

Coincidence of spinal tumor (astrocytoma) and non-specific encephalomyelitis

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Submitted: 2012-05-13 *Accepted:* 2012-09-01 *Published online:* 2012-12-28

Key words: **intra-medullary tumors; demyelination; neuromyelitis optica;
acute transversal myelitis; magnetic resonance imaging;**

Neuroendocrinol Lett 2012; **33**(8):769-772 PMID: 23391981 NEL331012C04 ©2012 Neuroendocrinology Letters • www.nel.edu

Abstract

The aim of this paper is to demonstrate that differential diagnostics of intra-medullary spinal lesions can sometimes be very difficult, even when the latest complement examinations are used, including magnetic resonance imaging. The particular case dealt with here was complicated by the presence of two different pathological processes.

We present the case report, where the case history, clinical course and results of the paraclinical examinations, including the magnetic resonance imaging, suggested an intra-medullary inflammatory/demyelinating process. The post-mortem histological finding was a surprise, because besides signs of non-specific encephalomyelitis, it also displayed signs of a spinal tumor (histological character of diffuse astrocytoma grade II-III).

We would like to emphasize some important facts in our discussion, especially from the perspective of the magnetic resonance imaging. Finally, we would like to ask if the presence of both pathologies (astrocytoma and nonspecific myelitis) was coincidental, or if the myelitis had an iatrogenic etiology (by therapy, by infection during the lumbar punctions).

INTRODUCTION

There are several cases where differential diagnostics according to the information from MRI scans gives us a wide range of possible diagnoses, especially in those cases where the clinical symptoms and other paraclinical tests do not help to specify this wide differential diagnostics. The situation is complicated by the fact that spinal cord biopsy is not usual, because of its high risk of iatrogenic damages. This case report documented difficulty to distinguish spinal cord neoplasm from inflammatory/demyelinating process.

CASE REPORT

A healthy 32-year-old patient presented with paraesthesia irradiating into the lower limbs. Later, paraparesis with sensitive loss in all qualities appeared and developed into a transversal spinal lesion with a sensitive borderline in the Th4 segment. An MRI showed a longitudinally extensive intra-medullary lesion (C5–Th7) with contrast enhancement at the level of Th5 (Figure 1A–C). Cerebrospinal fluid (CSF) revealed slightly increased protein, cytology – lymphocytes 17%, monocytes 19%, and neutrophil leukocytes 63%; no evi-

dence of oligoclonal bands. Brain MRI was normal. In the differential diagnostics, the radiologist mentioned both possible diagnoses, spinal cord inflammation, and also neoplasm. There was a rapid extensive progression in follow-up MRI after seven days with haemorrhagic component (Figure 1D) and more likely an inflammatory etiology was suspected – an acute transverse myelitis, acute disseminating encephalomyelitis. Repeat CSF showed increased pleocytosis (lymphocytes 30%, neutrophil leukocytes 65%) and haemorrhagic component in accordance with MRI. Virological and bacteriological examinations and tests of the autoimmune diseases were negative, excepted anti-aquaporine 4 antibody positivity in the only sample of the CSF, neuromyelitis optica (NMO) was suspected. The patient was treated with a pulse dose of corticosteroids and four plasma exchanges. He made a good recovery and was dismissed. This treatment, followed by the physiotherapy, led to a relatively stabilised status. One month after dismissal intense pain of the cervical and upper thoracic spine occurred and worsening mobility of the lower limbs developed in plegia.

After three month the course of the disease became complicated as a result of non-obstructive hydrocephalus with intracranial hypertension symptoms, meaning

