Fibrocartilaginous embolism: a comprehensive review of an under-studied cause of spinal cord infarction and proposed diagnostic criteria

Mahmoud A. AbdelRazek, Ashkan Mowla, Salman Farooq, Nicholas Silvestri, Robert Sawyer, Gil Wolfe

Department of Neurology, State University of NY at Buffalo, Buffalo, NY, USA

Background: Most spinal cord infarctions are due to aortic pathologies and aortic surgeries. Fibrocartilaginous Embolism (FCE) has been reported to represent 5.5% of spinal cord infarctions. Some believe that FCE is more common than presumed and is rather under-diagnosed due to vagueness surrounding its clinical presentation.
Method: A literature search was conducted for case reports of FCE published before August 2014. PubMed, the Cochrane Central Register and Google Scholar were searched for different combinations of the key words "fibrocartilaginous, "nucleus pulposus", "embolism", "spinal cord", "inter-vertebral disc", "infarction", "stroke", "paraplegia", "quadriplegia", "myelopathy".
Result: Fifty-five case articles were reviewed, ten of which were translated from foreign languages. A total of 67 cases of FCE were found, 41 tissue-confirmed and 26 clinically suspected. A comprehensive summary of the clinical anatomy, patho-physiologic mechanisms, epidemiology, diagnosis and treatment of FCE is described, along with the conflicting opinions on its incidence and relevance after reviewing all of the related literature. The 41 tissue proven cases are summarized and a schematic approach to the clinical diagnosis of FCE, deducted from their clinical findings, is presented.
Conclusion: FCE of the spinal cord, often mis-diagnosed as transverse myelitis, may be more common than presumed. Future research into FCE, including the development of a chondrolytic therapy that can be given empirically upon its clinical suspicion to acutely reverse its symptoms, may be of value.

Keywords: Nucleus pulposus, Paraplegia, Transverse myelitis, Stroke, Idiopathic myelopathy

Introduction

Fibrocartilaginous Embolism (FCE) refers to the migration of fibrocartilaginous nucleus pulposus material through the nearby vasculature to embolize into one of the spinal cord vessels. It was first described by Naiman[1] in 1961 in a 15-year-old boy who developed quadriplegia shortly after suffering a trivial fall on his back during a basketball game. Since then several cases have been reported. FCE has been shown to cause embolic infarction most commonly to the spinal cord, but also to the lung,[2] brain,[3] vertebrae and ribs.[4] FCE is also well described in the veterinary literature, most commonly occurring in dogs, and sporadically in other species.[5] FCE to the spinal cord in humans has been reported in 41 histo-pathologically confirmed (Table 1) and 26 clinically suspected[3] cases.

Correspondence to: Mahmoud AbdelRazek, 100 High St, Neurology Dept, State University of NY at Buffalo, Buffalo, NY 14203, USA. Email: meabdelr@buffalo.edu

Clinical Anatomy

Despite being referred to as "the largest avascular structure in the body," the inter-vertebral disc can indeed be the source of embolic material as evidenced by histopathologic sectioning and staining in all cases mentioned in Table 1. The inter-vertebral disc is classically divided into an outer mesodermally derived annulus fibrosus, and a central endodermally derived nucleus pulposus.[6] In neonates, the inter-vertebral disc is a highly vascular structure with large thin walled blood channels running mainly in the cartilage end plate.[6,7] This vascular tissue quickly starts to regress after 2 months and throughout the first decade of life. Eventually, by age 11–16 years will have completely disappeared.[7]

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Table 1 Histo-pathologically confirmed cases of FCE of the spinal cord

