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Case report

Idiopathic herniation of the thoracic spinal cord

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Abstract. Since 1974, when Wortzman et al were the first to describe a case of idiopathic spinal cord herniation (ISCH), the number of reported cases has increased owing to magnetic resonance imaging (MRI) now is routinely available for patients with myelopathy and spinal surgeons are becoming more familiar with this clinical entity. This extremely rare herniation occurs exclusively in the thoracic spine, causing slowly progressive myelopathy. Diagnosis is based on ventral displacement of the spinal cord in the thoracic spine. MRI is the technique of choice to exclude a posterior arachnoid cyst, the most common mistaken diagnosis, and to recognize a spinal cord herniation when an anterior dural defect is present. A case of ISCH is reported and a Literature review of this clinical entity often mis-diagnosed has been obtained. (www.actabiomedica.it)

Key words: Idiopathic Spinal Cord Herniation, Brown-Séquard Syndrome, Magnetic Resonance Imaging

Introduction

Since 1974, when Wortzman et al were the first to describe a case of idiopathic spinal cord herniation (ISCH) (1), the concept of ISCH has gradually been appreciated, and the number of reported cases has increased owing to magnetic resonance imaging (MRI) now is routinely available for patients with myelopathy and spinal surgeons are becoming more familiar with this clinical entity. This extremely rare herniation occurs exclusively in the thoracic spine, causing slowly progressive myelopathy. Diagnosis is based on ventral displacement of the spinal cord observed on sagittal MRI. Surgical reduction of the herniated spinal cord usually improves the myelopathic condition (2). The idiopathic or spontaneous aetiologies are different from documented traumatic or postoperative causes (3). Despite the existence, to our knowledge, at least 133 ISCH cases (Table 1) have been reported in the international Literature, whose 14 in the radiological Literature, misdiagnosis and delayed diagnosis remain a major concern (1-60).

With this article we report another case of ISCH of the thoracic tract and provide a thorough review of the Literature about the clinical-radiological correlation to better recognize and characterize this entity.

Etiopathogenesis

ISCH can be classified on the basis of its aetiology into 4 groups: idiopathic (12, 14, 21, 23, 26, 27, 30, 32, 34, 39, 40), iatrogenic (29, 61, 62), post-traumatic (12, 26, 63, 64), and post-inflammatory (37).

The primitive etiopathogenetic mechanism consists in pre-existing dural defect trough which an arachnoid cyst causing pressure. Actually, the real etiopathogenesis still is unknown (14, 17, 25, 38, 65-67). There have been many hypotheses about the cause of the dural defect as it exists congenitally: a dorsally existing arachnoid cyst causing pressure (23), an unrecognized traumatic event (23, 32, 46, 53, 60), and compression by a thoracic disc prolapse (60).

TABLE I. Summary of ISCH cases reported in the Literature.

