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Case Report

A stroke mimic with left-hemispheric leptomeningeal vessel paucity and hypoperfusion ☆

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ABSTRACT

In the neuroimaging workup of a suspected ischemic stroke, the involvement of more than one arterial territory without an anatomical substrate should raise the suspicion of a stroke mimic. We report the case of a 61-year-old male with a new-onset headache and transient phasic disturbances who presented a pattern of vascular abnormalities characterized by left-hemispheric leptomeningeal vessel paucity and hypoperfusion.

Introduction

Acute neurological disorders are diagnostic emergencies. In many cases, they are caused by ischemic strokes, characterized by occlusion of the arteries supplying brain regions by blood clots [1]. The identification of ischemic stroke signs is therefore paramount in neuroimaging, including nonenhanced brain computed tomography (NECT), CT angiography (CTA), and perfusion CT (CTP) [2]. Besides, the radiologist should be aware of the potential pitfalls of CTP interpretation, such as upstream flow restriction and arterial anatomical variants [3]. Nevertheless, stroke mimics may represent up to 25%-30% of cases [4,5], with various etiologies [6]. The following case of a headache and transient neurological deficit emphasizes the importance of recognizing the pattern of arterial and perfusion abnormalities and brings to attention some similar imaging findings in the literature.

Case history

A 61-year-old male patient was brought to the emergency room because of incoherent speech, difficulties finding words, and impaired comprehension. He presented slight somnolence and photophobia, but his neurological examination was otherwise unremarkable. His vital signs were correct (blood pressure: 130/80 mm Hg; heart rate: 74 bpm; oxygen saturation: 98%). He complained of progressive holocranial tension-

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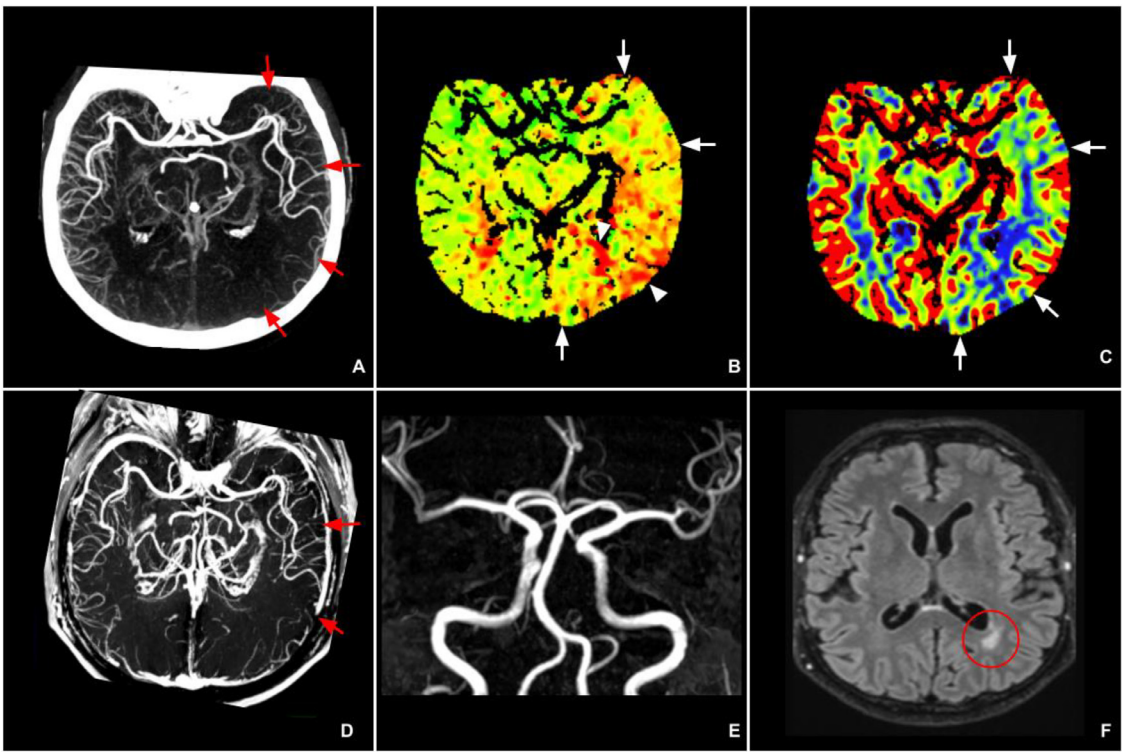