Case-Author-Year	Age-Sex	PMH	Suspected Trigger	Time From Trigger to Onset/Time to Symptom Peak/ Time to death	Pain-Radiation	Paralysis	CSF	Level of Infarction	Arterial vs Venous (Territory)	Degenerative Disc Ds / Level	Schmorl's Node
1-Naiman-1961[1]	15-M	_	Falling on buttocks	20 m/1 hr/3 hr	Neck, back, shoulder	Quadriplegia	N	MO-C2-T7	A(ASA)	No	_
2-Laterre-1962[8]	31-F	None	None[9]	-/2 hr/3 mo	Mid Back	Paraplegia	N	C6-T1	A(ASA)	No	No
3-Feigin-1965[10]	49-F	C discectomy 2 yrs prior	_	- /3 wk/6 wk	Left shoulder	Paraplegia, UE Monoparesis	N	C6-T1	V(ASA)	Yes/C	Yes
4-Feigin-1965[10]	39-F	None, Diarrhea 2 days prior	_	-/30 m/ 4 mo	Left shoulder	Paraplegia, UE Monoparesis	N	C4-T2	A-V(PSA)	Yes/-	Yes
*5- Feigin-1965[10]	55-M	Portal Cirrhosis	Portal Hypertension	_	None*	None*	_	Sacral*	V*	_	_
6-Bodechtel-1968[11]	28-F	Pregnancy,7th mo	_	-/ 12 hr/ 6 d	_	R Hemiplegia	N	MO-Conus	Venous **	Yes/-	No
7-Bruno-1969[12]	47-F	Htn, Obesity	_	-/24 hr/ 142 d	Lt chest, neck, mid back	Paraparesis, UEMonoparesis	N	C1–7	ASA(A and V)	Yes/T6 (& T6 Fracture)	-
8- Lvovskiy-1969[13]	21-M	?	Extended Neck	?/ 4 hr/ 5 d	Neck	?	?	C2-C7	A(ASA)	?	?
9-Jurkovic-1970[14]	66-F	None	Shopping &heavy lifting with 2arms	1 d /few days/ 18 d	Lower Back to LE's	Paraparesis	_	C7, T4, T8, T10, L2	V(ASA)	_	Yes/L (4 nodes)
10-Kepes-1973[15]	38-F	None	MVA 3 wks prior	3 wk/1 hr/ 40 d	R posterior neck	Quadriplegia	N	MO-C4	A-V(ASA)	No	Yes
11-Hubert-1974[16]	63-F	Obesity	Episode of coughing[17]	minutes/?/ Some Hrs	Neck	Quadriparesis except R LE.	N	C3-C7	A-V(ASA)	No	No
12- Hubert-1974[18]	17-F	_	Years of riding	-/ 48 hr/ 11 d	Neck	Quadriplegia	N	C4-T6	A(ASA)	No	No
13-Roitzsch-1975[19]	69-F	_	Fall on back[17]	Minutes/- / 11 mo	_	Paraplegia	N	T12-L4	A-V(ASA)	No	_
14- Peiffer-1976[20]	36-M	_	Fall forward[17]	-/ 48 hr/ 2 mo	LowerBack,Rhip	Paraplegia	N	?	A-V **	Yes/C5–6	No
15-Hanski-1977[21]	51-M	Smoker, PAD	Loading Goods[17]	-/ -/ 4 d	Chest pain	Paresis of UE's	↑Ptn (47)	C5-T1	A(ASA)	No	No
16- Schairer-1977[22]	19-F	Obesity	_[17]	-/ 3 hr/ 9 mo	No mentioning	Quadriplegia.	N	C4-T-L	A(ASA)	_	_
17- Budka- 1979[23]	49-F	LBP[24]	_[17]	-/ 10 hr/ 14 wk	Lower back	Paraparesis.	N	T11-Conus	A-V(PSA)	No	No
18-Ho-1980[9]	22-F	None	Stooping	15 m/30 m/9 d	Back of neck	Paraplegia	N	C3-T1	A(ASA)	No	No
19-Bots-1981[17]	29-F	_	None	-/2–5 hr/ 15 d	Back	Quadriplegia	N	MO,C1-T5	A	No	_
20- Bots- 1981[17]	32-F	_	_	/3 hr/ 11 mo	Shoulders	Quadriplegia	N	MO, C1-T5	A(ASA)	No	_
21- Bots- 1981[17]	77-M	DM	_	-/ hrs/ 22 hr	Back of neck	Quadriplegia	N	C1-C4	A(ASA)	No	_

