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Spinal Cord Ischemia: Clinical and Imaging

Patterns, Pathogenesis, and Outcomes in 27 Patients

THESE

Préparée sous la direction du Professeur Julien Bogousslavsky jusqu'au 26 avril 2006, puis sous la direction du Professeur Paul-André Despland,

et présentée à la Faculté de Biologie et de Médecine de l'Université de Lausanne pour l'obtention du grade de

DOCTEUR EN MEDECINE

par

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Lausanne

2007

REMERCIEMENTS

L'auteur tient à remercier chaleureusement le Dr Alain Carruzzo pour son aide intense et ses précieux conseils tout au long de la réalisation de ce travail.

RESUME

Introduction: l'histoire naturelle et la physiopathologie des infarctus de la moelle épinière restent largement inconnues. En effet, la plupart des études cliniques portent sur des patients qui ont souffert d'infarctus médullaire secondaire à des chirurgies aortiques ou des hypotensions prolongées.

Méthode: ce travail analyse les données cliniques, le laboratoire, l'imagerie (IRM) et l'évolution de 27 patients souffrant d'infarctus de la moelle épinière admis dans le service de Neurologie du CHUV. Parmi ces patients, il y avait 11 hommes et 16 femmes (âge moyen de 56 ans, tranche d'âge de 19 à 80 ans).

Résultats: dix patients (37%) souffraient d'infarctus de l'artère spinale antérieure, 4 (15%) d'infarctus unilatéraux antérieurs, 4 (15%) unilatéraux postérieurs, 3 (11%) d'infarctus centraux, 2 (7%) d'infarctus des artères spinales postérieures, 2 (7%) d'infarctus transverse tandis que 2 patients présentaient des tableaux cliniques inclassables. Vingt patients (74%) n'avaient pas d'étiologie identifiable. Les patients avec infarctus centraux ou transverses présentaient fréquemment (40%) des artériopathies périphériques et tous les infarctus transverses survenaient à la suite d'hypotensions artérielles prolongées. Le début de tous les autres types d'infarctus était associé à des facteurs mécaniques ($p=0.02$) et ces patients avaient fréquemment des pathologies du rachis ($p=0.003$) au niveau de la lésion médullaire. Dans ces cas, les données cliniques suggèrent une lésion d'une racine nerveuse au niveau de l'infarctus médullaire compromettant mécaniquement le flux de son artère radiculaire. L'évolution clinique était généralement favorable, seuls 13 patients (48%) présentaient une atteinte significative de la marche à la sortie de l'hôpital.

Conclusion: ce travail montre qu'il existe 2 types principaux d'infarctus de la moelle épinière : d'une part les infarctus dans le territoire d'une artère radiculaire (infarctus de l'artère spinale antérieure, des artères spinales postérieures et infarctus unilatéraux) et d'autre part les hypoperfusions régionales globales de la moelle épinière (infarctus centraux et transverses). Chacune de ces 2 catégories d'infarctus ont des caractéristiques cliniques, radiologiques, physiopathologiques et pronostiques distinctes.

ORIGINAL CONTRIBUTION

Spinal Cord Ischemia

Clinical and Imaging Patterns, Pathogenesis, and Outcomes in 27 Patients

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Background: The natural history and pathogenesis of ischemic spinal cord infarction remain largely unknown because most clinical studies have included mostly patients with ischemic lesions associated with aortic surgery or prolonged arterial hypotension.

Objective: To assess the pathogenetic mechanisms and outcomes of these cord infarctions based on clinical findings and spinal vascular anatomy.

Design: Retrospective review.

Patients: We analyzed the clinical, laboratory, imaging, and outcome data for 27 patients with acute spinal cord infarction admitted between 1990 and 2003. There were 11 men and 16 women (age range, 19-80 years [mean age, 56 years]).

Results: Ten patients had anterior spinal artery patterns, 4 each had anterior and posterior unilateral patterns, 3 had central patterns, and 2 each had posterior spinal artery patterns, transverse syndromes, and unclassifiable clinical pictures. Twenty patients had no identifiable etiology. Patients with a central or transverse infarct showed a high frequency of peripheral vascular disease, and all transverse infarcts occurred following prolonged arterial hypotension. The onset of all other infarcts was associated with mechanical triggering movements ($P=.02$), and these patients frequently had diseases of the spine ($P=.003$) at the level of the spinal lesion, with the clinical data suggesting root involvement at the level of the spinal cord lesion and pointing to mechanical injury of a radicular artery. The outcomes were favorable, with only 13 patients showing significant gait impairment on leaving the hospital.

Conclusions: There are 2 main types of spinal cord ischemia: (1) radicular artery territory infarct (bilateral anterior or posterior spinal artery infarcts and unilateral infarcts) and (2) extensive spinal cord hypoperfusion (central and transverse infarcts). Each type has characteristic clinical, imaging, pathogenetic, and prognostic features.

Arch Neurol. 2006;63:1113-1120

SPINAL CORD INFARCTION IS much less frequent than cerebral infarction, accounting for only 1% of all strokes.[1] Because of this, few large clinical investigations have been carried out, and most have dealt with spinal cord ischemia due to aortic surgery; therefore, the pathogenesis and natural history of spontaneous or nonsurgical spinal cord infarctions remain largely unknown. The objective of this study was to assess the pathogenetic mechanisms and outcomes of these cord infarctions based on clinical findings and spinal vascular anatomy.

