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(CASE REPORT)

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Schneider syndrome (post-traumatic Central cord syndrome) associated with trigeminal spinal nucleus damage: A case report

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Abstract

Post-traumatic Central cord syndrome (Schneider's syndrome) is an intra-medullary contusion arising in the course of cervical trauma, and was first described by Schneider in 1954. It is characterized by motor impairment predominantly in the upper limbs, with a motor ASIA score disparity of at least 10 points between upper and lower limbs, and variable sensory and bladder dysfunction. We report the case of a patient treated in our department who presented with Schneider's syndrome following a high-kinetic trauma in a degenerative spine context, with the particularity of a tactile and thermo-algesic sensory deficit of the left part of the face, as well as its functional evolution.

Keywords: Schneider syndrome; Central cord syndrome; Tetraplegia; Trigeminal; Bladder-sphincter disorders; ASIA score

1. Introduction

Post-traumatic central cord syndrome (Schneider syndrome) is a form of intra-medullary contusion that occurs after cervical trauma and gives rise to clinical symptoms of varying severity, the most severe of which is tetraplegia, which may or may not be associated with impaired respiratory function [1,2]. It was first described by Schneider in 1954 [3]. It is characterized by motor impairment predominantly in the upper limbs, with a disparity of at least 10 points in the motor ASIA score between the upper and lower limbs, and variable sensory and bladder-sphincter impairment [4, 5]. It occurs most often in elderly subjects in the context of degenerative cervical myelopathy, although it may occur in young subjects following cervical spine trauma [2, 6].

2. Observation

Mr. A.A, aged 45, married with 2 children, previously autonomous, lives on the 3rd floor of a building without a lift, and has no particular pathological history. He was the victim of an assault by a wooden bat with a posterior cervical point of impact, with a brief initial loss of consciousness, after which he presented with tetraplegia with a deficit predominantly in the upper limbs. A cervical MRI performed in the emergency department revealed a compression fracture of the C3-C4 vertebrae, with a posterior wall recession making an impression on the anterior epidural space, with T2 spinal cord hyper intensity opposite. He underwent surgery on day 2 of the trauma to install an anterior plate and 4 screws, and was then referred to the Physical Medicine and Rehabilitation Department for rehabilitation.

The clinical examination on admission revealed a conscious patient with good orientation in time and space, with a motor deficit in all 4 limbs, predominantly in the upper limbs, with a motor ASIA score in the upper limbs of 27/50, compared with 38/50 in the lower limbs, and an ASIA D. Sensory examination revealed tactile and thermo-algesic

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hypoesthesia with a C2 sensory level, associated with tactile and thermo-algesic hypoesthesia of the left part of the face without corneal anesthesia or masticatory muscle deficit. The patient described poorly systematized neuropathic pain in the left upper limb, with numbness, electrical discharges and tingling (DN4 score: 3). Deep tendon reflexes were strong in all 4 limbs, Hoffman's sign was positive in the upper limbs and Babinski's sign was positive in both lower limbs, with no associated bladder-sphincter or anorectal disorders.

In terms of function, the patient was able to transfer to and from bed independently, feed and dress himself with assistance, and walk with a technical aid.

The MIF functional independence score on admission was 93/126, the SF12 short form survey (physical score: 30.3%, mental score: 46.11%), the ECM motor capacity scale in tetraplegics at 210: the walking index for spinal cord injury WISC was 15, and the motor capacity of upper limbs in tetraplegics (MCS) was: 164/224

The evolution was marked by an improvement in muscle testing in all 4 limbs. The patient still had a deficit in abduction of the left shoulder, muscle atrophy of the deltoid region and of the left arm, prompting an ENMG of the upper limbs, which revealed associated C5-C6 radicular damage. Spasticity appeared in all 4 limbs, with a global score of 2 on the modified Ashworth scale. An ENMG of the face was performed in the face of hypoesthesia of the left part of the face, showing abolition of the R2' response on the left and the ipsilateral R2 response on the right, pointing to post-traumatic damage to the ascending branch of the spinal contingent of the trigeminal nerve.

Spinal cord MRI revealed an extensive intramedullary lesion opposite C2-C3-C4 measuring 4x3.8mm in T1 hypointensity, T2 hyperintensity, unenhanced after injection of Gadolinium, indicating spinal cord injury (Figure 2), associated with signs of degenerative cervical myelopathy. Cerebral MRI showed no abnormalities.

