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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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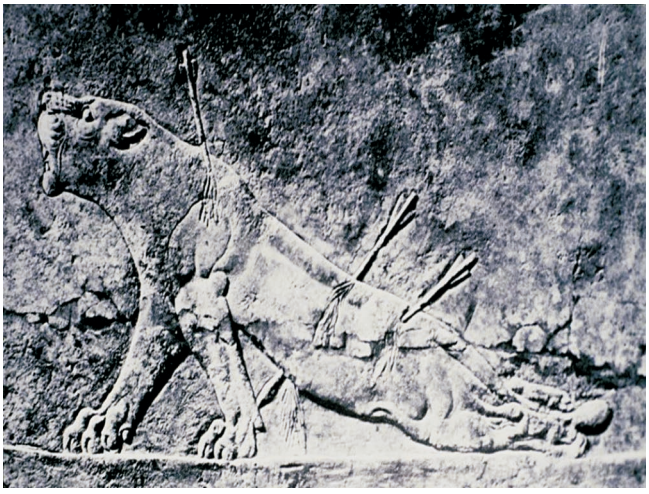

Figure 1. Paraplegia and quadriplegia due to spinal cord injury (following arrow shots) as depicted in the Edward Smith Surgical Papyruses, Case 31[8]. Egyptians were able to discriminate a spastic position of anterior extremities, when present (note the forelimbs) from the flaccidity of hindlimbs, as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

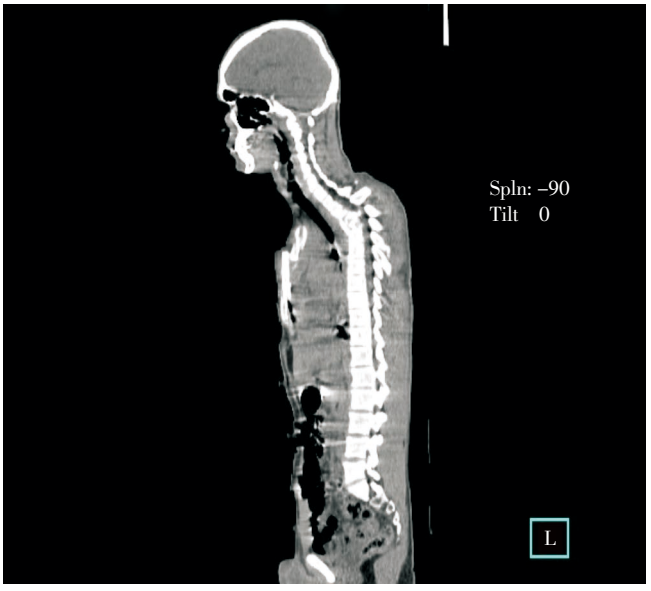

Figure 2. A case of traumatic paraplegia, with sternal fracture and comminuted fractures of third and fourth bodies of thoracic vertebrae.
Note the abnormally angled vertebral column (CT scan images, sagittal reconstruction).

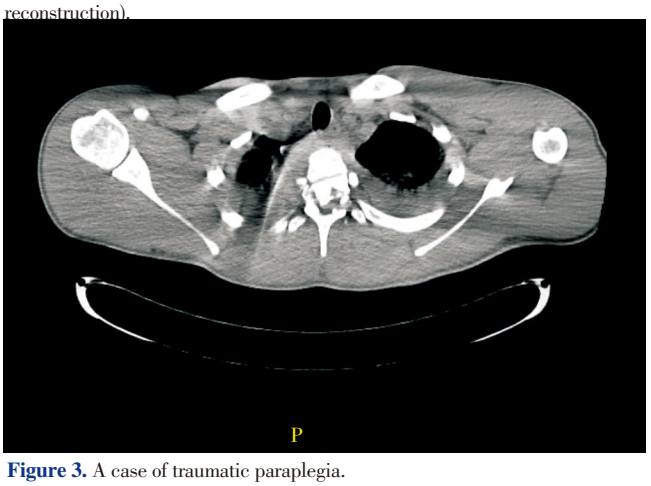

Figure 3. A case of traumatic paraplegia.
Axial CT images, demonstrating the comminuted fracture of the vertebral body, whose osseous fragments already have invaded the spinal channel and provoked the spinal shock.

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.

The second case is a sixty-two years old female patient suffering from neck pain and diffuse articular pain since years. The complaints were considered as of arthritic and osteoporotic nature. After a minor effort during raising a certain load, she felt numbness in both legs and loss of motor control within hours.

