CLINICAL NOTE

Extensive spinal cord infarction due to a herniated intervertebral disk

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Anterior spinal artery syndrome may be caused by a variety of events, such as lesions secondary to bleeding and ischemia affecting the anterior portion of the spinal cord. The usual clinical picture involves diffuse or radicular pain below the level of the lesion, with loss of sensitivity on the surface but preservation of proprioception and sensitivity to vibration, plus paraparesis and loss of sphincter control. We report the case of a 39-year-old man who developed sudden neck pain radiating to the arm within a few hours of suffering tetraplegia with compromised airways. Images showed a herniated posterolateral cervical disk and extensive infarction of the cervical spinal cord. The possible causes of anterior spinal artery syndrome, including the relationship between spinal infarction and a herniated intervertebral disk, are discussed. [Emergencias 2009;21:389-392]

Key words: Cervical spinal cord infarction. Herniated intervertebral disk.

Introduction

Anterior spinal artery syndrome was first described by Spiller (1909) in a patient with thrombosis of that artery, and necropsy showed cervical-thoracic spinal cord infarction.

The arterial system of the spinal cord is formed by an anterior spinal artery and two posterior arteries with major branches arising from the vertebral, deep cervical, intercostal and lumbar arteries[1]. The anterior cerebral artery provides 70% of the vascularization of the cord. Infarction of the anterior spinal artery is more frequent than infarction of the posterior ones.

The incidence of spinal cord infarction is not known, but the event is generally considered infrequent[1-3]. In the last decade, there has been a significant increase in the number of cases published in world literature, which may be explained by a better understanding of this clinical entity and availability of magnetic resonance imaging (MRI) to confirm the diagnosis. Occlusion of the anterior spinal artery due to fibrocartilaginous embolism related to disc herniation is extremely rare. We describe a case of spinal cord infarction in the territory of the anterior spinal artery in relation to disc extrusion at the cervical level; we also review the incidence and discuss pathogenic mechanisms, diagnosis and diseases that trigger this disorder.

Case report

The patient was a 39-year-old male bricklayer, occasional smoker, with no other background of interest, or previous trauma, except for two brothers with Steinert's disease. He consulted our emergency department for severe pain of sudden onset in the cervicothoracic region on getting up that morning, radiating to the right arm, with loss of right arm dexterity which progressively spread to the left.

Neurological examination showed the patient to be conscious and oriented; he showed no alteration of cranial nerves or myotonia of the tongue, but there was progressive paresis of the right half of the body, only minimally observed in the left half. Basic laboratory test was normal. Computed tomography (CT) scan was normal. Cervical MRI showed corrected cervical lordosis with degenerative multidiscopathy and posterior disc osteophyte

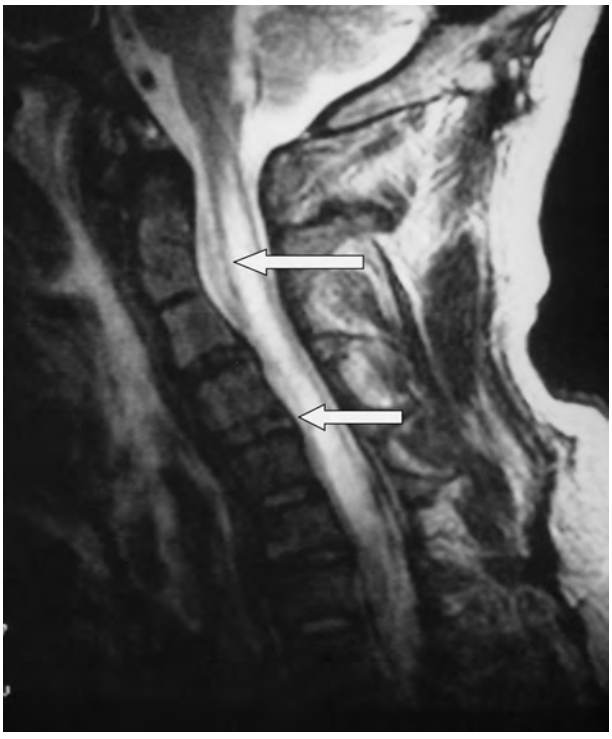

Figure 1. Magnetic resonance image 9 days after symptom onset, showing diffuse thickening of the spinal cord (arrows) from C2 to C7, with T2 hyperintense lesion.

