increases the activity of the sympathetic and parasympathetic autonomous nervous system in an attempt to increase the stroke volume and the BP to a level exceeding the pressure on the brainstem. The purpose of this process is to overcome the vascular resistance to cerebral blood flow caused by increased ICP.[11–15] This physiological response to elevated ICP is called the Cushing reflex and it is described clinically by the triad of arterial hypertension, bradycardia, and irregular breathing, indicators of poor clinical prognosis. In the case we describe, doctors detected bradycardia, but no arterial hypertension or irregular breathing.

Headaches that last more than one week after lumbar administration of epidural anaesthesia, stop responding to postural change, or appear with focal neurological signs should alert us to the possibility of an acute intracranial process. Symptoms of such processes no longer reflect CSF hypotension — the typical feature of PDPH — but rather intracranial hypertension, mass effect, and displacement of intracranial structures caused by SH.

Reference

1. Reina M, López A, Benito-León J, Pulido P, María F. Hematoma subdural intracraneal y espinal, una complicación poco común de la anestesia epidural y subaracniodea. Rev Esp Anestesiol Reanim. 2004;51:28–39.
2. Guenot M. Chronic subdural hematoma: historical studies. Neurochirurgie. 2001;47:461–3.
3. Trotter W. Chronic subdural haemorrhage of traumatic origin, and its relation to pachymeningitis hemorrhagica interna. Br J Surg. 1914;2:271–91.
4. Machurot PY, Vergnion M, Fraipont V, Bonhomme V, Damas F. Intracranial subdural hematoma following spinal anesthesia: case report and review literature. Acta Anaesthesiol Belg. 2010;61:63–6.
5. Amorin JA, Canuto dos Anjos Remígio DS, Damázio Filho O, Guerra de Barros MA, Nicole Carvalho V, Moraes Valença M, et al. Hematoma subdural intracraneal postanestesia subaracnoidea: relato de dos casos y revisión de 33 casos de la literatura. Rev Bras Anestesiol. 2010;60:344–9.
6. International Headache Society. International Classification of Headache Disorders (ICHD-II). 2nd ed. Cephalalgia. 2004;24 Suppl. 1:1–160.
7. Peiró CM, Caballer N, Errando CL, Moliner S. Hematoma espinal y cerebral postpunción lumbar diagnóstica con evolución letal. Rev Esp Anestesiol Reanim. 2003;50:481–5.
8. Acharya R, Chhabra SS, Ratra M, Sehgal AD. Cranial subdural haematoma after espinal anaesthesia. Br J Anaesth. 2001;86:893–5.
9. Reynolds F. Dural puncture and headache: avoid the first but treat the second. Br Med J. 1993;306:874–6.
10. Van de Kelft E, Bosmans J, Parizel PM, Van Vyve M, Selosse P. Intracerebral hemorrhage after lumbar myelography with iohexol: report of a case and review of the literature. Neurosurgery. 1991;28:570–4.
11. Gamal Hamdan Suleiman MD. Trauma craneoencefálico severo. Medicrit. 2005;2:107–48.
12. Jeremitsky E, Omert L, Dunham CM, Protetch J, Rodríguez A. Harbingers of poor outcome the day after severe brain injury: hypothermia, hypoxia and hypoperfusion. J Trauma. 2003;54:312–9.
13. Marion DW, Obrist WD, Carlier PM, Penrod LE, Darby JM. The use of moderate hypothermia for patients with severe head injury: a preliminary report. Neurosurgery. 1993;79:354–62.
14. Busto R, Dietrich WD, Globus MY, Valdés I, Scheinberg P, Ginsberg MD. Small differences in intraischemic brain temperature critically determine the extent of ischemic neuronal injury. J Cereb Blod Flow Metab. 1987;7:729–38.
15. Gilo Arrojo F, Herrera Muñoz A, Anciones B. Hipertensión intracraneal aguda. Neurología. 2010;25 Suppl. 1:3–10.

