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Sudden Paraplegia due to an Anterior Spinal Artery Syndrome during the Course of *Staphylococcus aureus* Septicemia

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Introduction

Spinal cord infarcts are rare, accounting for only 1% of all strokes, and the most recent information available concerns case reports describing clinical and radiological features, or unusual causes [1, 2]. Blood supply to the spinal cord arises from the vertebral arteries and the radiculomedullary arteries from the segmental branches of the aorta. The extrinsic intraspinal arterial network consisting of three longitudinal arteries (one anterior and two posterior) is connected by a spinal arterial plexus (vasa coronae) and supplied by a number of radiculomedullary arteries. Their number and location vary among individuals. In the adult only a few of the 31 pairs of segmental arteries are preserved as radiculo-medullary arteries that truly supply the spinal cord [3]. The anterior spinal artery and its branches – the sulcocommissural arteries – have a much less efficient collateral supply than the posterior spinal arteries, in particular in the lumbar region where, in the majority of cases, only one radiculomedullary artery exists (great radicular artery of Adamkiewicz). This territory is therefore more vulnerable to the effects of vascular disease. The typical presentation of this syndrome is a pronounced para- or tetraparesis or plegia, areflexia below the level of the lesion, atonic urinary bladder and paralytic ileus, and impaired perception of pain and temperature but preservation of sensation of touch, vibration and position [3]. Spinal cord infarction is uncommon and accounts for 5–8% of all myelopathies [4, 5]. Among the various etiologies of spinal ischemia are: (1) vessel occlusion or embolism caused by aortic diseases or surgery, vasculitis, coagulopathy or cardiac embolism, (2) watershed infarction in the case of generalized ischemia as in cardiac arrest or severe anemia, and (3) spinal cord compression following trauma, hematoma, tumor or abscess [3, 5]. We report here a case of a 62-year-old woman who presented an anterior spinal artery syndrome during the course of a *Staphylococcus aureus* septicemia.

Case Report

A 62-year-old woman, known to have hypertension, obesity and common uncomplicated low back pain visited her general practitioner complaining of a new episode of acute low back pain

with no irradiation to the lower limbs. Her doctor noticed no neurological deficits and proposed an NSAID gluteal intramuscular injection. The patient noticed no relief but 3 days later developed fever, diarrhea and dyspnea for which she was admitted to the emergency ward of our hospital. On admission, she presented with fever (38°C), tachyarrhythmia (atrial fibrillation), hypertension (220/140 mm Hg), tachypnea and dyspnea. Chest auscultation and percussion revealed a left basal pneumonia and pleural effusion. Spinal percussion was painless. Paraclinical examinations confirmed left basal pneumonia (chest X-ray) and showed severe inflammatory syndrome (C-reactive protein: 400 mg/l, 3,809 nmol/l). The full blood count demonstrated leukocytosis (21.5 [10⁹/l]) but with normal numbers of red blood cells and platelets. Prothrombin time (PT) was low (60%, INR: 1.2) and activated partial thromboplastin time (aPTT) was high (31 s). Blood cultures taken at that time subsequently confirmed a *S. aureus* infection. Empirical antibiotic therapy (ceftriaxone and clarithromycin) was initiated. A thoraco-abdominal computed tomography confirmed left basal pneumonia and bilateral pleural effusion but no other infectious sites in particular in the spine. The pleural effusion was drained and the same bacteria were found. No anticoagulation treatment was initiated for the atrial fibrillation because it resolved itself within the first 24 h. Transesophageal echocardiography revealed no signs of endocarditis or intracardiac thrombus.

On the third day after admission, the patient developed a sudden paraplegia associated with impaired perception of pain and temperature and areflexia below the T5 level as well as sphincter dysfunction. Sensation of touch, vibration and position was preserved. A MRI scan was obtained quickly and demonstrated an extensive centromedullary hypersignal on T₂-weighted images from the T₂ level to the medullary cone associated with epidural contrast enhancement but no spinal compression by an epidural abscess or spondylodiscitis (fig. 1). Lumbar puncture displayed marked pleocytosis (906 cells/mm³, 62% polymorphonuclear leukocytes, 12% lymphocytes, 17% monocytes) with elevated total protein (30.3 g/l) and decreased glucose CSF/blood ratio (0.22) suggesting bacterial meningitis. Probably because of the treatment with antibiotics initiated 3 days earlier, the cerebrospinal fluid culture remained sterile. At that time, the treatment was changed to vancomycin, amikacin and aciclovir and associated with high-dose intravenous methylprednisolone. Since no epidural abscess was found there was no indication for surgical decompression or drainage. However, the inflammatory syndrome persisted and no neurological improvement took place. Therefore, a second MRI scan was obtained at day 23 which showed a persistent extensive centromedullary hypersignal on T₂-weighted images as well as epidural abscesses both anteriorly and posteriorly (fig. 1) and a spondylodiscitis at the C7-D1 level.

