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ORIGINAL ARTICLE

Perilesional myeloradiculopathy with tethered cord in post-traumatic spinal cord injury

R Gross[1], O Hamel[2], R Robert[2] and B Perrouin-Verbe[1]

Study design: A retrospective series of cases.
Objective: To identify, among post-traumatic myelopathies, a specific entity in which clinical and radiological features are not extensive but are strictly limited to the perilesional zone.
Setting: The data set of the Regional Spinal Cord Injury Department of Nantes, France.
Methods: A systematic analysis of all traumatic spinal cord injury (SCI) patients who presented with a neurological aggravation delayed from initial injury, without syringomyelia or extensive myelomalacia.
Results: Twelve patients presenting with this type of complication were identified (that is, four tetraplegics and eight paraplegics). The neurological worsening consisted in weakness of the muscles close to the motor level in five patients, and in isolated at-level neuropathic pain in seven patients. A tethered cord was evidenced by the magnetic resonance imaging (MRI) results in all of the patients. Roots were involved by the tethering on the MRI results in eight cases. Surgery, with untethering and expansile duraplasty, was performed in all cases. Surgery allowed motor recovery in patients who presented with a motor loss (motor score gain range = 1–7 points; median = 3) and decreased pain in all pain patients (decrease on the 10-point numerical rating scale: range = 1–6 points; median = 4).
Conclusions: In traumatic SCI patients, a tethered cord could be responsible for clinical and radiological changes, which are strictly localised to the perilesional area. The term perilesional myeloradiculopathy is proposed for this complication, which requires cord release surgery.
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Keywords: spinal cord injury; post-traumatic myelopathy; tethered cord

INTRODUCTION

The development of extensive lesions in the spinal cord after a traumatic spinal cord injury (SCI) is known as post-traumatic myelopathy (PTM).[1–7] Cord tethering, defined as attachment of the spinal cord to the wall of the bony spinal canal,[8] is known to be a major factor in the occurrence of such a complication.[3–5,9,10] The long-term SCI patient follow-up highlights that PTM is a frequent complication among these patients.[2] In addition to urinary, respiratory, vascular or cutaneous complications, PTM should be screened for and treated. PTMs, which can be either cystic (post-traumatic syringomyelia) or non-cystic (post-traumatic progressive myelomalacic myelopathy), are referred to as progressive pathologies.[3,5,6,11] The possibility that cord tethering could induce late lesions that would not be extensive but remain strictly limited to the injured area has not been evidenced in the literature. Our experience allowed us to identify patients who presented with such a pathology of the spinal cord.

PATIENTS AND METHODS

This study was conducted within the national reference centre for SCI patients at the university hospital of Nantes, France. Within our cohort of around 1500 patients, we selected all of the traumatic SCI patients who presented with changes in their neurological status (worsening) that were associated with a tethered cord on the magnetic resonance imaging (MRI), and who underwent surgery because of this deterioration. Patients with syringomyelia or extensive myelomalacia,[8] as well as those who presented with a worsening within the first month post-SCI, were not included.

Clinical, radiological and therapeutic data were assessed retrospectively at the following four key follow-up periods: immediately after trauma (T_0); before the worsening (T_1); after the worsening (T_2); and 6 months after the surgical treatment (T_3). A standardised clinical evaluation was reproduced at T_0, T_1, T_2 and T_3. The neurological examination was performed according to the International Standards for Neurological Classification of Spinal Cord Injury.[12] The American Spinal Injury Association (ASIA) Impairment Scale (AIS), motor and sensory levels, and motor and sensory scores were established in all of the patients. When pain was present, its typology (nociceptive and/or neuropathic) and location were reported according to the International Spinal Cord Injury Pain Basic Data Set.[13] Pain intensity was assessed by the average pain intensity in the last week evaluated with a Numerical Rating Scale (NRS).[13] The drugs used were identified and their efficacy was assessed on pain intensity as abovementioned. All patients had their analgesic drugs left unchanged between T_2 and T_3.

In addition to these clinical data, the following data were evaluated:

At T_0, the type of spinal injury was specified using the AOSpine Fracture Classification.[14] The type and delay of the surgical stabilisation procedure performed after the trauma was specified if performed.