bars causing stenosis of the canal, especially at C6-C7 which showed left posterolateral disc herniation with mild spinal deformity. These findings did not explain the current picture. The evolution of the patient in ED was unfavourable; tetraplegia ensued with respiratory compromise twelve hours later, which necessitated transfer and admission to the resuscitation unit for respiratory support. The existence of thoracic aorta aneurysm as a precipitating cause was ruled out by CT angiography.

In cervical MRI (ninth day post-infarction) there appeared in T1 a diffuse thickening of the spinal cord from C2-C7 with a hypointense linear image within it, and in T2 a hyperintense signal from C5-D1, which confirmed the diagnosis of a large cervical spinal cord infarction (Figure 1).

Neurophysiological study, performed with electromyelography (EMG) 12 days post-infarction, showed extensive involvement of C5-D1, with conservation of posterior cords confirmed by somatosensory potentials of the arms, and no signs of denervation (EMG). Lumbar puncture was strictly normal.

During his stay he received hemodynamic support with vasoactive drugs and high-dose crystalloids, medical rehabilitation treatment, with slight motor improvement of the left arm and spontaneous breathing in the fourth week of evolution. He required permanent catheterization. The patient is totally dependent for daily living activities.

Discussion

Vascular diseases of the spinal cord are less frequent than brain ischemic episodes. Vascular events are of sudden onset, disabling over time4, and prognosis is correlated with the size of the deficit at the start of the picture[5].

The etiology of spinal cord infarction is varied and unclear[6,7]. The most common cause is aortic atherosclerosis in individuals between the sixth and eighth decades of life with a history of hypertension and diabetes mellitus, and aortic surgery[8-10] (Table 1). The underlying mechanism may be the release of atheromatous material[9] and/or increased spinal fluid pressure secondary to aortic clamping[10].

Spinal cord infarction due to nucleus pulposus embolism is extremely rare[11-13]; predominantly in females (69%), with a bimodal age distribution (22 years and 60 years) and mainly cervical (69%)[11]. The pathogenesis is still unclear with the main mechanisms considered to be minor trauma[13], Valsalva manoeuvres[13], sudden movement, flexion or extension[14], although, as in our patient, there are case reports that do not include these events[15]. It has been suggested that compression of spinal cord radicular arteries, or of the spinal artery itself, against the wall of the spinal canal due to lateral herniation of the intervertebral disc may be associated with a route of entry of fibrocartilaginous material into the artery. In this context, a transient increase in intra-vertebral pressure may cause disc herniation that exceeds the mean arterial pressure, and be sufficient for part of the disc material to enter arterial circulation[12,13,16,17].

The difficulty of establishing an early diagnosis requires acting on a suspected diagnosis of spinal cord infarction due to fibrocartilaginous disease in patients with rapid onset of flaccid paralysis with areflexia for pain and temperature, with sensory preservation of arthro kinetic and vibratory stimuli in the context of a history of Valsalva manoeuvre or minor trauma, and also with unremarkable radiographic findings, which initially rules out aortic disease with a history of hypertension, diabetes mellitus, or severe trauma[17]. In children it is often associated with post-vaccination or post-infection myelopathy, or genetic disease[18], but some cases have been associated with nucleus pulposus embolism[19]. In our case, laboratory tests ruled out

Table 1. Causes of ischemia and infarction in the region of the anterior spinal artery

