Anterior spinal artery infarction: study of a case in the neurology department of the CHU Ignace Deen in Conakry

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ABSTRACT

Introduction: Spinal cord infarction corresponds to all the clinical manifestations secondary to ischemia of part or all of the spinal cord, whether sudden or progressive. It is a rare condition with a difficult diagnosis affecting only 1.2% of patients admitted to the neurovascular pathologies department. It is most frequently located in the territory of the anterior spinal artery (66.7%).

Observation: 43-year-old patient with a lifestyle of repeatedly carrying heavy weights on the head, presenting with neck pain radiating to the upper limbs and tetraparesis. The clinic is characterized by anterior spinal artery syndrome, a lesional and sub-lesional syndrome and the spinal MRI revealed an extensive T2 hyper signal localized at the level of the anterior horn producing the characteristic appearance of snake eyes evoking an infarction in the territory of the anterior spinal artery.

Conclusion: It is a diagnosis which remains difficult and is based on the comparison of clinical and para-clinical data.

Keywords
Infarction, Anterior spinal artery, Neurology, Ignace Deen University Hospital of Conakry.

Introduction
Spinal cord infarction is the set of clinical manifestations secondary to ischemia of part or all of the spinal cord, occurring suddenly or progressively [1]. Spinal cord infarctions represent approximately 1.2% of all ischemic strokes and less than 10% of acute non-traumatic myelopathy [2] leading to devastating neurological sequelae for patients [3]. The clinic is variable; the onset is often marked by spinal pain, sometimes following even minor trauma. However, the installation can be rapidly progressive in a “step of stairs” associating a lesional and sub-lesional syndrome; sometimes accompanied by sphincter disorders, more inconstant signs may exist, such as disorders of the sense of position and vibration sensitivity [4]. The diagnosis is made by performing an emergency MRI; after formation of the infarction, the typical appearance is that of two well-defined homogeneous nodular hyper signals affecting the anterior horn of the gray matter on each side giving a classic appearance of ”snake eyes” or ”owl eyes” in sequence T2 reflecting damage to the anterior vascular network [5]. Regardless of the treatment of the cause, the treatment of spinal cord infarctions is primarily symptomatic [4], it is also based on rehabilitation and nursing care [6]. The prognosis of anterior spinal artery infarction is poor [7], mortality in the acute phase is approximately 20%, and severe sequelae are present in 50 to 60% of patients [4].

We report the study of a case of infarction of the anterior spinal artery diagnosed in the neurology department due to the rarity of spinal infarctions, the diagnostic difficulties, the multiplicity of etiologies and the poor prognosis it confers on patients.
**Case Presentation**

43-year-old woman teacher admitted for intense neck pain radiating to the upper limbs accompanied by weakness in all 4 limbs, evolutoin three (03) months. With an unremarkable personal and family history, the symptomatology would appear suddenly, marked by the occurrence of neck pain radiating to the upper limbs, tingling in the lower limbs followed by an abnormal stair-like gait then occurs for 2 days later a weakness of all 4 limbs. Faced with the worsening of the initial clinical picture; and on the advice of her daughter, she finally decided to consult our department for support.

The parameters on admission (See table) were within normal limits; the integuments and conjunctiva are normal in color with a preserved general condition.

**Painting**

Patient Parameter Value.

<table>
<thead>
<tr>
<th>Settings</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vital</strong></td>
<td>Blood pressure = 140/80 mmHg.</td>
</tr>
<tr>
<td></td>
<td>Temperature = 36.8°C.</td>
</tr>
<tr>
<td></td>
<td>Respiratory frequency = 15 cycles/min.</td>
</tr>
<tr>
<td></td>
<td>Heart rate = 72 beats/min.</td>
</tr>
<tr>
<td></td>
<td>Random blood sugar at $1.20$ g/L.</td>
</tr>
<tr>
<td><strong>Anthropometrics</strong></td>
<td>Weight: 59kg.</td>
</tr>
<tr>
<td></td>
<td>Height: 1m50.</td>
</tr>
</tbody>
</table>

The neurological examination carried out revealed a conscious patient oriented in time and space, uncooperative, with:

- an anterior spinal artery syndrome including neck pain, flaccid paraplegia, the presence of Babinski’s sign, thermo algic anesthesia going back to the C5 dermatome but preservation of proprioceptive sensitivity.

