

THORACIC OUTLET SYNDROME DUE TO HYPEREXTENSION-HYPERFLEXION CERVICAL INJURY

Alexandre A.; Buric J.; Corò L.; Azuelos A.

OBJECTIVE:

To establish the brachial plexus entrapment due to thoracic outlet syndrome as a physical basis for the prolonged complaints of patients affected by the hyperextension-hyperflexion, non structural, cervical injury due to vehicular, slow-speed, rear-end accidents.

METHODS:

Analysis of 24 patients seen after a mean period of 11 months (2-48 months) post-injury. The patient population included those patients that previously to the accident did not suffer signs or symptoms of cervical and/or brachial plexus lesions.

RESULTS:

All patients in the study referred symptoms of pain, paresthesias and slight weakness possibly related to a brachial plexus entrapment. In fourteen patients there were also neurological signs showing brachial plexus entrapment. Electroneurophysiological, summary index, testing was positive for a brachial plexus involvement in all cases.

Seventeen patients were operated on. Sixteen had a neat improvement on a six month to one year follow-up. One patient did not improve. Seven patients refused surgery; of these, six patients had clinical worsening on the same follow-up period while one remained unchanged.

CONCLUSIONS:

All the patients whose clinical symptoms and signs do not reverse after some time post-injury should be investigated for a possible brachial plexus entrapment. Surgical procedure or, eventually, conservative measures, resolve the problem in the majority of cases.

MATERIAL AND METHODS

The patient population included those patients that have suffered mild car accidents prevalently in rear-end modality. Only those patients that reported no other injuries than cervical were included in the analysis. Concomitant head injury, back injury or arm injury were factors for exclusion of the patient from the analytic process. The data were taken from a careful patient examination as well as from emergency unit documentation.

Patients that have suffered neck pain and stiffness or had symptoms of brachial plexus entrapment previously to the injury, were excluded from the study as well as those patients whose MRI or/and CT scans revealed disc herniation at the level or of the size that could be responsible, even if partially, for the symptoms accused.

Patients with postural factors and anatomic anomalies seen on radiological investigation were also excluded from the study.

All patients included in the analytic process had had an X-ray of the cervical spine and possibly MRI or CT scan done as well as the electrodiagnostic testing by summary index for the upper extremities on both sides.

RESULTS

The time lap between the injury and the moment that the patients came to the authors attention was 11.4 months (min 2-max 48). There were 20 female and 4 male patients. The age range was between 19 and 57 years with an average of 36.5 years. There was no significant difference in age between the male and female group.

The brachial plexus entrapment was seen on the right side in 13 patients, on the left in 9 patients and in 2 patients the entrapment was seen bilaterally.

The superior trunk alone was affected in 5 patients, the inferior trunk in 3 patients while 8 patients had the involvement of all three trunks. Other combinations of trunk involvement were superior and middle in 4 patients and superior and inferior in 4 case. Middle trunk was never affected alone.

Clinical symptoms

All of the patients complained of neck pain and stiffness. Of these, 6 complained also of headache alone or in association with shoulder and/or arm pain. 6 patients referred shoulder pain associated with neck pain and/or headache. Arm pain was present in 15 patients. Most of the patients whose neurophysiological findings indicated the primary superior trunk involvement referred pain in the interscapular territory on the side of affection. Tingling in the arm and the hand was frequently reported and was accentuated during the sleep. Dizziness was present in just two patients while one had also a blurred vision.

Neurological findings

All of the patients showed tenderness and various grade of muscular stiffness in the supraclavicular region as well as reduced motion amplitude of the neck in all directions. The Adson and "signe du plateau" test was positive in all patients. In 17 patients there were signs of neurological disturbances while 5 patients were free of any of neurological sign to be correlated with a brachial plexus entrapment. When present, the neurological findings were almost always slight and depended largely on the brachial plexus trunk affected. If just the superior trunk was involved than the most common finding was the hypesthesia of the skin over the deltoid muscle and in the interscapular region and the force decrease of the supraspinatus muscle. When also the middle trunk was involved than the hypesthesia of the skin on the outer side of the forearm and a slight decrease of force in the triceps muscle and the extensor communis of the fingers was seen. When the inferior trunk was affected than the most common neurological finding was a slight decrease of the force in the hypothenar muscle group and interosseal muscles as well as mild hypesthesia of the inner two fingers and the inner side of the forearm and, occasionally, the arm. When more than one trunk was involved than there was a various degree of combination of the aforementioned signs.

Radiological findings

All patients had an X-ray of the cervical spine done immediately after the injury. 8 patients presented a straightening of the cervical vertebral column. No patient had an inversion of the curve, while 16 patients had a normal X-ray exam. MRI and/or CT scanning was performed in 22 patients. In 16 patients the scanning was negative for disc disease and/or other traumatic or degenerative problems that could explain the symptomatology. In 6 patients a disc herniation was found, however, it did not correspond to the level of radicular symptoms or could not explain completely the radicular symptoms referred by the patient. Two patients did not perform CT or MRI imaging exams as their neurological signs clearly indicated the presence of

a brachial plexus entrapment.

