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CME ARTICLE

Clinically suspected fibrocartilaginous embolism: clinical characteristics, treatments, and outcomes

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Objective: To study the frequency, demographics, clinical characteristics, and outcomes of patients with an antemortem diagnosis of fibrocartilaginous embolism (FCE), a rare cause of spinal cord and cerebral infarction because of the presumed embolization of nucleus pulposus material into the vascular circulation.

Methods: We retrospectively reviewed the institutional experience of patients who received an antemortem diagnosis of FCE by their treating physician at the Mayo Clinic (Rochester, MN, USA) from 1997 to 2009. All patients underwent laboratory, radiological, and clinical exclusion of other possible and related diagnoses.

Results: Of 164 patients with acute spinal cord infarction seen during the study timeframe, 9 (5.5%; 95% CI 2.5, 10.2%) met inclusion criteria for high likelihood of FCE (6 men, 3 women; median age 46 years old, range 21–64). All patients were severely affected (median modified Rankin Scale 4, median Barthel index 45; mean time to evaluation 57 days). One patient (1/9) experienced concomitant cerebral infarction. No patients had noticeable improvement from steroid treatment.

Conclusion: The diagnosis of FCE in life is common at this referral center, accounting for 5.5% of all cases of acute spinal cord infarction seen. Although FCE is a postmortem diagnosis, we propose clinical criteria for FCE in life to better characterize the relatively high number of patients with unexplained ischaemic myelopathy.

Introduction

Fibrocartilaginous embolism (FCE) is an unusual cause of spinal cord and cerebral ischaemia, most definitively diagnosed at autopsy. Embolization of nucleus pulposus fragments interrupts the vascular supply, presumably because of retrograde movement through the spinal arteries [1,2]. Severe spinal cord injury or death may be the devastating result. Most cases of FCE have been reported in women [3] and document a sequence of lifting, physical exertion, minor trauma, or Valsalva maneuver before severe spinal cord infarction [1–4].

Initially, all cases of FCE were discovered at autopsy. Naiman *et al.* [5] described the first case of FCE in a young man who developed quadriparesis and respiratory failure after minor trauma whilst playing basketball. Autopsy showed diffuse embolic material of the nucleus pulposus in the anterior spinal cord artery and basilar arterial system. Since then, nearly 40 autopsy-proven cases have been reported [4,6–11] In 1991, the first diagnosis of FCE was made in a patient who survived [12]. Although there are no formal diagnostic criteria for FCE in life, a number of authors have suggested specific clinical features that make the diagnosis likely [1,3,4,13]. These include antecedent minor trauma, absence of vascular risk factors, spinal cord imaging consistent with evolving infarction, normal cerebrospinal fluid (CSF) analysis, and exclusion of other possible etiologies. The number of clinically suspected cases exceeds the number of autopsy-proven cases reported in the literature over the past decade.

Fibrocartilage as an embolic cause of spinal cord ischaemia is assumed to derive from the intervertebral disk and Schmorl's nodes. Although the intervertebral disk is often avascular, fibrocartilaginous disk material may travel to the spinal bone marrow through communicating sinusoids and venules. As a result of a valveless peripheral venous system, conditions resulting

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in high internal pressure, such as a Valsalva maneuver or trauma, may permit retrograde flow of emboli through anastomoses with the spinal arterial system. An arterial infarct can result [4]. The neuroimaging features of this process can include extensive T2 hyperintensity on magnetic resonance imaging, minimal contrast enhancement, terminal cord hemorrhagic necrosis, and bone marrow infarction [9].

Given that FCE may be an important cause of otherwise unexplained spinal cord ischaemia, we retrospectively reviewed the institutional experience of patients with an antemortem diagnosis of fibrocartilaginous embolism at the Mayo Clinic over a 12-year period. Patients' clinical and imaging features as well as treatment outcomes were characterized. Finally, we counted the patients with suspected FCE and compared this number to all patients who presented with spinal cord infarcts over the same time period to assess the rarity of this disorder.

Methods

A search for the keywords 'fibrocartilaginous embolism' was performed on all patient records from the Mayo Clinic (Rochester, MN, USA) from the dates 1 January 1997 to 28 February 2009. All clinical, imaging, and laboratory records of patients who received the diagnosis of FCE were reviewed. Patients with a clinical diagnosis of FCE had exclusion of other possible and related diagnoses, including aortic dissection, hypercoagulable states, intracardiac embolic source, dural arteriovenous fistula, rheumatologic disorders, demyelinating disease, infectious and metabolic etiologies of spinal cord disease (e.g., syphilis, human immunodeficiency virus, vitamin B12, or copper deficiencies), and/or systemic malignancy as appropriate.