Continued

Table 1 *Continued*

Case-Author-Year	Age-Sex	PMH	Suspected Trigger	Time From Trigger to Onset/Time to Symptom Peak/ Time to death	Pain-Radiation	Paralysis	CSF	Level of Infarction	Arterial vs Venous (Territory)	Degenerative Disc Ds / Level	Schmorl's Node
22- Bots- 1981[17]	60-F	_	None	-/ -/3 mo	_	Paraplegia	_	T10-T11	A(ASA)	_	_
23- Srigley-1981[25]	46-M	DM,Hypothyroid, Sarcoid	Extended neck during shaving	Minutes/ 17 hr/ 1 mo	Neck	Quadriplegia	↑Ptn (45)	C1-C4	A(ASA)	Yes/C	_
24-Kase-1983[26]	23-F	_	None	-/minutes/ 12 d	Occipital HA	Quadriplegia	N	MO-C5	A(ASA)	No	_
25-Barz-1986[27]	56-F	_	Jump from 80 cm window	15–30 m/2 hr/ 13 d	_	Paraplegia	↑Ptn (50)	T12 -Conus	A(ASA)	Yes	Yes/T12
26-Barz-1989[28]	77-F	Yrs of LBP	Shopping exercise/ Strain	4 hr/8 hr/14 d	Back then R leg	Paraperesis	N	S-Conus	A(ASA)	_	No
27-Banerjee-1989[29]	21-M	None	None	-/2 hr/2 d	Neck to Lt arm	Quadriplegia	_	C2-C6	A(ASA)	No	-
28- Kestle-1989[30]	43-M	LBP × 3 wk	_	-/2 d/ 2 wk	Butocks& thigh	Paraparesis	↑Ptn (50)	T12-Conus	A(ASA,PSA)	Yes/T9-L1	Yes/T9-L1
29-Bockenek-1990[31]	20-M	_	MVA & hearing a snap in his spine	24 hr/1 hr/6.5 yr	Neck to 4 limbs	Quadriplegia	_	C4-T4	Vessels	No	_
30-Scully -1991[32]	13-M	MVA × 1 yr	Somersaults	10–20 m/ 6 hr/-	Neck	Hemiparesis.	_	C2–4	A(ASA)	No	_
31-Scully -1991[32]	61-F	Htn × 10 yrs	Grocery Shopping	1–2 hr/3 hr/-	No mentioning	Paraplegia	N	T11- L1	A-V(ASA)	_	_
32-Moorhouse-1992[33]	63-F	_	Strain with defecation	10–20 m/30 hr/hrs	Mid& Lower Back	Paraplegia	N	T11-L1	A(ASA)	Yes/L4–5	No
33-Mikulis- 1992[34]	61-F	Htn, Obesity, LBP × 4 mo 24	_	_	Lower Back	Paraplegia	N (only ↑RBC 68/ mm3)	T11-L1	A-V(ASA)	Yes	No
34-Toro -1994[24]	16-F	None	Stooping to milk a cow	1–10 m/15 m/6 wk	LBP to thighs	Paraplegia	↑Ptn	L1	A-V	No	No
35-Yousef -1998[35]	14-F	Obese (86 kg, 1.6 m, 33.6 BMI)	Leaning forward to pick up an object	Immediate/ <24 hr/ 5 d	Mid back	Paraplegia	N	T5–7	A(ASA)	No	Yes
36-Freyaldenhoven -2001[36]	19-M	None	Hit on the back by a large door	2 d/-/-	Back	Quadriplegia	N	C	A	No	_
37-Alexander-2003[37]	60-F	Chronic LBP	Swimming	minutes/- /17 d	Funny lower back sensation	Paraplegia	N	T9–11	A(ASA)	Yes/L3-L5	Yes/L4
38- Uppal -2004[38]	41-F	Pregnancy, 25th wk	_	-/ <30 m/22 d	_	Paraplegia	N	T6–7 to Conus	A(PSA)	Yes/C3–5	Yes
39- Duprez-2005[39]	78-M	Htn	Minor fall	Few days/6 hr/28 d	_	Paraplegia.	↑Ptn (58)	Conus	A	Yes	Yes
40-Meyer-2005[40]	66-M	_	Paravertebral C5–6 injection	2.5 hr/2 d/61 d	_	Quadriparesis	_	C2–3	A(ASA)	No	_
41-Piao- 2009[41]	23-M	_	Trivial strike to neck and back	10 d/-/3 mo	Mid Back	Quadriplegia.	Cells 20/ mm^3, MBP 22 nmol/l	MO - C7	A-V(ASA)	Yes/C7-T1	_

