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CLINICAL ARTICLE

Post-traumatic thoracic outlet syndrome

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Abstract
Background To evaluate the clinical presentation, diagnostic and therapeutic management and outcome of 27 cases of post-traumatic thoracic outlet syndrome (PT TOS).
Methods Retrospective chart analysis.
Results Nineteen women and eight men were included in this study. Involvement in a traffic accident was the most common scenario. Fracture of either the first rib or the clavicle was reported in eight patients at the time of injury; in others, the diagnosis was cervical or shoulder soft tissue trauma. Upon presentation at our clinic at a mean 41 months after injury, four patients had bilateral symptoms and 17 reported decreased function of either the arm or hand. Two patients presented with severe lower trunk deficits including one who had received surgical intervention at both the cervical spine and elbow before diagnosis of TOS was made. Sixteen and 15 patients were suffering from some degree of anxiety and/or depression. Upon diagnosis of neurogenic TOS, the two patients with atrophy of the hand musculature were treated surgically. Conservative treatment was applied to all other patients. Six months after presentation to our clinic, nine patients demonstrated a significant improvement. The remainder that reported incapacitating symptoms were offered surgical treatment. Three patients declined the latter. Fifteen patients received surgical treatment via an anterior supraclavicular approach with resection of the anterior scalene muscles. Eleven patients had resection of the middle scalene muscle while five had resection of an osseous structure (partial claviculectomy, C7 transverse process or a cervical rib). The two patients with atrophy of the hand only slightly improved their motor deficit but had a notable relief of symptoms of pain. Postoperative improvement occurred in 80% of surgically treated patients.
Conclusions The majority of patients suffering from a post-traumatic TOS present a neurogenic, usually subjective syndrome. Prompt therapeutic management is necessary, addressing both physical and psychological complaints. Most patients are cured or well improved by conservative and/or surgical treatment.

Keywords Brachial plexus · Nerve compression syndrome · Post-traumatic TOS · Scalenus muscle · Subclavian artery · Whiplash

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Introduction

Thoracic outlet syndrome (TOS) is defined by a set of symptoms related to compression/irritation of the brachial plexus and/or subclavian vessels in the cervical area. According to Wilbourn [71], TOS can be either venous, arterial, or neurogenic, depending on whether the conflict predominantly involves the subclavian vein, subclavian artery, or brachial plexus. Pure vascular TOS is rare, accounting for only about 10% of diagnosed syndromes. The vascular forms of TOS are generally accepted in all medical circles. On the other hand, neurogenic TOS, which is by far the most frequent, is more difficult to diagnose because there is no standard objective test to confirm clinical impressions. Most patients with TOS complain of pain in the arm and shoulder, often made worse by elevation of the arm (disputed neurogenic TOS of Wilbourn [71]); other common symptoms include arm and hand

paresthesias, tiredness, paleness, and numbness. Common signs on physical examination include weakness of adduction and abduction of the fifth finger and tenderness on thumb pressure over the supraclavicular area [60]. A subset of patients may have notable hand atrophy (true neurogenic TOS of Wilbourn [71]). Thus TOS remains a highly controversial subject. Lack of objective confirmatory tests leads some clinicians to doubt its existence. Its exact incidence is not known: it is said to be underdiagnosed by some [54] and overdiagnosed by others [72]; overall, TOS could explain around 10% of painful arm syndromes [33].

TOS usually presents as a spontaneous nerve entrapment syndrome; TOS can also develop following trauma to the neck or shoulder area [1–3, 7, 8, 11, 15, 16, 18, 24, 26, 37, 49, 54, 57, 58, 63]. The post-traumatic TOS is the source of much discussion and debate among clinicians and medical experts around the issues of diagnosis and optimal management.

The incidence of whiplash-related injuries has increased over the last 20 years [38, 42, 44, 69] in part due to the compulsory use of seatbelts in motor vehicles. Hyperextension injury of the neck can cause neck pain, either immediately or within 1 or 2 days; arm and hand symptoms develop in a small percentage of patients, rapidly or within a few weeks because of cervical disc herniation, uncinate arthritic changes, brachial plexus traction, or decompensation of the thoracic outlet area [27, 32, 45, 64, 66, 70]. In 1991, Schwartzman [59] who had seen over 2,000 whiplash patients, described the classical history of what he calls a *brachial plexus traction injury*: the patient involved in a car accident develops stiffness and pain in the neck that soon radiates to a C2 distribution as well as across the trapezius ridge and the arm. The patient is seen in an emergency room and told he has suffered from a cervical sprain after C-spine X-rays demonstrated negative. The patient is given a cervical collar and non-steroidal anti-inflammatory drugs. Most patients improve within 6 weeks to 3 months; others still suffering from neck and arm pain are restarted on different medications and will consult up to five or six physicians before the correct diagnosis of posttraumatic TOS (PT TOS) is made.