At T_1, spinal canal stenosis was measured using the method previously described by Perrouin–Verbe *et al.*[2] The most recent MRI was examined to

[1]Department of Physical and Rehabilitation Medicine, University Hospital of Nantes, Nantes cedex, France and [2]Department of Neurotraumatology, University Hospital of Nantes, Nantes cedex, France
Correspondence: Dr R Gross, Department of Physical and Rehabilitation Medicine, University Hospital of Nantes, 85, rue Saint Jacques, 44093 Nantes cedex, France.
E-mail: raphael.gross@chu-nantes.fr
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identify the post-traumatic spinal cord lesions using the criteria defined by Wang *et al*.[8]

At T_2, the MRI that was performed because of the neurological worsening was assessed using the abovementioned criteria.

Surgical treatment of the tethered cord was performed in all of the patients. The procedure was initiated with a laminectomy on at least three vertebral levels. The treatment of a residual spinal canal stenosis was necessary in two patients. Then, the dura mater was opened on its midline. A meticulous dissection of the arachnoiditis allowed untethering the cord and roots. The procedure concluded with an expansile duraplasty with a supersized synthetic tissue (Neuro-Patch, B-BRAUN Medical, Melsungen, Germany). The duraplasty was tacked up to the paraspinal muscles laterally, and to the spinous processes at both extremities of the zone of laminectomy.

This study was performed in agreement with the Helsinki Declaration relative to patients' rights and in accordance with the law on data protection (last version no 2004–801, 6 August 2004). Because the data were collected retrospectively and patient management was not modified and according to French law (last version no 2004–806, 9 August 2004), this study did not require approval by a research ethics committee.

RESULTS

Twelve patients met the inclusion criteria described above and were analysed in this study.

Sociodemographic data, type of initial vertebral lesions and post-traumatic neurological status (T_0)

Clinical and radiological data concerning the initial traumatic SCI are detailed in Table 1. Four patients had lesions at the subaxial cervical spine (from C3 to C7) that caused tetraplegia. Eight patients had lesions at the thoracolumbar junction that caused paraplegia. All patients benefited from spinal surgery in the acute phase, which aimed to decompress the spinal cord and to realign and stabilise the spinal lesions. Two patients had a delayed surgical treatment because of their initially unstable haemodynamics (see Table 2).

Clinical and radiological evolution of the patients before worsening (T_1)

All of the tetraplegic patients presented with changes in their motor level and motor score, which indicated spontaneous motor recovery. The initial median progression of the motor score in these patients was 7 points (min = 6; max = 10, see Table 3).

A major residual stenosis caused by intracanalar bony fragments was observed on the postoperative computed tomography scan in two patients.

A MRI of the spinal cord was performed at T_1 in 8 out of the 12 patients. A focal cyst was noted in six cases and a focal myelomalacia in two cases (see Table 4).

Clinical and radiological changes after worsening (T_2)

Clinical changes. Five patients complained about the onset of motor weakness (two cases were associated with pain), and seven patients complained about isolated pain. Therefore, these two groups of patients are discussed separately. Detailed data concerning the changes in the neurological status of patients between T_1 and T_2 are presented in Figure 1.

Patients with motor loss: Five patients (that is, four tetraplegics and one paraplegic) underwent motor loss in their most caudally preserved muscles. The affected muscles were located at the motor level, just above, or just below (that is, the zone of partial preservation in AIS A patients). In all of these patients, the motor loss was confirmed by two assessments made by at least two examiners. Four of these patients benefited from an electromyographic study, indicating intense acute denervation in the weakened muscles (Table 5). The delay between the initial trauma and the onset of motor loss ranged from 6 to 40 months (median = 16.5 months) in four patients. The median loss on the motor score was 4 points (range: 1–8). Two patients also exhibited neuropathic pain with the same dermatomal distribution as motor loss.

Pain patients: Seven paraplegic patients presented with isolated pain, which had the features of at-level neuropathic pain[13] in all cases. The onset of pain ranged from 1 to 4 months (median = 2 months). The pain intensity was high with a median evaluation of 8 on a 10-point NRS (see Figure 1).