An urodynamic assessment was carried out during his hospitalization, showing: a bladder of normal capacity, normal bladder compliance, with no detrusor hyper-activity. Uroflowmetry showed a bell-shaped curve, with no significant post-micturition residue and a maximum flow rate of 29ml/s, but revealed detrusor-sphincter dyssynergia (DSD). (Figure 1)

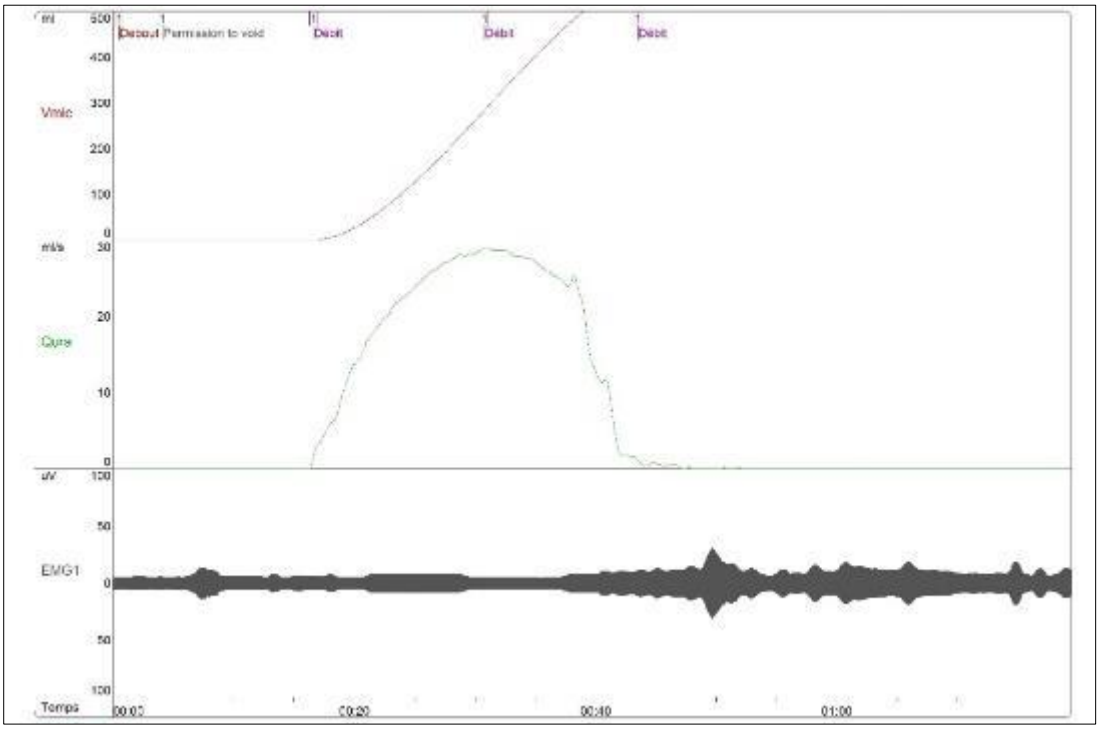

Figure 1 Flow measurement showing a roughly bell-shaped curve, a maximum flow rate of 29ml/s, micturition time: 36s, detrusor-sphincter dyssynergeia.

After 12 weeks in hospital, the patient was able to walk without technical aids, with a motor ASIA score of 38/50 for the upper limbs and 46/50 for the lower limbs. He recovered in terms of fine and global gripping, with a MCS score that

rose to 215 (vs. 164), the WISC score to 20 (vs. 15), the ECM to 232, the SF 12 (physical score 32%, mental score 56%) and the MIF to 106 (vs. 93).

3. Discussion

Post-traumatic central cord syndrome has been described for many years. According to studies, a 10-point difference in the ASIA motor score between the upper and lower limbs is necessary to confirm the diagnosis [4,7]. The associated sensory deficits are variable, ranging from purely subjective isolated disorders to disorders of thermo-algesic and epicritic sensitivity below the level of the lesion. The central cord syndrome lesion interrupts the spinothalamic tract pathways, which decompose into the spinal cord. There is therefore a suspended thermo-algesic sensory deficit which respects proprioceptive sensitivity [6,8,9]. Detrusor-sphincter disorders (DSD) result from reflex function of the medulla underlying the lesion, as part of the sub-lesion syndrome, freed from supramedullary inhibitory control. When spinal cord injury is incomplete, the neurourological picture may be less obvious, and neurological examination is not sufficient to predict bladder behavior. Urodynamic assessment is the only test that can identify TVS in these patients.

The age distribution of Schneider's syndrome is bimodal [2], with a first peak in young patients suffering from high-velocity trauma, and a second peak in elderly patients suffering from low-energy trauma to a degenerative spine [1]. In our patient, we found a combination of both situations: a high-velocity trauma in a context of degenerative cervical myelopathy (Figure 2).