From 1996 to 2008, 498 patients were diagnosed as suffering from TOS in our clinics according to our computerized database; 358 were managed conservatively while 140 were operated on because of failure of medical treatment or the presence of neurological deficit (unpublished data). Among the 498 patients, 27 developed their symptoms following trauma to the neck or shoulder region. This subgroup of patients is here analyzed. The influence of both physical and psychosocial factors on the outcome following a whiplash-type of injury has been extensively studied [66]. Psychological, social, cultural, and professional elements interact with the musculo-tendinous condition; hence we have tried to analyze all the factors that could play a role in the outcome of a PT-TOS.

Methods

The diagnostic criteria of TOS were the presence of pain in the cervical/trapezius region and upper extremity symptoms and signs not explained by a cervical spine or peripheral nerve disease. The upper extremity symptoms were dermatomal or diffuse pain, numbness and tingling. Weakness complaints were either diffuse or specifically directed to fine hand movements. Symptoms were to be reproduced by the hands-up test and/or supraclavicular deep palpation. The syndrome was considered to be PT when it developed following a trauma to the neck or shoulder in a person without prior medical history of TOS, or shoulder or neck injury. In some cases, we observed a two-stage evolution with initial cervical/trapezius muscle pain and later upper extremity symptoms. A continuity of complaints had to be well documented to consider TOS as PT in those cases.

Data collected retrospectively in the charts included the age and sex of the patient, past medical history, past professional history, clinical presentation, results of diagnostic complementary examinations, conservative therapeutic measures and operative management, and the medical and professional outcome.

The second author, CL, a fourth-year student in physical therapy, thoroughly analyzed all the charts, sent a questionnaire to all the patients, and had telephone contact with most of them. General, physical, and psychological aspects were assessed with the Disability of Arm-Shoulder-Hand index (DASH), the Hospital Anxiety and Depression scale (HAD), and the Medical Outcome Study 36-Item Short Form Survey (SF-36).

DASH is an autoquestionnaire of 30 items developed in 1997 in order to evaluate the functionality of upper extremities [12]: 21 questions are related to difficulties in daily physical activities performances; five questions assess the degree of pain at rest or during activity, paresthesias, weakness, and stiffness; four questions analyze repercussions of the upper extremities limitations/symptoms on social life, professional life, sleep, and psychological condition. The level of difficulty of each activity is graded from 1 to 5. The total is converted into a 0–100 scale, where 0 means a normal upper extremity function and 100 means a maximal upper extremity impotence.

The HAD scale was developed in 1983 to evaluate the level of symptoms of anxiety and/or depression in a given pathology [65]. Each of the 14 items (seven concerning anxiety and seven concerning depression) is graded from 0 to 3. The final score for each of the two studied domains stands between 0 and 21. Anxiety or depression is either absent, doubtful, or present when the score is respectively below or equal to 7, between 8 and 10, or equal or above 11.

The SF-36 analyzes self perception of one's general health, physical and psychological health, and well-being. Statistical

analysis was performed using Student's *t* test to compare data between the surgical group and the medical group.

