levels of such markers of inflammation as C-reactive protein or erythrocyte sedimentation rate.

Although the pathophysiology of the condition is not well understood, several trigger factors have been identified; these include recent trauma or a history of viral respiratory tract infection.[2–4]

Neuroimaging studies are essential, especially neck CT, which shows calcific deposits in the longus colli muscle, typically in the retropharyngeal space at the C1-C2 level, and prevertebral oedema of the soft tissues.[5]

It is crucial for neurologists and otorhinolaryngologists at emergency departments to be aware of this underdiagnosed entity. Differential diagnosis of longus colli tendinitis includes such severe neurological diseases as meningitis, spinal disc herniation, vertebral artery dissection, and spondylodiscitis, and other conditions that may be treated surgically, such as retropharyngeal abscess.[6]

The condition follows a benign clinical course, with treatment based on relative rest and the use of non-steroidal anti-inflammatory drugs, combined with opioids and corticosteroids in refractory cases. Symptoms usually resolve within 1-3 weeks.

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References

1. Hartley J. Acute cervical pain associated with retropharyngeal calcium deposit. A case report. J Bone Jt Surg Am. 1964;46:1753–4.
2. Jiménez S, Millán JM. Calcific retropharyngeal tendinitis: a frequently missed diagnosis. Case report. J Neurosurg Spine. 2007;6:77–80.
3. Coulier B, Macsim M, Desgain O. Retropharyngeal calcific tendinitis-*longus colli* tendinitis – an unusual cause of acute dysphagia. Emerg Radiol. 2011;18:449–51.
4. Roldan CJ, Carlson PJ. *Longus colli* tendonitis, clinical consequences of a misdiagnosis. Am J Emerg Med. 2013;31, 1538.e1-2.
5. Zibis AH, Giannis D, Malizos KN, Kitsioulis P, Arvanitis DL. Acute calcific tendinitis of the *longus colli* muscle: case report and review of the literature. Eur Spine J. 2013;22 Suppl. 3:S434–8.
6. Artenian DJ, Lipman JK, Scidmore GK, Brant-Zawadzki M. Acute neck pain due to tendonitis of the *longus colli:* CT and MRI findings. Neuroradiology. 1989;31:166–9.

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Posterior reversible encephalopathy syndrome: a case report☆,☆☆

Síndrome de encefalopatía posterior reversible: a propósito de un caso clínico

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity characterised by the acute or subacute onset of headache, altered level of consciousness, visual alterations, seizures, nausea, and vomiting; it also causes neuroimaging alterations, which are generalised, reversible, and predominantly posterior.[1–3] It usually manifests in the context of systemic diseases; in children, it has been identified in the context of kidney failure, immunosuppressant treatment and chemotherapy, such autoimmune diseases as systemic lupus erythematosus, and idiopathic arterial hypertension (AHT), among others.[1,4,5] Approximately 70%-80% of patients present moderate to severe AHT.[2,6,7] Brain magnetic resonance imaging (MRI) is essential for diagnosis as it identifies the presence of oedema surrounding the white matter bilaterally, mainly in the posterior area (parietal and occipital lobes).[2,3,8,9] The pathophysiology of PRES is unknown; several mechanisms have been suggested, and probably coexist in some cases: loss of autoregulatory vascular tone causing hyperperfusion, systemic vasoconstriction with hypoperfusion, and dysfunction or endothelial injury with lesion to the blood-brain barrier.[9,10] Symptoms fully resolve when the underlying cause is corrected early; otherwise, however, the condition may result in such irreversible damage as cortical blindness or death. MRI abnormalities disappear in follow-up examinations performed after the proper treatment is administered.[11–13]

Our patient is a 6-year-old girl with no relevant personal history, who attended the emergency department due to a 4-day history of headache, vomiting, abdominal

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☆☆ This study was presented in poster format at the 15th Portuguese National Congress of Paediatrics.

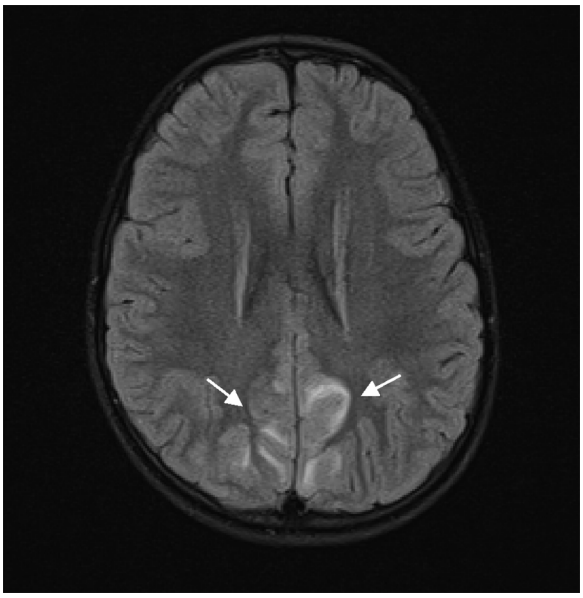

Figure 1 Axial T2-weighted FLAIR MRI sequence showing bilateral occipital and posterior parietal cortico-subcortical hyperintensities, which are nearly symmetrical (arrows).