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Opalski syndrome: A variant of lateral-medullary syndrome☆

Síndrome de Opalski: una variante del síndrome medular lateral

Dear Editor:

Lateral-medullary syndrome is often found in patients with vertebrobasilar vascular lesions. On rare occasions, the syndrome is associated with ipsilateral hemiparesis; this is known as Opalski syndrome.[1]

We present the case of a male smoker aged 67 with a history of arterial hypertension, peripheral artery disease, and trigeminal neuralgia. He was undergoing treatment with enalapril, carbamazepine, and baclofen. He was examined following a 12-hour episode of dizziness, nausea, vomiting, and difficulty walking. The initial assessment showed right-sided facial paralysis, mild paresis, dysmetria of the right upper limb, and ataxic gait with lateropulsion. Blood tests (including serum levels of carbamazepine) and cranial CT yielded normal results. The patient was admitted with a diagnosis of cerebral infarct in the vertebrobasilar region which was probably atherothrombotic in origin. Cardioembolic origin was ruled out, and the patient began treatment with antiplatelet drugs. The patient's clinical

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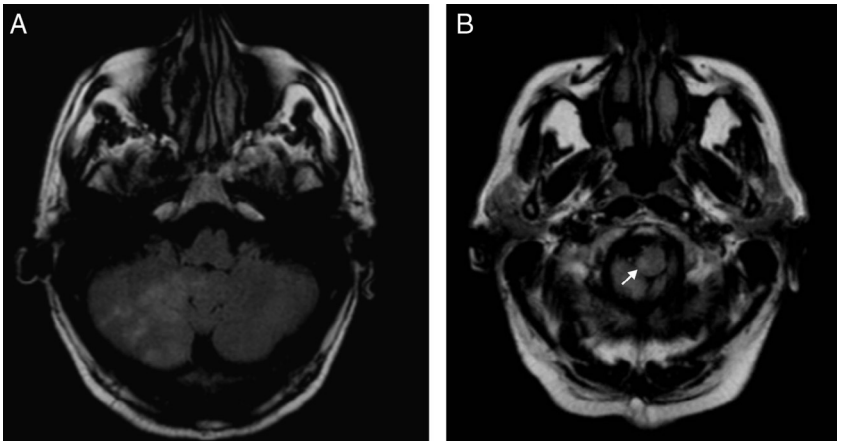

Figure 1 MRI: axial slices, FLAIR sequence. Ischaemic lesion in right cerebellar hemisphere (A), extending to the cervical region of the spinal cord (B).

condition worsened in the first 24 h after admission. The examination revealed horizontal-rotary nystagmus, right-sided Horner syndrome, hemiparesis and dysmetria in right upper limbs, and hemihypaesthesia affecting the trunk and left extremities. Arthrokinetic reflex remained intact. Doctors performed a cranial CT that ruled out the presence of haemorrhagic lesions. The patient began anticoagulant treatment, which gradually improved his symptoms. Upon discharge, he still presented right-sided mild nuclear facial paralysis, slight paresis of the right upper limb, left hemihypaesthesia, and ataxic gait. Cranial MRI and MR angiography of the supra-aortic trunks were also performed. Cranial MRI showed acute ischaemic infarct in the bulbar region and right cerebellum, reaching the right cervical region of the spinal cord (Fig. 1A and B). MR angiography of the supra-aortic trunks revealed asymmetries in the diameter of the vertebral arteries. The left vertebral artery was thinner (asymptomatic), especially at its origin and the distal end. The right vertebral artery presented fewer irregularities.

Opalski syndrome is a rare variation of lateral medullary syndrome (Wallenberg syndrome) in which the lesion is associated with ipsilateral hemiparesis.[1] An infarct in this region may result from any of several causes, including differences in the diameter of the vertebral arteries,[2,3] dissection of the vertebral artery,[4] and atherosclerosis, which is the most frequent cause[1] and the suspected aetiology in our case.

