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Acute flaccid paraplegia: neurological approach, diagnostic workup, and therapeutic options

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ABSTRACT

Acute flaccid paraplegia is a clinical occurrence with extreme importance, due to the dramatic presentation, the severity of the underlying disorder, and the generally poor prognosis that follows such a condition. Among etiological factors, the traumatic events are of particular interest, with the clinical treating dealing with a severely ill patient, following fall from height, motor vehicle collisions, and direct shocks applied over the vertebral column. The non-traumatic list is more numerous; however the severity of the acute paraplegia is not necessarily of a lesser degree. Viral infections, autoimmune disorders, and ischemic events involving feeding spinal arteries have been imputed. However, chemical and medications injected during procedures or accidentally intrathecal administration can produce acute flaccid paraplegia. A careful neurological assessment and complete electrophysiological and imaging studies must follow. In spite of the poor prognosis, different therapeutic options have been proposed and applied. Neurosurgical and orthopedic interventions are often necessary when trauma is present, with high dose glucocorticoids treatment preceding the intervention, in a hope to decrease edema-related compression over the spinal cord. Immunoglobulins and plasmapheresis are logical and helpful options when a polyradiculoneuritis produces such a clinical picture. The role of decompression, as neurosurgical exclusivity, has been considered as well.

1. Introduction

A flaccid paraplegia is defined as a clinical syndrome, with rapid and symmetrical onset of weakness in both lower limbs, progressing to a maximum severity within several days to weeks[1]. Depending on the gravity of the disorder and the level of neuraxial injury, muscles of respiration and swallowing might be interested; when this happens as a rule, the patient is already quadriplegic. The flaccidity of the occurrence means that a deep muscular hypotonia will already be present on examination. In spite of the nature of the underlying disorder, lower extremities might show hyperesthesia and hyperalgesia. Sabin and Wright have made a meticulous description of this clinical picture more than eighty years from now, with their case showing characteristically an absence of knee jerks, with a negative Babinski sign, and with no meningeal irritation[2].

Obviously the medical casuistics, even the remote one, is florid on situations and etiologies, with several of them identified and reported decades before, such as spinal lesions, aortic dissection, herpetic infections and the postoperative setting[3-6]. In a highly professional opinion paper, Clarke illustrated in 1908 his experience with paraplegic patients through describing cases with poliomyelitis, toxic polyneuritis and myelitis[7].

Acute flaccid paraplegia and quadriplegia of traumatic origin were well known to Egyptians even millennia before, as the Edward Smith Surgical Papyruses have incontestably proved. If we scrutinize the image reproduced in Figure 1, we might easily understand that ancient Egyptians were able to differentiate a spastic from a flaccid paralysis: just note the obvious extended position of the forelimbs, when compared with the drooping hindlimbs of the lion, shot with arrows.

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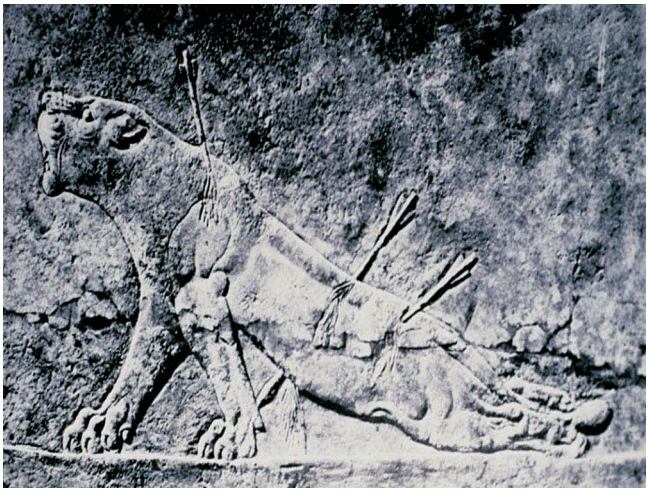

Figure 1. Paraplegia and quadriplegia due to spinal cord injury (following arrow shots) as depicted in the Edward Smith Surgical Papyruses, Case 31[8]. Egyptians were able to discriminate a spastic position of anterior extremities, when present (note the forelimbs) from the flaccidity of hindlimbs, as depending from the level of traumatic injury (arrow shots).

With the clinician dealing a highly dramatic condition, the need for a prompt identification of the etiological factor and the initiation of an ad hoc therapy, is extremely important. The patient might present in two grossly divided situations: a: patient who is in coma, with paraplegia generally due to two principal diagnostic events, namely, due to a recent nervous (cranial; medullar) trauma of certain severity, frequently in a polytraumatized patient; or when the clinical picture of an acute post-infectious encephalomyelitis is seen; b: lucid patient, such as when a compressive event is taking place adjacent or within spinal structures; or much more frequently when a polyradiculoneuritis of the ascending form is diagnosed.