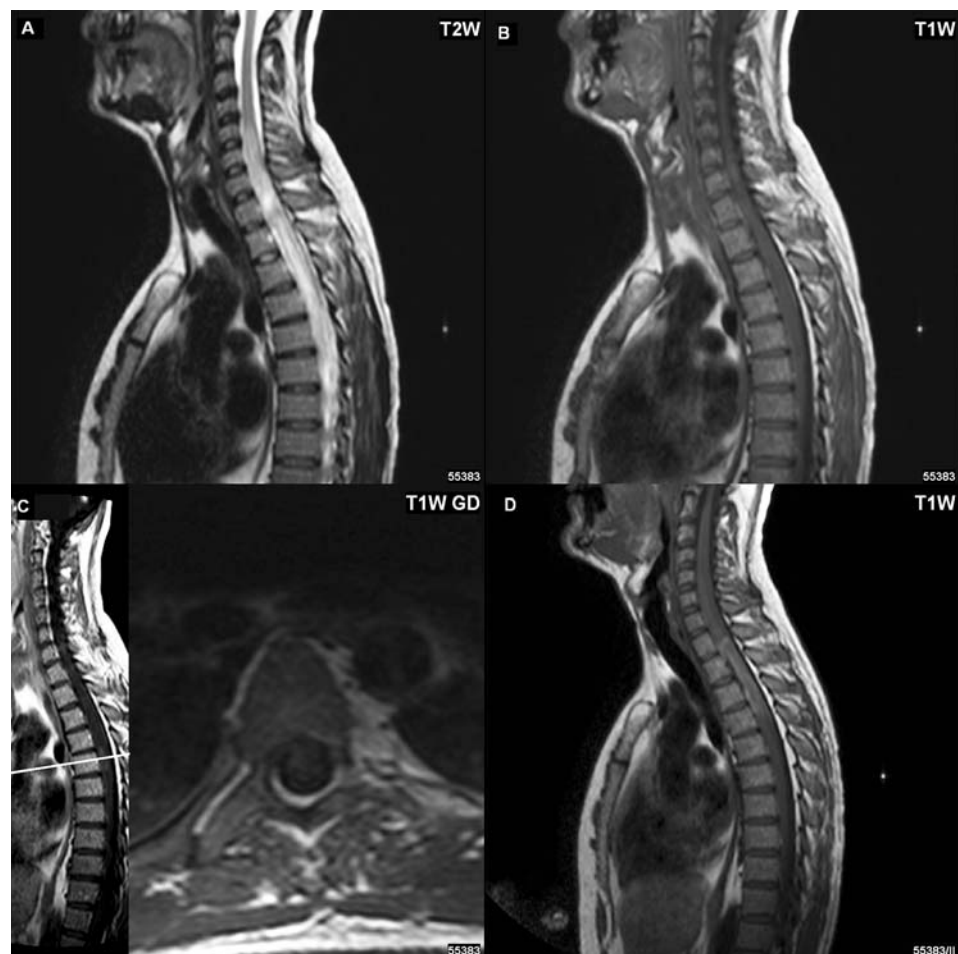


Fig. 1. A–C: MRI of the spinal cord. A significant widening of the spinal cord in the area from C5 to Th7, with hypersignal in T2WI, without significant correlation in T1WI. Postcontrast T1WI showed the enhanced oval lesion at the level of Th5. **D:** Repeat MRI after 7 days. MRI revealed hypersignal in T1WI as new finding (haemorrhagic component).

that a ventriculoperitoneal shunt had to be implanted, and most of the symptoms of intracranial hypertension disappeared. An MRI of the brain five days after the implantation of the shunt demonstrated regression of the hydrocephalus. Hypersignal lesions in T2WI in cerebellum encircling the fourth ventricle were interpreted as inflammatory changes.

Clinically, there was a gradually worsening of mobility of the upper limbs, there was a practical plegia in lower limbs, and pain in the C and Th spine particularly were presented (opiates were applied). Finally, the process spread to the brain stem (Figure 2A–D) and patient developed bulbar symptoms (respiratory problems, dysphagia) and died a few days later.

Post-mortem histological findings described the tumorous lesion – a diffuse astrocytoma in combination with a non-specific encephalomyelitis without detailed signs, massive oedema, and haemorrhage were also seen (Figure 3A,B).

DISCUSSION

MRI has become a dominant diagnostic modality for the spinal cord. In the first phase, MRI allows us to reliably distinguish the intra- and extra-medullary pro-

cesses. These two clinical units usually have different clinical management. The differential diagnostics of intra-medullary lesions is very wide-ranging; the most important aim is to distinguish tumorous from non-tumorous lesions (Brinar *et al.* 2006). It is often not easy, sometimes enhancement pattern and brain MRI can help.

Intramedullary tumors are quite rare lesions and they represent only 5–15% of all tumors of the spinal structures (Van Goethem *et al.* 2004). The most common intramedullary tumors in childhood are astrocytomas, followed by ependymomas; in adults the order is the opposite. Astrocytomas – on the MRI scans, the lesion extends the spinal cord; there is a typical increased signal in T2WI and they are iso- and hypointense in T1WI. Their localization is usually eccentric and they are spread into other segments of the spinal cord. In comparison with intracerebral tumors, most intramedullary astrocytomas show spotted irregular postcontrast enhancement. Ependymomas – MRI scans show the focus of the increased signal in T2WI and lower signal in T1WI, cysts are presented in 50% of all cases, while calcification is very rare in spinal tumors. Other histological forms of intramedullary tumors are not so common (hemangioblastomas, ganglioneuroblasto-



Fig. 2. A-C: MRI of the spinal cord. The spinal cord was still enlarged with hypersignal in T2WI, inhomogeneous signal in T1WI. Postcontrast T1WI showed extensive enhancement of spinal cord. **D:** Progression of the local changes in the medulla oblongata was observed.