N°	Author	Year	Journal	Cj/ Rj	N. of Cases	Age/ Sex	Symptoms	Level	mRX	CTm	MRI
1	**Cobb**	1973	j Neurosurg	Cj	NAA						
2	**Hoffman**	1973	j Neurosurg	Cj	NAA						
3	**Wortzman**	1974	J Neurosurg	Cj	1	63/m	BS	D7	X		
4	**Masuzawa**	1981	J Neurosurg	Cj	1	36/f	BS	D4-5	X		
5	**Chan***	1985	Neurosurgery	Cj	NR	NR	NR	NR	NR	NR	NR
6	**Mizuno**	1986	No Schinkei Geka	Cj	1	55/m	SpP	C6-7		X	
7	**Alvisi***	1987	j Neurosurg	Cj	17	-	-	-	-	-	-
8	**Andrews***	1988	j Neurosurg	Cj	5	NR	NR	D	X	X	X
9	**Oe***	1990	Nippon Seikeigeka Gakkai Zasshi	Cj	1	61/m	SpP	D4-5	-	-	-
10	**Isu**	1991	Neurosurgery	Cj	2	43/f	BS	D5-6			X
11						45/f	SpP	D5-6			X
12	**Tronnier**	1991	Nuerosurgery	Cj	1	45/f	S Def	D3-4		X	?
13	**Nakazawa**	1993	Spine	Cj	2	43/f	BS	D2		X	X
14						39/f	BS	D4-5		X	X
15	**White**	1994	J Neurol Neurosurg Psychiatry	Cj	2	61/f	BS	D4	-	-	-
16						39/m	SpP	D8	-	-	-
17	**Borges**	1995	Neurosurgery	Cj	3	68/f	BS	D7-8		X	X
18						69/m	BS	D2-3		X	X
19						48/f	BS	D7-8		X	X
20	**Batzdorf**	1995	Neurosurgery	Cj	1	23/f	BS	D6-7	-	-	-
21	**Kumar**	1995	J Neursurg	Cj	1	38/m	BS	D7-8		X	X
22	**Sahl**	1995	Rofo	Rj	NAA	-	-	-	-	-	-
23	**Hausmann***	1996	Neuroradiology	Rj	4	56/f	BS	D6	-	-	-
24						36/m	SpP	D6-7	-	-	-
25						51/f	BS	D6-7	-	-	-
26						49/m	SpP	D4-5	-	-	-
27	**Matsumura**	1996	Rinsho Schinkeigaku	Cj	1	63/f	BS	D3-4		X	X
28	**Miura**	1996	*Neuroradiology*	Rj	1	49/m	SpP	D5-6	X	X	X
29	**Sioutos**	1996	Spine	Cj	1	34/f	SpP	D6-7			X
30	**Slavotinek**	1996	*Neuroradiology*	Rj	1	22/f	BS	D5			X
31	**Urbach**	1996	*Nueroradiology*	Rj	1	44/m	S Def	D5-6			X
32	**Baur**	1997	*Eur Radiol*	Rj	1	66/f	BS	D10	X	X	X
33	**Lee***	1997	British journal of neurosurgery	Cj	1	19/m1	Paraplegia	-			X
34	**Takahashi**	1997	Spine Spinal Cord	Cj	3	57/m	BS	D2-3	-	-	-
35						56/f	SpP	D3-4	-	-	-
36						68/m	SpP	D7-8	-	-	-
37	**Henry**	1997	Arch Phys Med Rehabil	Cj	1	30/f	BS	D7			X
38	**Uchino**	1997	*Eur Radiol*	Rj	2	71/f	BS	D4-5		X	X
39						61/f	BS	D6		X	X
40	**Dix**	1998	*AJNR*	Rj	1	44/f	BS	D7-8		X	X

(*continued*)

