Neurología. 2018;33(9):614—628

SOCIEDAD ESPAÑOLA DE NEUROLOGIA

NEUROLOGÍA

www.elsevier.es/neurologia

LETTERS TO THE EDITOR

Primary central nervous system lymphoma mimicking cerebellopontine angle tumour☆

Linfoma primario del sistema nervioso central aparentado lesión del ángulo pontocerebeloso

Dear Editor:

Introduction

Primary central nervous system (CNS) lymphomas constitute a rare form of extranodal non-Hodgkin lymphomas involving the central nervous system exclusively. Primary CNS lymphomas may appear both in immunosuppressed and in immunocompetent individuals, and account for less than 2% of all intracranial tumours[1,2]; they are infratentorial in only 10%-20% of cases.[3]

Clinical case

Our patient was a 60-year-old man, with no relevant medical history except for left-sided hearing loss, developing over the course of several months. The patient came to our hospital due to a 4-week history of subacute left-sided facial paraesthesia and hypoaesthesia, mild ataxic gait with a tendency to sway to the right, and worsening of hearing loss. The patient had not lost weight, and a complete blood count did not indicate anaemia or any other relevant alteration. A brain MRI scan (Figs. 1—3) revealed an expansive lesion in the left cerebellopontine angle (CPA), measuring 20 × 20 × 15 mm (CC × AP × T); the lesion was nodular, extra-axial, hypointense on T1-weighted sequences, and moderately hyperintense on T2-weighted and FLAIR sequences. We observed perilesional oedema extending across the cerebellar peduncle to the left cerebellar hemisphere, left side of the pons and midbrain, and left cerebral peduncle. The whole area displayed intense, homogeneous gadolinium uptake. We considered several diagnostic possibilities, including vestibular schwannoma, meningioma, and metastasis, and the less likely diagnoses of vascular malformation, abscess, or chronic granulomatous disease. Further testing, including tumour markers and a chest and abdomen CT scan, showed no signs of malignancy or alterations in the lymph nodes; treatment was started with moderate doses of oral corticosteroids (0.5 mg/kg/day). Despite initial improvement, symptoms reappeared and worsened after corticosteroid dosage was reduced. The patient was readmitted for evaluation of his eligibility for surgery. In addition to the symptoms mentioned above, our patient displayed left-sided peripheral facial paralysis (House—Brackmann grade III), persistent horizontal nystagmus, left-limb dysmetria, moderate dysarthria, global hyperreflexia, and marked ataxic gait. A complete blood count revealed mild macrocytic anaemia. Serology tests for HBV, HCV, and HIV yielded negative results. An MRI scan performed 3 months after the first scan showed that the lesion had increased in size (27 × 18 × 26 mm), displayed similar characteristics, and exerted a more pronounced mass effect on the medulla oblongata and pons. The patient underwent surgery, given his clinical and radiological progression. The lesion was

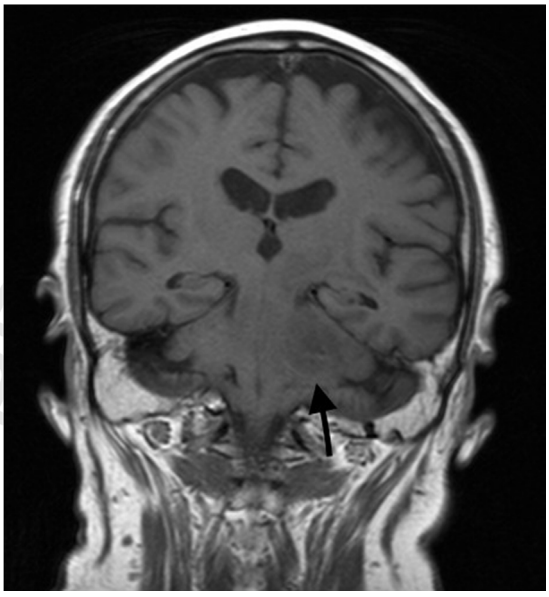

Figure 1 Brain MRI, coronal T1-weighted sequence showing that the lesion originated in the pons.