Fig. 1. (– A) CTA - maximum intensity projection: marked paucity of left MCA and PCA leptomeningeal vessels (red arrows). (B) CTP - TTP: hypoperfusion of left MCA and PCA territories (white arrows), predominantly in the superficial watershed and the deep periventricular regions (white arrowheads). (C) CTP - CBV: moderate decrease in the corresponding areas (oligemia). (D) MRI - 3DT1-GE-Gd: leptomeningeal vascular paucity. (E) MRI - TOF: normal caliber of moderate-to-large intracranial arteries. (F) MRI - FLAIR: white matter hyperintensity focus in the deep left peritrigonal region.

like headaches for 3 weeks with a flareup in the last 48 hours but had no history of migraine. He also presented an infected wound on the leg without fever that was being treated with antibiotics. His medical history consisted of well-substituted hypothyroidism and smoking (20 cigarettes/d).

Due to the suspicion of stroke, emergent NECT, CTA, and CTP were performed. No signs of acute ischemic or hemorrhagic stroke, arterial occlusion, or stenosis were found. However, CTA disclosed a marked paucity of leptomeningeal vessels in the left middle cerebral artery (MCA) and posterior cerebral artery (PCA) territories without involvement of medium-to-large cerebral arteries (Fig. 1A). Of note, the left PCA was not of fetal origin. CTP demonstrated diffuse hypoperfusion in the corresponding territories, visible on all parametric maps, including time-to-peak (TTP; Fig. 1B) and a moderate decrease in local cerebral blood volume (CVB; Fig. 1C), compatible with oligemia. The alterations were most prominent in the MCA/PCA superficial watershed territory and the deep periventricular region. Due to the discrepancy between the extent of hypoperfusion and clinical symptoms, notably the absence of motor symptoms, intravenous fibrinolysis was not administered. Magnetic resonance imaging (MRI) showed persistent leptomeningeal vascular paucity in the same territories on day 6 on the gadolinium-enhanced 3D T1-weighted gradient echo sequence (3DT1-GE-Gd; Fig. 1D). Medium-to-large intracranial arteries were normal on 3D time-of-flight (TOF) angiography (Fig. 1E). A small new lesion in the left peritrigonal white matter was also found, as a T2-weighted fluid-attenuated inversion recovery (FLAIR) hyperintensity (Fig. 1F) and as a hypodensity on the NECT coupled with positron emission tomography performed on day 10. No signs of acute ischemia, cerebral edema, pathological enhancement, venous thrombosis, or metabolic abnormality were found.

Blood and cerebrospinal fluid (CSF) analyses showed mild serum hyperleukocytosis (13,200/mm^3 of which 8410 neutrophils) without increased C-reactive protein, as well as mild proteinorachia (0.63 g/L) without pleocytosis. Serial electroencephalograms (EEG; days 3, 4, 6) revealed intermittent and diffuse slow delta/theta-band activity, sometimes paroxysmal but without ictal activity and unmodified by photic stimulation or hyperpnea.

The patient remained slightly somnolent and complained of mild headaches. The symptoms gradually subsided from day 4, before Keppra was initiated to treat the possibility of a seizure-like phenomenon. The patient reported no ulterior headaches or impairments.

Discussion

In the context of headache, dysphasia, and signs of oligemia in more than one vascular territory without an anatomical substrate, the diagnosis of a stroke mimic was proposed. We

discuss the differential diagnoses and their imaging findings hereunder.