- an injury syndrome: bilateral cervico-brachial pain;
- a sub-lesional syndrome: flaccid paraplegia, the presence of Babinski’s sign, the abolition of abdominal skin reflexes. In addition to the localization capability of clinical examination, magnetic resonance imaging (MRI) with and without contrast is the radiological method of choice for evaluating a patient suspected of anterior cord syndrome.

the MRI revealed:

In conjunction with the imaging, the biological assessment was unremarkable: The lumbar puncture performed after the MRI to rule out other diagnoses (myelitis) and to provide arguments for certain infectious or neoplastic causes independently revealed normal cerebrospinal fluid, Doppler ultrasound for ASD and MRI angiography were unremarkable. Faced with this bundle of clinical arguments (anterior spinal artery infarction syndrome, lesional syndrome and sub-lesional syndrome) and paraclinical arguments (appearance of “snake eyes” on MRI) we have retained the diagnosis of: Anterior spinal artery infarction. She benefited from 5 days of boluses of corticosteroid therapy based on methylprednisolone 600 mg per day then an oral relay and physiotherapy sessions. The evolution after one week was marked by the regression of neck pain, ample movements of the upper limbs and slight movements of the left foot, no change for the right foot.

**Discussion**

Spinal cord infarction (SCI) is one of the uncommon causes of acute non-traumatic myelopathy and ischemic stroke [8]. This rarity is explained, on the one hand, by the high level of collaterals which maintain the vascular supply to the spinal cord [9]. On the other hand, spinal arteries are less sensitive to atheroma formation than their cerebral counterparts [10]. Anterior spinal artery (ASA) syndrome is an uncommon cause of acute ischemic spinal cord...
infarction. It results from occlusion or hypoperfusion of the anterior spinal artery supplying the anterior two-thirds of the spinal cord. The anterior spinal artery formed by the union of the two branches of the intracranial vertebral arteries at the level of the foramen magnum and descends to the conus medullaris along the anterior median groove of the spinal cord. Along its course, the ASA supplies the ventral cord and the anterior two-thirds of the spinal cord. The caliber of the artery is variable with the narrowest segment in the thoracic region, considering it an area vulnerable to ischemia. The paired posterior spinal arteries most often arise directly from the vertebral arteries and pass through the posterolateral groove of the spinal cord. Throughout the course, they give off penetrating branches to supply the posterior columns and the dorsal gray matter. The anterior and posterior spinal arteries join to form an anastomotic loop at the conus medullaris. The radiculomedullary arteries, arising from certain segmental vessels, pass through the intervertebral foramina and run along the ventral and dorsal roots to reinforce the longitudinal spine [11]. The spinal cord receives an unevenly distributed blood supply along the different regions. A larger diameter and numerous radicular arteries made the superior cervicothoracic and thoracolumbosacral segments richly vascularized. Where a single anterior radicular artery (Adamkiewicz artery) between T4 and T8 made the intermediate or mid-thoracic part poorly vascularized [12]. This lesser anastomosis between the ASA and the artery of Adamkiewicz makes the ASA a terminal artery. In contrast, PSA has numerous posterior radicular arteries at this level, providing an extensive collateral system. Therefore, PSA occlusion generally does not cause significant clinical dysfunction. This regional variation explains the vulnerability of the T4T8 region of the spinal cord to ischemia, particularly hypoperfusion due to hypotension. Anterior spinal artery syndrome is a rare cause of acute ischemic spinal cord infarction, which occurs due to complete occlusion or hypoperfusion of the anterior spinal artery. Clinical features include motor paralysis, bowel-bladder incontinence, bilateral loss of pain and temperature sensation, with intact proprioception and vibration sense due to sparing of the posterior column [13]. The clinical presentation of our case corroborates with that of Rahman et al. who reported that the presentation the most common is sudden, severe back pain, which may radiate caudally and patients may present without pain, but more than 80% of spinal infarctions are painful, which makes an exciting and unexplained difference from infarction painless brain [11]. In addition to this pain, almost all patients typically have other neurological deficits arising from the injury to the spinal pathways located in the anterior two-thirds, including bilateral weakness, paresthesias, and sensory loss. Loss of sphincter control with difficulty evacuating the bowel and bladder becomes evident within a few hours. Depending on the level of spinal cord injury, motor weakness can range from bilateral leg weakness to quadriplegia. Indeed, these cases of quadriplegia following interventions on the posterior fossa have been reported. These were generally patients operated on in a sitting position, but this complication can also occur after an operation carried out in the prone position. The generally accepted mechanism is the impact on the vascularization of the spinal cord of hyperflexion of the neck. A vascular mechanism and positional factors could also be involved in the neurological worsening observed after cervical laminectomy. However, no history of surgery was reported in our patient [14]. Despite the absence of vesico-sphincter disorders in our patient, it should be noted that intestinal and bladder complications following ASA syndrome can appear but nevertheless also differ depending on the evolution of the lesion. Immediately after injury, patients typically experience urinary retention due to loss of urination due to spinal shock. In this phase, incontinence results from the distended bladder and is manifested by continuous dribbling. As the spinal cord regains its function, the urination reflex becomes independent without interruption from the higher center. This uncontrolled urination reflex contributes to overactive bladder and urge incontinence. In this phase, patients typically have intermittent urinary loss rather than continuous dribbling, which results from disruption of inhibitory neurons in the spinal micturition center [11]. Diagnosis is based on imaging and any acute spinal cord syndrome requires emergency spinal cord MRI [15]. MRI makes it possible, from the acute phase,
to exclude certain causes of acute spinal cord syndrome such as acute spinal cord compressions or hematomyelia. At this stage, the differential diagnosis with myelitis is difficult [16].