Summary of cases

Clinical presentation The first presentation at the neurosurgical consultation of the first author occurred at a mean post-injury time of 41 months (2.5 months to 9 years). The clinical data are summarized in Table 1. There were eight males and 19 females, ranging from 17 to 53 years old (mean: 36.4). The most common traumatic event was a traffic accident, occurring in 17 cases (63%). Nine accidents (33.3%) involved litigation concerning workers' compensation (n=7) or automobile liability (n=2). Twenty-three patients (85.2%) complained of neck or shoulder pain within minutes, hours, or 1 day. Seventeen (63%) sought medical attention in an emergency department or from their family doctor on the day or the following day. Eight patients (29.6%) had a clavicle or first rib fracture. A *diagnosis of cervical sprain* was made in 11 cases (42.3%). Eleven patients (42.3%) wore a rigid or soft cervical collar for a few days or weeks. Symptoms radiating down the arm developed within days (n=2, 7.4%), weeks (n=9, 33.3%), months (n=9, 33.3%), or years (n=7, 25.9%). Upper arm pain (n=20, 74.1%) was ill-defined in seven patients and localized at the ulnar side of the upper extremity in 13 patients. The symptoms were aggravated with use of the upper extremity in 19 (70.4%). Weakness of arm and hand was reported in nine patients (33.3%) while two patients (7.4%) presented with severe wasting of hand muscles. Frequent complaints were painful arm tingling (n=16, 59.3%), arm heaviness (n=8, 29.6%), arm numbness (n=8, 29.6%) at night, during repetitive or in elevation arm movements. On physical examination, nine patients (33.3%) were overweight. Breast hypertrophy was not present in any of the women. A drooping shoulder posture was noted in four patients (14.8%). Neck and trapezius muscles were tender on palpation in 21 patients (77.8%). Pressure on supraclavicular and scalene muscles was painful in 12 patients (44.4%) with pain radiating down the arm in three (11.1%). During the hands-up exercise test or Roos test (repetitive fist movements in the 90-degree abduction and external rotation shoulder position), 22 patients (81.5%) complained of arm pain (n=10, 37%), heaviness (n=10, 37%), numbness (n=4, 14.8%). A motor and/or sensory deficit was present in 17 patients (63%), mainly in the distribution of C8. A special case illustrating the occasional clinical evolution in two stages is shown in Fig. 1: the patient first complained of left little finger paresthesias a few months after a heavy ski fall on the left shoulder. She developed, years later, a rapidly progressive sensory and motor deficit of the left upper extremity.

Radiological studies The radiological findings are summarized in Table 2. Cervical spine X-rays (n=27, 100%) showed degenerative changes in four cases (14.8%); an hypertrophied C7 transverse process or a cervical rib was present in eight patients (29.6%). Eight patients had clavicle or first rib fracture (29.6%). CT scan (n=8, 29.6%) and/or MRI (n=22, 81.5%) of the cervical spine were entirely normal (n=23, 85.2%) or showed uni or multi-level degenerative changes (n=4, 14.8%) without signs of root compression. MRI of brachial plexus (n=4, 14.8%) were all normal. Significant flow reduction in the subclavian artery was seen in eight of the 22 patients (36.4%) studied by Doppler examination with provocative maneuvers.

Electrophysiological studies The majority of the patients (n=21, 77.8%) underwent electromyographic and nerve conduction studies. They included needle EMG examination and evaluation of motor and sensory conduction velocities of median and ulnar nerves. Medial antebrachial cutaneous nerve was not studied routinely. Electrodiagnostic tests were entirely normal in 11 patients (52.4%). A denervation pattern at the C8-T1 level was present in four cases (19%). Reduction in the amplitude of the medial antebrachial cutaneous

Table 1 Demographic/epidemiologic/clinical data of the study group (n=27)

Description	Number
Women/men	19/8
Mean age (range)	36.4 (17–53) years
Occupation at time of injury	
Teacher/secretary	8
Mason/workman/mechanic	8
Shop assistant	7
Nurse/home assistant	5
Unknown	1
Etiology of trauma	
Motor vehicle accident	17
Fall	5
Weight fell on shoulder/arm	2
Lifting injury	3
Mean interval trauma/arm symptoms (range)	9.5 months (3 days–3 years)
TOS side (patients)	Dominant: 12
	Non-dominant: 11
	Bilateral: 4
Complaints	
Neck/trapezius muscle pain	21
Upper arm pain	20 (13 radicular, 7 non-radicular)
Motor deficit	10
Sensory deficit	17
Medicolegal issue	9