Unknown	Cervical subluxation
Polyarteritis	Dorsolumbar sympathectomy
Giant cell arteritis	Use of epidural morphine
Drepanocytosis	Post-irradiation
Postinfection	Hypotension
– Pneumococcus	– Cardiac arrest
– Varicella zoster	– Aortic rupture
– Schistosomiasis	– Aortic dissection
– Syphilis	
Post-vaccination	Angioma
Angiography	Cocaine abuse
Umbilical artery catheterization	Phenol neurolysis
Aortic atherosclerosis	Intervertebral disc herniation
Aortic and cardiac surgery	Arteriovenous malformations
Surgical Procedures	Esophageal sclerotherapy
– Hyperlordosis	
– Sedestation	
Aortic balloon counterpulsation	Pelvic vein thrombosis
Myeloptic neuropathy	Mitral diseases
Spinal metastases	Retrograde aortography
Trauma	Axillary plexus blockade

possible infectious, inflammatory, immune and oncologic causes.

In the past, only necropsy was used to confirm the diagnosis. Today MRI has become a first line diagnostic test[17,20-24], but has the disadvantage that in the early stages it rarely shows abnormalities[17,19,23]; the signs indicating infarction usually begin to appear between five and fifteen days after the event[17], although there are reports of cases describing MRI changes 9 hours after infarction. In the acute period, hyperintense intra-cord images can be seen in T2, attributed to the presence of spinal cord edema secondary to ischemia. In the subacute stage, there may be visible cord thickening on T1-weighted images, while T2-weighted sequences show longitudinal hyperinsity[17,22,23]. These findings usually persist over time, along with the emergence of atrophic myelomalacia areas. The possibility of early diagnosis has been suggested, by component analysis of muscular potentials, showing loss of F wave within a few hours, reflecting the decreased cellar excitability[24].

Recovery from spinal cord infarction varies widely, and several studies indicate that it largely depends on the severity of the initial deficit[5,6,8], with the greatest motor deficit appearing immediately after injury, followed by slow recovery in 60% of cases. Complete restoration of motor function is, however, unusual. Evolution tends to be more favourable in patients with unknown aetiology or secondary to an infectious process, and more dramatic in cases due to aortic diseases8. The mortality rate for patients with anterior spinal cord syndrome is 18.3%[8].

There are no studies indicating specific therapeutic measures for patients with spinal cord ischemia or infarction; to ensure consistent treatment, the physiopathology of this entity must be better understood. Animal studies suggest that hypothermia, steroids and barbiturates are effective in spinal cord protection[6,29,30], but this treatment should begin before or shortly after the ischemia to achieve the protective effect. The mechanism is uncertain: it could be attributed to conservation of cell function, maintenance of cord self-regulation or reduction of free radicals. Agents such as doxycycline that inhibit leukocyte adhesion, thus reducing reperfusion damage in the spinal cord, may reduce infarction size[31], but should be used immediately after onset of ischemia.

For reducing neurological deficits, magnesium and naloxone have been indicated; the latter drug can increase spinal cord blood flow and prevent the entry of calcium into nerve cells after spinal cord ischemia[29,32]. Rehabilitation should be carried out as early as possible[33].

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Infarto medular cervical extenso por herniación de un disco intervertebral

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El síndrome de la arteria espinal anterior posee una etiología muy variada por lesiones secundarias a cuadros hemorrágicos e isquémicos que afectan a la porción anterior de la médula espinal. La presencia de dolor difuso o radicular con pérdida de la sensibilidad superficial y conservación de la sensibilidad propioceptiva y vibratoria, junto a paraparesia y pérdida del control de esfínteres a nivel infralesional es la clínica típica del cuadro. Se presenta el caso de un varón de 39 años que presenta de forma súbita cervicobraquialgia e instauración en pocas horas de tetraplejia con compromiso respiratorio. Las pruebas de imagen demostraron la existencia de hernia de disco posterolateral cervical e infarto medular cervical extenso. Se describe la posible etiología, patogenia y la relación entre el infarto espinal y la herniación de un disco intervertebral. [Emergencias 2009;21:389-392]

Palabras clave: Infarto medular cervical. Herniación disco intervertebral.