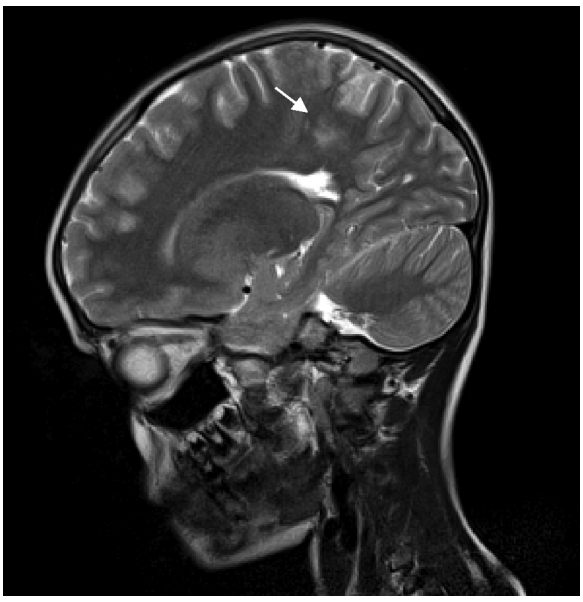

Figure 2 Sagittal T2-weighted brain MRI sequence showing temporo-parietal cortico-subcortical hyperintensities.

pain, and lack of mobility. During the examination, the patient was drowsy but easily awakened, with the neurological examination detecting no other alterations (eye fundus with no papilloedema). During the first hour after admission, she presented weak mastication and empty gaze, and blood pressure values were observed to be persistently above the 95th percentile. A head computed tomography scan revealed cortico-subcortical hypodensities in the left anterior parietal and parasagittal regions, and an electroencephalography showed a slow, poorly reactive, and ill-defined rhythm, suggesting cortico-subcortical involvement. A brain MRI scan showed multifocal, T2-hyperintense lesions symmetrically surrounding the white matter of the temporo-parietal and occipital regions and the left frontal lobe, suggesting PRES (Figs. 1 and 2). Other such diagnostic hypotheses as stroke, cerebral venous thrombosis, central nervous system vasculitis, and encephalitis were ruled out. For the aetiological diagnosis of AHT and its consequences, we requested the following studies: urine sediment analysis, kidney function test, serum electrolyte study, lipid profile, immunology study, 24-hour urine catecholamines, renal ultrasound, an echo-Doppler study of the renal arteries, and an echocardiogram; all tests yielded normal results without identifying any secondary cause of AHT.

Blood pressure was controlled on day 9 of hospitalisation using enalapril and hydrochlorothiazide plus amiloride; subsequent clinical progress was favourable (with no de novo symptoms). The imaging study performed 9 days after treatment onset revealed almost complete recovery of the lesions diagnosed by MRI. The patient was discharged on day 12 and referred to the neuropaediatric outpatient consultation. Antihypertensives were suspended after 8 weeks of treatment; 12 weeks after the initial episode, the patient presented normal blood pressure, with a brain MRI scan showing complete resolution of the lesions.

PRES is a rare syndrome in children; however, given the low level of suspicion, its incidence may be underestimated.[13] This case presents atypical manifestations, since the symptoms appeared in a previously healthy girl in the context of sudden-onset AHT, with no identified aetiology and with symptoms resolving in 8 weeks.

Although the majority of the cases described in the literature are associated with systemic diseases,[6,14] this syndrome has been reported in association with idiopathic AHT.[2] Favourable clinical progression was observed after blood pressure control, with complete resolution of the lesions after 12 weeks. Furthermore, the AHT episode resolved without aetiology being confirmed.

Most authors recommend performing a new imaging assessment after symptom resolution, although there is no consensus on the ideal time for the study since resolution is observed between 8 days and 17 months after the initial episode.[1,14]

The progression time necessary for lesions to become irreversible is not well determined.

With this case report, we hope to raise awareness of a reversible clinical and radiological entity and to highlight the importance of timely diagnosis and early treatment of the underlying cause to prevent permanent neurological sequelae.

References

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494—500.
2. Bartynski WS. Posterior reversible encephalopathy syndrome. Part 1: Fundamental imaging and clinical features. Am J Neuroradiol. 2008;29:1036—42.
3. Bartynski WS, Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. Am J Neuroradiol. 2007;28:1320—7.
4. Javed MA, Sial MSH, Lingawi S, Alfi A, Lubbad E. Etiology of posterior reversible encephalopathy syndrome (PRES). Pak J Med Sci. 2005;21:149—54.