The spinal cord is supplied by 1 anterior and 2 posterior spinal arteries, which extend longitudinally in a variable fashion. Rostrally, these arteries originate from the V4 region of the vertebral arteries and anastomose at the level of the cone.[2] At many levels, they receive supply from the radicular arteries, which enter the canal along with the nerve roots. The anterior spinal artery gives rise to the central arteries, each of which enters the spinal cord to supply the anterior horn and the anterior part of the lateral column on the left or right side at each level. The spinal arteries are connected by a pial plexus that surrounds the spinal cord, and the posterior spinal arteries may be linked together[3,4] (**Figure 1**). Each radicular artery supplies a separate functional region of the spinal arteries, particularly the anterior spinal artery.[2] The first region extends from C1 until T3 and is supplied at the C3 level from the vertebral arteries[5] and at the level from C6 until C7 from the cervical ascending arteries.[3,5] The second region extends from T3 until T7 and sometimes receives a branch from the intercostal

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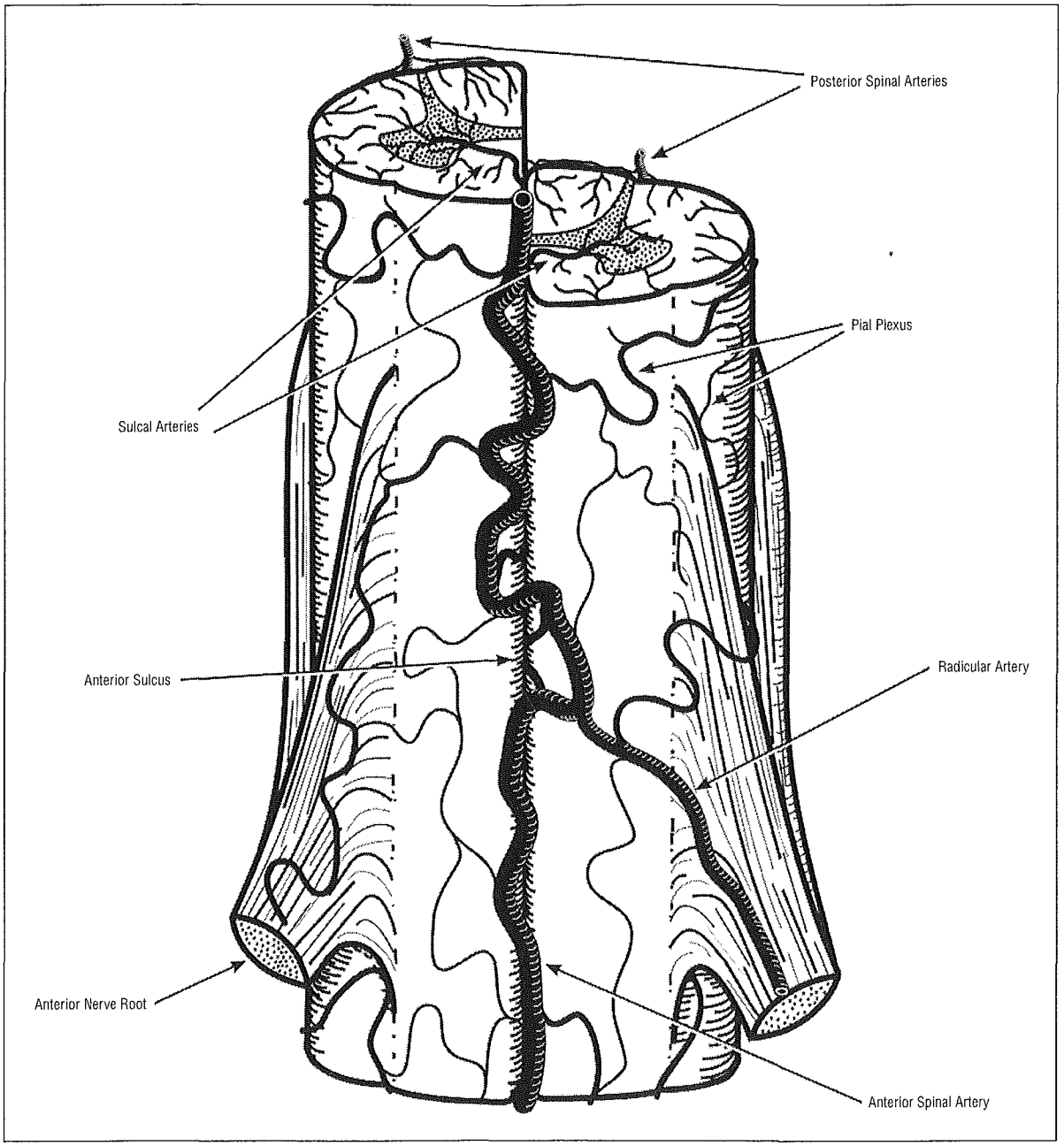

Figure 1. Regional vascular anatomy of the spinal cord.

artery at the T7 level.[1] The third region extends from T8 to the cone and receives a branch (Adamkiewicz artery) from the intercostal artery, most frequently between T9 and T12.[6] There is sometimes a cone artery originating from the internal iliac artery (Desproges-Gotteron artery) at the L2[7] or L5[8] level (**Figure 2**).

METHODS

We studied 72 patients admitted for investigation of acute myelopathy between 1990 and 2003. The patients underwent complete etiologic investigations.

The laboratory workup consisted of complete blood cell count; erythrocyte sedimentation rate; electrolyte, glucose, creatinine, and cholesterol levels; liver function test; protein electrophoresis; coagulation tests; electrocardiogram; and thoracic radiography. The infection workup consisted of screening serologically for syphilis (VDRL test), Lyme borreliosis, herpes viruses, human immunodeficiency virus, and human T-cell lymphotropic virus. Patients were screened for antinuclear and antineutrophil cytoplasmic antibodies. The cerebrospinal fluid was analyzed for cell count; glucose and protein levels; and oligoclonal bands and was screened for Lyme borreliosis, syphilis, and herpes virus antibodies.

Spinal magnetic resonance imaging (MR imaging) (Siemens Vision and Symphony 1.5-T imager; Siemens, Erlangen,

Germany) was performed on all patients. The patients were screened for aortic disease (computed tomographic scan or ultrasonography) and vertebral dissection (angio–computed tomographic scan or angio–MR imaging) based on the level of involvement. Cardiac ultrasonography was used to screen the patients for a cardioembolic source.