The lady was emergently consulted for an acute and flaccid paraplegia. A total spine MRI suggested diffuse arthrosic changes in the entirety of the vertebral column; however, loss of intervertebral disc height and extramedullary collection was detected at the level of the fifth to sixth cervical intervertebral space (Figure 4).

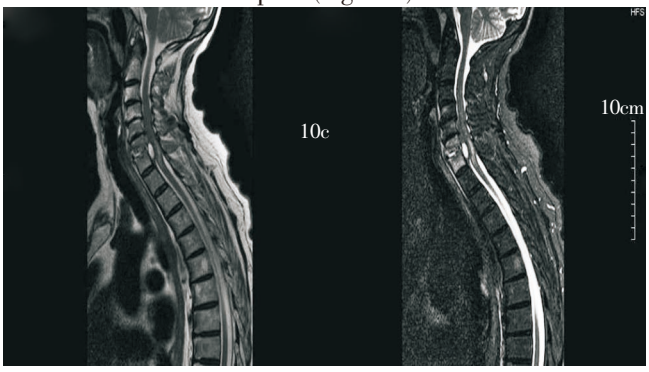

Figure 4. Cervical discitis with anterior extramedullary collection and edematous changes in the adjacent medulla, with a considerable loss of height at the discal space between the fifth and the sixth cervical vertebrae.
Left inset: T1 flair sagittal MRI images; Right inset: T2 MRI sagittal images of the spinal column).

Of note was the fact that she was still able to command effectively both upper extremities, in spite of edematous changes all along the spinal cord adjacent to the collection. A neurosurgical consultancy suggested a decompressive laminectomy that was refused from the patient. Opting for a conservative treatment, low-dose dexamethasone was applied (4 mg twice daily) for ten days, and a supportive cervical immobilizing corset was put in place. The patient was dismissed home with antalgics and continuous positioning of the cervical immobilizing corset; on follow-up one month later she had gained but very few in the motor ability of lower extremities, and was referred to a rehabilitative facility.

The third case is a fifty four years old lady, suffering from diabetes since three years ago, and on oral hypoglycemic therapy, was consulted for a subacute loss of motor function in both lower extremities. Initially considered as a diabetic neuropathy, she was suggested a tight glycemic control and physiotherapy; however she lost completely the ability to walk two days before hospitalization.

An abolition of ankle and knee reflexes, together with a decrease in muscle tone and loss of any voluntary movement were all present. Overflow urinary incontinence warranted a catheterization; an electrophysiological study attested the presence of a discrete sensory neuropathy, but otherwise the neurography could not explain the presence of a total and subacute paraplegia.

A total body scan was performed, with the suspicion of a paraneoplastic disorder, mainly orientated from the conspicuous loss of weight in the last month (the lady referred losing twelve kilograms in less than five weeks). A parasagital-midline meningioma of the cerebral falx was seen, and the patient was transferred to a neurosurgical facility (Figure 5).

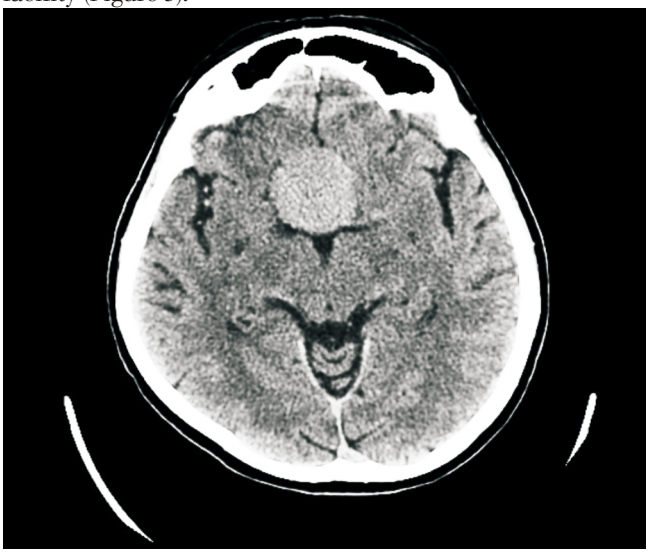

Figure 5. Unenhanced axial CT scan images of the head: a midline parasagital meningioma of the cerebral falx, leading to a subacute paraplegia in a diabetic patient.