N°	Author	Year	Journal	Cj/ Rj	N. of Cases	Age/ Sex	Symptoms	Level	mRX	CTm	MRI
41	**Miyake**	1998	J Neursurg	Cj	2	45/f	BS	D3-4		X	X
42						53/m	BS	D2-3		X	X
43	**Watters**	1998	*AJNR*	Rj	1	55/f	BS	D3-4		X	X
44	**Abe**	1999	j orthop sci	Cj	1	58/	BS	D7-8	X	X	X
45	**Brugieres**	1999	*AJNR*	Rj	2	54/f	BS	D6			X
46						70/m	BS	D5-6			X
47	**Marshman**	1999	Neurosurgery	Cj	1	55/f	BS-SpP	D8			X
48	**Vallee**	1999	Acta Neurochir (Wien)	Cj	4	28/f	BS	D3-4		X	X
49						58/f	BS	D4-5		X	X
50						40/f	BS	D5-6		X	X
51						49/f	BS	D4-5		X	X
52	**Verny**	1999	Neurochirurgie	Cj	2	28/f	SpP	D3-4			X
53						58/f	BS	D4-5			X
54	**Bartolomei**	2000	Neurosurgery	Cj	1	61/f	BS	D3-4	-	-	-
55	**Ewald**	2000	Neurosurgery	Cj	1	51/f	BS	D5-6			X
56	**Martin**	2000	J Clin Neurosci	Cj	1	31/f	BS	D8			X
57	**Tekkok**	2000	Neurosurgery	Cj	1	49/f	BS	D3-4		X	X
58	**Wada**	2000	Spine	Cj	3	59/m	BS	D4-5		X	X
59						63/f	BS	D3-4		X	X
60						48/m	BS	D5-6		X	X
61	**Adams***	2001	*Neuroradiology*	Rj	1	NR	BS	NR	NR	NR	NR
62	**Aizawa**	2001	Spine	Cj	3	44/m	BS	D8-9	X	X	X
63						60/f	BS	D4-5		X	X
64						59/f	BS	D4-5		X	X
65	**Berbel**	2001	Rev Neurol	Cj	1	56/m	BS	NA			X
66	**Eguchi**	2001	Neurol Med Chir	Cj	1	54/f	SpP	D4-5		X	X
67	**Kawachi**	2001	Neurology	Cj	1	53/m	BS	D10		X	X
68	**Miyaguchi**	2001	Spine	Cj	1	54/f	BS	D3-4		X	X
69	**Morokoff**	2001	J Clin Neurosci	Cj	1	33/f	BS	D8		X	X
70	**Pereira**	2001	Acta Neurochir (Wien)	Cj	1	55/m	BS	D2-3			X
71	**Watanabe**	2001	J Neurosurg 95	Cj	9	43/f	BS	D4		X	X
72						39/f	BS	D3		X	X
73						54/f	BS	D4		X	X
74						71/f	SpP	D4		X	X
75						49/m	BS	D4		X	X
76						47/f	BS	D5		X	X
77						78/f	SpP	D4		X	X
78						56/m	BS	D6		X	X
79						47/f	SpP	D3		X	X

(*continued*)

N°	Author	Year	Journal	Cj/ Rj	N. of Cases	Age/ Sex	Symptoms	Level	mRX	CTm	MRI
80	**Barbagallo***	2002	J Neurosurg	Cj	2	28/f	SpP	D6	-	-	-
81						64/m	SpP	D8	-	-	-
82	**Cellerini**	2002	Acta Neurochir (Wien)	Cj	2	53/m	BS	D8		X	X
83						37/f	BS	D4-5			X
84	**Iyer***	2002	Br J Neurosurg	Cj	1	NR	NR	NR	NR	NR	NR
85	**Massicotte**	2002	Spine	Cj	8	63/m	BS	D5-6			X
86						39/f	BS	D6-7			X
87						50/m	S Def	D4		X	X
88						44/f	SpP	D5-6			X
89						33/f	BS	D7-8			X
90						57/f	SpP	D6			X
91						27/m	BS	D9			X
92						46/f	BS	D4	-	-	-
93	**Inoue***	2003	J Neurosurg	Cj	1	21/m	Headache	-	-	-	-
94	**Nakagawa**	2003	J Spinal Disord Tech	Cj	1	77/f	BS	D6-7		X	X
95	**Sagiuchi**	2003	Neuro Med Chir (Tokyo)	Cj	1	48/m	BS	D7-8		X	X
96	**Sasaoka**	2003	J Spinal Disord Tech	Cj	1	57/m	BS	D2-3		X	X
97	**Aquilina**	2004	Ir Med J	Cj	1	37/f	BS	D4			X
98	**Najjar**	2004	Surg Neurol	Cj	1	32/m	SpP	D8-9		X	X
99	**Rivas**	2004	Neurocirugía (Asturias, Spain)	Cj	1	49/m	BS	D6.7		X	X
100	**Saito**	2004	j orthop sci	Cj	1	66/f	Paraplegia	D6-7			X
101	**Spissu***	2004	Nuero Sci	Cj	1	-/f	BS	-	-	-	-
102	**Srinivasan**	2004	Nuerology	Cj	NAA	-	-	-			
103	**Maruichi**	2004	No Schinkei Geka	Cj	1	53/f	BS	D4-5		X	X
104	**White**	2004	Br J Neurosurg	Cj	3	61/m	BS	D7			X
105						62/f	BS	D6-7			X
106						66/f	SpP	D7			X
107	**Ammar***	2005	Neurosurgery	Cj	3	-	-	D-			X
108						-	-	D-			X
109						-	-	D-			X
110	**Sugimoto**	2005	J Spinal Disord Tech	Cj	1	48/m	BS	D4-5		X	X
111	**Karadeniz-Bilgili**	2005	Journal of Clinical Imaging	Rj	1	36/f	BS	D2-3			X
112	**Maira***	2006	j Neurosurg Spine	Cj	5	-/f	-	-	-	-	-
113						-/f	-	-	-	-	-
114						-/f	-	-	-	-	-
115						-/f	-	-	-	-	-
116						-/m	-	-	-	-	-