We included 27 patients with spinal cord ischemia. These patients displayed acute myelopathy (onset within minutes to a few hours, in some cases); the workup excluded other causes (compressive, traumatic, infectious, or inflammatory). In most cases, the MR imaging showed a well-defined lesion corresponding to arterial territory. The combined clinical and imaging data were used to determine the level and extent of the infarct. We analyzed the clinical features of the different stroke syndromes and correlated them with the imaging data.

The median follow-up time to discharge from the hospital was 12 days (range, 1-42 days). Fisher exact test was used to examine statistical correlations. Statistical significance was set at $P<.05$.

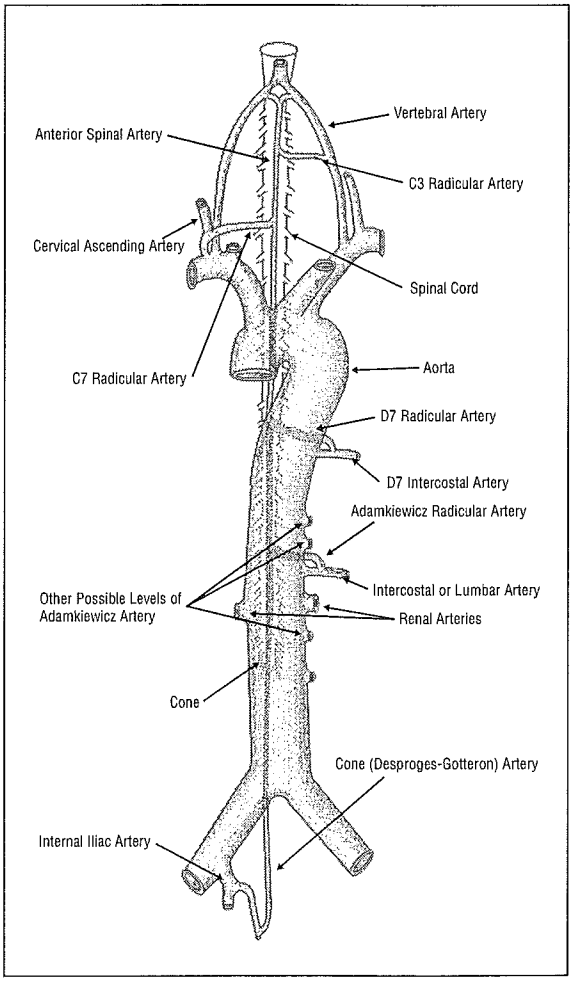

Figure 2. Arteries supplying the spinal cord.

RESULTS

PATIENTS

Our 27 patients (11 men and 16 women) had a mean age of 56 years (age range, 19-80 years). Patient details about sex, age, and cardiovascular risk factors are summarized in **Table 1**. Fewer than one third of the patients had cardiovascular risk factors.

CLINICAL FINDINGS

Of the 27 patients, 10 (37%) had anterior spinal artery patterns, 4 (15%) had anterior and 4 (15%) had posterior unilateral patterns, 3 (11%) had central patterns, 2 (7%) had posterior spinal artery patterns, and 2 (7%) had transverse syndromes, while 2 (7%) had unclassifiable clinical pictures. The manifestations usually developed within about 2 minutes (within a few hours in a few cases). In 5 patients, they appeared in 2 acute stages. Two of these 5 patients had a deficit that was initially unilateral and then became bilateral, while in another patient the deficit increased secondarily. The 2 remaining patients experienced transient ischemic attacks (TIAs) before spinal cord infarction (1 patient experienced several TIAs per year for 6 years). The TIAs had the same motor features as the definitive stroke. In these cases of progressive stroke or TIAs, the infarct was frequently localized at the cervical level (4 of 5 cases).

Sixteen patients (59%) reported back or neck pain at the onset of symptoms, and this was always localized at the level of the spinal cord lesion. In 13 (81%) of these patients, the pain radiated in a radicular fashion (bilaterally in 11 and unilaterally in 2). Three patients with posterior bilateral or unilateral syndrome experienced paresthesia. The initial back pain and paresthesia resolved spontaneously within days.

The topography of the lesions was associated with specific clinical pictures. These are summarized in **Table 2**.

Table 1. Demographics and Cardiovascular Risk Factors Among 25 Patients

Variable	Anterior Spinal Artery Infarct (n = 10)	Anterior Unilateral Infarct (n = 4)	Posterior Unilateral Infarct (n = 4)	Central Infarct (n = 3)	Posterior Spinal Artery Infarct (n = 2)	Transverse Infarct (n = 2)
Male-female ratio	3:7	1:3	1:3	3:0	1:1	2:0
Mean age, y	55	64	57	50	68	44
Cardiovascular risk factors						
Arterial hypertension	3	1	1	1	0	1
Hypercholesterolemia	2	1	2	0	0	1
Tobacco	0	1	1	1	1	0
Type 2 diabetes mellitus	1	1	1	0	0	0

Table 2. Clinical Features Associated With the Stroke Syndromes

Stroke Syndrome	Feature
Anterior spinal artery infarct	Bilateral motor deficit with spinothalamic sensory deficit
Anterior unilateral infarct	Hemiparesis with contralateral spinothalamic sensory deficit
Posterior unilateral infarct	Hemiparesis with homolateral lemniscal sensory deficit
Central infarct	Bilateral spinothalamic sensory deficit without motor deficit
Posterior spinal artery infarct	Bilateral motor deficit with lemniscal sensory deficit
Transverse infarct	Bilateral motor deficit with complete sensory deficit

LABORATORY AND IMAGING FINDINGS

The laboratory workup did not show any abnormalities except for previously identified diabetes mellitus and hypercholesterolemia. Cerebrospinal fluid analysis revealed an increased protein concentration in 12 patients (44%), but none had pleocytosis or intrathecal synthesis of immunoglobulins. There were no cases of vertebral dissection or a cardioembolic source. One patient had an aortic endoprosthesis.

