treatment. In addition, the symptomatic efficacy of amantadine in other types of chorea[16] is also well known.

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Primary sciatic nerve lymphoma

Linfoma primario del nervio ciático

Dear Editor:

Neurolymphomatosis is a well-known complication of lymphoma that involves the infiltration of cranial nerves, spinal roots or peripheral nerves by lymphoma cells from a systemic or central nervous system (CNS) lymphoma[1]. However, a lymphoma arises in the peripheral nervous system (PNS) only infrequently. In a recent paper, Descamps et al.[2] described a patient with a primary lymphoma of the sciatic nerve and reviewed the literature. They found references of only eight patients with sciatic nerve lymphoma[3-9] and another five who had primary condition of other peripheral nerves. Since the publication of that article, we have found only one case of primary lymphoma of the sciatic nerve[10]. To these ten cases, we add a new patient who presented a mononeuropathy of the common sciatic nerve and was diagnosed with diffuse B cell lymphoma of the sciatic nerve.

This was an 89-year-old man with a history of arterial hypertension, hyperlipidemia, diabetes mellitus and a smoking habit. In March 2005, he began to feel pain in his left leg and a tingling sensation in his left toes. Subsequently, he noticed increasing difficulty in mobilising his left foot. He underwent an ultrasound of the calves and the popliteal bursa, which was normal, at another centre. Three months later, he was treated at our Neurology Clinic; the exploration showed distal paresis of the left lower extremity affecting foot dorsiflexion, 0/5; plantar flexion, 2/5; foot eversion, 0/5; foot inversion, 0/5, and extension of the first toe, 1/5. Knee flexion was preserved; ankle reflexes were abolished. Algesic and tactile sensitivities were decreased in the lateral side of the left leg and the dorsum of the homolateral foot. The left plantar cutaneous reflex was indifferent. A paresis of the left common sciatic nerve was suspected.

A blood test showed the following values: glucose, 171 mg/dl; variant surface glygoprotein (VSG), 86 mm first hour; lactate dehydrogenase (LDH), 282 IU/l. A neurophysiological study showed a disease of the internal and external popliteal nerves and signs of denervation in the muscles depending on the common sciatic nerve, with the exception of the long and short heads of the biceps femoris. A pelvic magnetic resonance imaging (MRI) was normal; an MRI of the left thigh showed a fusiform tumour of 16 x 3 cm, located in the posterior side of the thigh, depending on the sciatic nerve, hyperintense on T2 and short TI inversion recovery (STIR) sequences and that captured contrast homogeneously after gadolinium administration (Fig. 1).

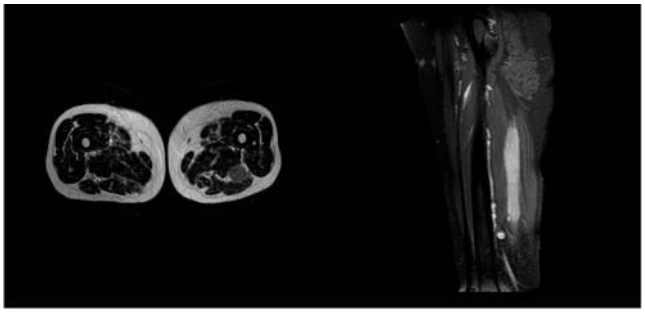

Figure 1 Magnetic resonance image of the left thigh. Axial plane T2 sequence showing a rounded tumour with slight hyperintensity with respect to muscle, with well-delimited edges, dependent on the common sciatic nerve (left). T1 sequence with fat suppression (SPIR) in sagittal plane after gadolinium administration showing an intense homogeneous enhancement of the lesion, with a fusiform morphology and approximate dimensions of 16 × 3 cm in craniocaudal and transverse direction, respectively (right).

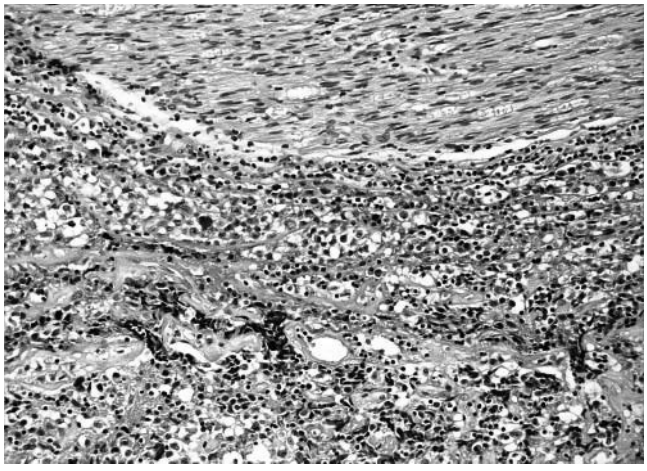

Figure 2 Diffuse lymphoproliferative process comprised by large cells that infiltrate the peripheral nerve (top part of the image). H-E stain, original × 400.

An intraoperative tumour biopsy showed a lymphoproliferative process. The definitive diagnosis was diffuse large B cell lymphoma (Fig. 2). Cervical, thoracic and abdominal computed tomography (CT) scans were normal. A positron emission tomography with 18-fluoro-2-deoxy-D-glucose (FDG-PET) showed a hypermetabolic mass in the upper third of the left thigh and another in the region of the external iliac artery. Cytology of bone marrow cells showed lymphocytes with normal appearance. A flow cytometry of peripheral blood showed lymphopenia with no clonality or lymphoma populations.