Based on neuroradiology and autopsy findings, we determined that the lesion was located caudal to the pyramidal decussation.[1–3,5,6] Impairment of that structure may explain the ipsilateral paresis, but this causal mechanism is not universally accepted because it is not always accompanied by abnormal reflexes such as the Babinski sign. Some researchers therefore believe that infarct may result from hypotonia secondary to spinocerebellar tract injury, and not from a pyramidal lesion.[7] Doctors also disagree as to which artery causes this disorder because of anatomic variability,[4] but the vertebral arteries are the ones most frequently involved.[3]

We present a patient who suffered lateral spinal infarct with paresis ipsilateral to the lesion. This profile suggests Opalski syndrome, a rare presentation of a spinal infarct with a characteristic radiological profile that correlates to the clinical symptoms. Since Opalski syndrome was first described in 1946,[8] there have been 15 cases published in the English- and Spanish-language literature.[1–10] Nine include cranial MR imaging studies that show lateral medulla oblongata impairment; only 3 also present cerebellar impairment, as in the case of our patient.[1,3,6] MR angiography studies are available from 6 cases; vertebral artery involvement was the most frequent finding in these studies.[1–5,10]

Reference

1. Bailon O, Garcia PY, Logak M, Timsit S. Opalski syndrome detected on DWI MRI: a rare lateral medullary infarction. Case report and review. Rev Neurol (Paris). 2011;167:177—80.
2. Montaner J, Alvarez-Sabín J. Opalski's syndrome. J Neurol Neurosurg Psychiatry. 1999;67:688—9.
3. Kim HY, Koh SH, Lee KY, Lee YJ, Kim SH, Kim J, et al. Opalski's syndrome with cerebellar infarction. J Clin Neurol. 2006;2:276—8.
4. García-García J, Ayo-Martín O, Segura T. Lateral medullary syndrome and ipsilateral hemiplegia (Opalski syndrome) due to left vertebral artery dissection. Arch Neurol. 2009;66:1574—5.
5. Kimura Y, Hashimoto H, Tagaya M, Abe Y, Etani H. Ipsilateral hemiplegia in a lateral medullary infarct — Opalski's syndrome. J Neuroimaging. 2003;13:83—4.
6. Sanahuja J, Begue R, García-Cerecedo T. Infarto isquémico en territorio de la arteria cerebelosa posteroinferior con afectación medular (síndrome de Opalski). Neurologia. 2003;18:117—8.
7. Hermann DM, Jung HH, Bassetti CL. Lateral medullary infarct with alternating and dissociated sensorimotor deficits: Opalski's syndrome revisited. Eur J Neurol. 2009;16:72—4.
8. Opalski A. A new sub-bulbar syndrome: partial syndrome of the posterior vertebrospinal artery. Paris Medical. 1946:214—20.
9. Nakamura S, Kitami M, Furukawa Y. Opalski syndrome: ipsilateral hemiplegia due to a lateral-medullary infarction. Neurology. 2010;75:1658.

10. Cheng MY, Sung CY. Opalski syndrome. Acta Neurol Taiwan. 2005;14:162—3.

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Neurological focal signs as a first manifestation of cardiac myxoma☆

Focalidad neurológica como primera manifestación de un mixoma cardiaco

Dear Editor:

Cerebral infarcts are the third most common cause of death in Spain and the leading cause of physical disability in adults. Twenty-five per cent of all strokes occur in people younger than 65.[1,2] Cardiac tumours are a very rare disease with an incidence that ranges from 0.001% to 0.28% in post-mortem studies. Half of such tumours are of myxomatous origin. They are an infrequent cause of cerebral embolism and account for less than 1% of all strokes.[3] Although they are rare, they must be considered in the differential diagnosis of all young patients with cerebral ischaemia. We present the case of a patient who experienced acute neurological deficit and prior episodes of transient cerebral ischaemia.