MRI scan data at T_2. All of the patients benefited from a spinal MRI at T_2 (see Table 4). A tethered cord was found in all cases. The data were compared with those obtained at T_1 in the eight patients who

Table 1 Patient population, type of vertebral injury and initial neurological status (T_0)

Patient number	*Patient gender*	*Spinal injury*	*Motor level (R/L)*	*Motor score (R/L)*	*Sensory level (R/L)*	*Sensory score (light touch/ pinprick)*	*AIS*
1	M	Tear drop, C4 (C2.3)	C3/C3	3/1	C4/C4	19/19	B
2	M	Tear drop, C5 (C2.3)	C5/C5	4/5	C5/C5	16/14	A
3	F	Luxation fracture, C4-C5 (C2.2)	C4/C4	3/1	C4/C4	66/22	A
4	F	Luxation, C6-C7 (C3.3)	C6/C7	10/10	C6/C6	76/76	B
5	F	L1 complete axial burst fracture (A3.3.3)	T12/T12	2/2	T12/T12	102/104	A
6	M	L1 Burst-split fracture (A3.2.1)	T12/T12	25/25	T12/T12	68/72	D
7	M	L1 burst-split fracture (A3.2.3)	L1/-	7/24	T12/-	101/102	D
8	M	T10-T11 oblique fracture (C3.2)	T9/T9	0/0	T9/T9	68/72	A
9	M	L1-L2 anterior subluxation associated with a complete burst fracture (B1.2.3 + A3.3)	T10/T10	0/0	T10/T10	74/70	A
10	M	T12-L1 complete rotational burst fracture (C1.3.3)	T10/T11	0/1	T10/T11	74/75	C
11	M	T12 complete flexion burst fracture (A3.3.2)	T11/T11	2/10	T11/T11	88/86	A
12	M	T12 flexion spondylolysis associated with an inferior incomplete burst fracture (B2.3.2 + A3.1.3)	T11/T10	0/0	T11/T10	74/72	A

Abbreviation: AIS: American Spinal Injury Association impairment scale.
The type of injury is given in accordance with the AOSpine Fracture Classification.[14] Motor and sensory scores and levels, as well as the AIS grade are given according to the International Standards for Neurological Classification of Spinal Cord Injury.[12] Motor scores are indicated for the upper limbs in tetraplegic patients (upper extremity motor score), and for the lower limbs in paraplegic patients (lower extremity motor score).

Table 2 Initial spinal lesions and their surgical treatment

Patient	*Vertebral injury*	*Delay of initial surgery (days)*	*Surgical procedure*	*Fused segments*	*Specific findings*
1	Tear drop, C4 (C2.3)	0	C4 corporectomy, arthrodesis with a graft, anterior osteosynthesis	C3-C5	
2	Tear drop, C5 (C2.3)	0	C5 corporectomy, arthrodesis with a graft, anterior osteosynthesis	C4-C6	
3	Luxation fracture, C4-C5 (C2.2)	0	C4 and C5 corporectomy, arthrodesis with a graft, anterior osteosynthesis	C3-C6	
4	Luxation, C6-C7 (C3.3)	0	C7 corporectomy, arthrodesis with a graft, anterior osteosynthesis	C6-T1	
5	L1 complete axial burst fracture (A3.3.3)	0	L1-L2 laminectomy, posterior osteosynthesis	T11-L3	
6	L1 burst-split fracture (A3.2.1)	0	Posterior osteosynthesis	T11-L3	No laminectomy performed
7	L1 burst-split fracture (A3.2.3)	7	T12-L1 laminectomy, duraplasty, posterior osteosynthesis	T11-L3	Posterior dura rip
8	T10-T11 oblique fracture (C3.2)	0	posterior osteosynthesis	T9-L2	No laminectomy performed
9	L1-L2 anterior subluxation associated with a complete burst fracture (B1.2.3 + A3.3)	16	L1-L2 laminectomy , expansile duraplasty, posterior osteosynthesis	T11-L4	Posterior dura rip with conus and roots display
10	T12-L1 complete rotational burst fracture (C1.3.3)	0	T12-L1 laminectomy, posterior osteosynthesis	T10-L3	Posterior dura rip
11	T12 complete flexion burst fracture (A3.3.2)	0	T11-T12 laminectomy, posterior osteosynthesis	T10-L2	Posterior dura rip
12	T12 flexion spondylolysis associated with an inferior incomplete burst fracture (B2.3.2 + A3.1.3)	0	L1 laminectomy, posterior osteosynthesis	T10-L2	Posterior dura rip

had previously undergone MRIs. Only one patient exhibited changes of his spinal cord lesions (slight extension of a cyst).

