its use, suggesting that it also increases the risk of cardiac and thromboembolic events in the long term[8].

In the case presented here, thrombosis was identified by CTA in two different vascular territories—in the lung (pulmonary embolism) and heart (coronary thrombosis). Our finding of hypoperfusion of the left ventricular myocardium, which is supplied by the right coronary artery, reflects acute coronary occlusion and corresponds to ST segment changes on the ECG. Although ECG-gated CT is usually the noninvasive method of choice for evaluating coronary artery disease, non-ECG-gated CT of the chest may suffice as a means of providing diagnostic information regarding the patency of the coronary arteries in some cases. In addition, CTA is a widely available method of evaluating the pulmonary arteries and thoracic aorta, highlighting the unique role of CT in patients who are treated with cisplatin and suspected of having experienced a vascular event.

REFERENCES

1. Dasari S, Tchounwou PB. Cisplatin in cancer therapy: molecular mechanisms of action. Eur J Pharmacol. 2014;740:364–78.
2. Berliner S, Rahima M, Sidi Y, et al. Acute coronary events following cisplatin-based chemotherapy. Cancer Invest. 1990;8:583–6.
3. Karabay KO, Yildiz O, Aytekin V. Multiple coronary thrombi with cisplatin. J Invasive Cardiol. 2014;26:E18–20.
4. Herrmann J, Yang EH, Iliescu CA, et al. Vascular toxicities of cancer therapies: the old and the new—an evolving avenue. Circulation. 2016;133:1272–89.
5. Iliescu CA, Grines CL, Herrmann J, et al. SCAI Expert consensus statement: evaluation, management, and special considerations of cardio-oncology patients in the cardiac catheterization laboratory (endorsed by the Cardiological Society of India, and Sociedad Latino Americana de Cardiología Intervencionista). Catheter Cardiovasc Interv. 2016;87:E202–23.
6. Zahir MN, Shaikh Q, Shabbir-Moosajee M, et al. Incidence of venous thromboembolism in cancer patients treated with cisplatin based chemotherapy – a cohort study. BMC Cancer. 2017;17:57.
7. Moore RA, Adel N, Riedel E, et al. High incidence of thromboembolic events in patients treated with cisplatin-based chemotherapy: a large retrospective analysis. J Clin Oncol. 2011;29:3466–73.
8. Gietema JA, Meinardi MT, Messerschmidt J, et al. Circulating plasma platinum more than 10 years after cisplatin treatment for testicular cancer. Lancet. 2000;355:1075–6.

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The Heidenhain variant of Creutzfeldt-Jakob disease

Dear Editor,

A 78-year-old man presented with a two-month history of progressive spatial disorientation and altered color perception, without significant behavioral changes or seizures. An ophthalmologic examination showed no alterations. Serological tests for HIV and syphilis were negative. On magnetic resonance imaging (MRI) of the brain, fluid-attenuated inversion recovery (FLAIR) sequences showed a hyperintense signal in the cortical region, most pronounced in the parietal and occipital lobes, together with restricted diffusion (Figure 1). There were no signs of involvement of the white matter or basal ganglia; nor was there any contrast enhancement. A diagnosis of Heidenhain variant of Creutzfeldt-Jakob disease (HvCJD) was suggested, and that hypothesis was corroborated by electroencephalography, which showed acute, periodic triphasic waves, predominantly in the posterior areas.

CJD, also known as transmissible spongiform encephalopathy or prion disease, is a rare, rapidly progressive neurodegenerative disease with no predilection for gender, preferentially affecting patients between the fifth and eighth decades of life. It can be sporadic, which is the most common form, accounting for 85% of cases; inherited, by various mutations in the prion protein gene; iatrogenic, caused by inoculation of prions with contaminated materials; or in a variant form, which usually results from the transmission of bovine spongiform encephalopathy to humans, usually through the consumption of contaminated meat[1–3]. The typical clinical findings include a rapid decline in cognitive function, followed by myoclonic jerks and akinetic mutism. However, in HvCJD, the classic manifestation is cortical blindness due to involvement of the parieto-occipital