As part of a separate study at this same institution [14], a search was performed for all patients who were diagnosed with 'spinal cord ischaemia,' 'spinal cord infarct,' 'ischaemic myelopathy,' 'anterior spinal artery syndrome,' or 'spinal stroke' over the same timeframe. A neurologist or neurology resident reviewed each spinal cord infarction case to ensure the patient had the stated diagnosis. Patients who had a vascular malformation of the spinal cord, hemorrhage (hematomyelia), or transient symptoms (i.e. spinal cord transient ischaemic attack) were excluded from this total count.

Amongst patients with a clinical diagnosis of FCE, those that met the following three inclusion criteria (determined by the authors prior to chart review) were included in our analysis. These criteria are based on the approximately 40 autopsy-diagnosed [4–11] and 14 clinically suspected antemortem cases [1,4,12,13,15–23] reported to date.

- Rapid evolution of symptoms consistent with a vascular etiology, with or without antecedent minor trauma or Valsalva maneuver.
- Magnetic resonance imaging (MRI) changes consistent with ischaemic myelopathy, including T2-weighted hyperintensity on initial imaging and temporal evolution consistent with infarction, with or without evidence of disk herniation, Schmorl's nodules, or other degenerative disk pathology.
- No more than two vascular risk factors (including age >60 years old, diabetes mellitus, untreated hypertension, active smoking, untreated dyslipidemia, and previous vascular event).

Measures of functional independence included the Barthel index [24] and modified Rankin Scale. Treatment outcomes were determined by retrospectively reviewing the medical records for documentation of subjective or objective neurologic examination improvements from the point of maximal disability.

Basic statistics including median, mean, proportions, and 95% confidence intervals were used to describe the patient group. The Mayo Clinic Institutional Review Board approved this study.

Results

Between January 1997 and February 2009, nine of the patients with a presumed diagnosis of FCE fulfilled our inclusion criteria. No patient with a diagnosis of FCE had a history of a previous vascular event, recent surgery, intracardiac thrombus, or antecedent major trauma. An additional 30 patients had the keywords fibrocartilaginous embolism appear at least once in the differential diagnosis or diagnosis section of their medical records, but ultimately had an alternative diagnosis and/or did not meet our stated criteria of high likelihood. In this same timeframe, there were 164 cases with an acute spinal cord infarct evaluated at this institution. Of the nine patients with FCE, there are six men and three women (median age 46 years old, range 21–64). The clinical characteristics of these patients and results of diagnostic testing are shown in Table 1. Six patients were older than 40 years old at the time of onset. Most (7/9, 78%) reported a potential precipitating event in the 24 h prior to symptom onset. These included motor vehicle accident without recognized back injury ($n = 2$), heavy lifting ($n = 3$), physical exertion ($n = 1$), and bending over ($n = 1$). Symptom onset occurred within six hours of the recognized antecedent event in five patients (5/7, 71%; range: immediate onset to 15 h). The time from symptom onset to maximal weakness was 4 h or less in eight patients and <12 h in all patients (range minutes to 11 h). Back or neck pain was a prominent initial

Table 1 Clinical characteristics and results of diagnostic testing for nine patients with a clinical diagnosis of spinal cord infarction because of fibrocartilaginous embolism