A=arterial; ASA=anterior spinal artery; C=cervical; d=days; DM=diabetes mellitus; Ds=disease; F=female; FCE=fibrocartilaginous embolism; HA=headache; Htn=hypertension; hr=hours; L=lumbar; LBP=lower back pain; LE=lower extremity; Lt=left; M=male; m=minutes; mo=months; MO=medulla oblangata; MVA=motor vehicle accident; N=normal; PAD=peripheral arterial disease; PSA=posterior spinal artery; Ptn=protein (mmol/L); R=right; S=sacral; T=thoracic; UE=upper extremity; V=venous; wk=weeks; " -- "= no report in the literature to confirm or deny, "?"= unclear translation.

*FCE found incidentally in sacral veins on autopsy with no adjacent cord pathology. ** Later reevaluated by Schairer to be arterial.[17,22]

Neo-vascularization reappears in the normal adult inter-vertebral disc at the circumferential edges at around 50 years of age.[7] In individuals with degenerative disc disease, this revascularization has been demonstrated to occur much earlier and is more pronounced.[6] It has also been postulated that remnants of vascular channels can persist in the inter-vertebral disc beyond the second decade of life.[6]

The vertebral bodies and the spinal cord, in contrast, have a fixed blood supply throughout life. One anterior and two posterior spinal arteries run longitudinally on the surface of the spinal cord from medulla to conus. They are reinforced throughout their course by transverse radicular arteries that arise from the vertebral, inter-costal and lumbar arteries through their dorsal then spinal branches (Figure 1). The spinal branches, in addition to giving radicular branches to reinforce the longitudinal spinal arteries, they also give anterior branches to supply a large portion of the posterior aspect of the vertebral bodies.[42] This common arterial supply between the spinal cord and vertebral bodies is important in understanding the postulated mechanisms of FCE (Figure 1).

Another relevant component to the understanding of FCE mechanisms are Schmorl's nodes, which are focal masses of fibrocartilage found within the bone of vertebrae, and thus lie in close proximity to the vascular supply of the vertebral body. Schmorl's nodes are a common occurrence, present in 38% to 79% of the adult population[43, 44] and are thought to have developed due to herniation of nucleus pulposus material into the body of the adjacent vertebra as a consequence of degenerative disc changes.

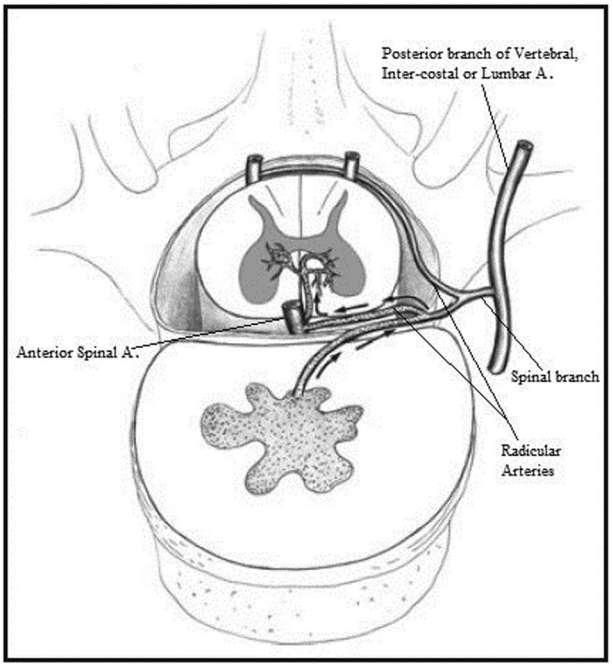

Figure 1 A diagram of the clinical anatomy and mechanism of FCE. Reproduced from[56] with permission from John Wiley and Sons. Labels were added by the authors of this paper.

Mechanisms

The fibrocartilaginous disc material gains vascular access via any of three pathways: (1) revascularization of the inter-vertebral disc by normal aging or degenerative disc disease especially herniation;[6,7] (2) formation of Schmorl's nodes;[43,44] or (3) persistence of inter-vertebral disc vasculature into adulthood.[45] It is postulated that the initial trigger for break off of fibrocartilaginous nucleus pulposus material is increased intra-disc or intra-vertebral body pressure by axial loading forces applied to the spine, such as heavy lifting, straining, falls or minor traumatic events to the neck and back.