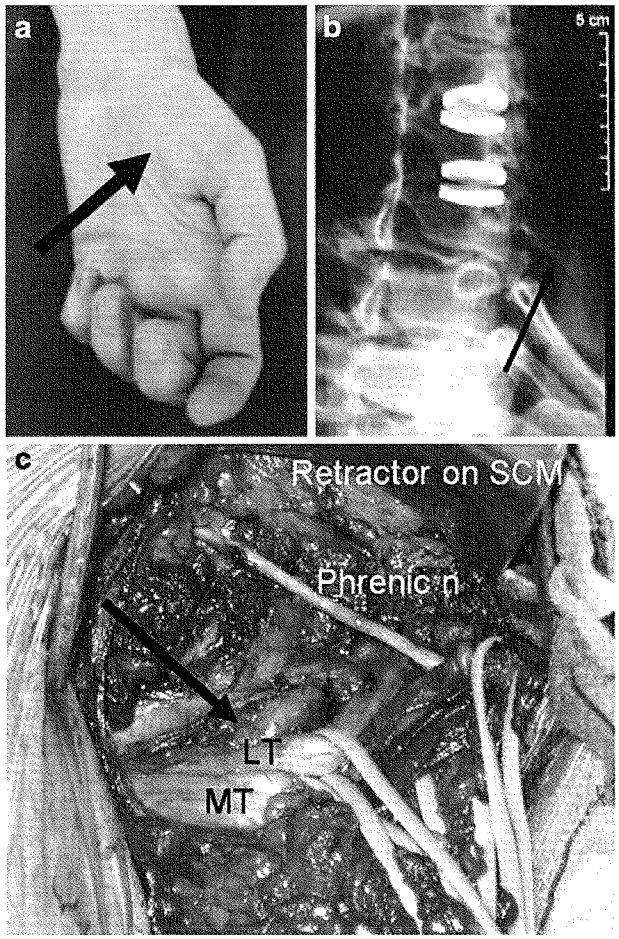

Fig. 1 Picture of the atrophic left hand (**a**), oblique C spine X-rays (**b**), and operative picture of brachial plexus decompression by anterior approach (**c**). The clinical history of this 48-year-old female patient illustrates the occasional evolution in two stages of PT-TOS (first disputed then true neurogenic TOS) and frequent misdiagnoses leading to poorly justified surgeries. (1) The patient first complained in 2001 of left fifth digit paresthesias following a ski fall on the left shoulder; she had a very large left periscapular hematoma. She progressively developed C8 left cervicobrachialgia; initial electrical evaluations were non-contributive. In retrospect, the patient was probably suffering at that time from disputed TOS. CT-scan of the cervical spine showed degenerative changes at C5–C6 and C6-C7. The patient underwent in 2006 double discectomy and disc prostheses placement. (2) The patient progressively developed in 2007 weakness and atrophy of left forearm and hand. Electrical studies showed motor changes involving the median nerve. Curiously, the patient was then operated on for ulnar nerve neurolysis at the elbow. The motor deficit continued to progress, leading to severe atrophy of interossei, hypothenar, and thenar muscles (**a**, *arrow*). Pain and paresthesia were still present at the ulnar side of the upper extremity. C-spine X-rays showed rudimentary cervical ribs (**b**, *arrow*). The patient was then sent to us by a neurologist who suspected brachial plexus compression. The patient had been out of work (secretary) for 18 months. The patient was operated on within days of presentation to us by the classical anterior supraclavicular approach: the C8 spinal nerve and lower trunk (*LT*) appeared severely compressed by the supernumerary osseous element and the large musculature inserting on it (**c**, *arrow*; *MT* middle trunk; the retractor on SCM medially retracts the sterno-cleidomastoid muscle). At 12-month follow-up, the patient was free of pain and her neurological status was stabilized. She had resumed her professional activity 2 months after the operation

Table 2 Radiological and electrophysiological data of the study group (n=27)

Description	Number
C-spine/shoulder X-rays	27
Clavicle or first rib fracture	8
Hypertrophied C7 transverse process	8
C-spine CT-scan and/or MRI	8–23
Normal or mild degenerative disc changes	27
Brachial plexus MRI	4
Normal	4
Subclavian vessels Doppler	22
Reduced flow on provocative maneuvers	8
Electroneuromyography	21
Normal	11
C8/T1 denervation signs	4
Reduced medial antebrachial cutaneous nerve sensory amplitude	2
Non-specific neurogenic signs	4

nerve was recorded in two patients (9.5%). The study showed non-specific radicular neurogenic signs in four patients (19%) and distal ulnar nerve entrapment in one patient (4.8%). No somatosensory evoked potentials were used.

Initial conservative treatment All patients were treated initially with analgesics, anti-inflammatory and muscle relaxant drugs; 11 (42.3%) wore a cervical collar for a period of a few days to months. When arm complaints developed, most patients received another course of the previous treatment associated to B vitamins, neurogenic pain medicine, and antidepressants. All patients were on sick leave for a period extending from 0.5 to 42 months.

