

## ***Delayed onset diplopia associated with a stab wound dural cerebrospinal fluid fistula***

### **Diplopia de aparecimento tardio associada a fistula dural de liquido cefalorraquidiano por facada dorsal**

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#### **Abstract**

Abducens nerve palsy is the most commonly described intracranial neuropathy secondary to the intracranial hypotension syndrome (usually spontaneous or iatrogenic-especially after lumbar puncture or epidural anaesthesia). Nonetheless, it is a rarely reported complication in the literature. Stab wounds to the spinal cord are relatively uncommon, but even rarer is the presentation of such an injury in a delayed fashion. We report a case of a 31-year-old male who presented with diplopia 6 days after a dorsal stab wound injury. This case highlights the need of an early and accurate assessment on the depth of stab wounds particularly near the vertebral spine and despite the absence of clinical evidence of myelopathy. The non-operative management of such an injury is discussed, with a review of the literature.

**Key words:** Spinal cord injury; Stab wound; Penetrating spinal injury; Dural fistula; Intracranial hypotension.

#### **Resumo**

A paresia do motor ocular externo é a neuropatia intracraniana secundária à síndrome de hipotensão intracraniana mais frequentemente descrita (habitualmente espontânea ou iatrogénica, especialmente depois de punção lombar ou de anestesia epidural). Mesmo assim, trata-se dum complicação pouco frequentemente descrita na literatura. As lesões por facadas na medula espinhal são relativamente infrequentes, embora ainda mais infrequente é a sua apresentação clínica de forma atrasada. Apresentamos o caso dum homem de 31 anos de idade que começou com diplopia 6 dias depois de ser esfaqueado na região lombar. Este caso clínico mostra a necessidade dum avaliação precoce e eficaz da profundidade da facada, particularmente quando a lesão está perto à coluna vertebral, ainda sem sinais de mielopatia no exame neurológico. O tratamento não cirúrgico desta patologia é discutido, com revisão da literatura.

**Palavras-chave:** Lesão medular espinhal; Ferida por facada; Lesão penetrante espinhal; Fistula dural; Hipotensão intracraniana.



## Introduction

Spinal stab wounds (SSW) account for only 8-11% of all penetrating spinal injuries in most North American series, with virtually all of these presenting acutely<sup>1</sup>. The largest experience with SSW comes from the South African literature, with the three largest series reporting over 900 cases<sup>2-4</sup>. The vast majority of such injuries present in an acute fashion, and the injury is usually obvious. However, in rare cases, patients do not seek medical attention acutely and, instead, present in a delayed fashion<sup>5,6</sup>.

## Clinical report

A 31-years-old man was attended at our Emergency Room after being attacked with sticks and knives. His vital signs were normal. He had a Glasgow scale score of 15. He presented several blunt cranial traumas. He also had several stab wounds localized at the frontal region of the head, at the left lateral cervical region and at the back. One stab wound in the back seemed to be deeper in the midline region, next to the D9 spinous process. The neurological exam was normal at that time. A CT scan was performed and no intracranial lesions or signs of cranial fracture were detected. All stab wounds were sutured as they seemed to be superficial not affecting structures other than the skin. The patient was discharged asymptomatic.

Five days later, the patient began to suffer from bioccipital headache. While at rest and laying down, the patient was fully asymptomatic. The headache appeared after rising up and tended to have a holocranial irradiation. Laying down was the only strategy to alleviate the headache.

On the sixth day, the patient noticed horizontal diplopia presenting in various gaze positions. He was unable to perform his duties because of this disabling symptom. The headache was getting progressively worse.

Eight days after the aggression, the patient returned to

the ER because of the progressive worsening of the symptoms. The neurological examination disclosed a bilateral abducens nerve palsy. General examination, including vital signs and aspect of the sutured stab wounds was normal. The patient was admitted on the clinical suspicion of an intracranial hypotension syndrome. Conservative treatment was initiated with bed rest, IV fluids, on demand analgesia and alternant ocular occlusion.