Magnetic resonance imaging showed an ischemic lesion defined as a well-demarcated T2-weighted hyperintensity matching an arterial territory of the cord (**Figure 3**) in 18 patients (67%). Pathologic MR imaging findings were more frequent in the patients with severe weakness (less than M4) than in those with less severe weakness ($P=.01$). Only 1 patient (with a posterior spinal artery infarct) had a vertebral body infarction, at the upper limit of his cord lesion. No patient displayed signs of arteriovenous fistula or malformation.

Except in patients with central and transverse infarcts, the infarcts were grouped in the following 4 distinct spinal segments: from C1 until C3, C4 until C7, T3 until T7, and T8 until L1. None of the patients had spinal cord infarction between T1 and T3.

ETIOLOGY

No identifiable cause was found in 20 patients (74%). Three patients had experienced a prolonged episode of arterial hypotension just before the infarction. The other most common factor was disk prolapse or herniation (3 patients), diagnosed in all cases by MR imaging; in 1 patient there was a median disk prolapse at the level of the infarct, with no compression of the spinal cord (**Figure 4**A), and in 2 patients the disk herniation was lateral and compressed the root at the level of the arterial lesion (Figure 4B). Among the other potential causes, 1 patient had an aortic endoprosthesis, and the 2 patients with a transverse infarct had experienced prolonged arterial hypotension.

The cases in which the spinal arteries were clearly exposed to mechanical tension associated with spine mobility, spinal diseases, and infarct-triggering movements and those with prolonged hypotension and symptomatic arteriopathy (peripheral or coronary) are summarized as a function of stroke syndrome in **Table 3**. Spinal disease, seen in 12 patients, was associated with chronic back or neck pain with radicular radiation in 5 patients and with spine compression fractures in 4 patients, while the other 3 patients showed spondylolisthesis, chronic arachnoiditis, or chronic cervical disk protrusion. In 11 of these 12 patients, the spinal disease was at the level of the cord infarct.

The ischemic symptoms developed immediately after a movement in 13 patients (48%). In 7 patients, this was a movement of the back (an extension in 4 of them), while in 3 patients it was an arm movement, and in the remaining 3 patients it was a Valsalva maneuver or a gait initiation. The level of the spinal cord lesion corresponded with the level of mechanical stress in the spine.

Anterior and posterior spinal artery infarcts and unilateral infarcts were associated with mechanical triggering movements ($P=.02$) and with acute and chronic spinal disease ($P=.003$). None of the patients with central or transverse infarcts reported a triggering movement or spinal disease, but these patients had a higher frequency of underlying arteriopathy (2 [40%] of 5 patients) than patients with other stroke syndromes (1 [5%] of 20 patients).

OUTCOMES

The outcome was generally favorable, with complete or incomplete recovery in 19 (70%) of 27 patients. Only 13 patients (48%) showed significant gait impairment on leaving the hospital. The clinical outcomes of the patients are summarized in **Table 4**. Motor deficits showed a higher frequency of recovery (17 of 19 patients who showed recovery) than sensory or sphincter deficits.

The 3 patients with an anterior spinal artery infarct at the cervical level and bibrachial paresis regained normal function in both arms. One of these patients developed unilateral amyotrophy localized in the territory of the C8 nerve root.

The patients received various treatments. Eight patients had no specific treatment, while 9 patients were treated with antiplatelet therapy (300 mg/d of aspirin), 5 patients with corticosteroids, 3 patients with antiplatelet therapy plus corticosteroids, and the remaining 2 patients with anticoagulation therapy.

COMMENT

IMAGING

The MR imaging showed a cord lesion in 18 (67%) of our patients. Sagittal and axial T2-weighted images were considered most useful. The radiological patterns for the different stroke syndromes are shown in Figure 3. These patterns correlated well with data from autopsy and imaging studies.[9-13] Infarction of a vertebral body has been described as a useful confirmatory sign of cord infarction,[14,15] but in our series it was rare (1 of 18 patients). Vertebral lesions are probably more frequent in surgical series in which the patients undergo resection of multiple branches of the aorta.

