Split cord malformation incidentally discovered

We present here the case of an 80-year-old woman admitted to hospital for pain in the dorsal spine that was resistant to medical treatment, but with no neurological deficits. The patient had a congenital hair patch in the lumbar region and she also suffered from osteoporosis. Magnetic resonance imaging (MRI) of the dorsolumbar region (Fig. 1) showed the expected D7–D8 vertebral body compression fractures, but also an unexpected L4 diastematomyelia with associated tethered spinal cord and syringomyelia. The split cord malformation was considered inoperable because of the patient’s advanced age and lack of neurological deficit. Thus, we performed D7–D8 vertebroplasty, using a posterior left intercostals—transverse approach (Fig. 2), while bearing in mind the need to cause as little trauma as possible to avoid spinal cord damage, which has been described in association with spinal surgery or trauma. There were no complications, and the patient was able to walk 6 h later; she reported satisfactory pain control within 24 h and her status remained unchanged during the following 3 months.

Diastematomyelia, or split cord malformation, is a congenital anomaly consisting of sagittal division of the spinal cord into two hemicords separated by a spur [1]. It is usually treated with surgery, and even prophylactically in asymptomatic patients, as many authors have noted that, once a neurological deficit appears that can be related to growth, spinal surgery or trauma, there is only a small chance of complete recovery [2]. Although this malformation was considered rare in the pre-MRI era [3], nowadays, the true incidence of this pathology is not known [4].

The case we report here suggests that diastematomyelia may be less unusual than we thought and may even be an incidental finding in adults. Our case also confirms that percutaneous vertebroplasty is a safe and effective method for treating vertebral compression fractures [5], even in those patients who have asymptomatic, untreated split cord malformations.

Figure 1  MRI: sagittal T1 and T2-weighted (A, B) and axial T2-weighted (C) views of the D7–D8 vertebral body compression fracture, and the L4 diastematomyelia associated with a tethered spinal cord and syringomyelia.
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Figure 2  Fluoroscopy images showing lateral (A) and anteroposterior (B) views of the D7–D8 vertebroplasty, using a posterior left intercostals – transverse approach.

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References


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Two cases of vertebral artery dissections with late stroke recurrences

Case 1: a previously fit 30-year-old man was admitted because of sudden cephalalgia with vertigo, nausea and vomiting triggered by defecation. Diffusion-weighted magnetic resonance imaging (DWI) showed bilateral cerebellar infarctions. Both digitalized angiography and magnetic resonance angiography (MRA) showed smooth tapering of the right vertebral artery. No intramural thrombus or double lumen were observed. Analysis of the cerebrospinal fluid was normal. Electrocardiogram (ECG) and transesophageal echocardiography (TEE) were normal. Examination of cervical and cerebral arteries by ultrasound and fat-suppressed T1WI was normal. Symptoms improved quickly and the patient was discharged with a daily dose of aspirin. Three months later, he presented sudden severe cephalalgia, vertigo and vomiting. DWI showed a new recent bilateral cerebellar infarct. Angiography showed the tapering of the right vertebral artery (Fig. 1A) but with a thrombus in the lumen of the artery, which was compatible with an arterial dissection. Intravenous heparin followed by oral anticoagulation was administered. A follow-up angiography (Fig. 1B), performed 4 months later, showed a false aneurysm. The anticoagulant was replaced by an antiplatelet drug and no further neurological deterioration was observed.

Case 2: a healthy 19-year-old man was admitted with repeated transient ischemic attacks during and after a soccer match. He complained of right and left hemianopsia, left hemiparesis and dysesthesia with a mild headache. DWI showed a recent right occipital infarct. Ultrasound examination of cervical arteries and MRA were normal. Fat-suppressed T1WI was not performed. The blood results showed a low platelet count (113,000/mm³) but no other coagulation or auto-immune abnormalities. ECG and TEE were normal. The patient was discharged with a daily dose of aspirin. Five months later, he presented successively right and left dysesthesia, and dysphasia. MRI showed a new left occipital infarction. Angiography showed occlusion of the left vertebral artery and stenosis of the basilar artery. Antiocoagulation therapy was initiated. A follow-up angiography after 3 months gave regressive findings. Oral anticoagulation was stopped after 4 years, but shortly afterwards the patient complained of transient vertigo, ataxia, dystarhria and facial paresis. Angio CT showed a new stenosis of the basilar artery. Angiography showed stenosis of the left vertebral artery with pearl and string sign (Fig. 2), compatible with left vertebral artery dissection. Endovascular occlusion of the artery was performed. An antiplatelet drug was administered and there was no ischemic recurrence over the following year.