the tender temporo-mandibular joint was always included in this algic pattern. Coccigodinia was present in 140 patients (50%). An amplified cutaneous reactivity, as for example enhancement of the stria alba or stria rubra after gentle cutaneous stroke was always evident.

Local clinical evaluation

All patients had a postural lateral tilt of the head toward the injured side and Tinel's sign with irradiation along the ulnar or radial side of the limb by tapping on Erb's point.

Neurological evaluation

All patients presented hypesthesia with radicular distribution, in 8 patients in the C5-C6 region and in 62 patients in the C8–T1 region. Two patients presented a severe interosseous muscle atrophy.

Strumental evaluation

X-rays of the cervical spine showed bilateral cervical rib in 28 cases. EMG demonstrated interosseous muscle denervation in two cases. Doppler sonography was performed in 88 cases and was positive only in one, showing digital artery flow impairment. Angiography was performed in 28 cases and a slow-down of the flow of the subclavian vein was always demonstrated. An analogous finding was seen in 60 cases on enhancement-CT. In no other case did the strumental diagnosis confirm TOS diagnosis but in all cases was valuable for exclusion of other pathologies.

Surgical indication

All our patients with TOS diagnosis were referred to physiotherapeutic treatment for at least three months. Persisting symptoms

were an indication for interventional therapy. Patients had to have brachial pain for more than 6 months to be operated. Subjective symptoms should be severe enough to disturb life style, objective symptoms must comprise three cardinal signs: 1) dermatomeric hypesthesia, 2) positive supraclavear Tinel's sign, 3) positive brachial plexus tension test (Elvey's test).

Surgical findings and etiopathogenetic correlations

During our study we encountered a number of anatomical vagaries: 28 cervical ribs, 12 scalenus minimus, 4 strengthening of Sibson's fascia, 12 anomalies of the attachment of the scalenus medius, 8 neurovascular conflicts between the lower trunk and the arteria transversa profunda colli. However, these anatomical variations do not have the same pathogenetic weight. While we put emphasis on the cervical rib (28 cases) or on the arteria transversa profunda colli (8 cases) for plexus injury, we are not sure that the other observed anomalies play a role in the pathogenesis of TOS. In another 244 patients we did not have immediate evidence of any compressing or offending structure and therefore we should look for the shape of the attachment of the scalenus anterior muscle on the first rib and its angle of inclination. In 120 patients out of this group, scalenus anterior lies near or merges with the scalenus medius in the caudal attachment. In such a situation the interscalenic triangle becomes progressively thin up to a suspended eyelet from which the neurovascular plexus hangs up thus complicating the clinical pattern. In 200 patients out of 244, verticalisation of the first rib reduced the angle between the scalenus anterior and the first rib with pinching of the subclavian artery and of the C8–T1 trunk. The lower trunk is more at risk due to its location: there is the pulsating artery in front and the firm posterior pillar of the tunnel entry located behind; i.e. the edge of the bare first rib or the scalenus medius at its attachment. This latter feels very taut when probing with palpating finger. Therefore in 212 cases out of 244 we assumed that a neurovascular conflict was evident. When adding to these 212 patients the first 36, we would find that in 248 out of 280 patients (88%) compressing or pulsating forces played a role in the pathogenetic mechanism. In 32 patients no feature of compression was found at all. In all patients we found a fibrillar net, bridging the interscalene gap and strangling the neurovascular plexus inside. This tangle of fibrillar lacinia was visible both in micro and in eye vision. In all cases (100% of patients) it was possible to see for example how the trunks of the plexus were stretched by pulling the scalene muscles with forceps: this test provides evidence that distraction also plays an important role in the pathogenetic mechanism. Traction force accounted for plexus injury in all cases and may be considered the chief offender in those 32 patients without evidence of compression.

Results

All patients within few hours after awakening from the operation had complete remission of the neurogenic pain. Two hundred and eight patients with follow-up of more than 1 year were assessed. In 52 patients (24%), vascular pain disappeared suddenly and totally after awakening, In 164 patients (76%) pain endured but at a more reduced distribution, scarcer and more tolerable than before. Eventually, after 3–4 weeks, all patients had complete remission of the vascular pain. Myofascial pain was completely remitted after the operation in 20 patients (10%), dragged on for weeks or months in 88 patients (40%) and endured until 1 year in 108 patients (50%), however, easier to bear than before. In all patients paresthesia and hypesthesia resolved within a few days following surgery. Patients with muscular atrophy did not show EMG and ENG evidence of improvement but fist grip and precision grip were more effective. It must be stressed that in all patients daily performances and mood improved.