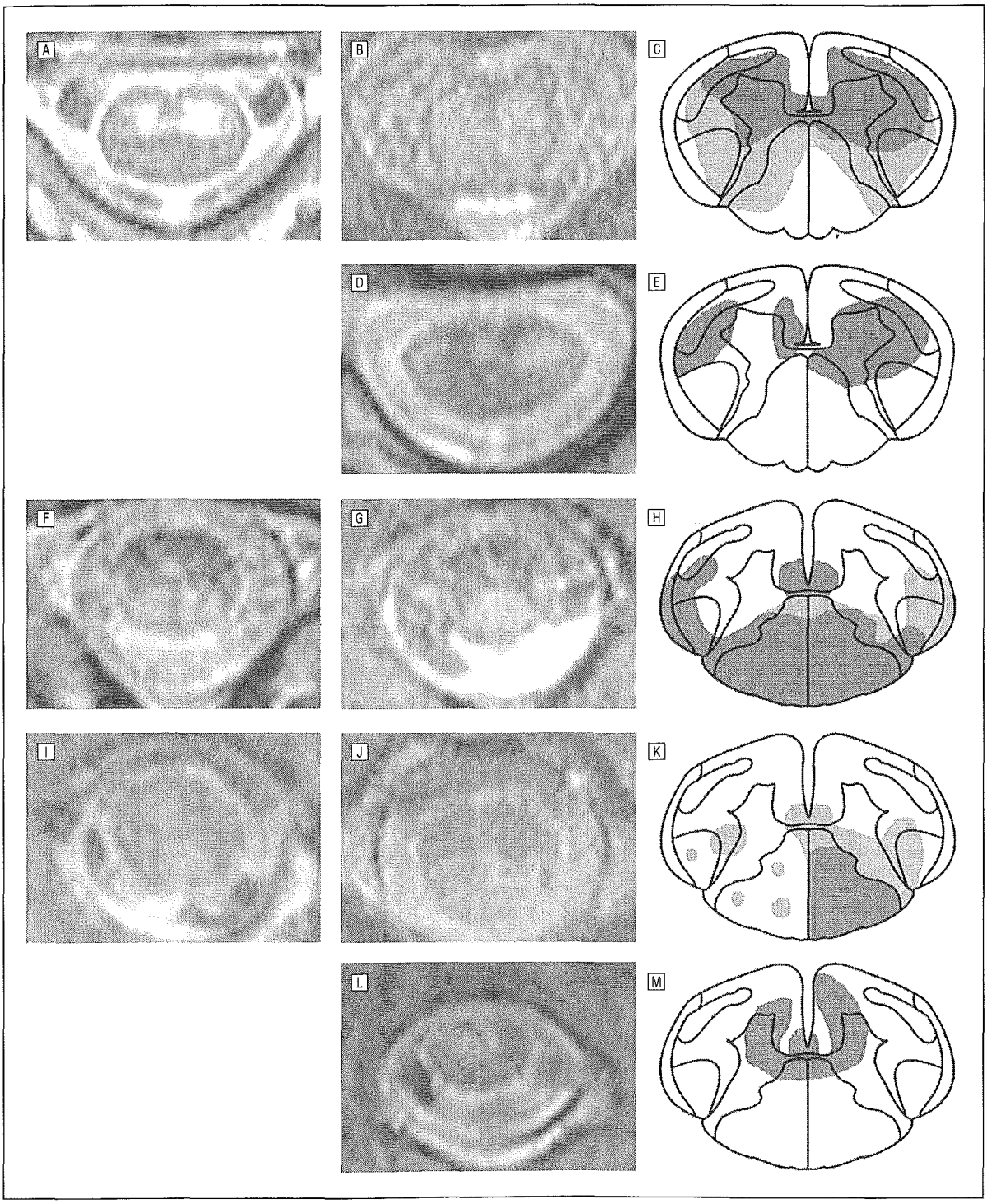

Figure 3. Transverse T2-weighted fast spin-echo magnetic resonance imaging sequences (repetition time, 4000-5000 milliseconds; echo time, 99-125 milliseconds; 3-mm [cervical] and 4-mm-[thoracic] section thicknesses; number of signals required, 2-8; with a 512×300-pixel matrix) of the different spinal cord infarction syndromes and their anatomic locations. The extension of the lesions for each syndrome is represented schematically on the right (C, E, H, K, and M). A through C, Anterior spinal artery infarct. The lesion may be limited to the anterior horns and the surrounding white matter (A [dark gray in C]) or extend to the posterior horns (B [light gray in C]). D and E, Anterior unilateral infarct. The patient first displayed an anterior spinal artery syndrome with respiratory failure, which evolved to a unilateral syndrome with normal breathing. In addition to the left-sided lesion (D), there were small anterolateral T2-weighted hypersignals on the left and right sides of the spinal cord (dark gray in E). F through H, Posterior spinal artery infarct. The lesion may be limited to the posterior columns (F [dark gray in H]) or extend into the posterolateral regions (G [light gray in H]). I through K, Posterior unilateral infarct. The lesion may be limited to the posterior column (I [dark gray in K]) or extend into the posterolateral region (J [light gray in K]). The patient had numerous spinal transient ischemic attacks, and the main lesion was surrounded by smaller scattered lesions (J [light gray in K]). L and M, Central infarct. The lesion (L) was restricted to the border of the anterior sulcus (dark gray in M).