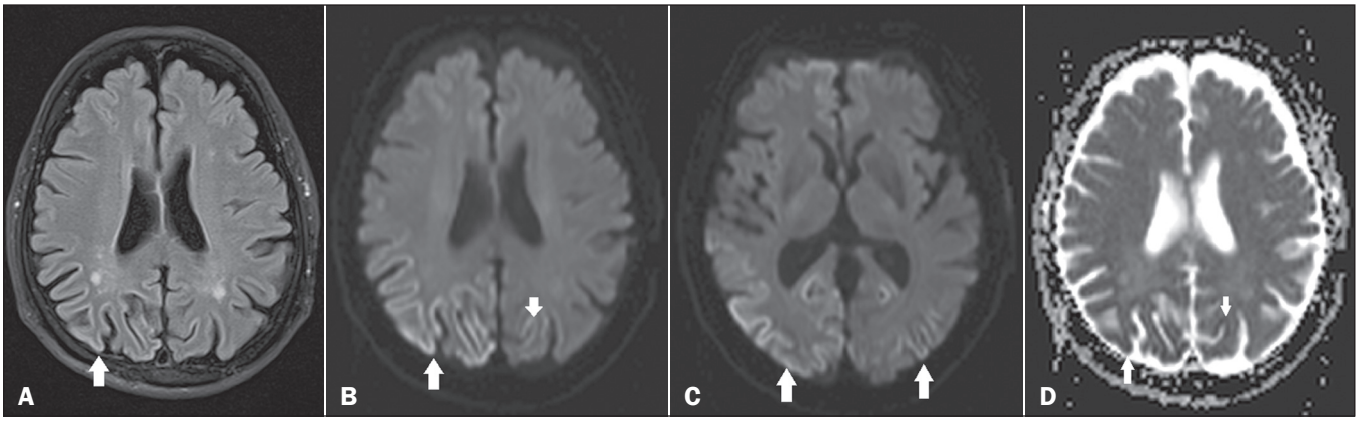

Figure 1. A: Axial FLAIR MRI sequence showing a hyperintense signal in the bilateral parieto-occipital cortex (arrow), more evident on the right, sparing the subcortical white matter. **B:** Axial diffusion-weighted MRI, at the same level depicted in **A**, showing restricted diffusion in the parieto-occipital cortex (arrow). **C:** Axial diffusion-weighted MRI, at the level of the basal ganglia and thalami, showing no changes in signal intensity. Note the restricted diffusion in the bilateral parieto-occipital cortex (arrows). **D:** Axial MRI, with apparent diffusion coefficient mapping, at the levels depicted in **A** and **B**, showing low signal intensity, confirming the restricted diffusion, in the cortical lesions.

cortex, which can be accompanied by myoclonus and progressive dementia[1,3].

MRI studies have come to play an ever more important role in the evaluation of patients with neurological diseases[4–7]. On MRI, the sporadic and inherited forms of CJD usually present areas of high signal intensity in T2-weighted and FLAIR sequences, with restricted diffusion, in the cerebral cortex and the basal ganglia, especially the striatum, in a focal or diffuse, symmetric or asymmetric form, sparing the region around the rolandic cortex and the thalami[3]. Classic signs such as the pulvinar sign and the "hockey stick" sign are typical of the variant form and are characterized respectively by hyperintense signals in T2-weighted and FLAIR sequences of the posterior and posteromedial thalami[8,9].

In HvCJD, there is invariably involvement of the parietooccipital cortex, including the primary visual cortex, characterized on MRI by hyperintense signals in T2-weighted and FLAIR sequences, together with restricted diffusion, typically with preservation of the subcortical white matter and of the basal ganglia. It is noteworthy that restricted diffusion can precede the clinical manifestations of CJD[3].

In HvCJD, the electroencephalogram typically shows acute, periodic triphasic waves, predominantly in the posterior areas[10]. Analysis of the cerebrospinal fluid can reveal elevated 14-3-3 protein levels[3]. Histopathological analysis is the gold standard diagnostic method, showing marked neuronal loss, spongiform changes, intense astrogliosis and immunoreactivity to the abnormal pathogenic isoform of the prion protein[11]. The prognosis is bleak, and death usually occurs within one year[2,9].

It is important to make the differential diagnosis of HvCJD. The main differential diagnoses are frontotemporal dementia, status epilepticus, hypoxic-ischemic encephalopathy, severe hypoglycemia, immune-mediated autoimmune encephalopathy, posterior cortical atrophy, and hyperammonemia[3]. Although rare, HvCJD should be borne in mind in the differential diagnosis of visuospatial deficits, especially when MRI shows areas of high signal intensity in T2-weighted and FLAIR sequences, together with restricted diffusion, in the cortical region of the occipital lobes.