Case/age (years)/sex	Vascular risk factors	Potential precipitating factor, time to symptom onset/time to maximal weakness	Location of SCI on MRI	Aortic imaging	Trans-esophageal echocardiogram	CSF protein[a] (mg/dl)	Coagulation studies[b]	Autoimmune/ inflammatory markers[c]	Acute Treatment	mRS/BI at last evaluation/ time to last neurologic evaluation, days
1/52/M	Past smoking	None, NA/few minutes	C2–7, central	NP	Normal	61	Normal (INR and aPTT only)	NP	Dexamethasone 4 mg IV q6 h × 9 days	3/**/18
2/46/M	Treated hypertension, treated dyslipidemia	12-mile bicycle ride prior to leg weakness, 1–2 h/10 h	T10-conus, central	CTA normal	Normal	64	Normal	Normal ESR, CRP, ANA, ENA, c-ANCA, p-ANCA, RF	IVMP 1 gm daily × 4 days, daily Aspirin initiated	4/60/17
3/52/M	Treated hypertension, untreated dyslipidemia	Heavy lifting, immediate/11 h	T8-conus, central	MRA normal	Tiny patent foramen ovale	54	Normal	Normal ESR, CRP, ANA, c-ANCA, p-ANCA, RF	IVMP 500 mg twice daily × 7 days, PLEX days 6–8, prednisone PO 20 mg day 8, IV heparin	4/60/27
4/42/M	None	None, NA/4 h	T8-conus, anterior	CTA normal	NP	58	Normal	Normal ESR, ANA, RF, CCP, SSA, SSB, dsDNA	IVMP, IVIG x 4 days, then PLEX	5/10/38
5/64/M	Past smoking	Heavy lifting, immediate/4 h	C4–T3, anterior	CTA normal	NP	NP	Normal	Normal ESR, CRP, ANA, ENA, RF, CCP	Anterior cervical decompression, IVMP 1 gm × 5 days	5/0/18
6/52/F	None	Weight lifting, a few hours/20 min	Lower thoracic, anterior	MRA normal	Tiny patent foramen ovale	53	Normal	ESR 30 (normal 1–29 s); normal CRP, ANA, dsDNA	'IV steroids: few doses' twice, low molecular weight heparin before transfer	4/**/195
7/31/F	None	Minor MVA, approximately 15 h/few minutes	T9-conus, anterior	CTA normal	Normal	Normal	Normal apart from high fibrinogen[c] (512)	Normal ESR, ANA, SSA	IVMP × 5 days, PLEX × 3 days, inferior vena cava filter placement	4/40–50/72
8/26/M	None	Bending over, immediate/2 h	T8-conus, central	NP	NP	90	Normal	Normal ESR, ANA, ENA	IVMP × 5 days, daily Aspirin initiated	4/35–40/90

Table 1 (Continued)

Case/age (years)/sex	Vascular risk factors	Potential precipitating factor, time to symptom onset/time to maximal weakness	Location of SCI on MRI	Aortic imaging	Trans-esophageal echocardiogram	CSF protein[a] (mg/dl)	Coagulation studies[b]	Autoimmune/ inflammatory markers[c]	Acute Treatment	mRS/BI at last evaluation/ time to last neurologic evaluation, days
9/21/F	None	Minor MVA, approximately 12 h/20 min	C5-T1 anterior	CTA normal	Normal	31	Normal apart from low fibrinogen[c] (148)	Normal CRP, ANA, CCP	IVMP × 5 days, PLEX × 3 every other day, PO steroid taper, daily Aspirin initiated	4/45/37

[a]Normal CSF protein 14–45 mg/dl; CSF cell counts and glucose were normal in all tested patients.

[b]Includes INR, aPTT, and a hypercoagulability panel except where indicated.

[c]Normal fibrinogen in women is 200–430 mg/dl; the isolated high fibrinogen in patient seven was attributed to an acute phase reaction; the isolated low fibrinogen in patient nine was attributed to recent plasma exchange.

**Insufficient data in medical record to calculate Barthel index.

NA, not applicable; SCI, spinal cord infarct; MVA, motor vehicle accident; MRI, magnetic resonance imaging; NP, not performed; CTA, computed tomography angiogram; MRA, magnetic resonance angiogram; CSF, cerebrospinal fluid; INR, international normalized ratio; aPTT, activated partial thromboplastin time; PLEX, plasma exchange; ESR, erythrocyte sedimentation rate; CRP, c-reactive protein; ANA, antinuclear antibody; ENA, antiextractable nuclear antigen antibody; c-ANCA, circulating antineutrophil cytoplasmic antibody; p-ANCA, protoplasmic-staining antineutrophil cytoplasmic antibody; RF, rheumatoid factor; CCP, anticitrullinated protein antibody; SSA, anti-Sjogren syndrome A antibody; SSB, anti-Sjogren syndrome B antibody; dsDNA, antidouble stranded DNA antibody; IVMP, intravenous methylprednisone; IVIG, intravenous immunoglobulin; PO, per os ('by mouth'); mRS, modified Rankin scale; BI, Barthel index.

symptom in six patients and accompanied by a radicular component in three patients. Four patients had vascular risk factors other than age.

Location of infarction by magnetic resonance imaging of the spinal cord was cervical alone (n = 1), cervicothoracic (n = 2), thoracic alone (n = 2), and thoracic to conus medullaris (n = 4). Serial MRI studies from case 9 demonstrate the evolution of MRI findings from herniated disk material on initial imaging to longitudinally extensive infarction on subsequent imaging (Fig. 1a–d). Posterior circulation cerebral infarctions, in addition to spinal cord infarction, were noted in this patient (1/9, 11.1%) (Fig. 2a–d). She was asymptomatic from these cerebral lesions.