Female patient aged 33 with a personal history of depressive syndrome and chronic anaemia that remained uncorrected after treatment with ionic iron. The patient came to the hospital due to a 2-hour transient episode of diplopia. She reported having had a similar episode previously. Upon arrival at the hospital, the patient presented no neurological symptoms. However, the examination revealed significant purpuric lesions on the thighs and crura, and on the distal parts of the upper limbs. During her stay in the emergency department, doctors performed laboratory tests, an ECG, and a cranial CT, none of which revealed pathological findings. Once the patient was admitted, doctors began the procedure described below for diagnosing stroke in young patients. (a) Haematological analysis, which revealed chronic anaemia disease with an increase in acute-phase reactants; immunology study (ANAs, ANCAs, anti-DNA, anticardiolipin and antiphospholipid, anti-Ro, anti-La, rheumatoid factor, and anti-ACh receptor antibodies) and hypercoagulation study (all results negative); (b) lumbar puncture showing absence of oligoclonal bands and negative Link and Tibbling index; (c) brain MRI showing old ischaemic lesions in cerebellar hemispheres; (d) vascular cerebral study by means of CT angiogram and neurosonology that revealed no significant haemodynamic disorders; (e) transthoracic echocardiogram (TTE) with a subsequent transoesophageal approach (TEE) showing a mass of 11 cm^2 in the LA that suggested atrial myxoma (Fig. 1); (f) heart MRI confirming the presence of a mass with irregular contours in the LA; and (g) biopsy of the cutaneous lesions, which showed intravascular thrombi consisting of angiomyomatous tissue.

Despite prophylactic treatment with anticoagulants, the patient presented a new episode of cerebral ischaemia during the diagnostic process. The episode was characterised by right-sided paraesthesia that lasted 30 minutes before resolving completely. Doctors resected the mass which was of myxomatous origin according to the histological study. Subsequent check-ups performed by the outpatient cardiology unit indicate that the patient has had no clinical or echocardiographic signs of a relapse.

Atrial myxoma is a benign tumour which mainly affects women (2:1) aged between 30 and 60. Although these tumours mainly arise sporadically, 7% of all cases have a familial component. Up to 10% of the cases may be asymptomatic, while the rest may present symptoms through 3 basic mechanisms: (a) mitral valve obstruction with symptoms of dyspnoea, heart failure or sudden death; (b) constitutional symptoms such as fever, arthralgia, or weight loss which may be related to interleukin 6 synthesis by the tumour; and (c) embolic phenomena in peripheral organs.[4—6] Neurological symptoms are present in between 26% and 45% of the patients, with cerebral infarct being the most prevalent condition.[6—9] Researchers have described completely asymptomatic cases of ischaemic lesions of different sizes and location that were detected by brain MRI.[10] Symptoms resulting from tumour infiltration are less common; researchers have described intracranial aneurysms which are secondary to the infiltration of the vascular wall, as well as tumours secondary to the parenchymatous infiltration. Neurological follow-up is therefore advisable, since these complications can occur years after resection of the tumour.[7,11]

Diagnosis is based on the echocardiogram, which has a diagnostic sensitivity of 100% according to different series.[7] The transoesophageal approach is the technique of choice as it provides a better view of the atrium and enables detection of smaller tumours.[12] Cardiac MRI may be useful, considering that ultrasound sometimes presents false negatives, underdiagnoses the extension of the tumour, or offers limited information about its type.[7] Additional tests, such as a cutaneous biopsy revealing angiomyomatous tissue in the capillary lumen, may help doctors establish a diagnosis.[13]

In a case of suspected cardiac myxoma, surgical resection should be performed as soon as possible given the high risk of recurring embolic phenomena.[4,14,15] When surgery is scheduled, secondary prevention may be a matter of debate. The use of antiplatelet or anticoagulation therapy has been recommended due to the embolic nature of this disease, but there is no consensus regarding the use of these drugs,[7] and embolic relapses may occur despite treatment.[8]

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