Treatment was initiated in January 2006 with cyclophosphamide 50 mg/day for 5 days and deflazacort 30 mg/day, with good clinical response. Radiotherapy was applied on the deposits described in the PET in the left thigh and left external iliac region between 20th February and 18th March 2006 (40 Gy in 20 sessions). The patient died in June 2006 due to a sepsis of renal origin. An abdominal ultrasound showed retroperitoneal lymphadenopathies. No autopsy was performed.

Although primary lymphoma of the peripheral nerve is rare, it seems to have a special predilection for the sciatic nerve. It has been postulated that B cells may be located or originate in the sciatic nerve or that this affinity is related to the expression of specific molecules for cell adhesion in the nerve that have not yet been identified[2]. In some patients with secondary neurolymphomatosis, there lymphoma cells also seem to have an affinity for the PNS[11].

We have described a new patient with lymphoma originating in the peripheral nerve. The PET-FDG images of extraneural hypermetabolism may correspond to regional lymph node condition. This test was carried out 9 months after the start of the clinical picture, so it does not rule the neural origin of the lymphoma. In fact, a pelvic MRI had been normal 6 months earlier.

Among the 10 cases previously described with primary lymphoma of the sciatic nerve, most were treated by chemotherapy with or without radiotherapy. In 3 patients, there was no evidence of disease after a follow up of 12[3], 30[4] and 48[2] months. The 48-month follow-up is the patient described by Descamps et al. who was treated with cyclophosphamide, doxorubicin, vincristine and prednisolone, along with rituximab. The patient described by Strobel et al.[10] presented a complete response assessed by PET/CT after receiving 6 cycles of chemotherapy, but the article does not specify the drugs used or the subsequent follow-up. Another patient was still alive after a follow-up of 57 months with residual local, stable disease[5]. Our patient presented a favourable initial clinical response but unfortunately the follow-up was short and there were no radiological controls of the injury, nor was permission obtained for necropsy to confirm the probable spread of the disease. The treatment of choice has not been established but, given the generally poor prognosis, chemotherapy at high doses would be indicated. Some authors also propose CNS prophylaxis and even amputation of the affected limb[8].

In conclusion, we can say that primary PNS lymphoma is a rare cause of mononeuropathy with a special predilection for the common sciatic nerve. In cases of patients with crural deficit of peripheral origin, pain and a lesion located outside the usual compression sites, as well as unfavourable evolution, imaging tests (preferably MRI) are obligatory. Atypical radiological signs, such as large size and homogeneous contrast uptake are not frequent in tumours of neural origin and force us to include primary PNS lymphoma in the differential diagnosis, especially if the sciatic nerve is affected.

Presentation

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Cranial nerve VI palsy and spontaneous intracranial hypotension

Parálisis del VI par craneal e hipotensión intracraneal espontánea

Dear Editor:

We report the case of a 40-year-old woman, with no personal history of interest, with a clinical picture of a 15-day frontal and occipital headache of oppressive evolution. It worsened with sitting and improved with analgesia and in supine position. Subsequently, she began to suffer from photophobia and diplopia. There was no history of trauma.

The general examination was unremarkable and vital signs were normal. The neurological examination highlighted a palsy of the left sixth nerve with horizontal diplopia to the left, which disappeared when looking to the right; there were no other abnormalities.

Diagnostic tests showed no abnormalities in chest x-ray, electrocardiogram, cranial computed tomography (CT), general analysis, microbiological culture, serology (syphilis, *Borrelia*, HIV and *Brucella*), angiotensin converting enzyme (ACE), adenosine deaminase (ADA), and cerebrospinal fluid (CSF) anatomopathology.

Cranial magnetic resonance imaging (MRI) with paramagnetic contrast cranial (Fig. 1) showed a thickening and intense leptomeningeal enhancement in the absence of subdural collections or tonsillar descent; the rest of the examination was normal. The lumbar puncture (LP) showed an opening pressure of 3 cm H_2O, clear CSF, glucose at 83 mg/dl and protein at 158.1 mg/dL; 28 erythrocytes/μl and 8 leucocytes/μl. The isotopic cisternoscintigraphy (Fig. 2) performed showed an image indicative of CSF leakage in the paraspinal region at the level of the cervicothoracic junction. The study was completed with a cervicothoracic MRI (Fig. 3), which showed an image of subdural CSF collection from C6 to D3, compatible with CSF fistula at the cervical level. The correction of physiological cervical lordosis and posterior osteophytosis with accompanying disk in the C5-C6 space indenting the anterior epidural space could also be appreciated.

Conservative measures were initiated due to the liquoral hypotension (abundant hydration, relative rest, analgesia and corticosteroids). When these proved ineffective, an autologous epidural blood patch at the lumbar level (20 ml) was administered; a progressive improvement in headache (70-80% improvement) and diplopia (30% improvement) took place after one week. Within a month, the headache had disappeared and a lower limitation of the abduction of the sixth cranial nerve was observed, with diplopia only in extreme lateral gaze to the left, which disappeared in subsequent months. After 4 months, spinal MRI showed no CSF collection, confirming the absence of CSF leakage, as could be expected from the clinical evolution.

Intracranial hypotension syndrome (IHS) is secondary to a depletion of CSF volume. CSF leakage is postulated as one of the major aetiopathological causes, producing a rostrocaudal descent of the brain, pulling on pain-sensitive structures, cervical roots and the oculomotor nerves. The causes are often iatrogenic (lumbar puncture, spinal anaesthesia, myelography, massive CSF drainages, cranial or spinal surgery) or secondary to trauma. It may also be observed spontaneously, from trauma or banal efforts that