The fourth case is a thirty years old male who presented for a medical visit following one week of fever, treated symptomatically as a flu-like condition. He was severely paraparetic and unable to move without support. He hospitalized, the next three days he became paraplegic, with an ascending deficit that did not reached cranial territories. Afebrile at that period, he underwent several medical examinations, including repeated neurography, lumbar tap, serology and total spine MRI imaging.

Lumbar tap performed the second day of hospitalization suggested an abnormally high protein concentration, but an otherwise normal cellular presence (a cell count <5/μL). Initial signs of a demyelinating process were met at the neurography on the third day of hospitalization; the confirmation of polyradiculoneuritis was clear in the follow-up neurography. A nerve biopsy, however, was not performed.

The patient received immunoglobulins five consecutively days; three months from the event he was able to walk without support, and a rehabilitative program was advised. Figure 6 shows the result of right tibialis posterior nerve electroneurography.

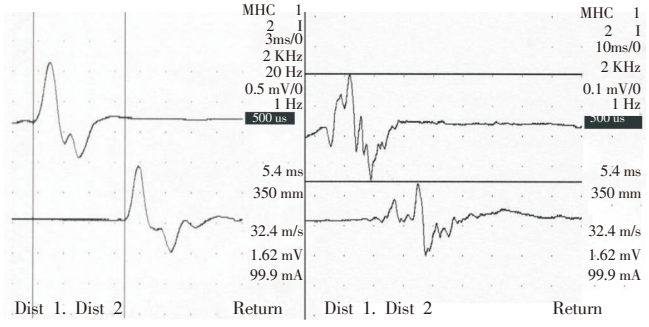

Figure 6. Right tibialis posterior nerve electroneurography.
Left inset: data received the third day of hospitalization. Although still a normal configuration is registered, the nerve conduction velocity is clearly below normal (32.4 m/second; the normal level is >40 m/second). Right inset: data received on the same territory, one month after discharge. Configurations of the potentials are highly disturbed, with dispersion of amplitudes (falling to the level of microvolts) and a further decrease in the nerve conduction velocity (actually 28.2 m/second).

3. Discussion

Once paraplegic, the future of the patient is gloomy, and challenges are enormous. Maybe due to the acuteness of the installation, the first hours of treatment could be decisive in blocking a severe paraparesis to become paraplegia. The different therapeutic options, discussed below, depend primarily from the nature of the underlying disorder, and from a dichotomist position in confronting paraplegia of traumatic nature, versus a non-traumatic one.

Another form of dichotomist evaluation and systematization relates to the fact if the paraplegia is merely of a peripheral form, or if the neuraxial structures are interested, thus rendering the event of a central nature. Table 1 below describes the main diagnosis based exactly on such a separation.

Table 1
Etiology of acute flaccid paraplegias.

Of a peripheral nature	Of a central nature
Non-compressive:	Acute transverse myelitis [17,18]:
Acute polyradiculoneuritis[17,18]	-Infectious myelitis
Acute polyneuropathy[19]	Neurotropic viruses
Acute anterior poliomyelitis[20]	Mycoplasmata
	-Non-infectious inflammatory myelitis
	Multiple sclerosis; neuromyelitis optica (Devic's disease) [25,26]
	Idiopathic transverse myelitis[27]
	Symptomatic transverse myelitis (connective tissue disorders) [18,28]
Compression of cauda equina:	Trauma:
-Epidural or subdural spinal hematoma[21]	-Vertebral and/or spinal traumatic event
-Epidural metastasis[22]	-Post lumbar puncture hematoma[29]
-Vascular malformations[23,24]	-Spinal ischemia following thoracic, cardiac and aortic surgery[30,31]
	Iatrogenic:
	-Inadvertent intrathecal injection of neurotoxic drugs[32,33]
	-Post vaccination paraplegic event[34]
	Acute non-traumatic medullary compression:
	-Malignant hemopathies[16]
	-Vertebral tumors or metastases[35,36]
	Spinal space occupying lesions:
	-Disc prolapse; discitis[34,37]
	-Epidural abscesses[37]
	Non-spinal disorders [34]:
	- Hyperkalemic or hypokalemic paralyses[38]
	- Parasagittal cortical syndrome[39,40]
	- Psychogenic paraplegic symptoms[41]

Although Table 1 includes a variety of diagnosis and events, it cannot be exhaustive. The list of causative factors is almost daily enriched with new occurrences of different nature, and this is particularly consistent when infectious agents are imputed. Thus, tuberculosis, hydatidosis and toxoplasmosis are among the infectious conditions reported[42-44]. Even other internal disorders, of a malignant nature (leukemia), or non-malignant such as related to the thyroid storm, might be found in the literature[45,46].