☆ Please cite this article as: Berrocal-Izquierdo N, Muñoz F, Bosch J, Molet J. Linfoma primario del sistema nervioso central aparentado lesión del ángulo pontocerebeloso. Neurología. 2018;33:614—616.

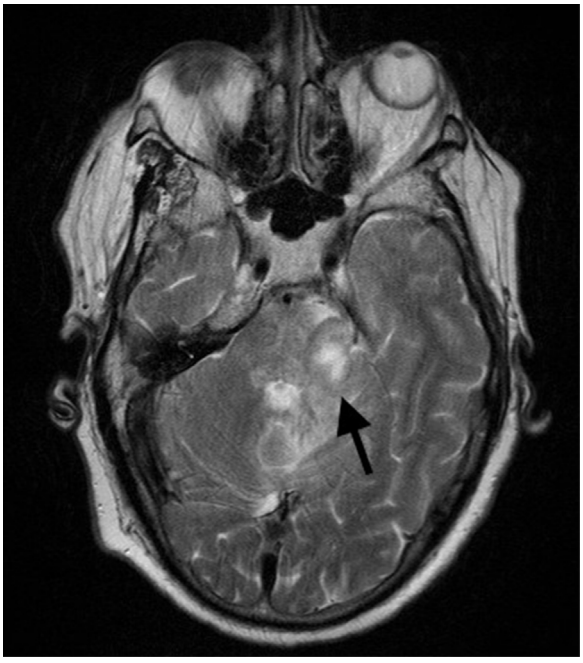

Figure 2 Brain MRI, axial T2-weighted sequence displaying the extension of the lesion across the cerebellum and peduncle.

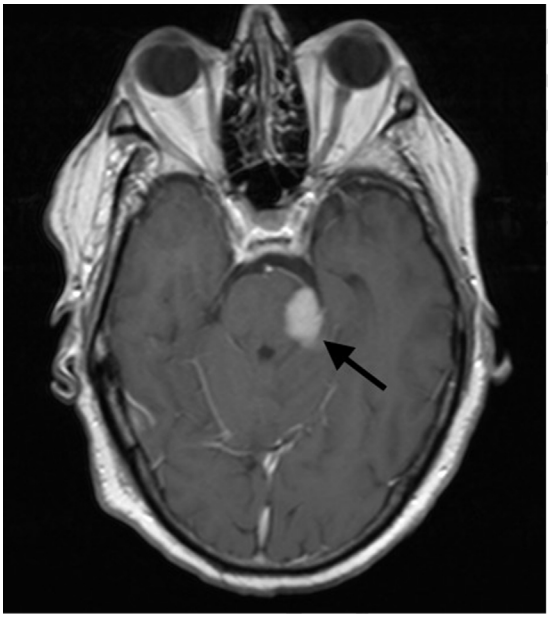

Figure 3 Brain MRI, gadolinium-enhanced axial T1-weighted sequence.

found to have originated in the pons. Anatomical pathology revealed proliferation of large, irregular lymphoid cells following a diffuse pattern, predominantly surrounding blood vessels but without infiltration; areas of necrosis; and a proliferation index >90%. The immunohistochemical study yielded positive results for CD10 and BCL6 and negative results for ALK; these findings are compatible with primary diffuse large B-cell lymphoma of the CNS. To date, 6 months after symptom onset, the patient has received 3 cycles of chemotherapy (carmustine, methotrexate, cytarabine, and rituximab), experiencing several adverse reactions; gait instability persists.

Discussion

Differential diagnosis of masses in the CPA should include vestibular schwannoma (70%-80%), meningioma (5%-10%), and epidermoid cyst (5%-7%). Primary CNS lymphomas presenting as a mass in the CPA are extremely rare. MRI is the technique of choice for studying these masses. It reveals intense, homogeneous contrast uptake, as in the case presented here. Marked response to corticosteroids is also characteristic of these tumours. To our knowledge, the literature includes only 16 cases of primary CNS lymphomas appearing as masses in the CPA.[4–10]

Funding

The study has not received any public or private funding.

