A. Lagares
P. A. Gómez
A. Ramos
J. F. Alén
R. D. Lobato

Traumatic cervical central cord syndrome due to intramedullary hemorrhage studied with MRI: case presentation

Received: 11 January 2001
Revised: 20 September 2001
Accepted: 20 September 2001
Published online: 14 November 2001
© Springer-Verlag 2001

A. Lagares (✉) · P.A. Gómez · J.F. Alén
R.D. Lobato
Department of Neurosurgery,
Hospital “12 de Octubre”,
Crta. de Andalucía km 5,400,
28041 Madrid, Spain
e-mail: algadoc@yahoo.com

A. Ramos
Department of Neuroradiology,
Hospital “12 de Octubre”, Madrid, Spain

Abstract Cervical central cord syndrome is characterized by disproportionately greater motor impairment of the upper than the lower extremities, predominantly distal motor dysfunction, varying degrees of sensory abnormalities, and bladder dysfunction. Although the original description stated that intramedullary hemorrhage is a possible cause of traumatic cervical central cord syndrome, recent studies have not demonstrated its presence on the basis of magnetic resonance imaging (MRI) or pathological evidence. The

authors describe and discuss a case of traumatic central cervical cord syndrome studied with MRI, which presented intramedullary hemorrhage. This supports hemorrhage as a possible cause of traumatic cervical central cord syndrome.

Keywords Traumatic central cervical cord syndrome · MRI

Introduction

Cervical central cord syndrome is characterized by disproportionately greater motor impairment of the upper than the lower extremities, predominantly distal motor dysfunction, varying degrees of sensory abnormalities and bladder dysfunction [13, 14]. Classically it has been stated that traumatic central cord syndrome may be caused by different types of medullary injuries, ranging from edema or concussion, through hematoma or contusion, to ischemia [12, 13, 14]. However, recent studies have not demonstrated the existence of intramedullary hemorrhage or blood products in cases of traumatic cervical central cord syndrome (TCCCS) studied with magnetic resonance imaging (MRI) or at autopsy [7, 11], and have therefore concluded that most probably hemorrhage is not a predominant feature of this syndrome.

MRI is the most sensitive diagnostic tool for studying parenchymal spinal cord injuries, and is even more sensitive in detecting blood, especially in the acute phases of injury. We present a case of traumatic central cord syndrome in which acute MRI evaluation showed intramedullary hemorrhage as the main finding.

Case report

A 70-year-old man was admitted to our department suffering from craniocervical trauma after falling down stairs. On examination he was conscious, oriented and, although he could hardly move his arms, he could move his legs normally. Strength was 3/5 proximal and 1/5 distal in his right arm and 4/5 proximal and 3/5 distal in his left arm. There was no clear sensory level, but he had pain in his shoulders that was elicited by touch. Reflexes were decreased in the upper extremities and normal in both legs. He was catheterized because of urinary retention. Cranial computed tomographic (CT) scans appeared normal. Cervical spine radiographs and helicoidal CT scans revealed a type II odontoid fracture (Fig. 1). Using a 0.5-T MR unit with a cervical coil, a T2-weighted gradient echo [600/28/6 (TR/TE/excitations); matrix, 224×128; flip angle 200°] axial cervical MRI was performed in the first 12 h after the trauma, revealing the existence of a low-intensity intramedullary lesion at the level of C2 (Fig. 2). The patient was operated on 5 days later, reducing the fracture by a posterior C1–C2 wiring. At discharge, the patient had nearly normal strength proximally in both arms, with 3/5 in both hands. Neurological examination 1 year after the trauma showed a minor deficit in flexoextension of the right hand fingers and mild hyper-reflexia. A cervical MR examination (Fig. 3) performed 1 year after trauma revealed the presence of a cystic cavity in the place where the hematoma had been.

Fig. 1 Cervical radiograph (A) and helicoidal computed tomographic scan (B) showing type II odontoid fracture

Fig. 2 A Sagittal T2-weighted magnetic resonance (MR) image showing odontoid fracture and cervical stenosis due to spondylosis. B Axial T2-weighted gradient echo image shows hypointense intramedullary lesion compatible with intramedullary hematoma

Discussion

The pathological basis of central cord syndrome is a matter of controversy. Schneider et al., in their original description of the syndrome, commented on a case described by Spiller of the autopsy of a man who had suffered a typical traumatic central cord syndrome, where hemorrhage was found in the cord [13]. Schneider also described, in three of his reported cases in which autopsy was performed, the existence of central cord destruction involving several segments of the affected cord [13, 14]. The fact that a large group of patients suffering from this syndrome recovered with mild deficits led to the idea that a major component of this syndrome might be edema, a condition that could resolve without leaving major damage to the cord. Various authors have also reported that the appearance of this syndrome after cervical trauma is more frequent in patients with cervical spondylosis.