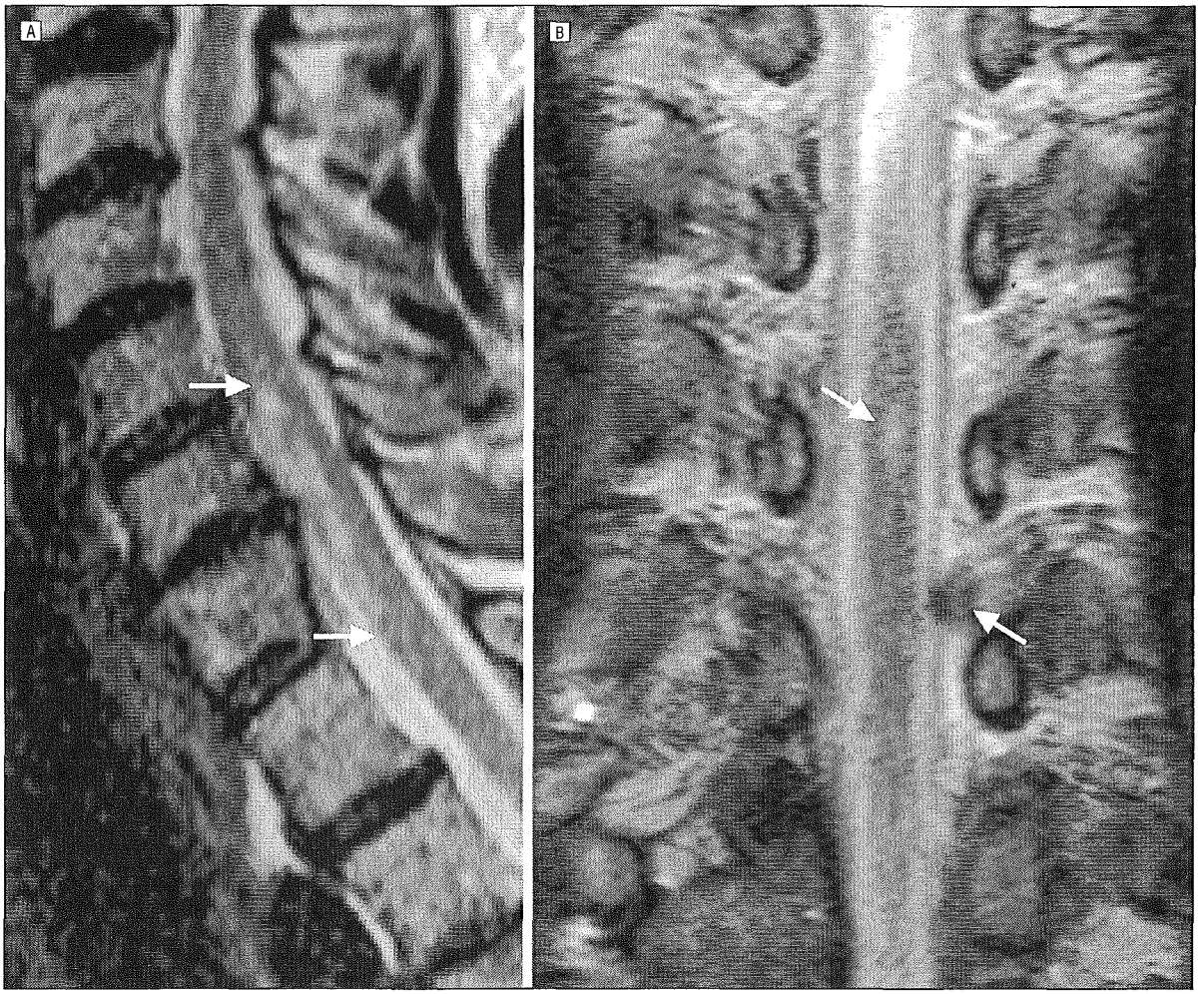

Figure 4. Compression of the radicular artery (anterior spinal artery syndrome caused by a median disk prolapse). A, Sagittal T2-weighted fast spin-echo magnetic resonance imaging sequence (repetition time, 4700 milliseconds; echo time, 112 milliseconds; 3-mm-section thickness; number of signals required, 3; with a 1024×360-pixel matrix) showed a lesion in the territory of the anterior spinal artery. Arrows indicate the extent of the infarct. B, Coronal section of another patient with a lateral herniated disk (right arrow) at the level of the radicular artery corresponding to the cord infarction (left arrow).

Table 3. Pathogenetic Characteristics of the Stroke Syndromes*

Characteristic	Anterior Spinal Artery Infarct (n = 10)	Anterior Unilateral Infarct (n = 4)	Posterior Unilateral Infarct (n = 4)	Central Infarct (n = 3)	Posterior Spinal Artery Infarct (n = 2)	Transverse Infarct (n = 2)
History of arteriopathy	0	0	1	1	0	1
Prolonged hypotension	0	0	1	0	0	2
Chronic spinal disease	7	1	2	0	2	0
Triggering movement	6	3	2	0	2	0

*Column totals may exceed the total number of patients in a group because some patients exhibited more than 1 characteristic.

Diffusion-weighted imaging offers promising techniques to detect early cord ischemia,[16,17] but the quality of echo-planar diffusion-weighted images is reduced because of susceptibility artifacts due to the heterogeneous magnetic environment of the spine that can create false-positive results. Line scan diffusion imaging is less sensitive to artifacts,[18,19] but this capability is available from only a single main manufacturer of MR imaging machines.

PATHOGENESIS

In our series of patients, 2 different pathogenetic mechanisms were distinguished. These included (1) those occurring after a mechanical triggering factor (bilateral anterior or posterior spinal artery infarcts and unilateral infarcts) and (2) infarcts occurring after prolonged hypotension or in arterial insufficiency (central and transverse infarcts).

Table 4. Outcomes for the Stroke Syndromes

Outcome	Anterior Spinal Artery Infarct (n = 10)	Anterior Unilateral Infarct (n = 4)	Posterior Unilateral Infarct (n = 4)	Central Infarct (n = 3)	Posterior Spinal Artery Infarct (n = 2)	Transverse Infarct (n = 2)
Complete recovery	0	0	0	1	0	0
Incomplete recovery	7	3	4	2	2	0
No improvement	2	1	0	0	0	2
Worsening	1	0	0	0	0	0
Aid required for walking	6	0	0	1	1	2

The most probable cause of infarcts occurring after hypotension or arterial insufficiency seems to be global hypoperfusion of the spinal cord. Zülch and Kurth-Schumacher[7] and Nagashima and Shimamine[20] showed that a transverse ischemic cord lesion is reduced to a central lesion at its superior and inferior extremities, so central and transverse lesions probably represent 2 poles of a continuum. The infarcts involve the territories of several spinal arteries in the thoracolumbar region. This vulnerability to hypoperfusion is explained by the local high density of motoneurons[21] and by the frequency of atherosclerosis in the aorta and iliac arteries. In an autopsy series, aortic atherosclerosis showed a correlation with lacunar infarction in the central region.[22]

The most probable cause of infarcts occurring after mechanical stress in our series was a lesion of the radicular arteries, as proposed by other authors.[11,23] In contrast to central and transverse infarcts, the localization of the infarction corresponded to the territories of the radicular artery and spinal arteries. Unilateral forms are explained by the incomplete linking of posterior systems[24] and by duplication of the anterior system.[3,4] The T1 to T4 region (which is not supplied by the radicular arteries) was spared, as in a previous study.[13] The initial pain showed a radicular distribution in many patients, sometimes with clear signs of a radicular lesion at follow-up (selective unilateral C8 amyotrophy). Vertebral body infarctions (which can be associated with cord infarction, as in a patient in our series) are another sign of radicular artery occlusion.[14,15]

Unlike the cerebral vessels, the spinal arteries run along a mobile structure, which makes them prone to mechanical damage.[10] The relevance of spinal diseases in our patients was difficult to assess because of the great variety of the pathologic conditions. In an asymptomatic population, Jensen et al[25] found that 27% had disk protrusion. However, if one includes chronic spinal diseases and infarcts due to disk lesions, mechanical factors were present in 15 (75%) of 20 patients with anterior and posterior spinal artery or unilateral stroke syndrome. This frequency is significantly higher ($P = .003$) than that in transverse and central infarcts. Moreover, the infarction coincided with the level of the spinal disease. Chronic compressive diseases induce foraminal and subarachnoid fibrosis, which reduces the collateral blood flow, endangering the mechanisms of compensation in the event of ischemia.[26] Although it is described as a classic mechanism of spinal cord damage in compressive myelopathy,[27] dysfunction of venous drainage leads to a subacute progressive course.[28] Therefore, it seems unlikely that this mechanism played a significant role in our acute cases.