(*continued*)

N°	Author	Year	Journal	Cj/ Rj	N. of Cases	Age/ Sex	Symptoms	Level	mRX	CTm	MRI
117	**Ellger**	2006	Clin Neurol Neurosurg	Cj	1	59/f	BS	D2		X	X
118	**Morley**	2006	Australas Radiol	Rj	1	28/f	BS	D5-6			X
119	**Roland**	2006	JBR-BTR	Rj	-	-	-	-	-	-	-
120	**Inoue**	2006	No Schinkei Geka	Cj	1	71/f	BS	D2-3		X	X
121	**Arts***	2006	Acta Neurochir (Wien)	Cj	2						X
122											X
123	**Barrenechea**	2006	J Neurosurg Spine	Cj	7	65/f	BS	D4-5		X	X
124						32/m	Urinary Dys	D7-8		X	X
125						54/f	BS	D2-3		X	X
126						60/f	BS	D2-3		X	X
127						59/f	BS	D5-6		X	X
128						34/m	NR	D7-8		X	X
129						72/m	BS	D4-5		X	X
130	**Bandai**	2006	No To Shinkei	Cj	1	63/f	SpP	D2-3		X	X
131	**Akaza**	2007	Internal Medicine (Tokyo)	Cj	1	56/m	BS	D2-3			X
132	**Yokota**	2007	Neurosurgery	Cj	1	33/m	Horner's Syndrome	C7-D1	X	X	X
133	**OUR CASE**	2007			1	61/m	BS	D6-7			X

Some authors also reported a spinal cord protrusion throughout a defect of the inner layer of a duplicated dura mater (20, 23, 26, 30, 53, 60).

However, to date, there is no radiological or pathological proof to confirm any of this theory.

Tekkok and Coworkers report that it is difficult to define criteria for distinguishing between traumatic and spontaneous cord herniation (51). Many patients, moreover, report a long time, often more than 30 years between the spinal trauma and the onset of symptoms and sometimes it is difficult to understand the relationship between traumatic event and the herniation (1, 54).

According to Isu et al (23) an intradural arachnoid cyst, causing pressure and erosion, migrates throughout a dural fissure arising from a congenital defect, a mild trauma, sometimes unknown or an erosion of the dura by a herniated and calcified disc (1, 26, 30). Degenerative disc prolapse with transdural rupture of disc material, often calcified, has been also proposed as a potential cause of the dural defect (20).

Miyaguchi et al (33) reported a case of ISCH with documented intervertebral disc herniated and calcified as the cause of the ventral defect. About the pathophysiological mechanism leading to spinal cord herniation, some authors (17, 29, 34) report the role played by factors as cardiac pulsations, respiratory movements, and the physiological spinal curve. These factors support the contact between spinal cord and dura mater, resulting, over the time, in a total adhesion with generation of a tear of the dura which will be almost totally blocked by the spinal cord.

