the first stroke; antiplatelet therapy following stroke plus low molecular weight heparin administered after surgery may have led to the fatal outcome. The dissection may have gone undetected by transthoracic echocardiography since the study did not focus on the aortic root. The aortic root and arch should therefore be studied in cases of stroke of unknown cause; this will help to rule out not only aortic dissection but also ateromatosis.

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Bilateral papilloedema as the initial manifestation of spinal ependymoma☆,☆☆

Papiledema bilateral como forma de debut de un ependimoma espinal

Dear Editor:

Most cases of secondary intracranial hypertension are due to intracranial processes.[1] Spinal tumours are a well-known yet infrequent cause of intracranial hypertension, and are usually associated with hydrocephalus.[2] These tumours rarely manifest with intracranial hypertension and without hydrocephalus. We present the case of a young man who developed intracranial hypertension without hydrocephalus and was subsequently diagnosed with a spinal tumour. Our case demonstrates the need to maintain a high level of suspicion in atypical cases of intracranial hypertension combined with normal neuroimaging results, in order to detect other, less frequent causes of intracranial hypertension.

The patient was a 19-year-old man with no relevant medical history. A year prior to admission, he began to experience lower back pain radiating to the lower limbs, and was initially diagnosed with bilateral sacroiliitis. Pain worsened progressively despite anti-inflammatory treatment. A month prior to admission, the patient began to experience oppressive holocranial headache, which worsened with Valsalva manoeuvres and was associated with blurred vision and horizontal diplopia. He also reported tinnitus and transient bilateral vision loss.

The neurological examination revealed bilateral papilloedema (Fig. 1), decreased visual acuity bilaterally, and paresis of both lateral rectus muscles. The pain had caused decreased muscle strength in the lower limbs and was more intense during the straight leg raise test. Stretch reflexes were absent in the lower limbs and normal in the upper limbs; sensitivity was preserved.

The results of a cranial CT scan were normal. An MRI scan of the dorsal and lumbar region showed a space-occupying mass in the conus medullaris, which was hypointense in T1 and highly heterogeneous in T2. The mass displayed contrast uptake and was partially intramedullary and partially intradural extramedullary (Fig. 2). These findings are compatible with ependymoma.

The tumour was resected; a biopsy confirmed the diagnosis. The patient remains asymptomatic after one year of follow-up.

Differential diagnosis of intracranial hypertension usually focuses on ruling out intracranial processes. The diagnostic criteria for idiopathic intracranial hypertension include absence of ventriculomegaly, masses, or intracranial structural or vascular lesions, but do not specifically mention extracranial processes. Though infrequent, intracranial

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☆☆ This study was presented in poster format at the 64th Annual Meeting of the SEN.

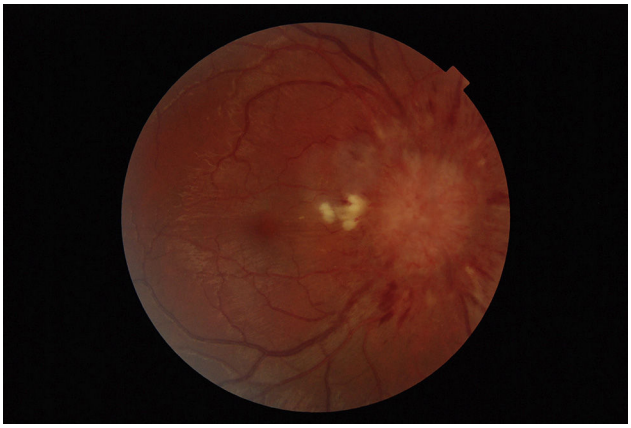

Figure 1 Papilloedema in the right eye.

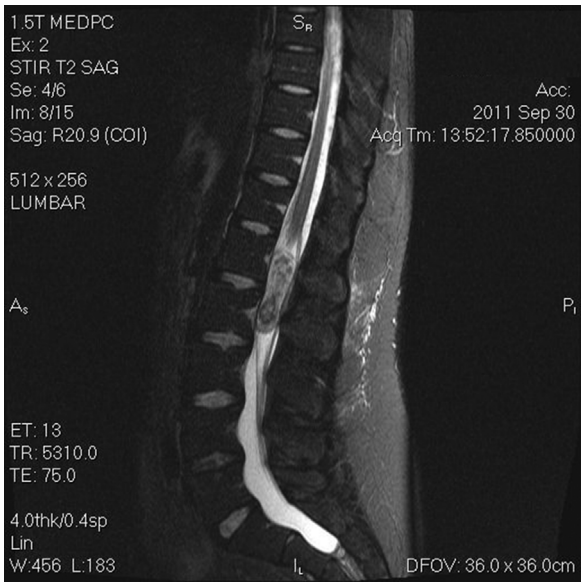

Figure 2 T2-weighted sequence showing the ependymoma.

