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Post-traumatic thoracic outlet syndrome (TOS)

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Summary

TOS is a compressive non-tumorous syndrome of the brachial plexus. It is possible, however, to consider as TOS the irritative and lesional plexus syndrome following trauma as long as compression (or traction) on the nerves is triggered by long-lasting pathological changes of the area after trauma. Overload work of judges and lawyers after traffic accidents does not help to remind the real victim's problem, that is stretching of the neck soft tissues during head acceleration-extension. This movement is due to a forward acceleration. Both the car and the victim's trunk are violently pushed forward while the head does not move fast enough so that it is actually pushed backwards. The mandibula is even slower than the head and this leads to an opening of the mouth with possible temporomandibular joint (TMJ) dislocation. If there is nothing stopping the neck extension, like an appropriate headrest, the momentum is only resisted by cervical soft tissue stretching. Prolonged antalgic contracture and motor neglect may contribute to connective tissue changes and development of microadherences. Final result is fibrosis of paraneurium. The pain-immobility-fibrosis loop is of basic importance in the development of this syndrome.

Keywords: Post-traumatic TOS; whiplash injury; temporomandibular joint dysfunction; paraneurium; entrapment.

Introduction

TOS is a compressive non-tumorous syndrome of the brachial plexus. In the sense proper it does not refer to traumatic damage of the plexus, therefore a direct injury on the posterior triangle of the neck with subsequent immediate deficit cannot be considered as TOS. It is possible, however, to describe TOS as irritative and lesional plexus syndrome following a trauma as long as compression (or traction) on the nerves is triggered by long-lasting pathological changes of the area after trauma. TOS can therefore be regarded not as a primary traumatic lesion but as a secondary one, and the onset of its clinical features can variably be delayed. As matter of fact, from a legal point of view, a post-traumatic TOS can begin after an asymptomatic period. The same concept has been accepted for ulnar nerve after elbow fractures. Palsy can arise immediately, early and late. In all cases the canalicular syndrome is considered a post-traumatic neuritis (Mumenthaler).

Discussion

If we consider the thoracic outlet as a canal, we properly distinguish in it inlet, walls and outlet. The interscalenic triangle is the inlet and the anterior and middle scalene muscles form the walls of the tunnel, the space between the clavicula and the first rib is the outlet. The neuro-vascular bundle runs inside the canal. This latter is made of soft and hard tissues. The outlet of the canal is entirely made of hard tissues. Among post-traumatic TOS we may categorize those due to compression at the outlet level, pathophysiologically quite different from the neurological ones, and those due to entrapment by the walls, very similar to neurological postural TOS.

Costo-clavicular space

There is usually no compression at this level in spontaneous syndromes (walls of the canal are usually involved in neurological TOS) but in cases of traumatic fracture of the clavicula the size of canal outlet can be reduced. A compression of plexus with related clinical features may follows in variable time. Clinical features of an enduring compression can be overshadowed by trauma sequelae and surgical indication is based on radiological findings only. Especially in

war lesions we must keep in mind the possibility of a post-traumatic fake of subclavian or axillary artery aneurysm. In this case of a space occupying lesion in an inextensible space, plexus compression symptoms appear at very short time.

Inlet and walls of the canal

Different from hard tissue outlet, soft tissue changes of walls during trauma are extremely frequent. In particular, soft tissues (scalene muscles) are involved in the acceleration-extension lesions of the cervical spine.

This pathology was first described (in the forties) in aviators during catapult assisted take-off from ships. These pilots presented with chronic cervical pain so severe that they were forced to retire from service. It is reported that some of them lost consciousness after take-off and crashed. The problem was solved by providing them with a headrest. In the fifties this pathology was observed again with the growing diffusion of cars. The aforesaid trauma is in fact very common because it is involved in a big number (20%) of traffic accidents. Acceleration-extension injury of cervical spine, as definition, has long been neglected by the more fashionable whiplash injury. The latter was more propagated owing to its truculent name but it is based on a wrong concept (as if the medulla was whipped by spine ligaments). Protean features of this syndrome arise from troubled peripheral receptors. Acoustic, vestibular and visual systems are involved. Complaints of the victims caused an increasing importance of this syndrome more in legal aspects than in scientific ones. Overload work of judges and lawyers after traffic accidents does not help to remind the real victim's problem, that is stretching of the neck soft tissues during head acceleration-extension. This movement is due to a forward acceleration. Both the car and the victim's trunk are violently pushed forward while the head does not move fast enough so that it is actually pushed backwards. Moreover the mandibula is even slower than the head and this results in an opening of the mouth with possible temporomandibular joint (TMJ) dislocation. If there is nothing to stop neck extension, such as an appropriate headrest, the momentum is only resisted by stretching of the cervical soft tissues. The reduction of the anterior-posterior length of the car during a crash is linked to an increase in height. The victim is pushed not only backward but also vertically. Very often, at the moment of thrust from behind the head is above the headrest, which so becomes useless. The situation may be worsened through the car seats' being pushed forward. Applied force is only one of the elements causing damage in a crash. Some other elements are: weight of the car, surface of the road (much worse if slippery), etc. Stretched among the neck soft tissues during an extension injury are the scalene muscles. However, it would not be correct to consider the scalene muscles as the only ones involved. All anterior muscles of the neck may be stretched and damaged. They may be found ruptured and bloodied after serious injuries. In case of less serious lesions they appear microscopically damaged but we don't have pathologic-anatomic reports. In a "whiplash injury" commonly shoulder, neck and upper limb pain is referred together with hand paresthesia, especially at the 2 last fingers. Obviously it is not due to root compression because vertebral foramens increase rather than decrease their size during extension of the cervical spine. All the above symptoms derive from entrapment of nerve trunks by contracted scalene muscles. The lesion is considered as TOS when this transitory situation turns into a permanent one. Many of our TOS patients experienced a traumatic onset. The temporomandibular joint dysfunction can also be the perpetuating factor. In case of a unilateral syndrome, it should be noticed that TMJ dysfunction is often asymmetric after a traumatic event in which the cervical spine has asymmetrically been extended. This can be attributed to the standing rotation of the spine at impact. TOS clinical features do not necessarily start immediately after the traumatic event. Often a long period follows during which different symptoms are evident (dysphagia, vertigo). It is possible also, to have a completely asymptomatic period before the onset of TOS. Complex post-traumatic TOS patients are different from spontaneous TOS patients. Even if most of them are females, leptosomic ones are more often involved. It is obvious that muscular strength is pivotal in resisting cervical spine extension. "Bull necks" are coping more easily than "swan necks" with this type of trauma. Apart from the different pathogenesis, psycho-physical background may be the same in both postural and post-traumatic TOS. Prolonged antalgic contracture and motor neglect may contribute to connective tissue changes and development of microadherences. Final result is fibrosis of paraneurium. As a consequence the nerve loses its gliding property. The pain-immobility-fybrosis loop is of basic importance in the development of this syndrome.

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