Even the pulsation of cerebral spinal fluid (CSF) on the dorsal side of the spinal cord, secondary to respiratory movements and cardiac pulsations, can contribute to generate the herniation, pushing the spinal cord into the extradural arachnoid cyst (Fig. 1) (30). Although Masuzawa et al describe this finding as an extradural arachnoiod cyst, Sioutos et al suggest that it should be classified as a meningeal diverticolum or an arachnoid pseudocyst (46). This pathogenetical mechanism could

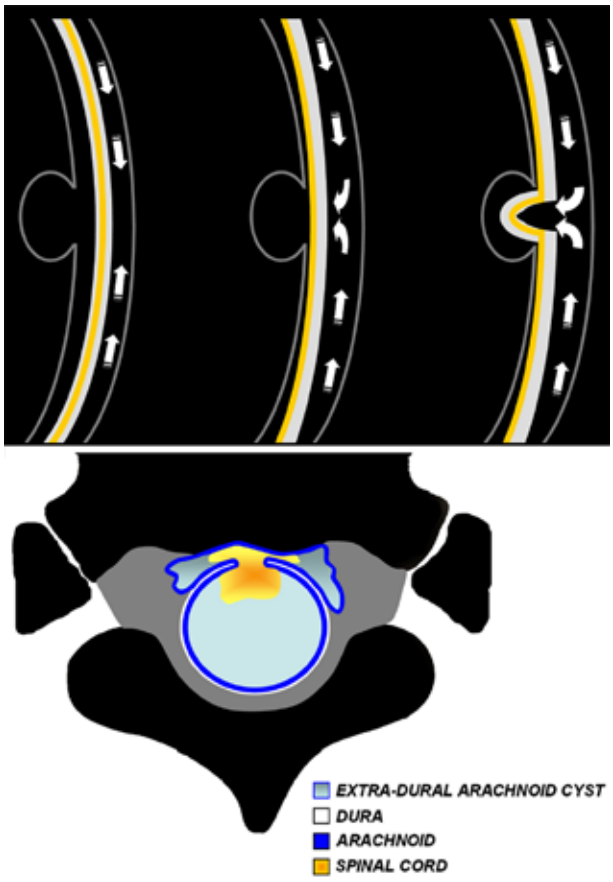

Figure 1. Drawings show, on axial and sagittal planes, the progression of the spinal cord herniation, through a dural defect, toward the epidural space.

result in a *type IIB* spinal meningeal cyst according to the nomenclature proposed by Kumar et al (26), modifying the classification of Nabors et al (68).

Some authors think that the pressure inside the dorsal arachnoid cyst, as it enlarges, could be enough to produce a progressive thinning of the ventral dura mater until a tear appears and the arachnoid herniates through it (Fig. 1) (21, 23). However, dorsal arachnoid cysts are reported only in 25% of ISCH so other mechanism have to be looked for the most part of the cases (21, 23, 32, 46, 47, 53, 60).

Moreover, as highlighted by White and Firth (60), an erosion of the dura due to the pressure by the arachnoid should be more common in intradural tumor than in arachnoid cysts. To strengthen the unknown trauma hypothesis, Tronnier et al (52), stress the presence of inflammatory changes observed in some of the cases reported by Literature, either in the epidural space (4, 52), or in the arachnoid membrane (60).

Other authors (12, 15, 31, 32, 39, 40, 55), think the herniation of the neural tissue through the dural ventral membrane as consequent to a congenitally duplication of the dura mater.

Clinical features

ISCH is an rare clinical entity with almost 90 cases reported in the Literature (66) which typically occurs in middle-aged adults. The median age in all cases reported was 49.9 years with a range from 21 to 78 years and female preponderance (ratio: 2:1) (12, 37, 44, 67).

The most common clinical presentation is a Brown-Séquard syndrome but spastic para-monoparesis, sensory dysfunction or sphincter dysfunction also can be observed (37, 41, 67). The symptoms are slowly progressive suggesting a long course resulting in an arachnoidal adhesions to the nervous tissue with secondary gliosis involving axons. Sudden onset of symptoms has been also reported (30, 60).