Evolution after treatment of the aggravation (T_3)

All patients who presented with motor loss benefited from surgery as a first-line treatment, with a median delay time of 2 months (min = 1; max = 4). In contrast, all of the patients who presented with isolated neuropathic pain were first treated with oral anticonvulsants. Because of failed medical treatment, surgery was later proposed and performed in all of these patients. The median time between the onset of pain and the surgery was 50 months (min = 6; max = 103) in these patients.

The results of the surgical treatment are presented in Figure 2 for those patients who presented with a motor loss. All of the patients were improved by surgery. The improvement on the motor score ranged from 1 to 7 points (median = 3 points). In three patients, this improvement allowed the patient to gain one motor level.

The results of the surgical treatment for the pain patients are presented in Figure 3. The median improvement on the NRS for these patients was 4 points (min = 1.5; max = 6). Three patients described a partial recurrence of pain within 2 weeks after surgery. In five patients, the drugs could be reduced from T_3, as pain was frankly reduced at this time.

DISCUSSION

This series of traumatic SCI patients is characterised by the delayed onset of perilesional neurological aggravation (motor weakness and/or at-level neuropathic pain), in the absence of syringomyelia or extensive myelomalacia. The MRI data indicated the presence of a tethered cord in all of the patients. The surgical treatment of this tethered cord allowed symptom improvement, which indicates an association between cord tethering and neurological worsening.

Table 3 Initial motor level (T_0) and evolution of the motor score for the patients from T_0 to T_3

Patient number	*T_0 motor level (R/L)*	*T_0 motor score (R/L)*	*T_1 motor score (R/L)*	*T_2 motor score (R/L)*	*T_3 motor score (R/L)*
1	C3/C3	3/1	6/5	4/3	5/5
2	C5/C5	4/5	8/8	6/6	7/7
3	C4/C4	3/1	6/4	5/4	8/5
4	C6/C7	10/10	13/14	12/14	13/14
5	T12/T12	2/2	7/7	5/1	8/5
6	T12/T12	25/25	25/25	25/25	25/25
7	T12/L2	7/24	22/25	22/25	22/25
8	T9/T9	0/0	0/0	0/0	0/0
9	T10/T10	0/0	0/0	0/0	0/0
10	T10/T11	0/1	3/9	3/9	3/9
11	T11/T11	2/10	18/19	18/19	18/19
12	T11/T10	0/0	0/0	0/0	0/0

Motor levels and scores are given according to the International Standards for Neurological Classification of Spinal Cord Injury.[12] Motor scores are given for the upper limbs in tetraplegic patients (upper extremity motor score), and for the lower limbs in paraplegic patients (lower extremity motor score). Scores are given separately for the right and left limbs (R/L).

The complication that patients in this series underwent can be considered a PTM; although, remarkable differences from the classical picture of progressive PTMs are present. The nosological framework of PTMs has been established by Edgar and Quail,[3] who distinguished between cystic and non-cystic myelopathies. Both entities have been considered progressive. In our patients, the aggravation, in relation to the clinical and radiological data, remained strictly confined to the perilesional zone. With respect to clinical signs, pain patients complained about neuropathic pain, which was strictly at-level. The motor loss only involved the most caudally preserved muscles. In four