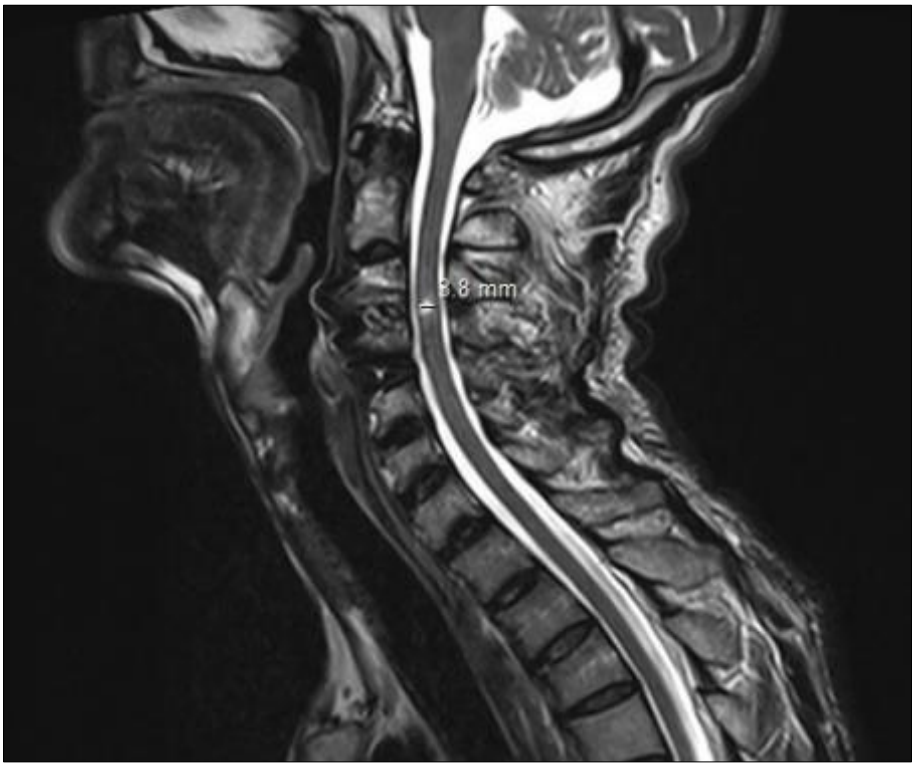

Figure 2 Spinal cord MRI showing a centromedullary hyperintensity opposite C3 to C4 measuring 4x3.8mm in relation to spinal cord injury, associated with an aspect of degenerative cervical myelopathy .

The explanation for the disparity in motor deficiencies between the upper and lower limbs is not sufficiently clear. This difference has been explained by the somatotopic organization of the corticospinal tract, where the nerve fibres of the upper extremities are more centered than those of the lower extremities [2, 3]. Jimenez et al. demonstrated a Wallerian degeneration of the corticospinal tract and suggested that the fibres of this bundle seem to be preferentially involved in motor control of the upper extremities rather than the lower extremities [10].

The sensory nucleus of the trigeminal nerve is a large structure extending along the entire length of the brain stem and into the upper cervical spinal cord. It is made up of three sub-nuclei: the mesencephalic nucleus, the pontine nucleus and the spinal nucleus. The trigeminal root, located in the cerebellopontine angle cisterna, has three contingents: the pars minor, superior-medial motor, the pars major, inferolateral sensitive and essentially thermo-algesic, and between

the two, the pars intermedia, essentially carrying corneal fibres. Sensory fibres in the root are somatotopic (Figure 3). Fibres conveying sensations of touch and pressure terminate in the nucleus pontinus. Those transmitting the sensations of pain and temperature end in the gelatinous nucleus, reaching their terminus by descending into the spinal bundle of the trigeminal nerve, a bundle of fibres located immediately on the surface of the nucleus. In the upper cervical spinal cord, the spinal bundle of the trigeminal nerve is continuous with Lissauer's area of the dorsal horn [11].