REFERENCES

1. Baiardi S, Capellari S, Ladogana A, et al. Revisiting the Heidenhain variant of Creutzfeldt-Jakob disease: evidence for prion type variability influencing clinical course and laboratory findings. J Alzheimers Dis. 2016;50:465–76.
2. Reis F, Palma ALG, Schwingel R, et al. Creutzfeldt-Jakob dementia. Radiol Bras. 2015;48:267–8.
3. Fragoso DC, Gonçalves Filho ALM, Pacheco FT, et al. Imaging of Creutzfeldt-Jakob disease: imaging patterns and their differential diagnosis. Radiographics. 2017;37:234–57.
4. Abreu PP, Muniz BC, Ventura N, et al. Intraventricular ganglioglioma with dissemination of cerebrospinal fluid. Radiol Bras. 2018;51:272–3.
5. Niemeyer B, Muniz BC, Marchiori E. Langerhans cell histiocytosis with isolated meningeal involvement: findings on magnetic resonance imaging. Radiol Bras. 2018;51:343–4.
6. Niemeyer B, Muniz BC, Ventura N, et al. Papillary tumor of the pineal region accompanied by Parinaud's syndrome: magnetic resonance imaging findings. Radiol Bras. 2018;51:202–4.
7. Niemeyer B, Muniz BC, Gasparetto EL, et al. Congenital Zika syndrome and neuroimaging findings: what do we know so far? Radiol Bras. 2017;50:314–22.
8. Collie DA, Summers DM, Sellar RJ, et al. Diagnosing variant Creutzfeldt-Jakob disease with the pulvinar sign: MR imaging findings in 86 neuropathologically confirmed cases. AJNR Am J Neuroradiol. 2003;24:1560–9.
9. Macfarlane RG, Wroe SJ, Collinge J, et al. Neuroimaging findings in human prion disease. J Neurol Neurosurg Psychiatry. 2007;78:664–70.
10. Güveli BT, Oktar AÇ, Çabalar M, et al. EEG and cranial MRI findings in Heidenhain variant of Creutzfeldt-Jakob disease. J Neurol Sci. [Turk]. 2014;31:218–23.
11. Kher M, Rao MY, Acharya PT, et al. Heidenhain variant of Creutzfeldt-Jakob disease: an autopsy study from India. Ann Indian Acad Neurol. 2009;12:48–51.

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Radiological findings in the liver of a patient with Rendu-Osler-Weber syndrome

Dear Editor,

A 57-year-old male patient with Rendu-Osler-Weber syndrome presented to the emergency department with a 24-h history of lumbar pain. A computed tomography scan of the abdomen showed liver alterations typical of the syndrome (telangiectasias, shunts, and arteriovenous malformations), which is also known as hereditary hemorrhagic telangiectasia. The examination showed opacification of the hepatic veins in the early arterial phase—a consequence of the arteriovenous shunts (Figure 1A). We observed heterogeneous opacification of the portal vein during the portal phase, with more pronounced enhancement in the intrahepatic branches—a result of portal venous shunt—as well as numerous prominent vessels near the hepatic hilum, corresponding to an arteriovenous malformation (Figure 1B). We also observed a confluent vascular mass, measuring 1.4 cm, located in segment II (Figure 1C). In addition, there were extensive areas of altered perfusion in the hepatic parenchyma, in a mosaic pattern, as well as increased caliber of the hepatic artery at its emergence from the superior mesenteric artery, which was also ectatic (Figure 1D).

Imaging exams have played an important role in the study of liver diseases[1–5]. Hereditary hemorrhagic telangiectasia is a dominant autosomal disease with a prevalence of 10–20 cases per 100,000 population[6]. It is a rare systemic fibrovascular dysplasia that makes the walls of blood vessels more vulnerable to trauma and spontaneous ruptures[7]. It affects multiple organs and systems, being characterized mainly by the presence of telangiectasias or vascular shunts in the liver, lungs, kidneys, central nervous system, or skin[8,9]. In adults, it typically manifests as recurrent epistaxis, mucocutaneous telangiectasias, digestive tract hemorrhage, and hemoptysis[9,10]. Telangiectasias appear gradually, the most common sites being the lips, tongue, palate, fingers, and face. The diagnosis of the syndrome is based on the presence of three of the four diagnostic criteria[8]: mucocutaneous telangiectasias, recurrent spontaneous epistaxis, visceral arteriovenous malformations, and a positive family history.

In Rendu-Osler-Weber syndrome, the liver is the organ most often affected, hepatic involvement being reported in 74% of cases. Hepatic involvement is typically diagnosed 10–20 years after the