Table 4 Data of the MRI of the spinal cord performed at T_1 and T_2

Patient number	*Injured vertebra(s)*	*Cord lesions on MRI (T_1)*	*Cord changes on MRI (T_2)*	*Tethering of the cord*
1	C4	Focal cyst at vertebral level C4 Thin myelomalacia upwards	Unchanged	Ventral
2	C5	Myelomalacia at vertebral level C4 and C5	Unchanged	Circumferential + C6 roots, bilateral
3	C4 + C5	NA	Focal cyst at vertebral levels C4 and C5	Ventral + C6 root, right-sided
4	C6 + C7	Focal cyst at vertebral level C6-C7	Focal cyst, slightly bigger	Ventral + dorsal
5	T11 + T12	NA	Focal cyst at vertebral level T12	Circumferential: cord + cauda equina
6	L1	Focal cyst in the conus medullaris	Unchanged	Dorso-lateral, left-sided
7	L1	Focal cyst in the conus medullaris	Unchanged	Circumferential: cord + cauda equina
8	L1	NA	Myelomalacia at vertebral level T10, T11 and T12	Conus medullaris-cauda equina junction, right-sided
9	T12 + L1	Myelomalacia from T10 to conus medullaris	Unchanged	Dorsal: cord + cauda equina
10	L1 + L2	Focal cyst in the conus medullaris	Unchanged	Dorsal: cord + cauda equina
11	T12	NA	Focal cyst at vertebral level T12	Dorso-lateral, left sided: cord + cauda equina
12	T10 + T11	Focal cyst at vertebral level T11-T12	Unchanged	Circumferential

Abbreviations: MRI, magnetic resonance imaging; NA: not available.
Spinal cord signal abnormalities are given according to Wang *et al.*[11]

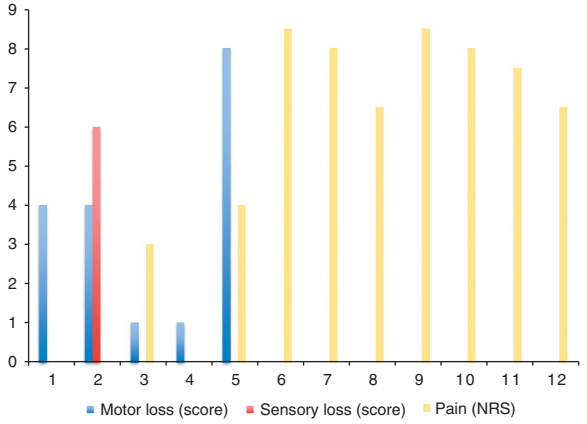

Figure 1 Motor and sensory score changes and pain evaluation, observed between T_1 (before worsening) and T_2 (after worsening) in the 12 patients of our series. Motor and sensory losses are expressed as points on the motor or sensory ASIA score.[12] Pain is evaluated using a 10-point NRS score.

out of five patients, aggravation led to the loss of one function level (3/5 muscle strength or greater), level which had recovered functional strength between T_0 and T_1. These patients, therefore, presented with a recovery/worsening sequence. Symptoms, such as spasticity increase, increased autonomic dysreflexia, bowel and bladder dysfunction, or below-level neuropathic pain, which are frequent in progressive PTM, have not been observed in our patients. In the pain patients of this series, the fair hindsight at surgery time (median = 50 months) suggests withdrawing the hypothesis of a progressive PTM beginning with at-level neuropathic pain.

Radiological data confirmed that the myelopathy in our patients was localised. The signal intensity changes in the cord, whether cysts or malacia, were confined to the immediate surroundings of the initial lesion. When previous MRI data were available (8 patients), the MRIs performed at T_2 were unchanged compared with the former MRI data in all of the patients but one (see Table 4 and Figure 4).

Therefore, it appears that among the PTMs, the pathological condition that is described in this series emerges. This focal entity consists in myelopathy and/or radiculopathy, which is strictly confined to the lesional and perilesional zones. A radiculopathy can be suspected because of an associated tethering of the roots in 9 out of 12 patients. We hypothesise that the term perilesional myeloradiculopathy (PMR) could be used to describe this condition. Although the natural evolution of a PMR to a progressive myelopathy is possible, this does not appear to be systematic, given our data. We, thus, consider PMR a distinct pathological entity rather than an incipient form of progressive PTM.

Progressive PTMs and PMR share the same pathophysiological mechanism, which is the cord tethering. Williams considered the presence of scars of the dura and arachnoid at the site of injury to be a critical factor in the genesis of a syrinx.[4] Edgar and Quail emphasised the role of the tethered cord in both forms of PTMs.[3] Many works have confirmed the causal link between arachnoiditis and syrinx[15,16] or myelomalacic myelopathies.[17] In our study, all of the patients had spinal lesions that were located at the subaxial cervical spine or at the thoracolumbar junction. The anterior cervical spinal fusion allows significant motion of the spinal cord. In thoracolumbar lesions, a short posterior fusion allows residual motion of the lumbar spine in relation to the above fused segments. The tethering in these mobile zones could have induced secondary lesions of the cord and/or roots due to traction forces during spinal movements.[18] These slight lesions were not visible on MRI scans but responsible for clinical and electromyographic changes.

