citriol. 25-OH-D_3 levels were equal to or below 20 ng/mL in 32 patients (72.7%), and were only above the optimal value of 30 ng/mL in one patient (2.3%). However, 1,25$(OH)_2D_3$ levels were above the lower limit of normality in all patients, with 17 (38.6%) presenting levels above the upper limit. Based on these data and the well-understood physiology of vitamin D, we may conclude that 1,25$(OH)_2D_3$ levels are increased due to secondary hyperparathyroidism caused by decreased deposits of 25-OH-D_3; as a consequence, this deficit may have no physiological or clinical impact, as the final pathway through which calcitriol acts is unaffected.

One issue arising from this analysis is the question of whether epidemiological studies exclusively evaluating the body's vitamin D deposits truly support the notion of vitamin D deficiency as a predisposing factor for MS. Specifically, all in vitro studies into the relationship between vitamin D and immune system regulation involve the action of 1,25$(OH)_2D_3$,[7] and studies into 25-OH-D_3 supplementation have been unable to reliably demonstrate changes in this regulation[9] or a reduction in relapse rates.[6,10]

In conclusion, given the current understanding of the physiology of vitamin D, we recommend vitamin D supplementation to achieve 25-OH-D_3 levels greater than 30 ng/mL in order to minimise the deleterious effects of secondary hyperparathyroidism on bone metabolism (osteomalacia). However, well-designed clinical trials are needed to justify long-term treatment with megadoses of vitamin D aiming to modify the clinical course of MS.

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Conflicts of interest

The author has no conflicts of interest to declare.

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Half and half syndrome as a presentation of multiple sclerosis☆

Síndrome del medio y medio como presentación de esclerosis múltiple

Dear Editor:

Half and half syndrome is a rare oculomotor disorder characterised by unilateral internuclear ophthalmoplegia (INO) and ipsilateral sixth nerve palsy. Its name refers to the impairment of half of the contralateral gaze (secondary to INO, which limits adduction of the ipsilateral eye) and half of the ipsilateral gaze (secondary to a fascicular lesion to the abducens nerve, with no damage to the abducens nucleus, allowing normal contralateral eye adduction).[1] This is an extremely rare INO plus syndrome, which may hinder differential diagnosis of other acute or subacute oculomotor disorders.

We present the case of a 47-year-old man with no relevant medical history who visited our hospital due to a 6-day history of progressive sensory impairment, right faciobrachial hemiparesis, and horizontal diplopia. The neurological examination revealed horizontal diplopia when looking to the left, with the false image being generated by the left eye; this finding is compatible with left sixth cranial nerve palsy. We also observed limited adduction of the left eye when looking to the right (with preserved conver-

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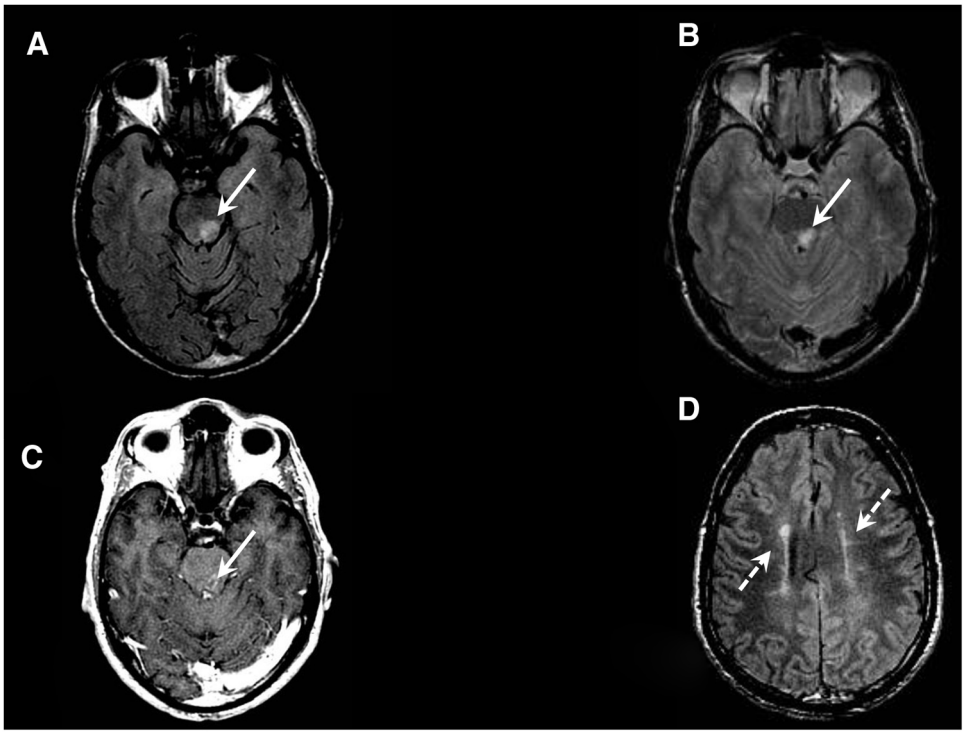

Figure 1 Brain MRI scan (axial plane). A and B) FSE T2-FLAIR and FSE T2 sequences at the level of the orbital apex showing a focal lesion to the left posterior parasagittal region of the pons (solid arrows). C) 3D FSPGR T1 sequence after intravenous administration of single-dose contrast, revealing open ring contrast enhancement around the lesion (solid arrow); this is a frequent finding in inflammatory/demyelinating lesions. D) FSE T2 FLAIR sequence showing more than 3 lesions to the supratentorial periventricular white matter, distributed along an axis perpendicular to the lateral ventricles; these findings are also frequent in patients with inflammatory/demyelinating lesions (dashed arrows).