Emergency spinal cord MRI is normal in approximately 17% of cases of spinal cord infarction [17]. Broadcast sequences are technically difficult to achieve. However, they are very sensitive in the early phases. The MRI characteristics of spinal cord infarctions are similar to those of cerebral infarctions. On T2 sequences, infarcts in the territory of the anterior spinal artery preferentially affect the gray matter. In the acute phase, the T2 hypersignal is fine, with a pencil appearance on a non-edematous initial marrow [16].

Regardless of the treatment of the cause, the treatment of spinal cord infarctions is symptomatic (particular attention to skin care intended to prevent pressure sores; sphincter care in the acute phase to avoid infectious phenomena: intermittent probing). Rehabilitation is the mainstay of treatment in these patients; drug and non-drug therapies aimed at limiting the extension of spinal cord infarction have never been the subject of randomized trials (hypothermia, anesthetic agents, calcium channel blockers, N-methyl-D-aspartate receptor antagonists, free radical scavengers, high doses of corticosteroids). It should be noted that the various drug and non-drug therapies used to limit the spread of the infarction have never proven their effectiveness. However, early implementation of appropriate rehabilitation is essential [4]. Thus, thrombolytic therapy for spinal cord ischemia is still under investigation. Few case reports demonstrate the success of thrombolytic therapy. The potential barrier to thrombolytic therapy in these scenarios is initial diagnostic uncertainty, delayed diagnosis beyond the treatment window. The concomitant presence of aortic dissection, vascular malformations, and recent surgical intervention further limits the use of thrombolytic treatment. Systemic corticosteroids have shown improved neurological outcomes in patients with acute non-penetrating traumatic spinal cord injury, where they have not yet been adequately studied in acute ischemic injury [18]. The favorable evolution marked by the regression of neck pain and treatment remains essentially symptomatic based on nursing care and rehabilitation. Its prognosis is poor, with high mortality and severe after-effects.

References