We hypothesise that PMR treatment must follow the same principles and techniques as treatment of progressive PTMs. The aim is to release the tethered cord and prevent relapse by creating an enlarged subarachnoid space (expansile duraplasty). If there is an associated compression of the cord due to spinal canal stenosis, for example, by bony fragments, this issue must also be treated. Surgical treatment led to partial motor recovery in two patients and complete recovery in three patients. In five out of the seven pain patients, surgery was followed by an improvement of at least three points on the 10-point NRS. These encouraging results and the lack of efficacy of the previous medical treatment in these pain patients provide arguments for performing surgery for pain patients with a PMR.

Cord release surgery is delicate. The arachnoidolysis must be performed carefully to prevent additional lesions of the cord or roots. No patient of our series exhibited postoperative worsening. However, three patients experienced partial recurrence of pain within the first two weeks after surgery, possibly caused by re-tethering of the cord

Table 5 EMG data in the patients who presented with a motor loss

Patient number	*T_1 motor score (R/L)*	*T_2 motor score (R/L)*	*Weakened muscles*	*EMG data (T_2): location of acute denervation*	*Intensity of spontaneous activity (fibrillations and/or positive waves)*	*Delay between trauma and EMG*
1	6/5	4/3	Elbow flexors (R + L) Wrist extensors (R)	Not available		—
2	8/8	6/6	Wrist extensors (R + L)	Wrist extensors (R + L)	+ + +	40 Months
3	6/4	5/4	Wrist extensors (R)	Wrist extensors (R)	+ +	18 Months
4	13/14	12/14	Triceps brachii (R)	Wrist extensors (R) Triceps brachii (R + L)	+ + + +	24 Months
5	7/7	5/1	Hip flexors (R + L) Knee extensors (R + L)	Knee extensors (R + L) (hip flexors not explored)	+ + +	30 Years

Abbreviation: EMG, electromyographic.
(R: right side. L: left side). The intensity of the acute denervation (fibrillations and/or positive sharp waves) is semi-quantified using the gradation from + (rare) to + + + + (intense).

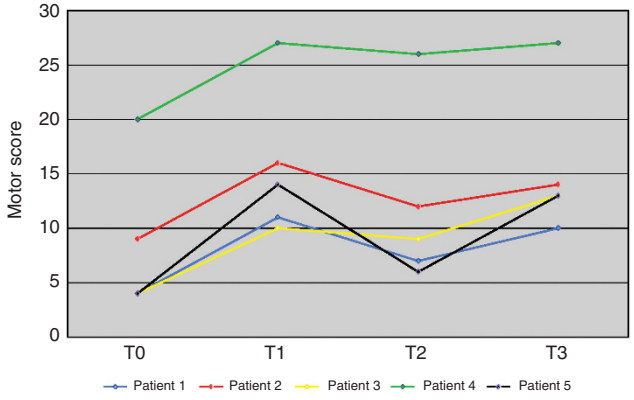

Figure 2 Evolution of the ASIA/ISCOS (International Spinal Cord Society) motor score for the five patients who presented with a motor weakness. Scores are given for T_0 (time of initial injury), T_1 (before worsening), T_2 (after worsening) and T_3 (after surgical treatment). The lower extremity motor score is given for patient 5.

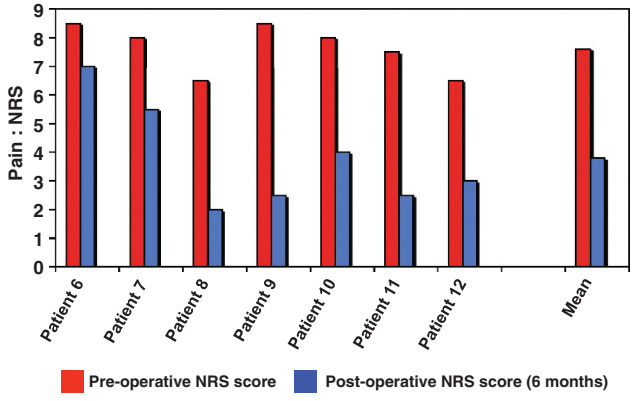

Figure 3 Effects of cord release surgery on pain in our paraplegic patients (mean NRS score).