Movement of the spine can lead to acute vascular compression, as demonstrated in 3 of our patients. In our series, the target of this compression appeared to be a radicular artery, but involvement of the anterior spinal artery[29] or lumbar artery[30] has also been demonstrated. New techniques of positional imaging should more easily demonstrate dynamic vascular compressions in the future.[31] In patients with damaged facets of the cervical or dorsal column leading to hyperlaxity, the spinal cord might be injured directly from mechanical compression. This usually occurs in traumatic settings or subacutely in predisposing conditions (eg, familial dystonia or spondylodiskitis sequela). In such cases, the imaging can be nonspecific, showing only spinal cord swelling.[32] Although none of our patients had a predisposing condition or a recent substantial trauma, we cannot exclude that such a mechanism might have played some role because we did not formally rule it out with dynamic imaging.[33]

Occlusion of spinal arteries and veins by fibrocartilaginous emboli has been reported after spine movement.[34] In this situation, one typically observes a free interval between the painful movement and the onset of neurological manifestations. The evolution is characteristically progressive without improvement and almost always lethal.

Transient ischemic attacks occurred at the cervical level in our patients. This region is more resistant to ischemia because of its intense vascularization. Transient ischemic attacks could result if vascular compromise resolves in this region. Spinal claudication, first described by Dejerine[35] in 1911, occurs if TIAs become repetitive, as in a patient in our series who experienced TIAs every time he worked with his arms (gardening), creating tension in his cervical spine. In another patient who experienced TIAs, the MR imaging showed a particular pattern with several small lesions surrounding the main infarction (Figure 3J). Transient ischemic attacks can also be observed in the progressive course of dural fistula.[36] In this case, these phenomena are attributed to venous hypertension[36] during a Valsalva maneuver or to arterial steal.[37] Although there were no clinical indications of dural fistula in our series (preexisting chronic myelopathy or worsening during the follow-up period) or any suggestive MR imaging sign, we cannot exclude that this particular mechanism may occasionally play a role.

COURSE AND OUTCOMES

Previous reports have shown that the prognosis of spinal cord infarction following aortic surgery is worse than that for other infarcts.[38-40] During hospitalization in our series, we generally observed a partial recovery, and gait

was infrequently compromised. Unilateral infarcts had a particularly favorable prognosis.

There are no clear guidelines for the treatment of spinal strokes. Animal investigations have shown some benefit with certain agents, such as prostaglandins, nimodipine, naloxone hydrochloride, adenosine, thiopental sodium, magnesium, *N*-methyl-D-aspartate antagonists, or corticosteroids,[41] but there have not been any prospective clinical studies, to our knowledge. de Sèze et al[42] did not note any difference in the clinical course in a retrospective study of patients treated with corticosteroids or anticoagulation in which all patients also received antiplatelet therapy for secondary prevention.

The long-term functional prognosis seems to be determined by the degree of spinal cone sparing.[43] Another important problem is the neurogenic pain that generally develops in the hypesthetic area after a few months. This phenomenon is seen most frequently with anterior spinal artery infarcts.[44]

When imaging results are negative, spinal cord infarction remains largely an exclusion diagnosis. In the future, new techniques should more clearly demonstrate the developing infarction in the acute phase. Early diagnosis would allow the consideration of treatments used in brain strokes, such as thrombolysis.

Accepted for Publication: March 8, 2006.
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REFERENCES