The mean duration of the symptoms was 4.25 years (range from 1-12 years) for patients who came in with a Brown-Séquard syndrome and 5.34 years for those presenting with spastic paraparesis (37, 51).

The first symptom is usually a progressive lower-extremity paraesthesia and weakness (54). According to the Literature (20, 34, 60). in the early period, not all of patients become aware of sensorial changes, so the majority of patients arrives at the clinical examination because of increasing difficulty in walking, frequent falls, progressive paraesthesia often preceded by a sensation of warm.

Sphincter dysfunction is reported only in a small percentage of the cases (26, 32, 46, 47, 53, 55, 56).

A increasing impotence is rare but may represent the onset of the symptoms (54). According to the Literature ISCH usually presents in more than 50 % of the cases with symptoms and signs as Brown-Sequard syndrome (3, 14, 26, 30, 34, 39, 41, 46).

The differential diagnosis of ISCH includes Demielinating Disorders, such as Multiple Sclerosis or a Transverse Myelitis (14, 20, 47).

Case Report

We report a case of ISCH in a 61-year-old man with history of a D7 explorative laminectomy for a suspicious of arachnoid cyst.

He was admitted to our institute with a clinical diagnosis of Brown-Séquard syndrome: weakness and paraplegia of the right lower extremity and paraesthesia of the controlateral lower extremity.

A neurological examination showed bilateral tendon reflexes hyperactive and the Babinski's sign was also bilaterally present. These data were suggestive of a spinal/medullar suffering below D9-D10.

The MRI of the dorsal spine T1- (performed before and after i.v. infusion of m.d.c.) and T2-weighted (Fig. 2 and 3) showed a focal atrophy with a right ventral displacement of the thoracic spinal cord at the D6-D7 intervertebral level and a straightforward mushroom-shaped herniation of the spinal cord at the D7 level. A dural cyst, cranially to the herniation, was also found.

The diagnosis of ISCH has been established on the basis of the thin-section MRI of the dorsal spine findings. Surgical intervention was performed (reduction of the herniated spinal cord and duroplasty) with a posterior approach. The patient's postoperative course was uneventful with rapid improvement of the symptoms of the lower extremities within few months.

Imaging

The most part of the ISCH reported have been founded between T2 and T10, with high predominance (79% cfr Brugieres et al) between T4 and T8, and symptoms may appear before than an herniation becomes demonstrable by MRI (15, 66, 67). The MR presentation of ISCH may be characterized by ventral displacement of the thoracic spinal cord.

The herniation through a dural defect may mimic a epidural tumor either ventral or ventrolateral. There is no contrast enhancement. A secondary enlargement of the dorsal subarachnoid spaces is also present. This sign may mimic a dorsal an arachnoid cyst.

A myelography, which led Wortzmann et al (1) to a surgical treatment of the first case of ISCH, can provide only approximate information on a anterior and/

Figure 2. Sagittal SE T1-weighted, enhanced T1-weighted, and T2-weighted magnetic resonance images. Coronal enhanced SE T1-weighted magnetic resonance image. MR images show a focal narrowing of the thoracic spinal cord and a displacement at the herniation level (D6-D7, arrow-heads).