Cerebrospinal fluid was analyzed in eight of the nine patients, with the only remarkable finding being elevated protein in six (range 53–90 mg/dl). No patients had oligoclonal banding, hypoglycorrhachia, xanthochromia, or other markers of inflammatory or infectious disease.

Given the clinical suspicion of spinal cord infarction, all patients underwent extensive diagnostic testing for alternative causes of spinal cord ischaemia. Imaging of the aorta by computed tomography (CT) or magnetic resonance (MR) angiography was negative for dissection or aneurysm in all tested patients (n = 7). Echocardiography was performed in six patients and revealed no potential sources of emboli in the heart or ascending aorta. CT or MR angiography of the spine was performed in five patients and showed no evidence of dural arteriovenous fistula or other vascular malformation. The one patient who had both cervical

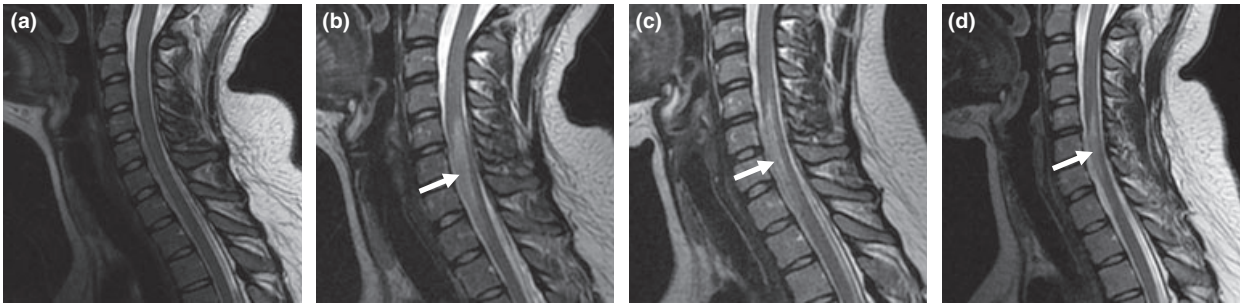

Figure 1 Evolution of ischaemic myelopathy of the cervical cord on T2-weighted imaging. Following a minor motor vehicle accident without recognized injury, this 20-year woman had an initially normal magnetic resonance imaging of the cervicothoracic spinal cord, with a small amount of presumed intravertebral disk material protruding at the C4–C5 interspace (a). Four days later, ischaemia of the anterior and right lateral cervical cord is noted extending from C5 to T1 levels, and the intervertebral disk protrusion is not longer seen (b). By 21 days (c) and 24 days (d) post-accident, the spinal cord lesion shows signs of regression with resolution of edema.

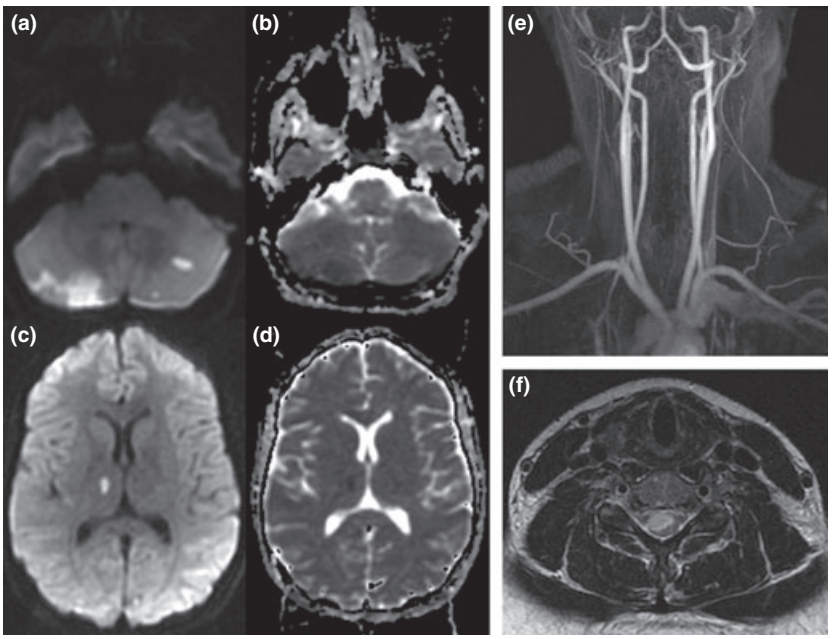

Figure 2 Bilateral cerebellar (a, b) and right thalamic ischaemic infarctions (c, d), as seen by brightness on diffusion-weighted imaging and corresponding darkness on apparent diffusion coefficient mapping, at one post-accident in the same 20-year-old woman. Magnetic resonance imaging of the neck (e, f) and intracranially (not shown) revealed no major vascular occlusion or stenosis.

spinal cord and cerebral infarctions (case 9) also underwent MR angiography of the head and neck; this too was negative (Fig. 2e,f).