Once in the vasculature, the fibrocartilaginous embolus can enter either an arterial or venous route to reach the spinal cord.

Arterial route

The fibrocartilaginous material travels retrograde through the arterial system supplying the spinal column, to reach the radicular artery which carries it into the spinal cord arterial system in a normal anterograde fashion (see Figure 1). This is supported by cases in which fibrocartilaginous material is found in the adjacent radicular arteries to the cord infarction.[35]

Venous route

The fibrocartilaginous material gain access to the venous system of the spinal column and travel initially in a normal anterograde fashion where they would enter the caval system, but then travel retrograde to the venous plexus of Batson and the parenchyma of the spinal cord. In his original article to explain metastatic lesions to the spine from breast, prostate and other cancers, Batson[46] demonstrated this retrograde flow in both animal and cadaveric models, especially in the setting of increased intra-thoracic or intra-abdominal pressures which promotes reversal of venous flow away from the heart. To support this theory, Kepes *et al*[15] demonstrated India ink in the spinal cord veins after it was injected into the vertebral body.

The initial retrograde travel in the arterial route and the eventual retrograde travel in the venous route is postulated to be aided by concomitant increases in the intra-thoracic or intra-abdominal pressure as may occur with lifting, straining, coughing or other activities

associated with Valsalva maneuver. The presence of both arterial and venous FCE in some cases can be explained by either concomitant arterial and venous embolization or arterio-venous shunts that are normally present in the epidural space.[47]

Epidemiology and Clinical Picture

In our review of tissue confirmed cases of FCE, there was some female predominance (63.5% female vs 36.5% male). The youngest patient was 14-years-old, the oldest 78 years, the average age was 41years (SD 20 years), there was no obvious age group predominance but worth mentioning is that nearly half of the patients were under 40 years of age (n = 20, 49%).

The typical clinical picture, as that of any spinal cord infarction, is the onset of dull transient neck or back pain followed by or accompanying a syndrome of myelopathy. This classically involves a sensory level, bladder and/or bowel dysfunction and paraplegia in case of thoraco-lumbar cord disease or quadriplegia with or without respiratory compromise in case of cervical cord disease. The characteristic finding of spinal cord infarction versus inflammatory cord disease is a rapid course of symptoms to nadir, typically over hours. A pathognomonic clinical finding for anterior spinal artery infarction is the sparing of proprioception and vibratory sensation below the sensory level. A characteristic clinical symptom that may point to FCE as a cause for this spinal cord infarction is a temporal correlation with a minor or even unnoticed incident that triggers the increased intra-disc or intra-vertebral body pressure as described above in the "Mechanisms" section. In our review of tissue diagnosed FCE, 61% of the cases presented following such an event. The duration between this trigger event and the onset of symptoms varied from minutes to days, but averaged at 2.4 days. The weakness was asymmetric in 15% of the cases. There was associated neck or back pain in 76%. Nearly 40% of deaths were due to preventable respiratory complications (pulmonary embolism 20%, pneumonia 17%, aspiration 2%). Further details are outlined in Tables 2 and 3.

Table 2 Clinical and imaging characteristics

Characteristics	Value
Temporal Relation to a Suspected Trigger n (%)	25 (61)
Mean, Standard Deviation (in days) for:	
Time from Trigger to Symptom Onset	2.4, 5.7
Time from Symptom Onset to Nadir	1.3, 4
Time from Symptom Onset to Death	126, 401
Characteristic Clinical Picture, n (%)	
Neck or Back Pain	31 (76)
Sparing of Vibration or Proprioception	6 (15)
Vascular Risk Factors, n (%)	
Hypertension	10 (24)
Diabetes mellitus	4 (10)
Active Smoking	2 (5)
Peripheral Arterial Disease	1 (2)
Age > 60 years	1 (2)
Prior Stroke	0 (0)
Two Vascular Risk Factors	4 (10)
Three Vascular Risk Factors	0 (0)
Degenerative Disc Disease (DDD), n (%)	
Total	13 (32)
At the site of lesion	4 (10)
Not at the site of lesion	9 (22)
Schmorl's Node (SN), n (%)	7 (17)
DDD or SN, n (%)	16 (40)
Distribution of Infarction, n (%)	
Medulla Oblungata	7 (17)
Cervical	25 (61)
Thoracic	23 (56)
Lumbar	12 (29)
Conus	6 (15)
CSF Analysis Reported, n (%)	33 (80)
Normal, n (% of cases with reported CSF analysis)	26/33 (79)
↑ Protein, n (%)	6/33 (18)
↑ Cells and Myelin Based Protein, n (%)	1/33 (3)
MRI Reported, n (%)	11
Unremarkable Initial MRI [35, 36, 38]	3 (27)
Gadolinium Enhancement MRI[34, 39]	2 (18)