Physical therapy assessment At time of presentation to us, the majority of the patients (n=25, 92.6%) were managed by a physical therapist well aware of the TOS pathology. General measures included weight reduction and proper breast support when needed, change in behavior predisposing to complaints and correction of provocative postures; medications such as muscle relaxants and tranquillizers or anti-inflammatory drugs were prescribed. The key point of the treatment was a *specific physical therapy program*, combining management with a physical therapist (10 to 100 sessions of physical therapy) and auto-exercises by the patient at home. The treatment also included relaxation techniques and exercises opening the costoclavicular pinch. Two patients (7.4%) were managed in a pain clinic center for a multidisciplinary treatment (medications, physical therapy, psychological support); no nerve block, scalene block, or Botox injection was used. Nine patients (33.3%) were improved or cured by medical treatment;

operative treatment was proposed to 18 patients (66.7%) and accepted by 15.

Post-medical treatment course (12=44.4% non-operated patients): at 6-month follow-up, nine of the 12 patients (75%) had a satisfactory, good, or excellent result; six (50%) returned to work. None of the three patients performing regular physical activity before PT TOS was able to resume it. The mean pre-treatment DASH score of 48 improved to 38. The percentage of positive patients dropped from 50% pre-treatment to 25% post-treatment at the HAD anxiety scale, and from 38 to 25% at the HAD depression scale. Following treatment, half of the items of quality of life (SF-36) returned within the range of the general population.

Surgical procedure Two patients were operated on for a *true neurogenic TOS* with hand atrophy and 13 for a *"disputed" neurogenic TOS*, at a mean time of 54 months post-injury. Two patients were operated on bilaterally. All patients were operated on by the anterior supraclavicular route. We used a horizontal skin incision one fingerbreadth above the clavicle from the clavicular head of sternomastoid to the anterior border of trapezius. We opened the platysma to reach the posterior triangle of the neck. The prescalenic fat pad was dissected and hinged laterally. Transverse cervical vessels were usually ligated and cut. The omohyoid muscle was moved down. The phrenic nerve was dissected and gently held away. The anterior scalene muscle was resected from its transverse processes attachments to the first rib; the medial part of the middle scalene muscle was resected in 11 cases, as well as any supernumerary muscle or fibrous band causing nerve compression. We also resected the cervical rib (n=1) or hypertrophied C7 transverse process (n=4) along with the bulky muscles attached to them. The clavicle was dissected at its superior and posterior borders. The costoclavicular space was grossly examined by palpation between the first rib and clavicle. When the clavicle had been fractured, it was dissected in order to place a sponge beneath it and to more carefully analyze the costoclavicular space. Part of the clavicular bone was sometimes drilled and part of the subclavius muscle was taken out. Histological examination of the scalene muscle (n=13) revealed fibrotic microscopic changes in only two cases (15.4%). The infraclavicular approach was not associated in any patient of this series.

Postoperative course No hematoma or infectious complication occurred. Transient hemidiaphragm paresis was seen in seven patients (41.2%). The follow-up ranged from 2 months to 10 years (mean: 37.5 months). Seven patients (46.7%) exhibited a good result with either complete relief or significant improvement in their preoperative pain. Five patients (33.3%) were partly improved while three patients (20%) were unchanged. No patient worsened. A long-term outcome (> 5 years) is available for 13 operated patients: ten (77%) obtained a satisfactory, good, or excellent result; eight (62%) returned to work; only one out of five was able to perform again his previous heavy physical activity. The operated patients improved their preoperative DASH score (68 to 49); the percentage of positive patients dropped from 67% preoperatively to 33% postoperatively for the HAD anxiety scale and from 67 to 44 % for the HAD depression scale. Quality of life (SF-36) scores stayed below the range of the general population.

Comparison between medically alone and surgically treated patients No difference was detected for age, sex, laterality, symptoms, and clinical signs, except for two patients with hand atrophy in the surgical group. A strong difference was apparent in the delay between trauma and the first consultation leading to TOS diagnosis: 4.5±5 years for operated patients versus 1.7±1.5 year for medically treated patients. A significant statistical difference exists between the two groups in the prior cervical and/or peripheral nerve surgeries (p=0.05): none of the conservatively treated patient had been operated on before TOS diagnosis and treatment, whereas three of the 15 operated TOS patients (20%) had been operated on for cervical disc prostheses and ulnar nerve neurolysis at elbow (one patient; see Fig. 1), ulnar nerve neurolysis at elbow two times (one patient) and shoulder calcifications (one patient). Doppler and ENMG studies were more often positive for surgical patients, but the difference did not reach statistical significance (p=0.18 and 0.1). Physical (DASH score) and psychological (HAD anxiety and depression scales) criteria were better pre-treatment in the medically treated group. The proportion of patients incapacitated more than 50% on the DASH scale was higher in the group of patients needing surgery (p=0.03). The mean DASH score before treatment significantly differed between the two groups (p=0.04). Following conservative and/or surgical treatment, no difference in the mean DASH score was no longer observed (p=0.48). Among the nine patients involved in a medicolegal procedure, seven were operated with marked, partial, or no improvement in five, one, and one patients, respectively; one of the conservatively treated patients had a good outcome and the other had a poor outcome.