hypertension secondary to spinal disorders is well documented in the literature. It is widely accepted that secondary intracranial hypertension can be caused by alterations in cerebrospinal fluid dynamics, which leads to secondary hydrocephalus.[2] Furthermore, spinal tumours may reduce the elasticity of the central canal of the spinal cord, increasing intracranial pressure.[3] Although several hypotheses have been proposed, the pathogenic mechanisms underlying intracranial hypertension in patients with spinal tumours are yet to be determined.[4] Intracranial hypertension without hydrocephalus, as in our case, is even less frequent. In these cases, the extracranial process may go undetected. Therefore, a high level of suspicion and a thorough evaluation are necessary to detect other less frequent causes of intracranial hypertension in these patients.

It is difficult to determine which patients should undergo a complete diagnostic study to rule out less frequent secondary causes of intracranial hypertension. Idiopathic intracranial hypertension mainly affects young women with obesity.[5] In patients not meeting this profile, the condition may have different features and be associated with a higher incidence of the less frequent causes of intracranial hypertension.[6] Based on the above, a possible approach would be to differentiate patients by phenotype. In patients with the classic profile (young women of childbearing age), additional emphasis should be placed on detecting less frequent causes of secondary intracranial hypertension. The condition is diagnosed when the patient shows no focal neurological signs other than those associated with intracranial hypertension. As a result, atypical neurological findings suggest presence of other less frequent causes of intracranial hypertension.

In the case of spinal tumours, we should pay attention to presence of lower back pain, lower limb weakness, sphincter dysfunction, or abnormal myotatic reflexes. Our patient, who experienced intracranial hypertension associated with normal neuroimaging findings, was a young man with a normal body mass index. He did not meet the classic patient profile. Furthermore, he had a one-year history of lower back pain and displayed abolished myotatic reflexes in the lower limbs. In the light of the above, we decided to study the spinal region, detecting an ependymoma.

Intracranial hypertension has been associated with a wide range of tumours,[2,7] including ependymomas,[8] primitive neuroectodermal tumours,[9,10] astrocytomas,[11,12] neurinomas,[13,14] and paragangliomas.[15]

In this case, the anatomical pathology study confirmed that the mass was an ependymoma. Tumour resection led to complete symptom resolution. It has been suggested that tumour resection restores central canal elasticity, leading to rapid resolution of intracranial hypertension.[3]

Conflicts of interest

The authors have no conflicts of interest to declare.

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An exceptional cause of sudden neurological deterioration and coma☆

Una causa excepcional de deterioro neurológico repentino y coma

Dear Editor:

We are writing about an unusual case, to our knowledge not previously reported, of sudden neurological deterioration and coma.

The case is that of a 25-year old woman who had given birth 3 months before after a managed full-term pregnancy without complications. She was treated with oral iron due to postpartum anaemia but had no other relevant medical history. This patient arrived at the ER presenting with a one-month history of headaches, apathy, and bradypsychia. The headaches had worsened in the last 48 hours with incomplete response to conventional analgesic treatment. Nausea and vomiting had appeared in the last few hours. She had no fever. While in the ER she had a sudden deterioration in level of consciousness together with a tonic—clonic seizure. She was intubated. An emergent CT scan showed signs of diffuse cerebral oedema and obliterated subarachnoid cisterns. Leptomeningeal and ependymal enhancement was present (Fig. 1A and B). These findings were suggestive of cerebritis accompanied by meningeal involvement and ventriculitis.

She was admitted to the ICU and 6 hours later suddenly developed non-reactive bilateral mydriasis. A decompressive wide bifrontal craniectomy with bilateral decompression of the frontal and temporal lobes was carried out as a compassionate treatment, and a biopsy of the right frontal lobe was performed. During the intervention, we observed the brain to be congested and of a hard consistency. After the procedure an intracranial pressure sensor was placed which initially recorded pressures below 15 mmHg. After intervention mydriasis was reversed and pupillary reflexes were restored. CSF analysis showed no cytochemical abnormalities and the results from the CSF culture were negative for bacteria, viruses, and fungi. The results of blood serology tests for autoimmune diseases were also negative. A brain MRI showed supra and infratentorial diffuse involvement, especially in the frontal lobes and corpus callosum. Small areas of contrast enhancement and signs of intracranial hypertension were also observed (Fig. 1C—F).

Clinical and radiological differential diagnosis included infectious entities such as progressive multifocal leukoencephalopathy and other forms of viral encephalitis; autoimmune diseases such as acute disseminated encephalomyelitis, vasculitis or connective tissue diseases; metabolic

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