Diagnosis

Currently FCE is diagnosed on clinical grounds, and confirmed only with biopsy for histo-pathologic analysis (see Figure 3), usually at autopsy with one exception in the literature.[34] Spinal cord infarction due to FCE and any other etiology is often mistaken for inflammatory cord lesions which are more common and more treatable. High likelihood for a clinical diagnosis of FCE is established with the above described clinical presentation along with suggestive CSF analysis and spine MRI findings. In spinal cord infarction, regardless of the cause, CSF analysis can be normal but usually shows elevated protein. It is different from inflammatory cord lesions in that it does not show pleocytosis or increased IgG index.[48,49] MRI of the cord in spinal cord infarctions typically show T2 hyper-intense lesions in a vascular distribution, and unlike those of an inflammatory cord lesion, they typically do not enhance with gadolinium and also can be delayed for 12–48 hours from symptom onset.[50] They can also be pathognomonicaly associated with similar radiologic changes in the posterior aspect of the opposing vertebral body,[51] due to a common blood supply. In FCE, these spinal cord MRI findings are often times opposite a Schmorl's node or a disc protrusion (Figure 2). For a comprehensive approach to diagnose FCE clinically, a step-wise scheme is provided (Table 3).

Table 3 Schematic approach to diagnosing FCE

Step 1-Establish the clinical syndrome of myelopathy, sensory level being most useful.[48, 49]

Step 2-Exclude traumatic and compressive etiologies of myelopathy by history and imaging using spine CT or MRI with and without contrast.

Step 3-Exclude inflammatory etiologies of myelopathy; Mainly[48, 49] by;

* Absence of pleocytosis or increased IgG index in CSF,
* Absence of gadolinium enhancement on MRI of the spine (although two cases of FCE showed enhancement[34, 39]).

Step 4-Establish the diagnosis of spinal cord infarction. This requires the above (Steps 1–3) *plus* one "Major" criterion or two "Minor" Criteria;

Major Criteria:

* Clear vascular distribution by exam, as sparing of proprioception or vibratory sensation[52]
* Clear vascular distribution on imaging modalities mainly axial views of MRI of the spine.[50]
* Radiologic changes, mainly MRI T2 hyperintensity, in the vertebral body or inter-vertebral disc adjacent to the infarction.[51]

Minor Criteria:

* Accompanying new onset neck or back pain[53] (in our review, 76% of FCE cases reported this).
* Symptom progression to nadir or near nadir in less than 4–8 hours[49, 52] (only 46% of FCE cases showed this and the mean time to symptom nadir for all cases was 1.3 days with standard deviation of 4 days).
* Initial unremarkable MRI of the spinal cord with subsequent evolution of an intra-parenchymal lesion[50]

Step 5- Establish the high likelihood of FCE. This requires the absence of other more common etiologies of spinal cord infarction, mainly being aortic pathologies s,[54, 55] *plus* the presence of one or more of the following;

* Temporal relation to heavy lifting or minor neck or back injury, or any event that can cause increased intra-disc or intra-vertebral pressure like axial falls, or events that can reverse the venous drainage of the spinal column away from the heart and to the spinal cord instead, like s Valsalva maneuver (this was evident in 61% of FCE).
* Presence of degenerative disc disease especially protrusions or Schmorl's nodes at or near the infarction.
* Absence of more than one vascular risk factor, as defined in Table 2 (in our review, 20% of FCE cases had one vascular risk factor, 10% had two and none had three or more).