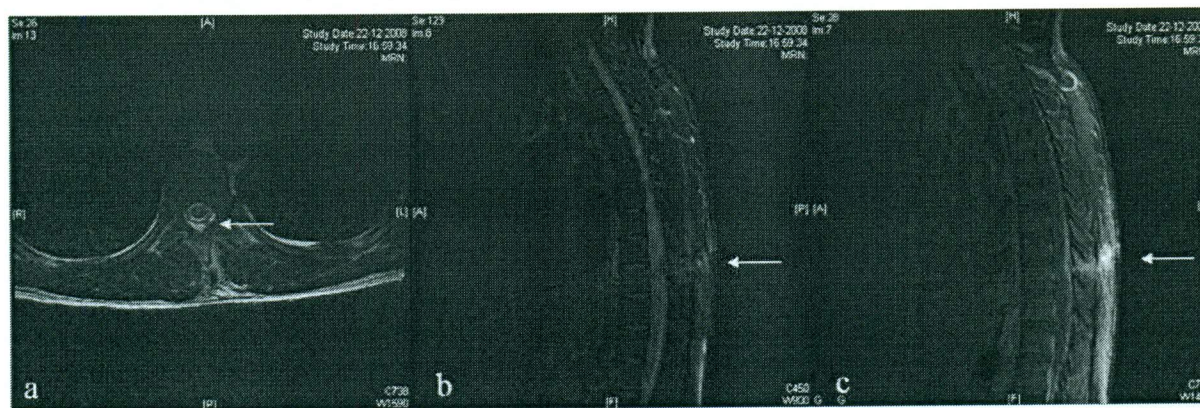
A Cranial Magnetic Resonance Imaging (MRI) Scan showed no intracranial abnormalities. The spinal MRI demonstrated a dural tear with an associated dural fistula at the D9-D10 level (Figure 1). This level corresponds to the stab scar next to the D9 spinous process. No signs of spinal cord lesions were detected.

Because of the 2 weeks gap between the date of the aggression and the one of the diagnosis of the traumatic dural fistula, and taking into consideration that there was not any clinical or radiological evidence of an active external fistula, the Neurosurgeon was prone to follow a conservative management of the situation.

Several weeks after being attacked, the patient continued to have diplopia. A gradual spontaneous improvement of the bilateral abducens palsy was observed during the next 4-5 months. At present, the patient is asymptomatic but he was not able to perform his usual activities of daily living for several months.

## Discussion

Bilateral sixth nerve palsy after trauma is rare and is highly associated with intracranial or cervical spine injury because is thought to indicate severe trauma to the head or spine<sup>7-8</sup>. The prevalence of post-traumatic bilateral sixth nerve palsies is low probably because the force needed to produce these lesions is generally incompatible with survival. Moreover, in these patients, we have to look for asso-



**Figure 1. a:** Axial T2-weighted MRI scan of the spinal cord at D9 level showing signal-intensity alteration of the soft paravertebral tissue on the left, and the posterior arc of D9, with an associated dural tear (white arrow) and absence of cord lesions. **b and c:** Sagittal T2-weighted MRI of the dorsal spinal cord showing the stab wound way from the skin to the dural canal at the D9-D10 level (white arrows). We can also document the presence of epidural and subdural cerebrospinal fluid collections at the cervical and dorsal levels with its maximum diameter at D9-D10 levels.



ciated injuries because of the high energy mechanism of the trauma.

The differential diagnosis for lateral gaze palsy after head trauma includes brain stem lesions<sup>9</sup>, peripheral nerve injury with or without basilar skull fracture<sup>10</sup>, and lateral rectus muscle injury or entrapment.

The mechanism of the acute neurological deficit that immediately follows an SSW can be due to direct penetration of the spinal cord neural elements, from spinal cord infraction, or rarely, from a spinal epidural haematoma. When the direct penetration of the spinal cord occurs, the patient is usually left with an incomplete spinal cord injury, most typically a Brown-Sequard variant<sup>11</sup>. Clinical findings in those cases can easily lead the diagnostic workup.

While delayed presentation of spinal stab wounds (SSW) is rare, it is important to consider and recognize this mode of presentation. Particular attention must be paid to a meticulous inspection and palpation of the neck and back region, concentrating not just on the midline but on the paravertebral and flank regions as well<sup>5</sup>.