Neurophysiological findings

The electrodiagnostic testing was performed by using the summary index test proposed by Robinson et al. that evaluates the summary results of nerve conduction studies and not the single tests.

The electrophysiological tests performed were the F wave from median and ulnar nerves, SSEP (N9) from median and ulnar nerves, motor and sensory nerve conduction studies from median, ulnar and medial antebrachial cutaneous nerves and electromyography.

The summary indexing of these parameters gave in all the patients the positivity for TOS.

A control study (submitted for revision) was done with patients affected by true neurological TOS and with normal control group. The results showed no difference between the true and whiplash group while the control group of patients shown normal parameters.

TREATMENT

Before coming to our attention, all of the patients had already done some conservative treatment addressed to the cervical spine. The majority of patients were treated by cervical dressing for a period from 1 week to 1 month and various modalities of physical therapy, postural corrections as well as analgesic and muscular relaxation drugs. Moreover, much of them were submitted to laser and ultrasound treatment of cervical paravertebral musculature. None of these treatment modalities brought to any significant, time lasting improvement.

Consequently, the surgery was proposed to all patients. 7 patients have refused surgery while 15 patients were operated on. The surgery performed was a supraclavicular approach with neurolysis of the offended nerve trunks. Occasionally, scalenotomy or other soft tissue removal was performed when found to be interfering with the nerve trunks. The intraoperative finding was that of a moderate to dense scar tissue surrounding completely the offended nerve trunks at the point of their exit from the posterior scalene space. When the superior trunk was involved, the scar tissue frequently incorporated also the origin of the suprascapular nerve. This fact explains possibly the pain in the interscapular territory. The middle trunk was less frequently involved and when it was, the scarring was less prominent. The inferior trunk was frequently in touch with the subclavian artery as the scar tissue made a sheet around both structures incorporating them together. This probably engendered a neurovascular conflict.

FOLLOW-UP

Of the 7 patients that were not operated on, 6 worsened on a six month to one year follow-up, while one patient remained unchanged. The worsening generally involved decrease in muscular force, increasing of pain and tingling in the radicular territory and arousing of unspecific disturbances as mental confusion, dizziness and, occasionally, face pain. The one patient that remained unchanged had initially just slight symptoms that remained as they were.

In the operated group, 16 patients on the six month to one year follow-up showed a neat improvement. Those operated earlier did better than those that had a shorter follow-up. This probably indicates that there is need for time for the nerve trunk to regain its normal function. The improvement primarily interested the symptoms and dysfunction signs of the neck and more proximal segments of the upper extremity while the distal parts tended to improve later. The two patients that referred dizziness preoperatively accused no more of this problem after the surgery although we have no idea as to why of this. The one patient that remained unchanged after the surgery had a repeated electromyography at three months after the surgery that showed no improvement in respect to the preoperative examination.

PATHOPHYSIOLOGICAL MECHANISM

When the car gets hit from behind, the body, supported by the seatback and seatbelt, tends to accelerate while the head, not supported, remains still. This makes that the head actually, runs backwards and the neck becomes hyperextended (4).

The headrest limits excessive movements and so prevents the structural damage to the column but it permits enough movement as to induce the stretching of the neck muscles and brachial plexus trunks.

The suspected pathophysiological mechanism is based on the fact that at the moment of the traumatic event two different, yet interrelated, events take place:

- 1) Sudden and forceful anterior neck muscles contraction due to their sudden and intense stretching
- 2) Brachial plexus trunks lengthening as to accommodate for the neck movement

In this way the sliding capacity of the nerve trunks inside the scalene space becomes limited due to muscular contraction and, consequently, two different nerve injury types develop:

- 1) A compression injury due to forceful muscular contraction (continued contraction)
- 2) A traction injury due to nerve trunks limited mobility at the compression site.

Moreover, it is well known that an intense and sudden muscular contraction leaves the muscles in a state of a prolonged contracture with the development of myofascial syndrome and trigger points (6,11). These present clinically with stiffness, pain and, on palpation, the presence of hard muscular strings. This post-traumatic, time-lasting contraction may eventually play a secondary role for a prolonged compression of the nerve trunks (3).