1. Sandson TA, Friedman JH. Spinal cord infarction: report of 8 cases and review of the literature. *Medicine.* 1989;68:282-292.
2. Lazorthes G, Poulhes J, Bastide G, Chancholle AR, Zadeh O. Spinal cord vascularization: anatomical and physiological study [in French]. *Rev Neurol (Paris).* 1962;106:535-557.
3. Turnbull IM, Brieg A, Hassler O. Blood supply of cervical spinal cord in man: a microangiographic cadaver study. *J Neurosurg.* 1966;24:951-965.
4. Romanes GJ. The arterial blood supply of the human spinal cord. *Paraplegia.* 1965;59:199-207.
5. Lazorthes G, Gouazé A, Bastide G, Santini JJ, Zadeh O, Burdin P. Cervical spinal cord arterial vascularization: study of substitutions anastomoses [in French]. *Rev Neurol (Paris).* 1966;115:1055-1068.
6. Lazorthes G, Gouazé A, Bastide G, Soutoul G, Zadeh O, Santini JJ. La vascularisation artérielle du renflement lombaire. *Rev Neurol (Paris).* 1966;114:109-122.
7. Zülch KJ, Kurth-Schumacher R. The pathogenesis of "intermittent spinovascular insufficiency" ("spinal claudication of Dejerine") and other vascular syndromes of the spinal cord. *Vasc Surg.* 1970;4:116-136.
8. Garcin R, Godlewski S, Rondot P. Clinical study of vascular myelopathies [in French]. *Rev Neurol (Paris).* 1962;106(6):558-591.
9. Perier O, Demanet JC, Henneaux J, Nunès-Vincente A. Does a syndrome of the posterior spinal arteries exist [in French]? *Rev Neurol (Paris).* 1960;103:396-409.
10. Gruner J, Lapresle J. Anatomopathologic study of vascular myelopathies [in French]. *Rev Neurol (Paris).* 1962;106(6):592-631.
11. Garland H, Greenberg J, Harriman DG. Infarction of the spinal cord. *Brain.* 1966; 89:645-662.
12. Hughes JT. Thrombosis of the posterior spinal arteries. *Neurology.* 1970;20:659-664.
13. Weidauer S, Nichtweiss M, Lanfermann H, Zanella FE. Spinal cord infarction: MRI imaging and clinical features in 16 cases. *Neuroradiology.* 2002;44:851-857.
14. Yuh WT, Marsh EE III, Wang AK, et al. MR imaging of spinal cord and vertebral body infarction. *AJNR Am J Neuroradiol.* 1992;13:145-154.
15. Faig J, Busse O, Salbeck R. Vertebral body infarction as a confirmatory sign of spinal cord ischemic stroke: report of three cases and review of the literature. *Stroke.* 1998;29:239-243.
16. Stepper F, Lövblad KO. Anterior spinal artery stroke demonstrated by echo-planar DWI. *Eur Radiol.* 2001;11:2607-2610.
17. Loher TJ, Bassetti CL, Lövblad KO, et al. Diffusion-weighted MRI in acute spinal cord ischaemia. *Neuroradiology.* 2003;45:557-561.
18. Bammer R, Herneth AM, Maier SE, et al. Line scan diffusion imaging of the spine. *AJNR Am J Neuroradiol.* 2003;24:5-12.
19. Robertson RL, Maier SE, Mulkern RV, Vajapayam S, Robson CD, Barnes PD. MR line scan diffusion imaging of the spinal cord in children. *AJNR Am J Neuroradiol.* 2000;21:1344-1348.
20. Nagashima K, Shimamine T. Anatomo-pathologic study of "pencil-shaped softening" of the spinal cord [in Japanese]. *Shinkei Kenkyu No Shimpo.* 1974; 18:153-166.
21. Duggal N, Lach B. Selective vulnerability of the lumbosacral spinal cord after cardiac arrest and hypotension. *Stroke.* 2002;33:116-121.
22. Fieschi C, Gottlieb A, De Carolis V. Ischaemic lacunae in the spinal cord of arteriosclerotic subjects. *J Neurol Neurosurg Psychiatry.* 1970;33:138-146.
23. Pou Serradell A. Acute spinal cord infarction: clinical study and MRI in 8 cases [in French]. *Rev Neurol (Paris).* 1994;105:22-32.
24. Mascalchi M, Cosottini M, Ferrito G, Salvi F, Nencini P, Quillici N. Posterior spinal artery infarct. *AJNR Am J Neuroradiol.* 1998;19:361-363.
25. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med.* 1994;331:69-73.
26. Yoshizawa H. Pathomechanism of myelopathy and radiculopathy from the viewpoint of blood flow and cerebrospinal fluid flow including a short historical review. *Spine.* 2002;27:1255-1263.
27. Mossman SS, Jestico JV. Central cord lesions in cervical spondylotic myelopathy. *J Neurol.* 1983;230:227-230.
28. Niino M, Isu T, Tashiro K. Nonhemorrhagic venous infarction of the spinal cord without spinal vascular malformation. *J Neurol.* 1999;246:852-854.
29. Hughes JT, Brownell B. Cervical spondylosis complicated by anterior spinal artery thrombosis. *Neurology.* 1964;14:1073-1077.
30. Rogopoulos A, Benchimol D, Paquis P, Mahagne MH, Bourgeon A. Lumbar artery compression by the diaphragmatic crus: a new etiology for spinal cord ischemia. *Ann Neurol.* 2000;48:261-264.
31. Weishaupt D, Schmid MR, Zanetti M. Positional MR imaging of the lumbar spine: does it demonstrate nerve root compromise not visible at conventional MR imaging? *Radiology.* 2000;215:247-253.
32. Wenger M, Adam PJ, Alarcon F, Markwalder TM. Traumatic cervical instability associated with cord oedema and temporary quadriparesis. *Spinal Cord.* 2003; 41:521-526.
33. Lee SW, Wong KW, Chan MK, Yeung HM, Chiu JL, Leong JC. Development and validation of a new technique for assessing lumbar spine motion. *Spine.* 2002; 27:E215-E220.
34. Tosi L, Rigoli G, Beltramello A. Fibrocartilaginous embolism of the spinal cord: a clinical and pathogenetic reconsideration. *J Neurol Neurosurg Psychiatry.* 1996; 60:55-60.
35. Dejerine J. Intermittent spinal cord claudication [in French]. *Presse Med.* 1911; 19:981-984.
36. Jellema K, Canta LR, Tijssen CC, van Rooij WJ, Koudstaal PJ, van Gijn J. Spinal dural arteriovenous fistulas: clinical features in 80 patients. *J Neurol Neurosurg Psychiatry.* 2003;74:1438-1440.
37. Taylor CL, Warren RS, Ratcheson RA. Steal affecting the central nervous system. *Neurosurgery.* 2002;50:679-689.
38. Cheshire WP, Santos CC, Massey EW, Howard JF. Spinal cord infarction: etiology and outcome. *Neurology.* 1996;47:321-330.
39. Salvador de la Barrera S, Barca-Buyo A, Monototo-Marqués A, Ferreiro-Velasco ME, Cidoncha-Dans M, Rodriguez-Sotillo A. Spinal cord infarction: prognosis and recovery in a series of 36 patients. *Spinal Cord.* 2001;39:520-525.
40. Foo D, Rossier AB. Anterior spinal artery syndrome and its natural history. *Paraplegia.* 1983;21:1-10.
41. de Haan P, Kalkman CJ, Jacobs MJ. Pharmacologic neuroprotection in experimental spinal cord ischemia: a systematic review. *J Neurosurg Anesthesiol.* 2001; 13:3-12.
42. de Sèze J, Stoykovic T, Breteau G, et al. Acute myelopathies: clinical, laboratory and outcome profiles in 79 cases. *Brain.* 2001;124:1509-1521.
43. de Sèze M, de Sèze M, Joseph PA, Wiart L, Nguyen PV, Barat M. Prognostic fonctionnel des paraplégies par ischémie médullaire: étude rétrospective de 27 cas. *Rev Neurol (Paris).* 2003;159:1038-1045.
44. Triggs WJ, Beric A. Sensory abnormalities and dysaesthesias in the anterior spinal artery syndrome. *Brain.* 1992;115:189-198.