Delayed onset of neurological deficits may result from retained weapon or fragment of one because all patient with history of stab wounds are at risk of harbouring a retained foreign body in or around the spinal canal. Other causes are infection, oedema, Cerebrospinal fluid (CSF) leak, granuloma formation or iron encrustation<sup>6,12</sup>.

The presence of a retained or impaled object should not obviate the undertaking of a complete trauma assessment because these patients are at risk of serious associated injuries. After appropriate initial trauma assessment and resuscitation, radiographic studies should be obtained to determinate if retained fragments are present.

CT scan can identify retained fragments, haematomas, bone fragments... but metal ones may produce an artefact that can decrease the CT studies quality. Magnetic resonance imaging has been advocated by some authors for neurodiagnostic workup in these patients. Moyed and colleagues<sup>13</sup> report that MR imaging is a "powerful tool" for identifying the injury tract, cord or root lesions, and associated lesions. The high sensitivity of MR imaging to retained foreign material, along with its ability to better characterize ligamentous and vascular injuries, has suggested its utility in the workup of these patients. This is somewhat controversial, however, because there is an increased risk of movement, and induction of currents in ferromagnetic or conductive materials when placed in an external magnetic field.

Definitive management for the delayed presentation of SSW must be individualized. Foreign bodies must be removed to decrease the neurological damages and do it

with utmost care to avoid causing undue injury during the surgery. In particular, movement of the fragment must be avoided, and electrocautery must not be used. The dura around the spinal cord must be closed to avoid CSF leak<sup>5</sup>. Adams et al.<sup>6</sup>, reported the case of an 18-year-old man stabbed with a screwdriver at T-9; he presented to the emergency department neurologically intact. After reviewing a plain x-ray film, the emergency physicians removed the screwdriver and discharged the patient home; he returned 5 weeks later with a postural headache, right sixth cranial nerve palsy, and MR imaging evidence of an active CSF leak. He was treated with autologous epidural blood patch and his symptoms completely resolved. Patients in whom surgical dural exploration is required following foreign body removal (or those in whom a CSF leak is suspected) should therefore be identified by qualified spine surgeons.

For patients who present with a stable neurological deficit, management is controversial. There is a relatively low rate of infection even in cases of retained fragments<sup>4</sup>. Intravenous steroid agent administration does not improve neurological function in patients with penetrating spinal cord injury and may be associated with an increased risk of infection<sup>14</sup>. It is therefore not recommended as a management option. Patients with wounds created by high-risk penetrating items and those that are significantly contaminated may benefit from a course of broad-spectrum antibiotic therapy with or without surgical debridement<sup>15</sup>.

The prognosis for patients with spinal stab wound is generally better than it is for those with either blunt spinal cord injury or gunshot wounds to the spine. Most patients present with only incomplete spinal cord injuries, and up to 66% have a good recovery<sup>3,4</sup>.

The spontaneous recovery rate of unilateral traumatic sixth nerve palsy has been estimated from 12% to 73% at 6 months. Of those patients who will spontaneously recover, the median time to recovery is 90 days. No large series are available for bilateral sixth nerve palsies of traumatic origin.

Acute symptomatic management of a sixth nerve palsy consists on occlusion of one eye to alleviate the symptoms of diplopia. Prisms lenses can also be used with this goal but are unsatisfactory in the long term for most patients. Botulinum toxin injections into the antagonist medial rectus muscle might eliminate its unopposed action and allow for single vision during the recovery period. It might also prevent contracture of the unopposed medial rectus.

Surgery, consisting in different eye muscle resections and transpositions, is currently indicated in those patients who have not recovered within 6 months to 1 year after injury.

The success rate of these surgeries ranges from 30% to 80%.

This case report illustrates the importance of early diagnosis and management of traumatic CSF leaks of traumatic origin. Even in the absence of acute neurological deficits, the emergency physician should be aware of the potential mechanisms that can lead to delayed highly incapacitating and potentially irreversible symptoms. ■

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