The causative mechanism of post-traumatic paraplegia is simpler under the semiotic point of view; it is generally related to a trauma of considerable intensity, such as during a fall from height or a motor vehicle collision[47]. Intentional stabbing into the spine has been reported as deliberately perpetrated for producing paraplegia, as another abhorrent method of torture[48]. The presence of air in the spinal canal has been exceptionally suggested as a particular form of traumatic paraplegia[49].

Since the list of circumstances and conditions is particularly numerous, a topographical approach might have classificatory value, when considering the primary noxious process as originating from inside the spinal cord (a primary medullary injury), or when the lesion is primarily extramedullary, with later extension into the spinal structures. Table 2 summarizes some of these conditions.

Table 2
Primary medullary *vs.* extramedullary injuries (Modified from [50-52]).

Primary medullary injury	Primary extramedullary, with extension into the spinal space
Acute:	
-Hematomyelia[51]	-Median disc herniation[54]
-Anterior spinal artery syndrome[52]	-Vertebral fractures[55]
Subacute:	-Extradural hematomas[56]
-Multiple sclerosis	-Spinal epidural abscesses[57]
-Acute and necrotizing myelitis[53]	

With such a variety of clinical pictures and background disorders, it is obvious that the treatment options vary accordingly. A review of all orthopedic and neurosurgical approaches is beyond the scope of this paper, but several stabilization procedures have been proposed and applied, together with the operative decompression of the cord and cauda, when paraplegia has been of a traumatic origin[58]. In spite of controversies, high-dose steroids have been widely used following spinal traumas, with the aim of reducing the medullar edema, but with unclear long-term outcomes. Methylprednisolone given intravenously is tried and applied with some efficacy, albeit no differences have been registered between patients receiving high-dose protocols (with a dose of approximately ten grams of methylprednisolone during the first day of treatment, separated in an initial loading bolus of 30 mg/kg and followed the same day by 5.4 mg/kg/hour for the next 23 h), and patients receiving lower doses of the same drug (such as one gram daily intravenously for ten consecutive days; or with another group of patients receiving a low-dose regimen of 100 mg of methylprednisolone daily for ten days). Complications of the high-dose regimen have been reported, and the results of three studies named NASCIS (National Acute Spinal Cord Injury I, II and III) have been published[59]. Other pharmacological options for the management of the acute spinal cord injury have been proposed, such as GM-1 ganglioside, and naloxone[59,60].

One of the very interesting orthopedic techniques suggested, once the paraplegia is installed and virtually irreversible, is the one proposed by Brunelli, who has tried to bypass the spinal cord, anastomosing the upper motoneurons directly to peripheral targets[61]. Although extremely interesting, the technique is far from being popular.

Pharmacological options are available as well when approaching a non-traumatic paraplegic patient. Obviously, infectious conditions need to be diagnosed and treated appropriately. Intravenous immunoglobulins and plasmapheresis are available treatments for the polyradiculoneuritis of a Guillain-Barré form, but their application has been widened to a much larger scope of neurological conditions[62,63]. The role of other measures such as decubitus ulcers' prevention, antalgics, anticoagulants and rehabilitative interventions, although to some extent secondary and supportive, need not to be underestimated[64,65]. Neurogenic bladder training is another important milestone of the rehabilitative programs[66]. The

same needs to be said with regard to the nutrition, as well as for the psychological support of the paraplegic patient[67].

4. Conclusions

Although a medical condition old as the humanity itself, acute paraplegia is a serious challenge for the nursing staff and the treating clinician, whatever the background disorder that leads to this syndromic picture. The literature, however, recognizes the lucky probability of a complete recovery or resolution of paraplegia, in certain circumstances[27,38,68,69]. In traumatic cases it seems that the first hours of medical care will be decisive in terms of restricting or limiting any further the remaining functional disability. Non-traumatic paraplegia has on the other side a very long list of potential causative factors; nevertheless pharmacology has made considerable advancements in the last decades. Intravenous immunoglobulins and plasmapheresis have gained citizenship in the treatment of polyradiculoneuritis and other peripheral nerve system disorders, but the same as in the traumatic event, the timeliness of the interventions is extremely important, for a neurological picture that otherwise has a long way towards its functional improvement, if any.

Conflict of interest statement

The authors report no conflict of interest.

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