Discussion

The most interesting issue emerging from this clinical report was the astonishingly large lot of patients in which symptoms started acutely. The sudden onset could easily be connected with traffic accidents in 20 cases and a change of occupational activity in 12 cases. However, the precipitating factor couldn't be recognized in 32 cases. The second interesting point was the presence and persistence of the myofascial pain pattern which should be correlated with surgical features. We assume that the pathogenetic mechanism is doomed to be misunderstood if we look for a compressing offender only. We believe that compression and distraction alternate in hurting neural primary trunks and that both forces are effective to a higher or lower degree for producing TOS. In the group of patients with cervical rib, anomalous compressive bone surely is the chief offender, but in the other patients it is tethering of the plexus by myofascial adhesions. We cannot say if these mechanisms are activated or enhanced during the day by movement or at night in a typical disturbed sleep. The presence of synechia between the plexus and scalene muscles is a constant pathological feature in the surgical field. We propose two theses regarding their nature: either they are physiological answers to a chronic nerve compression (starting from the thickened epineurium and going toward the fascia or vice versa) or they are offspring of a paraphysiological reaction of the connective tissue and therefore evident everywhere in the surgical field, on the platisma and on the homogeneous planes. The basic pathology, according to this thesis, would be a kind of connectivitis. Prior to surgery we found in our patients a diashesic tendency to sympathetic overactivity (long-standing stria alba and stria rubra); this could explain the enhancement of the pain-immobility-fibrosis loop [3]. For long time upon operation, our TOS patients presented an unchanged pattern of myofascial pain. This

impressively resembles in type and localization of tender points the fibromyalgic patients; fibromyalgia syndrome (FS) is a chronic pain disorder characterized by diffuse musculoskeletal soreness, non restorative sleep, psychological disturbance. It is more appropriate with an array: fibrositis, myo-fibrositis, fasciomyositis primaria (FMP), indicating a kind of pararheumatic illness; its histopathologic findings are a matter of debate particularly regarding the relationship with sleep. The overlapping of TOS and FS symptoms becomes more striking through the history (sleep troubles and bowel irritation are present in both). We have indeed the impression that the enduring myofascial pain, poorly modified by brachial plexus release, shows that this component of pain should not be a secondary phenomenon but a primary one. We stress that myofascial pain is able to independently rule among different symptoms related to plexus dysfunction. If we consider only this feature of pain in TOS patients, we find a striking analogy with pain arising from a cryptic form of pathology of the muscle or from a postural long-standing deviation (myofascial pain syndrome, MPS). We believe that the latter is very common in a large group of population, but if this coincides with a specific, particular anatomic situation, like a climbing first rib or a large basis of scalenus anterior, finally TOS could arise. To test this theory, we sampled within a few months other 240 patients (mostly female) who came to our department for pain symptoms in limbs, neck and trunk. Their general complaints (acrodynia, pectoral pain, no dermatomeric type brachialgia, sleep disturbances, bowel irritation, musculo-tensive cefalea) reflected analogous symptoms as in TOS operated patients, but the trigger points and myofascial pain pattern focused on FS. All patients of this type, also with minimal neurological signs indicating an irritative plexus were recruitable. In practice we easily found a large group of FS or MPS patients more or less bordering TOS. The high frequency of FS or MPS versus low frequency of TOS induces the authors to believe that these are the ''via finalis communis'' of different situations and that TOS is only a collateral, striking part of this phenomenon, an evolution regarding few patients only. It remains to explain how a ubiquitous MPS or FS with bilateral symptoms evolves in unilateral, upper quarter affecting TOS. We emphasize that both MPS and FS have actually symptoms prevailing one side and vice versa, many clear-cut TOS present a bilateral glove pattern of pain and paresthesia. Just the contralateral glove pattern of paresthesia which features a ubiquitous sympathetic overflow remitted quickly after unilateral operation. X-Ray finding showed a bilateral cervical rib in 28 patients. Also in this small group of patients this anatomical variant could not explain the one sided appearance of symptoms. We could track down in a few TOS patients the onset of clinical history to a whiplash injury, to a forcibly abducted shoulder or to a change of occupational activity, where the vascular nerve plexus was probably affected by unilateral stretching of soft tissues. But in many patients we did not find precipitating factors like these. We believe that in those cases trivial stretch or movement on overloaded muscles (e.g. scalene) could activate a latent trigger point and lock some fibres in ''taut bands''. So the thoracic outlet got suddenly more narrow by contracture on the scalene. In our opinion, this mechanism accounts for an abrupt onset of clinical history. Later on the transient early entrapment can be perpetuated by mechanical stress due to structural inadequacy or to postural imbalance. We report that all TOS patients had a lateral tilt of the pelvis, and we noted a close relation between the lateral down-slip of the pelvis and the side of arising symptoms. 248 patients (90%) had a homolateral TOS, 32 patients (10%) a contralateral TOS. We know that a tilted pelvis without rotation of the spine is due to an anatomical inadequacy for any of the body structures of the lower extremity kinetic chain, (anatomical length-limb difference – ''anatomical'' LLD-), and that meanwhile a tilted pelvis with rotation of the spine is due to muscular imbalance (postural scoliosis, or ''functional'' LLD).