Discussion

Incidence of post-traumatic thoracic outlet syndrome

The incidence of *whiplash injury* has increased over the last two decades [38, 42, 44, 69]. Most cases in the present series involved a high-energy trauma, often resulting from road accidents (63% of cases).

The incidence of TOS is difficult to establish as criteria for diagnosis are controversial [47]. Our diagnostic criteria of TOS were presence of pain in the cervical/trapezius region and upper extremity symptoms and signs not explained by a cervical spine or peripheral nerve disease, with symptom reproduction by the hands-up test and/or supraclavicular deep palpation. In 1978, Woods [75] reported the development of a TOS in 459 of 1,958 patients who sustained a *soft tissue cervical trauma*, which gives a ratio of 23%. At the same period, Capistrant [2], reporting 35 cases of TOS post-whiplash or cervical strain injuries, viewed TOS as the most common etiology for arm pain in cervical strain injuries. In Ide's [24] more recent publication, 45 of 119 (i.e., 37.8%) of patients injured in traffic accidents were diagnosed as having irritation of the brachial plexus; the criteria were persistent pain or paresthesia in the upper limb aggravated by some exercises, a positive Tinel's sign over the brachial plexus and reproduction of pain or paresthesia by maneuvers stressing the brachial plexus.

Pathophysiology

TOS usually develops as a *spontaneous nerve entrapment syndrome* under pathophysiological mechanisms similar to other such syndromes including combination of a predisposing narrow anatomy and external factors. The predisposing anatomy for TOS can be osseous or more often soft tissue variants; the "anomalies" at the thoracic outlet region are very frequent in the general population as shown in numerous studies [30, 34, 53]. These anatomical variations make the patient prone to develop brachial plexus entrapment under some circumstances. External factors leading to decompensation include obesity, breast hypertrophy, hypotonic shoulder musculature, psychological depression, and trauma to the neck or shoulder area.

Figure 2 illustrates the different physiopathological mechanisms discussed in the literature that can lead to a PT TOS. First, the brachial plexus can sustain a *mild direct traction injury* causing pain and sensory symptoms immediately noticed by the patient [24, 59]. In these cases, rapid radiological examinations are usually obtained in order to exclude root compression by a cervical herniated disc. Injury at a more distal, brachial plexus level, is suspected in front of normal C-spine MRI. This mechanism of mild BP traction injury, described by Ide et al. [24] and Schwartzman [59], was not present in any of our patients.

Secondly, TOS can develop following *clavicle, first rib, or C7 transverse process fracture*, as was the case in eight patients of our series [3, 35, 37]. Mulder [37] in 1973 reported seven patients developing different forms of TOS post-trauma to the clavicle, shoulder, first rib, or C-spine. According to Casbas et al. [3], TOS with acquired bone lesions, which they report in 13 patients, account for less than 5% of the overall TOS population. In that case, brachial plexus compromise is explained initially by hemorrhage and edema or direct compression from the bone fragments; in a later stage, bone callus can lead to progressive narrowing of the costoclavicular space.

Thirdly, TOS can arise from a brachial plexus entrapment in *fibrotic scarring tissue*: stretching of the *scalene muscles*, microhemorrhages, and ultimately muscle fibrosis lead to straightening and narrowing of the interscalenic space [1, 7, 8, 11, 15, 26, 49, 55, 75]. This physiopathological mechanism is the most commonly reported in the literature dealing with PT TOS, and yet it is the most controversial in the clinical and even more in the medico-legal settings. Crotti [7] believes that PT TOS is a secondary traumatic lesion under the same concept of the ulnar nerve entrapment after elbow fracture. As soon as 1935, Ochsner [41] reported six patients (all women) suffering from incapacitating arm and hand symptoms following trauma (mild trauma in three patients); the diagnosis of brachial plexus compression at the scalenic area was attained by a thorough physical examination, with no sophisticated diagnostic tests available at that time; four patients underwent surgery with scalene muscle resection. More recently, numerous studies have demonstrated histological modifications of the scalene muscles that were resected for PT TOS [55]. Histology of muscle specimens frequently demonstrate fibrosis and scarring in the anterior and medial scalene muscles, which fix the plexus and restrict its movement.