Platelet count, international normalized ratio (INR), and activated partial thromboplastin time (aPTT) were normal in all patients. Eight patients had additional special coagulation studies. Whilst two showed isolated abnormal fibrinogen levels, none were considered consistent with a hypercoagulable disorder. Erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP), antinuclear antibodies (ANA), and various other rheumatologic and/or vasculitic markers were negative in all tested patients ($n = 8$) with the exception of one patient who had a minimally elevated ESR. Antiphospholipid antibodies were negative in all tested patients ($n = 4$).

Testing for other infectious, inflammatory, and metabolic causes of myelopathy was performed in most patients but was not consistent from case to case. Examples of such testing included normal vitamin B12 ($n = 7$), homocysteine ($n = 7$), neuromyelitis optica antibody ($n = 6$), paraneoplastic antibody panel ($n = 5$), angiotensin-converting enzyme ($n = 5$), copper ($n = 5$; one with a mildly decreased level attributed to recent plasma exchange), and screening for infections such as Lyme disease ($n = 7$), syphilis ($n = 5$), Human Immunodeficiency Virus ($n = 4$), Human T-cell Lymphotropic Viruses ($n = 3$), and West Nile Virus ($n = 2$). Evaluations for non-ischaemic causes of myelopathy were rarely comprehensive given the rapid evolution of symptoms and resultant strong clinical suspicion of ischaemia in all patients.

All but one patient (case 4) received varying doses of intravenous (IV) steroids (range 4–9 days), all without any noticeable clinical improvement. Four patients (cases 3, 4, 7, 9) also received plasma exchange (range: one to three courses of a minimum of 3 days) – again without meaningful clinical improvement. Other treatments, including IV heparin (cases 3 and 6), a 4-day course of daily IV immunoglobulin (case 4), and emergent anterior spinal decompressive surgery (case 5), were all similarly unsuccessful.

Functional outcome varied from moderate to severe disability (median mRS 4, range 3–5; average time to evaluation 57 days range 17–195). Barthel index was assessed on seven patients and showed marked functional dependence (median 45, range 0–60). At the time of analysis, there was an average of 2.90 years since presentation. None of the patients in this series have died or had repeat events to our knowledge.

Discussion

Unexplained spinal cord ischaemia is not uncommonly diagnosed as FCE in living patients. Our series demonstrates that the range of cases presumed to be FCE by history, examination, laboratory studies, and magnetic resonance imaging can be consistent with the reported autopsy-proven cases of FCE. Current understanding of FCE is based on case reports, nearly all discovered at autopsy; therefore, the prognosis of

Table 2 Reported cases of pathologically confirmed fibrocartilaginous embolism causing cerebral stroke (1961-present)

Age (years), sex	Potential precipitating factor, time to symptom onset	Initial symptoms, time to maximal weakness[a]	Location of infarct	Time to death following symptom onset
15 M [5]	Fall whilst playing basketball, 20 min	Sudden back pain, 1 h	Medulla to T7	3 h
28 F [26]	Seven months pregnant with twins	Ascending paresthesias, 24 h	Medulla to T4	6 days
21 M[27]	Repeated forceful flexion of the neck against resistance, immediate	Neck pain, several hours	Medulla to T2	5 days
38 F [28]	Minor MVA, 3 weeks	Right posterior neck pain with left arm weakness, 1 h	Medulla to C4	5 weeks
29 F [29]	None	Sudden back pain with limb heaviness, 'hours'	Medulla to T4	15 days
32 F [29]	None	Sudden bilateral shoulder pain and arm tingling, 'hours'	Medulla to T5	11 months
23 F[30]	None	Sudden occipital headache, less than 1 h	Medulla to C5[b]	12 days
17 F [3]	Fall with brief loss of consciousness whilst playing basketball, 2–3 h	Left hemiparesis, immediate	Territory of right middle cerebral artery	3 days
23 M [11]	Strike to neck and back, 10 days	Sudden back pain and quadriparesis, immediate	Lower medulla through cervical cord	3 months

[a]Maximal weakness was characterized by quadriparesis with respiratory failure in all cases but #8 who had hemiparesis.
[b]Clinical suspicion of cortical blindness was attributed to transient bilateral occipital lobe ischaemia; autopsy reportedly showed no abnormalities of cerebral cortex.