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2173-5808/

Insulin neuritis or treatment-induced diabetic neuropathy☆

Neuropatía inducida por el tratamiento de la diabetes o neuritis insulínica

Dear Editor:

Treatment-induced diabetic neuropathy (TIDN), previously known as insulin neuritis, is a type of acute neuritis affecting diabetic patients after abrupt re-establishment of metabolic function. The condition was first described in 1933 by Caravati,[1] in a diabetic patient who developed symptoms of neuritis following rapid glycaemic control with insulin; symptoms resolved with insulin withdrawal. Few cases have been described to date; TIDN may be underdetected and is often misdiagnosed for other more frequent neuropathies in these patients. We present the case of a patient with diabetes who was admitted with acute neuropathy; symptoms were not initially identified as TIDN but were subsequently controlled.

The patient was a 25-year-old man with a family history of type 1 diabetes mellitus (grandfather and maternal uncle), who developed diabetic ketoacidosis in 2011. He was treated with insulin and informed about the disease and how to manage it. Follow-up was irregular over the first 2 years, as the patient did not attend follow-up appointments. He was admitted to the endocrinology department in 2014; he had discontinued insulin therapy for 3 months, displaying a glucose level >500 mg/dL, glycosuria, anorexia, postprandial fullness, and weight loss (BMI 15.5, indicating severe malnutrition). He was oriented and displayed no alterations in level of consciousness. The patient had ketonuria and an HbA1c level of 15.2%. Following rigorous metabolic correction, he was referred to the outpatient endocrinology department. Six weeks later, the patient began to experience pain in the knees and in the scapular and lumbar paraspinal muscles. Over a period of 7-15 days, the pain became burning, and stabbing in the proximal area of the lower limbs (''like having the flesh torn from my bones,'' as described by the patient); the pain was associated with dysaesthesias in the soles of the feet. The patient was readmitted due to intense pain (VAS: 8-9). The patient was extremely underweight and in a poor mood, but his level of consciousness, cortical function, and cranial nerve function were normal. Strength was preserved; all reflexes were hypoactive. Regarding superficial sensation, the patient showed hyperaesthesia and allodynia in the paraspinal muscles and in all 4 limbs. Deep sensation, cerebellar function, and gait were normal. The HbA1c level was 9%. High doses of NSAIDs, tramadol, and duloxetine achieved partial relief. The endocrinology department initially diagnosed radiating lower back pain, bone and muscle pain, and reactive depression. A lumbar MRI scan revealed diffuse degenerative changes and disc bulging affecting the entire lumbar segment. During admission, he displayed sustained tachycardia (101-117 bpm) and diastolic hypertension (86-92 mmHg), with normal systolic blood pressure (105-120 mmHg). EMG indicated demyelinating sensorimotor neuropathy, which did not meet the neurophysiological criteria for chronic inflammatory demyelinating polyneuropathy, and no signs of denervation activity (Table 1). A CSF analysis revealed high protein levels (89 mg/dL). A complete blood count ruled out systemic, liver, kidney, or thyroid diseases, and vitamin deficiencies (vitamins A, E, D, B_1, B_6, B_9, and B_{12}). The autoimmunity and serology tests for *Borrelia*, *Mycoplasma*, CMV, VZV, EBV, HSV, HIV, HBV, and HCV yielded negative results. The levels of thyroid hormones, ACTH, cortisol, iPTH, selenium, zinc, and copper were also within normal ranges. The urine albumin/creatinine ratio was normal. The patient was diagnosed with multifocal demyelinating polyneuropathy associated with poor diabetes control. A follow-up EMG was scheduled at 7 months; the patient refused. Pain progressively improved and the patient was discharged; 6 months later, he was nearly asymptomatic. During that period, the patient gained little weight (3-4 kg) and blood glucose levels were partially controlled. As TIDN was not suspected, autonomic function was not assessed; autonomic symptoms were overshadowed by the severe, disabling pain that led to the consultation with the neurology department.

☆ Please cite this article as: Hernández RC, Galindo AS, Acebes EM. Neuropatía inducida por el tratamiento de la diabetes o neuritis insulínica. Neurología. 2018;33:616—618.