Treatment

To date there are no available specific treatment options. Treatment primarily targets the prevention of complications and improvement of quality of life with pharmacologic and physical therapy. Throughout the literature patients have received oral and intravenous steroids, usually with the thought of treating transverse myelitis, with no improvement. Future research may target the development of intra-venous chondrolytic or fibrinolytic therapy to dissolve the FCE. This can be given empirically upon the clinical suspicion of FCE in the acute setting in an attempt to preserve spinal cord integrity before the ischemic injury occurs, much like thrombolytics are used for acute ischemic strokes of the brain.

Conclusions

Historically FCE is referred to as one of the rare causes of spinal cord infarction. Some clinicians believe this may not be true, but rather it may be under-diagnosed due to vagueness regarding its diagnosis in the clinical setting. A recent study[3] published in 2011 retrospectively reviewed 164 cases that were given a diagnosis of spinal cord infarction and found that 9 (5.5%) met inclusion criteria for high likelihood of FCE. We believe this also may be an underestimation for the following reasons;

- The aforementioned study[3] did not include cases given the diagnosis of idiopathic transverse myelitis (TM) to confirm that they all indeed met the criteria for TM.
- In clinical practice, most cases of clinical sensorimotor spinal cord dysfunction but unclear etiology, are given a diagnosis of idiopathic transverse myelitits (TM) or myelopathy. A large portion of these cases may be due to FCE, specifically those with no signs of inflammation in the CSF. This is highly likely given that MRI T2 lesions in 33% of cases with spinal cord infarctions

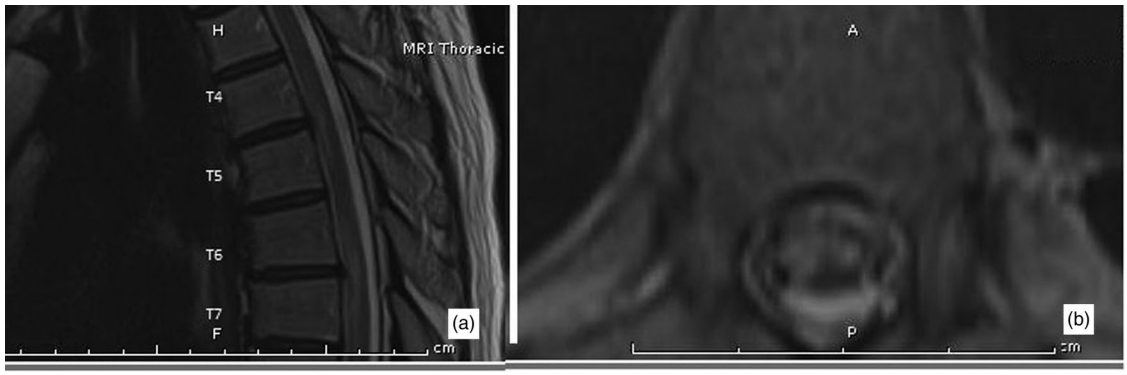

Figure 2 Sagital (a) and axial (b) T2 sequence MRIs showing hyper-intense lesions in the distribution of the Anterior spinal artery in a 63-year-old man clinically diagnosed with spinal cord infarction due to FCE. The lesions are characteristically opposite to disc protrusions at T4–5 and T6–7 thoracic levels.