Fourthly, PT TOS can occur if a *predisposed thoracic outlet*, already narrowed usually by a cervical rib, decompensates because of sudden large forces sustained by the region during the injury [11, 33, 59]. Decompensation can be rapid and produce what is called a true neurogenic syndrome, as was the case in two patients of our series.

Lastly, some authors advocate that *shoulder/neck muscle spasm and pain*, and bad shoulder posture usually following a whiplash type of injury can lead to the development of a PT TOS [2, 7, 11, 26, 46]. Not all individuals develop a PT TOS following a car accident; the occurrence may be related in

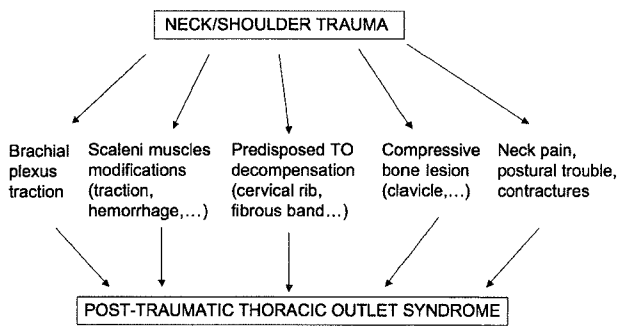

Fig. 2 Physiopathological mechanisms that led to development of a PT TOS in our patients. Multiple mechanisms can play a role in some patients

some cases to the anatomical conformation and pretraumatic posture and working habits. Prolonged antalgic contracture following a whiplash injury may lead to connective tissue changes and development of microadherences [51, 67].

Clinical presentation

According to the classification of Wilbourn [71], two patients in our series had a *true neurogenic* TOS, while 25 had a *disputed neurogenic* TOS; there was no case of venous or pure arterial syndrome. The true neurogenic TOS is defined by objective findings including hand atrophy, positive electrical studies (hopefully!), and presence of a convincing anatomical variant like a cervical rib. It is important to note that the two patients with true neurogenic TOS in our series first presented with a disputed TOS, defined by subjective complaints of pain and sensory changes. Obviously, unfamiliarity of the clinicians with the TOS led one of the patients to a very severe hand atrophy before brachial plexus decompression was achieved. This case, shown in Fig. 1, illustrates the fact that disputed and true neurogenic TOS can be two stages of the same disease, which is true for other nerve entrapment syndromes [21, 68]. This has been emphasized in the recently published article reporting our series of seven Gilliatt-Sumner hands [14]. Similarly, among the nine patients with severe hand atrophy reported by Gilliatt et al. [21] in 1970, seven first complained of pain and paresthesias.

The disputed neurogenic type of TOS is by far the most frequent form of both spontaneous and PT TOS [17, 26, 39]. The complaints can be vague, ill-systematized, or more often pain and sensory changes are present in a brachial plexus distribution, usually C8. Nocturnal pain and paresthesias are common. Motor deficit is seldom significant. Neurological disturbances are almost always slight: slight decrease of force in the hypothenar muscle group and interossei muscles, mild hypesthesia of the ulnar side of the hand, forearm, and occasionally arm. Schwartzman [59] emphasizes the physical examination. In our series, the common positive physical findings were trapezius pain on palpation, local pain and Tinel's sign on pressure in the supraclavicular area, arm heaviness and/or hand tingling at the hands-up test.

Complementary examinations

TOS, either spontaneous or PT, remains a *clinical diagnosis* and a *diagnosis of exclusion* [20, 22, 23, 25, 36, 48, 62]. Radiological and electrical work-up was entirely normal or showed mild degenerative C-Spine changes in most of our patients. Differential diagnoses include compressive syndromes, especially cervical radiculopathy and distal peripheral nerve entrapment, and non-compressive diseases at the cervical, brachial plexus, or more distal nerve level. C-spine MRI will exclude a disc disease; MRI can also demonstrate a disc herniation not explaining the patient's symptomatology, as was the case in nine of 48 MRI studied patients in Alexandre's [1] series. Woods [75] reported 15 patients who underwent cervical discectomy without improvement before the diagnosis and treatment of TOS were achieved. BP MRI was not routinely used in the present series, unlike the patients we currently care for. Indeed, some groups have reported that a carefully performed and interpreted MRI can show fibrous bands, anomalous muscles, and distortions of the plexus elements [5, 6, 9, 10, 43, 50]. In a post-traumatic context, one could hope for future demonstration of scalene muscle fibrotic changes by good-quality MRI. MR neurography has been reported to be more sensitive for detection of soft tissue abnormalities and abnormal course and signal of plexus elements in TOS.