Trauma without fracture or dislocation to an already compressed cord could lead to further damage due to edema or ischemia [6, 12, 13, 14]. Thus, it has been stated classically that several pathological entities such as edema, hemorrhage or ischemia, or a combination of them, could lead to the same clinical expression.

MRI has emerged as a very sensitive tool for detecting and differentiating spinal cord lesions in the acute trauma scenario [3, 9, 15]. The patterns of spinal cord injury on MRI resemble those seen in cerebral injury, and there is good correlation between different lesions seen on MRI and pathology. MRI is also very sensitive in differentiating the presence of blood and edema in the acute or subacute stages. Although various technical problems may hamper the use of MRI in patients suffering traumatic spinal cord lesions, various groups of investigators have demonstrated its diagnostic and prognostic utility, as lesions seen on MRI can be related to the degree of neurological deficit and to prognosis [3, 15]. Flanders et al., in

Fig. 3 Sagittal T2-weighted MR image 1 year after trauma. There is a cystic cavity where the hematoma was originally located. There is good reduction of the odontoid fracture

a series of 78 patients with acute cervical spine trauma, identified various patterns of spinal cord injury and related them to the degree of neurological injury [3]; all patients with hemorrhagic lesions in this series had complete neurological deficit, and the presence of hemorrhage indicates a poor prognosis. In a more recent MR study on patients with cervical spine injuries, Selden et al. presented similar findings in 55 patients [15]. However, although the presence of hemorrhage was related to worse neurological status and prognosis, not all patients with spinal cord hemorrhage showed complete injury, and some patients presented full neurological recovery, which was inversely related to the extent of the hematoma.

In a series of 21 patients with acute cervical trauma, Mirvis et al. [9] found that none of those presenting central cord syndrome had MR lesions compatible with hemorrhage, and all showed hyperintensity on T2-weighted images – a finding compatible with edema. More recently, Quencer et al. [11] reported a series of nine patients with traumatic central cord syndrome examined with MRI, three of whom also underwent necropsy studies. In none of them did MRI reveal images compatible with hemorrhage within the cord, even after employing specific sequences (T2-weighted gradient echo), and the pathological study showed no evidence of blood or blood products in the cord. The major finding in this study was the association of central cord syndrome with hyperintensity on T2-weighted images within the cord – a finding compatible with edema – which correlated with swelling and degeneration of large areas of the lateral corticospinal tract

and preservation of central cord areas. All these findings have been confirmed in a case presentation by Martin et al. [7]. The presence of MR findings compatible with intramedullary hemorrhage in our case confirms that, although perhaps infrequent, intramedullary hemorrhage could also be responsible for the production of this syndrome, as stated by Schneider et al. [13]. The limited extent of the intramedullary hematoma could relate to the incompleteness of the injury presented by our patient, and also to the recovery he experienced.

Prognosis after TCCCS has generally been considered good. Of the original series of patients in Schneider's series [13], five out of nine patients survived, and all five presented significant neurological improvement of their original situation. More recent series have confirmed these findings. Braakman and Penning [2], in a series of 57 patients with TCCCS, showed that of 32 patients presenting severe manifestation of the syndrome (paralysis of arms, paresis of legs), 13 patients experienced a complete recovery, 6 remained slightly handicapped, 3 had more severe spastic paraparesis, 4 died, and in 6 laminectomy had to be performed because of aggravation of neurological symptoms. Of the 25 patients with a moderate syndrome (marked paresis of hands or arms, slight paresis of legs), 15 recovered completely, 3 remained slightly handicapped, 1 remained more severely handicapped with spastic paresis, 1 died, and in 5 laminectomy was performed because of aggravation of neurological symptoms. It is not clear whether the nature of the pathological lesion (concussion, contusion or ischemia) responsible for the syndrome affects the final outcome. The presence of hemorrhage is related to bad prognosis in spinal trauma [15], but it is not clear whether this is also the case in TCCCS, as the experience of acute MR imaging of this syndrome is still limited.

The appearance and growth of a cystic cavity where the original lesion was located has been suggested in some cases to be the origin of later deterioration of these patients, even leading to surgical exploration of these cavities [4]. As a result of the injury, hematomyelia could resolve in liquefaction and myelomalacia, leading to the formation of a cyst [1, 16]. On MRI, the prevalence of cystic lesions in injured spinal cords is almost 50% [1]. These cystic cavities, in contrast to post-traumatic syringomyelia, are normally located opposite the fracture, and do not extend over more than one segment, nor enlarge [16]. The only symptom related to their presence is spasticity [1]. Surgical treatment is not recommended, as they are not a cause of deterioration and their direct puncture or decompression has not prevented their refilling nor changed the clinical findings, as shown in recent series [1, 16]. The presence of such a cyst in the late MR examinations in our patient confirms the affection of the medulla on the site where the hematoma was, and its presence could be related to the residual symptoms our patient experiences.