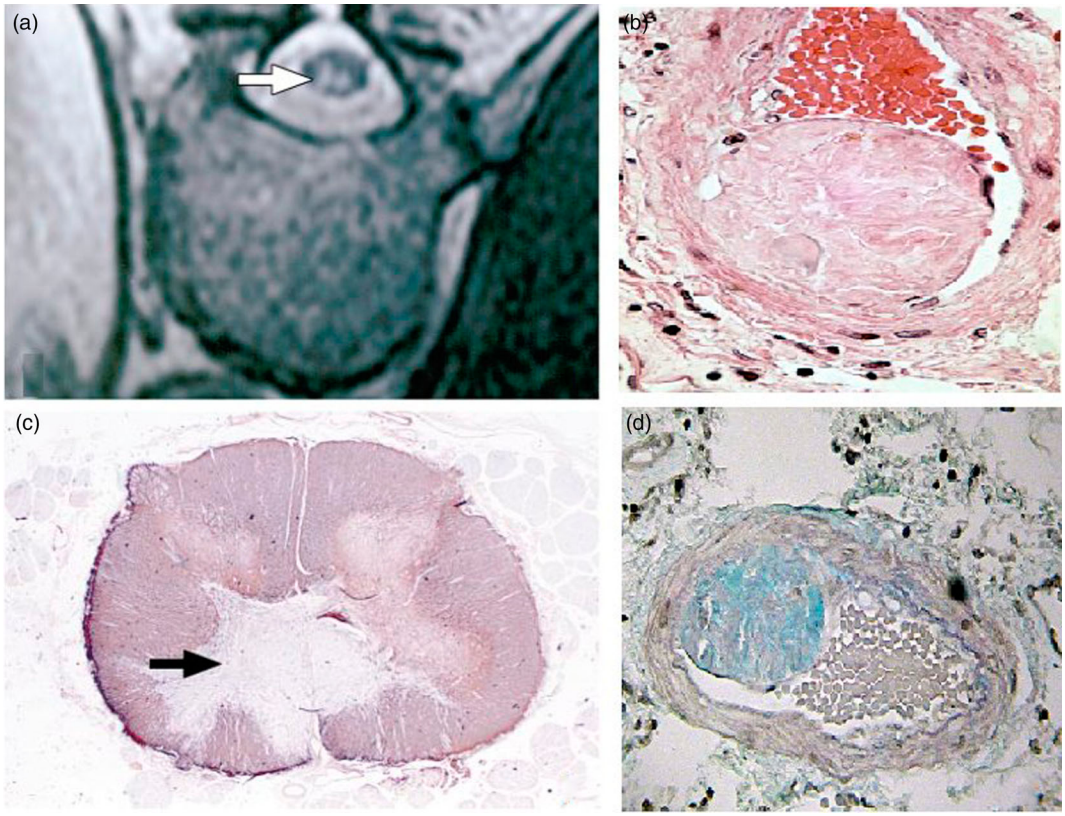

Figure 3 (a) Axial T2 sequence MRI showing hyper-intense lesions in the distribution of the Anterior spinal artery secondary to FCE[37] in a 60-year-old woman. (b) Gross pathology for the same lesion after autopsy. (c) FCE in the anterior spinal artery, Hematoxylin and eosin staining. (d) FCE in the anterior spinal artery, Alcian staining. (a) and (b) are reproduced from[37] with permission from Archives of Pathology & Laboratory Medicine. (c) and (d) are reproduced from[40] with permission from Elsevier.

do not follow an arterial territory[53] and thus are indistinguishable from TM.

- Several of the autopsy confirmed FCE cases were initially presumed to be idiopathic transverse myelitis, even in the absence of inflammatory signs in the CSF.[15,17,24,36,38,39]
- Despite the rarity of the overall reported cases in the literature, there are several incidents where multiple tissue confirmed cases are reported by the same team.[10,16–18,27,28,32] This supports the theory suggesting that when a clinical familiarity with the diagnosis of FCE is present, it is more commonly and accurately diagnosed.
- There have been reports of incidentally found FCE in the spinal cord vasculature on autopsy.[10]

In contrast, other clinicians argue that there is no evidence that these cases of idiopathic myelopathies are indeed spinal cord infarctions due to FCE, furthermore the empiric treatment of inflammatory myelopathy with a short course of intravenous steroids is relatively safe and the benefit largely outweighs the risks, even in cases of uncertain inflammatory myelopathy. Others argue that there is also no evidence against this theory of under-diagnosing FCE. If further research is performed to acutely reverse this condition by chondrolytic therapy, then this notion becomes important. To date there has been no large study that revised cases with established diagnoses of idiopathic TM or myelopathy to rule out that these may be spinal cord infarctions due to FCE. Until this study is performed, it may be reasonable to assume that FCE is more common than reported as this will open the door for research into safe acute medical interventions that can be offered to cases suspicious for FCE upon their presentation, as described above in the "Treatment" section.

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ORCID

Mahmoud A. AbdelRazek 0000-0002-2391-3502

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