In all eventualities, a prompt neurological consultancy, complimented with imaging and electrophysiological studies, is indispensable. Dealing with a patient in coma might be an extreme challenge, since the depth of the loss of conscience might be a sufficient factor to abolish spontaneous or reflective movement of the lower extremities. The initial care is of irreplaceable role; in fact, many unlucky cases had already undergone an emergency medical evaluation, which thereafter have been inconclusive or insufficient[9].

With a diagnosis of installed acute paraplegia, whatever the underlying cause, the prognosis will be reserved, if not infaust. The neurosurgical and orthopedic consultancies are among the first steps to be undertaken, even when trauma is not present in the history, since it might have been minor, trivial, neglected, or not involving directly the vertebral column and its structures, thus leaving little space to suspicion[10,11]. Once a traumatic event is completely ruled out, and appropriate imaging studies have documented its absence; the role of the neurologist and eventually of the infectious disease specialist will be of primary importance[12,13]. Electrophysiology, lumbar tap and serological examinations will be part of the diagnostic techniques[14-16]. Last but not least, when the panoply of examinations is inconclusive, a psychiatric consultancy might reveal interestingly the eventuality, albeit remote, of a malingering[12].

2. Illustrative case series

The first case is a young male aged 23 years old, who fell from a height of approximately ten meters while working.

He presented conscious, but deeply in pain in the emergency room. Subcutaneous emphysema, cutaneous crepitations and hematoma were seen in the parasternal region. The young adult was totally paraplegic and a vesical globe warranted the insertion of a urinary catheter. A total body scan was performed; the bone windows retrieved images of a fractured sternum, and comminuted fractures of the third and fourth thoracic vertebral bodies (Figures 2 and 3).

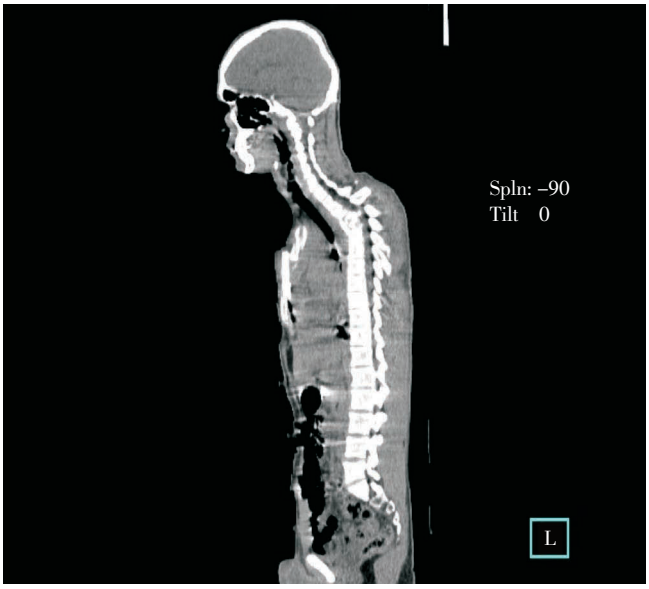

Figure 2. A case of traumatic paraplegia, with sternal fracture and comminuted fractures of third and fourth bodies of thoracic vertebrae.
Note the abnormally angled vertebral column (CT scan images, sagittal reconstruction).

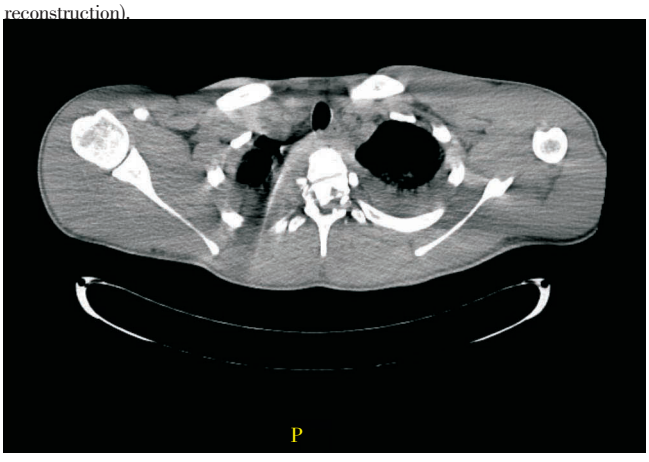

Figure 3. A case of traumatic paraplegia.
Axial CT images, demonstrating the comminuted fracture of the vertebral body, whose osseous fragments already have invaded the spinal channel and provoked the spinal shock.

Axial CT images showed the massive presence of osseous fragments within medullar channel, presaging a very poor prognosis for an already installed spinal shock (Figure 3). The patient received one gram of intravenous methylprednisolone for one week; then a corrective thoracic stabilization orthopedic intervention was made, aiming merely to enable the patient to stand in a sitting position in the future, since the ability to walk was permanently lost.