FCE may seem overwhelmingly pessimistic. Although FCE is a severely disabling form of non-traumatic spinal cord injury, the patients seen at this institution with a presumed diagnosis of FCE have all survived. Here, we have identified more patients with thoracic cord injury than cervical cord injury compared to collected case reports[3], and this may partially account for the better prognoses. We have not found a female predominance.

At our center, FCE is the clinical diagnosis of approximately 5.5% of spinal cord infarction cases, suggesting that the diagnosis is not rare. Because only autopsy can make the diagnosis definitively, it is possible that some of our reported cases of spinal cord infarction were not because of FCE and represent other unexplained causes of spinal cord ischaemia [25]. This concern relates to all reported antemortem cases. To minimize this possibility, we have employed strict inclusion criteria based on the existing literature. These include the time course of the event which make a vascular etiology most plausible, neuroimaging features consistent with ischaemic myelopathy, and exclusion of cases that have more than two vascular risk factors or other identifiable causes for a spinal cord disorder on neuroimaging. The presence of preceding trauma or Valsalva maneuver should further increase the clinical suspicion of FCE. Although these criteria are unlikely to identify all patients who have FCE, they are proposed to provide an estimate until clinico-pathological studies can be performed in groups. Older patients, who have multiple vascular risk factors, may also experience FCE, but our criteria have excluded patients with three or more vascular risk factors, a condition more likely in older patients.

Fibrocartilaginous embolism has been reported to involve other organs, including the lungs [26] and brain [3,5,11,27–31]. Amongst patients with presumed FCE, ischaemia involving the cerebral circulation is rare. Cerebral involvement has been reported in nine cases of autopsy-proven FCE in the available literature (Table 2). Ischaemia involved the medulla (supplied by the anterior spinal artery) and/or occipital lobes (supplied by the posterior cerebral circulation) in all but one of these patients and accompanied a longitudinally extensive cervical infarction in all patients. The outcome has been death because of respiratory failure, hemorrhagic ulcers, or brain herniation. Just one patient in our series had evidence of cerebral involvement and was asymptomatic from these lesions. This underscores the need to consider and screen for cerebral infarction in patients with cervical spinal cord infarction when FCE is in the differential diagnosis.

Common treatments for FCE have included intravenous steroids, intravenous heparin, and plasma exchange. Unfortunately, no treatment had a recognized impact on the patient's symptoms in the acute period. We cannot exclude the possibility that the provided treatments prevented further worsening or recurrence. The lack of available diagnostic criteria for FCE has otherwise prevented an accurate assessment of the benefit of a specific treatment. Isolated reports have found no benefit from intravenous steroids [21,22]. Steroids could be therapeutic in patients who suffer spinal cord edema related to either a vascular event or in other conditions that may present similarly to FCE. The administration of steroids, intravenous immunoglobulin, and plasma exchange in selected patients was most often performed to ensure that other possible and treatable diagnoses were addressed before the diagnosis of FCE was ultimately made. Differential diagnosis for FCE may include prolonged arterial hypotension, spinal disk prolapse or herniation, thrombo-occlusive aortic disease, vasculitis, systemic lupus erythematosus, antiphospholipid antibody syndrome, dissecting aortic aneurysm, hypertensive small vessel disease, sickle cell anemia, infections including tuberculosis, carcinomatous meningitis, and cholesterol and atheromatous emboli [25,32]. Despite this broad range of possibilities, up to 74% of spinal cord ischaemia cases have no identifiable etiology [25]. Better antemortem characterization of this potentially large group of patients may lead to improved assessment of whether regularly administered therapies have any real benefit to survivors.

Although we cannot rule out referral bias, the majority of patients at this center with a diagnosis of FCE in life are in middle age and survive with severe and disabling spinal cord injury. This is in contrast with the expectation that patients are usually young and die [3,4]. Cerebral involvement is rare but should be considered when high cervical cord injury is present. Finally, suspected FCE is not common but likely represents a significant proportion of patients who suffer from otherwise unexplained spinal cord ischaemia.

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