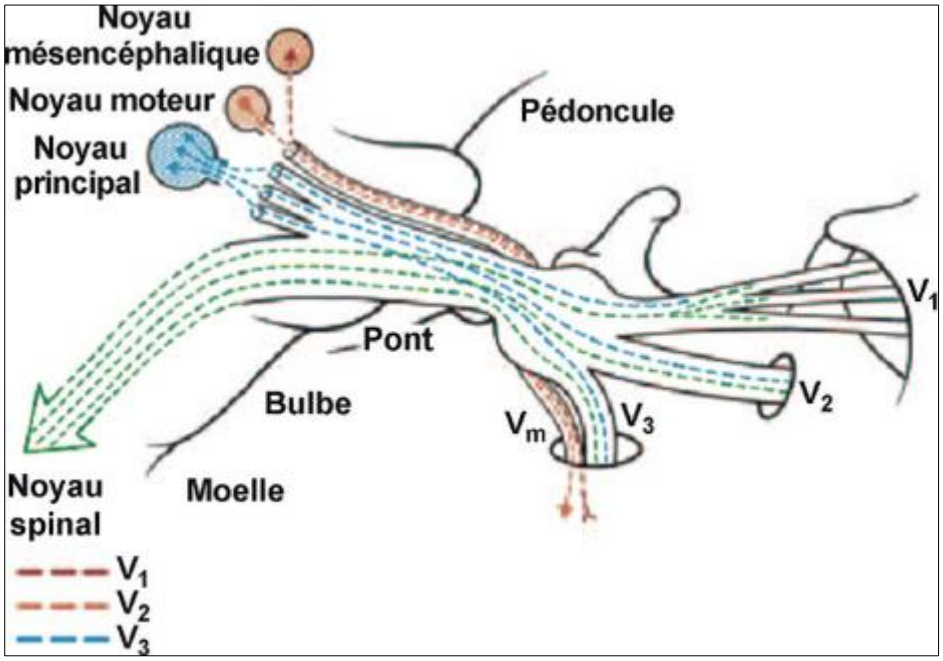

Figure 3 Diagram of the trigeminal root showing the 3 contingents (pars minor, pars intemedia and pars major) [11].

In our patient, we found proportional tactile and thermo-algesic hypoesthesia of the 3 cutaneous-mucosal territories of the left hemiface, without motor deficit, as shown by an ENMG of the face, raising the suspicion of traumatic involvement of the spinal contingent of the V.

The classic mechanism of injury is cervical hyperextension [4]. This hyper-extension causes acute narrowing of the spinal canal by anterior protrusion of the yellow ligament with anterior pinching of the spinal cord. The injury may be due to sudden spinal cord compression between an anterior disco-osteophyte component and a posterior osteo-ligament component [5]. We frequently find an associated osteo-ligamentous lesion, or a herniated disc [8, 12]. However, the disease may also occur without an osteo-disco-ligamentous lesion, in the context of a narrow cervical canal. The symptoms result from trauma-related centromedullary edema. It may also be related to hematomyelia. The latter is rarer and has a poor prognosis [1, 10, 13].

MRI is the reference technique for diagnosis, as it reveals the typical intramedullary hyperintensity in the T2 sequence. Standard X-rays and CT scans can be used to analyze bone structures and assess the anteroposterior diameter of the spinal canal.

There are two approaches to treatment: conservative treatment (cervical immobilization, early rehabilitation), and surgical treatment, which involves decompression of the spinal cord, with or without spinal stabilization [12].

Conservative treatment is reserved for patients with a moderate neurological deficit without osteoligamentous lesions compromising spinal stability. It consists of cervical immobilization, respiratory protection and maintenance of a systolic blood pressure ≥ 90 in order to limit secondary damage to the spinal cord [2, 7, and 8].

The optimal surgical approach is a matter of debate. As a general rule, the ideal surgical approach should target the predominant site of spinal cord compression: anterior, posterior or combined [1,6]. Current data do not suggest any difference between the approaches in terms of functional outcome [7].

Urgent decompression appears to be recommended in patients with incomplete tetraplegia or progressive neurological deterioration [14,15]. The benefits of early surgical decompression remain controversial, although some authors have

correlated it with a reduction in the length of stay in intensive care units and in the occurrence of medical complications [6,15]. Our patient was operated on after 48 hours using an anterior approach for spinal cord decompression, with placement of a plate to stabilize the cervical spine.

Several authors have focused on factors predictive of the recovery rate. A high level of education, a young age at the time of injury, a higher initial ASIA motor score on admission, the anteroposterior diameter of the spinal canal, the absence of comorbidities and the absence of spasticity correlate with good neurological recovery [9, 16,17]. The lower limbs tend to recover motor power first, bladder function second, and finally upper extremity strength, with the finest finger movements coming last [3]. In our case, the patient was able to walk independently without technical aids from the 4th week, but recovery was slower in the upper limbs, particularly in the intrinsic muscles of the hands, with a persistent facial sensory deficit at discharge.

4. Conclusion

Post-traumatic central cord syndrome (Schneider's syndrome) is a poorly understood pathology in our context, the diagnosis of which is sometimes difficult, although it is a fairly frequent form of incomplete spinal cord syndrome. Concurrent involvement of the trigeminal spinal contingent is exceptional.

Edema, hematomyelia, and Wallerian degeneration are common pathophysiological manifestations in these patients, causing variable neurological symptoms. MRI is the diagnostic tool of choice, showing a typical intramedullary T2 hyperintensity. Early surgical management is correlated with better functional recovery compared with conservative treatment, particularly in younger patients.

A higher initial ASIA motor score on admission and the absence of complications, in particular spasticity, favor a better long-term functional prognosis.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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