5. Yamamoto H, Natsume J, Kidokoro H, Ishihara N, Suzuki M, Tsuji T, et al. Clinical and neuroimaging findings in children with posterior reversible encephalopathy syndrome. Eur J Paediatr Neurol. 2015;19:672—8.
6. Lucchini G, Grioni D, Colombini A, Contri M, de Grandi C, Rovelli A, et al. Encephalopathy syndrome in children with hemato-oncological disorders is not always posterior and reversible. Pediatr Blood Cancer. 2008;51:629—33.
7. Onder AM, Lopez R, Teomete U, Francoeur D, Bhatia R, Knowbi O, et al. Posterior reversible encephalopathy syndrome in the pediatric renal population. Pediatr Nephrol. 2007;22:1921—9.
8. Chen TH, Lin WC, Tseng YH, Tseng CM, Chang TT, Lin TJ. Posterior reversible encephalopathy syndrome in children: case series and systematic review. J Child Neurol. 2013;28:1378—86.
9. Bartynski WS. Posterior reversible encephalopathy syndrome. Part 2: Controversies surrounding pathophysiology of vasogenic edema. Am J Neuroradiol. 2008;29:1043—9.
10. Sharma M, Kupferman JC, Brosgol Y, Paterno K, Goodman S, Prohovnik I, et al. The effects of hypertension on the paediatric brain: a justifiable concern. Lancet Neurol. 2010;9:933—40.
11. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: What's certain, what's new? Pract Neurol. 2011;11:136—44.
12. McCoy B, King M, Gill D, Twomey E. Childhood posterior reversible encephalopathy syndrome. Eur J Paediatr Neurol. 2011;15:91—4.
13. Gonzaga D, Correia T, Rios M, Pereira C, Matos P, Figueiroa S, et al. Síndrome de Encefalopatia Posterior Reversível (PRES). Nascer e Crescer. 2008;17:233—6.
14. Gumus H, Per H, Kumandas S, Yikilmaz A. Reversible posterior leukoencephalopathy syndrome in childhood: report of nine cases and review of the literature. Neurol Sci. 2010;31:125—31.

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Orthostatic tremor as the only manifestation of thyrotoxicosis following cerebral angiography☆

Temblor ortostático como manifestación aislada de tirotoxicosis tras arteriografía cerebral

Dear Editor:

The use of iodinated contrasts in diagnostic and therapeutic techniques has become increasingly frequent over the past 20 years. These techniques consist in the administration of iodine doses between 90 and several hundred times greater than the recommended daily intake. Iodine-induced thyrotoxicosis (IIT) presents a prevalence rate of 0.05% to 5%, mainly affecting patients with history of thyroid disease; most cases are caused by CT scans with contrast or cardiac catheterisation.[1,2] While such symptoms as cardiac arrhythmia, hyperthermia, tremor, or diarrhoea are frequent in thyrotoxicosis, orthostatic tremor (OT) is exceptional.

We describe an atypical case of acute-onset OT associated with hyperthyroidism secondary to a brain angiography study in a patient with no history of thyroid disease.

Our patient was an 81-year-old male former smoker with a history of hypertension, chronic obstructive pulmonary disease, and atherosclerotic ischaemic stroke of the middle cerebral artery secondary to stenosis of the ipsilateral internal carotid artery. He was being treated with acetylsalicylic acid, clopidogrel, omeprazole, atorvastatin, and amlodipine/hydrochlorothiazide/olmesartan. He was admitted to undergo a cerebral angiography, angioplasty, and stenting of the left internal carotid artery; no immediate complications were observed. Between 48 and 72 hours after the procedure, the patient reported a feeling of instability and presented tremor in all 4 limbs, triggered in the lower limbs by standing. The neurological examination revealed symmetrical postural tremor in the upper limbs, palpable tremor of the lower limbs during standing, and no other abnormalities; these findings are compatible with OT. Results from a systemic examination were normal and palpation of the thyroid revealed no nodules. No alterations were detected in heart rate or temperature, and the patient did not present diarrhoea or any other new symptoms. An analysis of thyroid hormone levels at symptom onset revealed primary hypothyroidism (TSH: 0.04 mU/L; free T4: 2.1 ng/dL; negative antithyroid antibodies). In the absence of other symptoms, the patient continued under clinical follow-up without treatment. A week later, he showed a progressive clinical improvement, with symptoms resolving spontaneously 10 days after onset. A follow-up laboratory test revealed normalised thyroid hormone levels (TSH: 0.30 mU/L; free T4: 1.61 ng/dL).

A typical contrast dose contains approximately 13 500 μg of iodide, which may be released as free iodine in the body. Under normal circumstances, iodine overload causes the Wolff-Chaikoff effect, a self-regulatory mechanism that inhibits iodine organification and thyroid hormone synthesis. Subsequently, at 7 to 10 days, an escape phenomenon occurs and hormone synthesis resumes. Sometimes, iodine overload saturates the self-regulatory mechanism and causes the Jod-Basedow effect, resulting in uncontrolled production of thyroid hormones and IIT. Iodine-induced thyrotoxicosis

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