Different explanations for the functional changes observed in the central cervical cord syndrome have been provided. Schneider and co-workers accepted Foerster's theory that the lateral corticospinal tracts are laminated, so that the fibers innervating the cervical segments lie medially to those subserving more caudal segments [13, 14]. Thus, a lesion lying in the center of the cord would affect mainly fibers innervating cervical segments. However, it has been demonstrated that there is a lack of somatotopic organization of the corticospinal tract in primates [5, 10]. An alternative explanation proposed by some authors is that the function of the lateral corticospinal tract is more important for hand and arm movement than it is for lower extremity use [6]. Thus, any lesion located in the center of the cord or more laterally, producing sufficient damage to the lateral corticospinal tract at the cervical level, would impair upper extremities function to a higher degree. Pathological evidence arising from more recent studies on this syndrome reveals that damage to the lateral corticospinal tract is a predominant feature, supporting this hypothesis [7, 11].

Some patients with this syndrome also present uncomfortable, painful, burning sensations over the shoulders, arms, and chest in varying degrees, which are induced by touch. Both hyperpathia and allodynia have been related to lesions of the spinothalamic tract at its decussation in the cord [5]. For a lesion to produce this clinical feature it should be located in a central cord position, as occurred in our patient.

On the other hand, clinical variations of this syndrome could also be due to the different nature of the underlying lesions leading to this condition [8]. As has been shown, the main feature of this syndrome is the lesion to the lateral corticospinal tract, which is responsible for the motor impairment that characterizes it. However, this can be produced by lesions with variable extensions in different locations through the medulla, capable of damaging other tracts and thus adding different clinical features to the syndrome [8].

References

1. Backe HA, Betz RR, Mesgarzadeh M, Beck T, Clancy M (1991) Post-traumatic spinal cord cysts evaluated by magnetic resonance imaging. *Paraplegia* 29:607–612
2. Braakman R, Penning L (1971) Injuries to the cervical spine. *Excerpta Medica*, pp 95–96
3. Flanders AE, Schaefer DM, Doan HT, Mishkin MM, Gonzalez CF, Northrup BE (1990) Acute cervical spine trauma: correlation of MR imaging findings with degree of neurological deficit. *Radiology* 177:25–33
4. Fox JL, Wener L, Drennan DC, Manz HJ, Won DJ, Al-Mefty O (1988) Central spinal cord injury: magnetic resonance imaging confirmation and operative considerations. *Neurosurgery* 22:340–347
5. Hopkins A, Rudge P (1973) Hyperpathia in the central cervical cord syndrome. *J Neurol Neurosurg Psychiatry* 36:637–642
6. Levi ADO, Tator CH, Bunge RP (1996) Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. *Neurosurgery* 38:179–185
7. Martin D, Schoenen J, Lenelle J, Reznik M, Moonen G (1992) MRI-pathological correlations in acute traumatic central cord syndrome: case report. *Neuroradiology* 34:262–266
8. Merriam WF, Taylor TKF, Ruff SJ, McPhail MJ (1986) A reappraisal of acute traumatic central cord syndrome. *J Bone Joint Surg Br* 68:708–713
9. Mirvis SE, Geisler FH, Jelinek JJ, Joslyn JN, Gellad F (1988) Acute cervical spine trauma: evaluation with 1.5-T MR imaging. *Radiology* 166:807–816
10. Nathan PW, Smith MC (1955) Long descending tracts in man. 1. Review of the present knowledge. *Brain* 78:248–303
11. Quencer RM, Bunge P, Egnor M, Green BA, Puckett W, Naidich TP, Post MJD, Norenberg M (1992) Acute traumatic central cord syndrome: MRI-pathological correlations. *Neuroradiology* 34:85–94
12. Rand RW, Crandall PH (1962) Central spinal cord syndrome in hyperextension injuries of the cervical spine. *J Bone Joint Surg Br* 44:1415–1422
13. Schneider RC, Cherry G, Pantek H (1954) The syndrome of acute central cervical spinal cord injury with special reference to the mechanism involved in hyperextension injuries of the cervical cord. *J Neurosurg* 11:546–577
14. Schneider RC, Thompson JM, Bebin J (1958) The syndrome of acute central cervical spinal cord injury. *J Neurol Neurosurg Psychiatry* 21:216–227
15. Selden NR, Quint DJ, Patel N, d'Arcy HS, Papadopoulos SM (1999) Emergency magnetic resonance imaging of cervical spinal cord injuries: clinical correlation and prognosis. *Neurosurgery* 44:785–793
16. Sgouros S, Williams B (1996) Management and outcome of posttraumatic syringomyelia. *J Neurosurg* 85:197–205