Electrodiagnostic studies had a surprisingly low yield in our series, even for patients with motor and/or sensory findings on examination. Electrical study of the brachial plexus is limited because of its very proximal location [28, 31, 73]. Hence, electrodiagnosis of TOS often becomes feasible only once Wallerian degeneration has taken place in the brachial plexus. In some cases, amplitude reduction of the medial antebrachial cutaneous nerve, depending of C8 and T1 spinal nerves, will be recorded at an early stage, by a good electromyographer, as was the case in two patients in our series [61]. Alexandre [1] reports that a very accurate electrophysiological testing was positive in all cases of his 54-patient series.

Conservative treatment

All but two patients in our series were first managed conservatively under the supervision of a specialist in physical therapy. One-third improved sufficiently and medical treatment was pursued; patients must realize that the medical treatment must be continued for a long time, with a home exercise program [19, 29, 40, 52]. The same attitude is offered to patients with back problems, who are frequently advised to attend the "school of the back" lessons.

Surgical treatment

Two patients were operated on rapidly because of hand atrophy; the other patients were offered surgery because of incapacitating symptoms lasting for a minimum of 4 to 6 months.

Currently, the surgical approach preferred by many teams for TOS is the *anterior supraclavicular route* with anterior and partial middle scalene muscle resection as well as removal of any compressive osseous or soft tissue element. This is especially true for the PT TOS, since the scalene muscles often play a role in its pathophysiology, as mentioned earlier [56]. The anterior approach allows perfect visualization of the

brachial plexus from the spinal nerve to the division level. The costoclavicular space is roughly analyzed by finger palpation. Morbidity is minimal. A phrenic nerve paresis frequently occurred in the present series, which resolved spontaneously within a few weeks or months.

TOS, either spontaneous or PT, is most of the time a *soft tissue disease*. We never remove the first rib by the anterior approach because we think like other authors that this is not necessary in most cases in order to achieve a good result and that it may be hazardous for the lower trunk [4, 8, 49]. Our preferred approach for first rib resection is the posterior subscapular approach, which is in our hands the safest for the lower plexus elements [13]. We propose this much heavier exposure to the few patients with a huge cervical rib or to those still complaining of incapacitating symptoms after the anterior approach. No patient in the present series was operated on by the posterior approach. We very seldom combine the supraclavicular with an infraclavicular approach for TOS, be it spontaneous or PT. The compressive elements for the brachial plexus and/or subclavian artery are most of the time located in the interscalenic space.

Post-operative management by a physical therapist is of paramount importance, as is the long-term postural program by the patient him or herself [74].

Conclusions

TOS is not a rare entity. Trauma to the neck or shoulder area can lead to the development of the syndrome by direct or indirect physiopathological mechanisms among which the scalene musculature usually plays the major role. The clinical picture of PT TOS resembles that of spontaneous TOS. The delay between trauma and first arm complaints is usually short. The diagnosis of PT TOS should be raised when a patient following trauma complains of neck and arm pain associated with symptoms such as heaviness, numbness, or tingling not explained by a cervical spine pathology. Patients can *easily be misdiagnosed* if the possibility of brachial plexus compromise is not borne in mind. Unfamiliarity of clinicians with the diagnosis and reluctance of clinicians and medical experts to accept the diagnosis at an early stage can lead to chronic pain syndromes, hand atrophy, or unnecessary cervical spine or peripheral nerve operations. The therapeutic attitude does not differ whether TOS is spontaneous or posttraumatic: conservative treatment supervised by a physical therapist and sometimes management in a pain clinic is the first line of treatment. Operative BP decompression should be offered rapidly in case of neurological deficit and to the patient complaining of incapacitating persisting symptoms despite optimal conservative management. Anterior scalenectomy will help most patients with minimal risk. Similarly to the lumbar disc operated patient, the TOS-operated patient must actively participate in a program of exercises and posture reeducation that will lessen the risk of failures and recurrences. Earlier diagnosis of PT TOS leading to psychological and physical therapeutic management could be helpful to reach a better result and quality of life.

Conflict of interest None.

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