Surgical decompression and drainage of the posterior epidural abscess was performed and rifampicin and trimethoprim-sulfamethoxazole were added to the ongoing treatment. The in-

Fig. 1. Magnetic resonance imaging at various times after symptoms onset on the sagittal and transversal planes (inserts). Upper row: T₂-weighted images (T₂-WI). Lower row: T₁-weighted gadolinium-enhanced images (T₁-WI + Gd). Immediately after onset of symptoms (day 0), an intramedullary T₂ hypersignal was described (white open arrowheads) associated with epidural contrast enhancement (white arrowheads). At day 23, the intramed-

ullary T₂ hypersignal was still present (white open arrowheads) and both anterior and posterior epidural abscesses (white arrows) and a spondylodiscitis at C7-D1 level (white asterisk) appeared. At day 43, note the persistence of the intramedullary T₂ hypersignal (white open arrowheads) of the anterior epidural abscess (white arrows) and of the spondylodiscitis (white asterisk) when the posterior abscess was correctly drained by multiple laminectomy.

flammatory syndrome resolved and some neurological recovery limited to movements against gravity of the distal right lower limb took place. A third MRI scan obtained at day 43 confirmed the centromedullary hypersignal, showed the resolution of the posterior epidural abscess but persistence of the anterior one, which, according to our neurosurgeon, required no further surgical treatment. The patient was therefore discharged to the neurorehabilitation unit.

Discussion

The main etiology of anterior spinal artery syndrome during sepsis is compression of the anterior spinal artery by an epidural abscess, the most common causative organism being *S. aureus*. Epidural abscesses occur mainly during the course of other infections as skin abscess, vertebral osteomyelitis or discitis and sepsis and after trauma or spine surgery. Diabetes mellitus is often an associated condition. Although conservative therapy has been shown to be safe and effective in selected cases, surgical

drainage combined with antibiotics remains the treatment of choice [6].

In the present case, the epidural abscesses were certainly not responsible for the anterior spinal artery syndrome, since spinal cord compression occurred days after the spinal ischemia developed (fig. 1). Atrial fibrillation has been shown to be a possible etiology in spinal cord ischemia [3], although it was of relatively short duration in the present situation. Coagulopathies have also been described as a cause of spinal infarction [3, 5], and it is interesting to note that the PT was spontaneously low, suggesting a coagulation cascade activation. Unfortunately, fibrinogen and fibrin monomers were not tested in the acute phase. Finally, as epidural inflammation occurred early in the course of the disease, as highlighted by epidural contrast enhancement on the first MRI scan (day 0, fig. 1), we could hypothesize that local vascular inflammation contributed to arterial thrombosis, as previously described in other cases of vasculitis [3]. In a recent series, the Study Group on Spinal Cord Infarction of the French Neurovascular Society demonstrated that a definite etiology was only found in 11 of 28 included patients (39%) with spinal cord infarction [2]. In the present case, it was also difficult to definitely establish the cause of the anterior spinal artery syndrome, which could have been induced by multiple mechanisms, but certainly not by the epidural abscesses.

There is no specific approved treatment for acute spinal cord infarctions mainly because of the absence of controlled trials due to the relative rarity of the condition [7]. The use of anticoagulation, antiplatelets, high-dose steroids or spinal cord revascularization has been proposed, but without evidence of definitive benefits in the acute phase. Nevertheless, adequate acute treatment of patients with spinal cord disease such as managing specific medical complications and initiating appropriate rehabilitation may be of value for the final outcome. In the present case, spinal infarction occurred prior to the development of epidural abscesses which explain why late drainage of the posterior epidural collection did not induce any improvement of the neurological symptoms. Factors of poor long-term functional prognosis are severity of the initial neurological deficits [2, 8], presence of proprioceptive impairment at onset [2] and extension of the infarction to the spinal cone [9]. All three were present in this case, which could explain the poor recovery and the limited value of the methylprednisolone treatment. Anticoagulation was not indicated because cardiac rhythm returned to sinus before 24 h after the onset of atrial fibrillation and not all the criteria for disseminated intravascular coagulation were present.

Although the main cause of anterior spinal artery syndrome during sepsis is compression of the anterior spinal artery by an epidural abscess, as in the present case, other etiologies have yet to be evoked. Neurological symptoms suggesting myelopathy in the context of sepsis require a rapid MRI imaging to exclude epidural abscess. Nevertheless, other causes such as atrial fibrillation, procoagulant states or local vasculitis should be evoked in the absence of evident signs of spinal compression. Further placebo-controlled multicenter studies are needed to evaluate new treatments dedicated to limit the extent and the consequences of spinal cord ischemia.

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Sequential Bilateral Medial Medullary Infarction due to Vertebral Artery Dissection

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Dissection of the vertebral artery (VA) can be a cause of medullary infarction [1–5]. Bilateral medial medullary infarctions usually occur simultaneously at the time of initial onset of a stroke [6, 7]. There has been one previous report of a case of sequential bilateral medial medullary infarcts with atherothrombotic etiology [8]. We present a patient with sequential bilateral medial medullary infarcts caused by VA dissection.

Case Report

A 55-year-old man was admitted due to acute vertigo with nausea and recurrent right-sided weakness. He was at that time a heavy smoker and had a long-standing history of hypertension. He became aware of abrupt right-sided weakness and nausea. Six