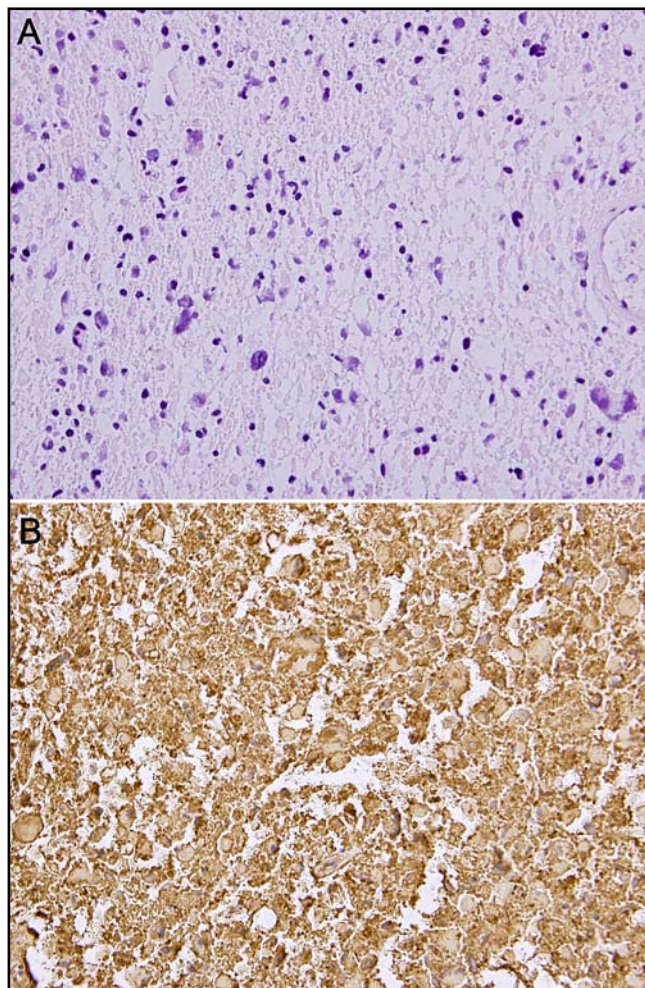


Fig. 3. A,B: Histopathology. Fibrillary and partially gemistocytic neoplastic cells with increased cellularity and occasional nuclear atypia without marked mitotic activity (A). Diffuse GFAP expression confirms the astrocytic origin of the tumorous cells (B).

mas, lymphomas), except perhaps metastatic lesions (Do-Dai *et al.* 2010). Neuromyelitis optica (M. Devic)-in the MRI scans, there are typical hypersignal lesions in the T2WI, occupying at least three spinal segments, which can have a post-contrast enhancement (Sheerin *et al.* 2009). Demyelinating/inflammatory lesions like multiple sclerosis (MS) was not so probable in our case, this was also due to negative findings on the MRI of the brain (which could have suggested the diseases mentioned above).

In retrospect of this case an inflammatory/demyelinating etiology was suspected more likely than tumour,

also due to the progression over time. Considering both the size and positive NMO-immunoglobulin G in the only sample of the CSF, NMO was considered, but the findings were not completely in accordance with the diagnostic criteria for NMO (Saiz *et al.* 2007).

Finally, we considered the diagnosis of acute transverse myelitis, even if it is a clinical diagnosis which has a wide-ranging etiology (demyelination, including multiple sclerosis, NMO, viral infections and other inflammatory disorders like lupus erythematosus, neurosarcoidosis, idiopathic, etc. (Jacob & Weinschenker 2008). Subsequently, because of the presence of inflammatory changes in the brain stem we finally gave the diagnosis of encephalomyelitis with a haemorrhagic component.

The biopsy was not indicated, biopsy, which it is not a part of the neurosurgical operation is not usual and we do not have a lot of experience of it. The results of the biopsy would have had no significant influence on the further course of the disease, although it could have led us to omit some therapeutic trials...

We still have some doubts concerning the coincidental presence of the astrocytoma and non-specific myelitis. Was it an accidental presence of these two pathological units, or was the myelitis iatrogenic (induced by the therapy, infection caused by the lumbar puncture etc.)?

ACKNOWLEDGEMENTS

The financial support was from the national research grant agency RVO-VFN64165/2012 and MSMTO21620849.

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