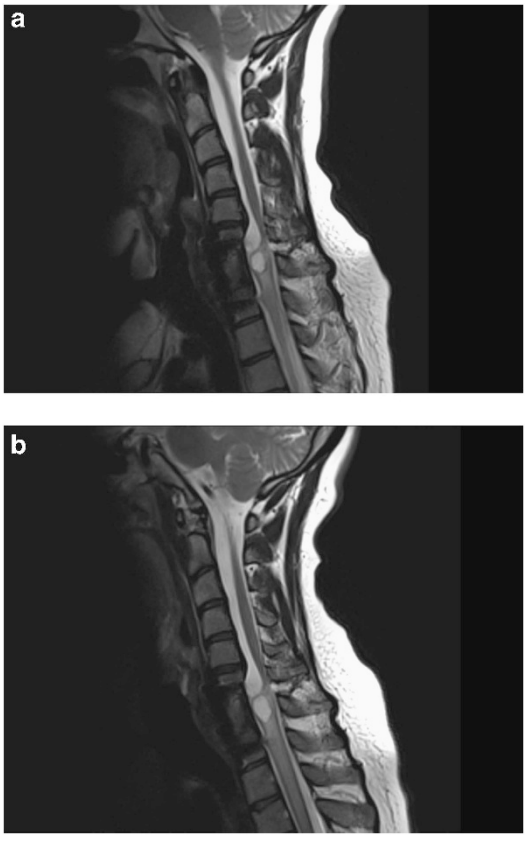

Figure 4 MRI scans of patient 4 before and after the neurological deterioration. (**a**) MRI scan performed at T1, before worsening. A focal cyst is visible at level C6-C7, and the cord is tethered ventrally and dorsally. (**b**) MRI scan performed at T2 (after worsening), 3 months later, showing the cyst, possibly slightly bigger and the tethering.

and roots, which has been previously emphasised.[5] Because arachnoiditis is a form of healing, the possibility to prevent postoperative arachnoiditis is poor. The prevention of postoperative re-tethering could be based upon the quality of the expansile duraplasty, which must be tacked up to the paraspinal muscles or spinous processes, as well as the absence of a subarachnoid haemorrhage. Lee *et al.*[5] also advocated the frequent turning of the patient from side-to-side into the prone position in the postoperative period, which could prevent the deposit of sediment in the recumbent position and the creation of a new tethering scar. Nevertheless,

preventing re-tethering after surgery of a tethered cord remains poorly controlled. Because the risk of pain recurrence, possibly due to re-tethering, is well known, the pertinence of performing a DREZotomy in association with the cord release should be discussed. DREZotomy has been shown to be effective in relieving pain in SCI patients with chronic at-level neuropathic pain. Some studies have demonstrated pain improvement in 68%[19] or 74%[20] of patients with this technique. Sindou *et al.* recently described a two-step technique for SCI patients with at-level neuropathic pain.[19] The first step was to release the cord and roots, and the second step was the DREZotomy. Our study supports the efficacy of cord release surgery alone. A prospective study that compares the results of cord release surgery alone versus cord release associated with a DREZotomy could help to identify the best treatment for at-level neuropathic pain in traumatic SCI patients.

CONCLUSIONS

PMR is a potential complication after a traumatic SCI, consisting in motor loss and/or at-level neuropathic pain. PMR can occur as soon as in the first 6 months post injury. It is caused by damage to the spinal cord and roots if these structures are tethered and submitted to traction, lengthening and compression constraints. This study stresses the necessity of performing an MRI for every SCI patient who presents with chronic neuropathic pain. An MRI can detect a curable cause, such as a tethered cord, for which surgical treatment is effective. This work suggests that to relieve symptoms and prevent further deterioration, early surgery should be proposed for patients who present with PMR.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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