The previously described events possibly induce a chronic compression of nerve trunks that interferes with the adequate blood supply of the same. This initially, induces changes in the small vessels of the endo and perineurium and consequently, changes in permeability of perineurium with swelling and oedema formation (5). As a consequence, there is the formation of perineurial and endoneurial fibrosis and, if enough long lasting, thinning of myelin sheath (1,9). The hypothesis is also that the ischemia primarily affects the orthograde and retrograde axonal transport that is very susceptible to oxygen deprivation and whose inadequate function interferes with adequate nervous conduction (1). The process at this point, becomes a vicious circle and, if not interrupted, deteriorates even if the muscles relax (1,7). During the surgery the Authors did, in fact, found the presence of the fibrotic scar tissue enveloping the nerve trunks and the site of greatest scarring was at the level of the lateral border of the anterior scalene muscle. This is in conformity with the statement that the maximal swelling and oedema formation is situated at the edges of the compression site (9).

Depending on the myelin and/or axonal derangement, the clinical expression of entrapment may present as pain and paresthetic disturbance or it may, at higher grades of axonal derangement, induce muscle wasting and fibrillation potentials in the wasted muscles (2,8,10). This pathological sequence of entrapment is well known for the surgeons of the peripheral nervous systems and was described already in 1964 by Weisl and Osborne (12). The process of entrapment is a progressive one and more time passes from the injury and beginning of entrapment more risk there is that it will reach a point where the changes become irreversible (1).

Why just some of the patients present the brachial plexus entrapment after this kind of injury is another important question. As the Authors see it, it may probably be related to the anatomical conformation and pre-traumatic posture and working habits. The brachial plexus, probably, becomes injured just in those patients that have some kind of anatomical variation and/or have daily working and posture attitudes with a faint equilibrium of the nerve trunks in the thoracic outlet and in whom the cervical injury is just what is needed to disrupt this homeostatic equilibrium.

Because all of the patients that we have seen have reached us at a late stage of their disease, there was little possibility as to cure them by conservative treatment, so we had to proceed with a surgical treatment that brought to a neat improvement in all except one case that remained unchanged. Nonetheless, we suppose that, if recognized in time, a conservative approach by postural training, revascularization, appropriate drug application and other physiotherapeutic and pharmacological measures addressed to brachial plexus treatment, could eventually resolve this problem in much of the patients.

The surgical treatment to be performed in these cases is a small supraclavicular incision and the smallest possible exposure of the nerve trunks. Once exposed, frequently is just the external neurolysis what one needs to perform. Big exposures are not indicated because of increased scar formation that might compromise the result of the surgery. Anyway, the surgical performance (neurolysis, scalenotomy, Sibson's ligament resection ecc..) is highly variable from patient to patient and must be suited as to perform just the minimally indispensable surgical act. The Authors are firmly convinced that, in these cases of TOS, there is no need for bone removal (clavicle or rib) and that the large exposure and bone removal may compromise the long-term results.

BIBLIOGRAPHY

Birch R, Bonney G, Wynn Parry CB: Traumatic lesions of the brachial plexus, in: Surgical disorders of the peripheral nerves. Churchill livingston eds, pp245-291, 1998.

Dahlin LB, Rydevik B, McLean WG: Changes in fast axonal transport during experimental nerve compression at low pressures. *Experimental Neurology* 84: 29-36, 1984

Frymoyer JW: Cervical acceleration injuries: diagnosis, treatment and long term outcome, in: The adult spine, 2° ed. Lippincott-Raven, vol 1, pp1235-1243, 1997

Larson SJ: Hyperextension, hyperflexion, and torsion injuries to the spine, in: Youmans JR – Neurological Surgery, 3th ed WB Saunders Company, vol 4, pp2392-2402, 1990

Macnab I: Acceleration extension injuries of the cervical spine. In Rothman RH and Simeone FA, eds: The Spine. Philadelphia, W.B.Saunders Co, pp 647-660,1982

Maigne R: Diagnostic et traitement des douleurs communes d'origine rachidienne. Une nouvelle approche. Expansion Scientifique Francaise, 1989.

Rydevik B, Lundborg G, Bagge U: Effects of graded compression on intraneural blood flow. *Journal of Hand Surgery* 6:3-12, 1981

Rydevik B, McLean WG, Sjostrand J,: Blockage of axonal transport induced by acute graded compression of the rabbit vagus nerve. *Journal of Neurology, Neurosurgery and Psychiatry* 43:690-698, 1980

Rydevik B, Lundborg G: Permeability of intraneural microvessels and perineurium following acute, graded experimental nerve compression. *Scand J Plast Reconstr Surg* 11:179-187, 1977

Thomas PK, Fullerton PM: Nerve fibre size in the carpal tunnel syndrome. *Journal of neurology, Neurosurgery and Psichyaty* 26:520-527, 1963

Travell JG, Simson DG: Myofascial Pain and Dysfunction. The Trigger Points Manual. Vol 1, Williams & Wilkins , Baltimora, 1983

Weisl H, Osborne GV: The pathological changes in rats' nerves subject to moderate compression. *Journal of Bone and Joint Surgery* 45B:297-306,1964