gence) and persistent horizontal-rotary nystagmus in the right eye (dissociated nystagmus), with reduced saccade velocity in the left eye when looking to the right. These findings are compatible with left INO. All complementary tests (complete blood count, CT scan, CT angiography, serology testing) yielded normal results. CSF analysis revealed a slightly elevated protein level (0.61 g/L) and no oligoclonal bands. T2-weighted brain MRI sequences revealed several hyperintense lesions to the periventricular white matter, genu of the corpus callosum, and left posterior parasagittal region of the pons; the latter region also displayed contrast uptake (Fig. 1). These lesions are suggestive of inflammation/demyelination; a spinal cord MRI scan revealed a central-dorsal lesion measuring 12 mm in length at the C2 level. The patient received corticosteroid therapy at standard doses for 4 days, showing good progression. He was diagnosed with half and half syndrome as the initial manifestation of multiple sclerosis, according to the 2017 McDonald criteria.[2,3]

Vertical and horizontal gaze are controlled by the brainstem. The abducens nucleus is the final common pathway for conjugate horizontal eye movements; it is located in the dorsal pontine tegmentum and connects with the nucleus of the contralateral third cranial nerve via the medial longitudinal fasciculus (MLF). Lesions to the abducens nucleus can cause complete ipsilateral conjugate gaze palsy (ipsilateral abduction and contralateral adduction), whereas lesions to the medial longitudinal fasciculus only affect ipsilateral abduction. Convergence, controlled by the midbrain, is usually preserved.[4,5]

INO is the best known syndrome of the MLF,[6] and is characterised by impaired adduction of the eye ipsilateral to the lesion and dissociated nystagmus in the contralateral abducting eye. Nystagmus is thought to be the result of an adaptive response to weakness of the medial rectus muscle ipsilateral to the INO (Hering law). The affected eye presents reduced saccadic velocity, predominantly in adduction. Oculocephalic reflexes were absent, which rules out the possibility of horizontal gaze pseudopalsy.

Several INO plus syndromes have been described, including one-and-a-half syndrome (lesion to the paramedian pontine reticular formation or the abducens nerve, associated with INO), wall-eyed bilateral INO (bilateral damage to the MLF), eight-and-a-half syndrome (one-and-a-half syndrome associated with seventh cranial nerve palsy), and half and half syndrome, which is extremely infrequent. The most common aetiologies are inflammatory (multiple sclerosis) in young patients and vascular in older individuals.[7]

Half and half syndrome is a rare combination of neurological signs. To our knowledge, this is the second reported case of half and half syndrome of inflammatory origin and the third reported case of the syndrome with any aetiology. This may partially be explained by underdiagnosis. As reported by Frohman et al.,[6] clinicians may not detect mild or incomplete forms of the syndrome, manifesting as slowing of adduction saccades in the affected eye.

Neuro-ophthalmological examination should include systematic assessment of smooth pursuit movements, saccades, the vestibulo-ocular reflex, and vergence movements; this increases diagnostic sensitivity for milder cases.

In conclusion, half and half syndrome should be included within the spectrum of INO and INO plus syndromes, which are frequent in multiple sclerosis, often presenting as the initial symptom; a high level of suspicion is therefore essential for early diagnosis.

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Anterior medullary infarction after bronchial embolisation☆

Infarto medular anterior tras embolización bronquial

Dear Editor:

Spinal cord infarction is a rare entity that accounts for approximately 1.2% of all central nervous system infarctions and explains 5%-8% of cases of acute myelopathy.[1] It may be caused by multiple factors, although the most frequent aetiology is atherothrombosis, in the context of invasive vascular procedures or thoracic-abdominal surgeries, during which detached emboli may migrate distally.[2]

We present the case of a 54-year-old Chinese woman with history of occasional haemoptysis due to a pseudonodular lesion to the right upper lobe and bronchial artery hypertrophy, for which she underwent successful embolisation in 2011. In 2018, she presented another episode of haemoptysis with haemodynamic instability, and underwent emergency bronchial artery embolisation; the procedure was successful. The patient's general status improved after 2 days at the intensive care unit, but she presented difficulty moving the right leg; this was interpreted as possible postoperative right femoral nerve neurapraxia. The intensive care unit requested a consultation with the neurology department 2 days later due to lack of clinical improvement. The neurological examination revealed an adequate level of consciousness and no alterations in cranial nerve function. The patient presented hypotonia in the right leg: she was only able to move the limb on the horizontal plane and presented 2/5 muscle strength in the right psoas, quadriceps, and hamstrings muscles and 4—/5 in the right tibialis anterior and gastrocnemius muscles. Muscle tone and strength were normal in all other limbs. The patient also presented tactile and thermal hypoaesthesia and hypalgesia in the left side of the body at the T6 sensory level, without reduced vibration sensitivity or alterations in arthrokinetic sensitivity, as well as lower limb hyporeflexia and absent plantar reflex in the right foot. Suspecting acute, incomplete anterior cord syndrome, we performed a spinal cord MRI scan, which revealed T2 and STIR hyperintensities and diffusion restriction in the anterior column of the upper thoracic segments (T2-T7), compatible with recent ischaemic lesions (Figs. 1 and 2).

The patient started rehabilitation treatment during hospitalisation, presenting acute urinary retention, which required placement of a urinary catheter, and frequent episodes of orthostatic hypotension associated with profuse sweating; these symptoms are compatible with autonomic dysreflexia. At discharge, the patient was able to walk short distances independently and presented mild right leg paresis with 4/5 muscle strength and tactile hypoaesthesia and hypalgesia in the left side of the body, at the T6 sensory level. After 3 months of outpatient rehabilitation treatment, the patient is independent in the activities of daily living, but considerable autonomic dysfunction persists.

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