LLD (structural inadequacy): this heading fits to sizable anisome. We found LLD in 128 patients (48%). The LLD about 1 cm or less, coupled with C-type scoliosis, in 88 cases, the LLD more than 1 cm, coupled with S-type scoliosis, in 40 cases. The technique used in our patients was the standing anterio-posterior X-ray examination, knee extended. We know that the muscular tone is greater in the ''short leg'' because the stance phase there is shorter, the swing phase is longer and the momentum of heel impact is greater. Therefore LLD accounts for unilateral raising of the muscular tone. Furthermore tilted pelvis corresponds with tilted shoulder girdle axis and the neck muscles must instantly compensate to maintain the head upright and eyes at level which leads to chronic overloading. In other cases (148 patients) we had a postural scoliosis. The overloading on the neck muscles was due to the

same mechanism. But in these cases we did not know the cause for which the unilateral overtonus had begun. Anyway, arising muscular imbalance applies torsional forces to the spine and results in lateral tilt of the pelvis. Bruxism was reported in 124 patients (48%) on admission. All examined patients were submitted to a dental check and a malocclusion was found in all cases. We believe that the temporo-mandibular joint dysfunction (TMJD), a very ancient muscular tension triggering mechanism, is the most important factor in raising muscular tone and in driving it toward one side. Perhaps the cause of producing torsional forces has not been acting for long, but the body maintains the same posture (postural neglect) from which the muscular overtone in the upper quarter is perpetuated.

Conclusions

We believe that in TOS patients two pain loop mechanisms, one specific for TOS the other a-specific, are interconnected. In the specific loop the pain is neurological and vascular and is maintained by compressing bone, band, or tethering fibrosis on the surrounding vascular nerve plexus soft tissues. We cope with vascular or true neurological TOS by severing this loop and the surgical removal of the offending cause is very successful. In the a-specific loop the pain is myofascial and is maintained by postural neglect with unilateral overtone. Unreliable response to surgery. In most of cases, the myofascial pain starts alone and heralds a pathological entity very difficult to define (''Disputed'' Neurological TOS) – and which borders with MPS are very blurred. Initially this is no indication for surgery. The problem can be settled with a correct conservative treatment. Without or despite this, minimal adhesions by chronic entrapment progressively build up around the nerve structures. Vascular and neurological pain loop mechanisms are eventually coupled. At this point we have to deal with a true TOS and must add the surgical treatment to the therapeutical panoply. However, we must inform the patients that the same pain symptoms remain because the myofascial loop mechanism cannot be severed by surgery. Therefore TOS, in which surgical treatment is mandatory, often is regarded a failure in preventive medicine, an example of a not timely corrected situation. TOS should be considered a multidisciplinary pathology, not only falling into the neurosurgeon's competence but also of other specialists such as rheumatologist, physiatrist and the maxillo-facialis. Their support is indispensable in caring for these patients. TOS needs to be approached without hubris by surgeons: neurological and vascular symptoms remitted easily after the operation; conversely myofascial pain may demonstrate to be very difficult to be resolved. We think that a poor chronic posture triggers continuously muscular tension anywhere in the upper quadrant or in hemibody and so myofascial pain has a self-maintained mechanism untouchable by surgery. Therefore a physiatric approach is fundamental because there are problems in this pathology which surgery does not fit. Interventional therapy should be only a step in the treatment. From this point of view we suppose that lacking awareness of this issue accounts for old and actual controversies in epidemiology, diagnosis, treatment, results of TOS.

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Correspondence: Prof. Francesco Maria Crotti, Clinica Neurochirurgica dell'Università di Sassari, Piazza Università, 07100 Sassari, Italy. e-mail: franc.crotti@tiscali.it