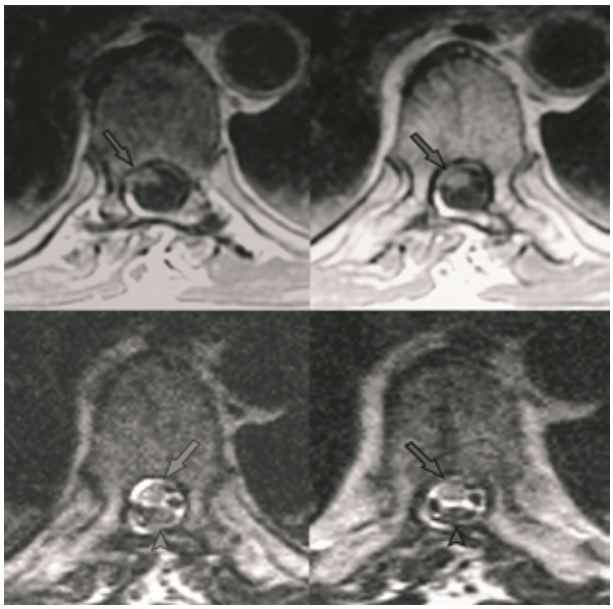

Figure 3. Axial enhanced T1-weighted MR images showing a focal atrophy of the spinal cord at the D7 level with the typical mushroom-shaped herniation through the dura mater.

or lateral displacement of the spinal cord. In the most part of the case, except for the case of Wortzmann et al (1) and White and Firth (60), reported in pre-CT era, a CT-Mielography (CTM) has been performed. The CTM, usually performed before a MRI study, demonstrate no filling defect dorsal to the spinal cord or retention of contrast medium along the ventral aspect of the dural sac. When performed after surgery, it can be useful to exclude the coexistence of a spinal cord herniation with a intradural spinal arachnoid cyst, as reported by Isu et al (2 cases) (23), Oe et al (1 case) (40), Borges et al (1 case) (14). Recently, advances in MRI have reduced the relevance of MCT in the ISCH diagnosis.

MRI typical findings of ISCH show on the sagittal scan an anterior S or C-shaped kink of the spinal cord with secondary enlargement of the dorsal subarachnoid space. On the sagittal plane a decreased spinal cord size (usually atrophic) can be also seen with spinal cord signal changes due to tethering.

The axial MRI images may show the dural defect in addition to the herniation but also arachnoid cyst and associated anomalies including scalloping of the vertebral body, spina bifida or other congenital deformities. Studying CSF dynamics by Phase-contrast cine MRI may be essential to detect a posterior compressing arachnoid cyst replacing MCT.

The most frequent misdiagnosis is: dorsal arachnoid cyst, enlargement of the dorsal subarachnoid space, extradural mass or compression, discal herniation or bulging with secondary spinal cord thinning.

The spinal cord appears typically abruptly deviated to the dorsal parts of the vertebral body at a localized area and the posterior subarachnoid space may be enlarged. These findings and the craniocaudal extent of the displacement are better shown on sagittal MRI scans (12). Hence, radiological techniques are crucial in ISCH diagnosis.

Management

Because of chronic progression of the symptoms, surgery represent the treatment of choice. The aim of surgery is to reduce the herniation, to repair the dural defect and to prevent recurrence.

After the herniation reducing, surgical treatment depends on type of dural defect. There are two main treatment strategies: a) closure of the defect if the nervous tissue is herniated in the epidural space or b) simply widening the aperture when a duplication of the dura mater or a ventral epidural cyst is present (3, 12, 14, 23, 56, 59).

Conclusions

We reported ISCH findings, surgically confirmed, in one case. In addition, we reviewed the Literature of the cases reported of this clinical entity often misdiagnosed. MRI findings are a ventral displacement of the spinal cord in the thoracic spine. MRI is the technique of choice to exclude a posterior arachnoid cyst, the most common mistaken diagnosis, and to recognize a spinal cord herniation when an anterior dural defect is present.

Due to its rare occurrence, its mild, non-specific, and slowly progressive symptoms, it is important to keep in mind this differential diagnosis to achieve an early diagnosis and surgical treatment to prevent major neurological dysfunctions.

Abbreviations: Cj: clinical journal; Rj: radiological journal; mRX: Conventional myelography; CTm: Computed Tomography Mielography; MRI Magnetic Resonance Imaging; m: male; f: female; BS: Brown–Séquard Sindrome; SpP: spastic paralysis; S Def: sensory deficit; Urinary Dys: Urinary dysfunction; D: dorsal spine; C: cervical spine; NAA: not abstract available; NR: not reported; -: not specified/not available; *: data derived from only abstract.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

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