- Our case shows similarities with a pediatric series of 16 stroke mimic patients with headaches and transient focal neurological deficits [7]. Hypoperfusion affects multiple arterial territories and is associated with a paucity of related arterial branches. The left MCA is most affected. In contrast, it is accompanied by an increased prominence of the corresponding leptomeningeal veins on MRI susceptibility-weighted imaging (SWI).
- HaNDL (Headache with Associated Neurological Deficits and CSF Lymphocytosis), characterized by paroxysmal headache and transient neurological deficits (confusion and aphasia among the most common), associated with leukocytosis and elevated protein in CSF [8]. The left hemisphere is more commonly affected [9]. Diffuse slowing on EEG may be observed in nearly half of the cases [8]. Neuroimaging abnormalities associated with HaNDL are uncommon and heterogeneous, including brain hypoperfusion and leptomeningeal signal abnormalities [8,9]. In addition, diffuse left-hemispheric hypoperfusion on CTP [10,11] and paucity of leptomeningeal veins on SWI [12] have also been reported. However, our patient did not demonstrate CSF lymphocytosis.
- Migraine with aura, where hypoperfusion of more than one arterial territory may also be seen, albeit with posterior predominance [13]. In this setting, increased prominence of veins on SWI in the affected territory has been reported [14]. However, our patient had no history of migraine or classic aura symptoms [15].
- Cryptogenic postictal origin, considering the perfusion abnormalities in atypical vascular distributions, which can be multilobar and holohemispheric [16], despite the absence of ictal activity detected on EEG or PET.

Other etiologies were deemed unlikely:

- Reversible cerebral vasoconstriction syndrome (RCVS), owing to the absence of a thunderclap headache history, vasoconstrictive trigger, subarachnoid hemorrhage [17], or medium-to-large artery involvement [18].
- Central nervous system vasculitis, due to the lack of ischemic or hemorrhagic lesions, white matter edema, pathological enhancement, or beading of arteries [19].

The pathophysiology of the association between headaches, neurological deficits, and vascular abnormalities remains unclear [7–14]. Of note, the appearance of a nonspecific white matter lesion in our patient may result from microischemic changes in the most severe focus of hypoperfusion. Moreover, SWI was not part of our patient's MRI protocol.

Conclusion

We highlight a pattern of vascular abnormalities in a stroke mimic with left-hemispheric leptomeningeal vessel paucity and hypoperfusion involving more than one arterial territory in the context of headaches and phasic disturbances. To our knowledge, this finding had not been reported *per se* in an adult. Careful examination of the arterial vasculature and accompanying neuroimaging abnormalities in the clinical context may help the radiologist to propose and exclude alternative diagnoses to ischemic stroke.

Patient consent

Written informed consent was obtained from the patient.

REFERENCES

[1] Phipps MS, Cronin CA. Management of acute ischemic stroke. BMJ 2020;368:l6983.
[2] Smith AG, Rowland Hill C. Imaging assessment of acute ischaemic stroke: a review of radiological methods. Br J Radiol 2018;91:20170573.
[3] Best AC, Acosta NR, Fraser JE, Borges MT, Brega KE, Anderson T, et al. Recognizing false ischemic penumbras in CT brain perfusion studies. RadioGraphics 2012;32(4):1179–96.
[4] Hand PJ, Kwan J, Lindley RI, Dennis MS, Wardlaw JM. Distinguishing between stroke and mimic at the bedside: the brain attack study. Stroke 2006;37(3):769–75.
[5] Pohl M, Hesszenberger D, Kapus K, Meszaros J, Feher A, Varadi I, et al. Ischemic stroke mimics: a comprehensive review. J Clin Neurosci 2021;93:174–82.
[6] Adam G, Ferrier M, Patsoura S, Gramada R, Meluchova Z, Cazzola V, et al. Magnetic resonance imaging of arterial stroke mimics: a pictorial review. Insights Imaging 2018;9(5):815–31.
[7] Lehman LL, Danehy AR, Trenor CC, Calahan CF, Bernson-Leung ME, Robertson RL, et al. Transient Focal neurologic symptoms correspond to regional cerebral hypoperfusion by MRI: a stroke mimic in children. Am J Neuroradiol 2017;38(11):2199–202.
[8] Al-Chalabi M, Hegde P, Asghar F, Aladamat N, Delcimmuto N, Gharaibeh K, et al. Transient headache and neurological deficits with cerebrospinal fluid lymphocytosis syndrome: a comprehensive systematic review of 93 patients from 57 studies. Cephalalgia 2023;43(4):033310242311576.
[9] Gomez-Aranda F. Pseudomigraine with temporary neurological symptoms and lymphocytic pleocytosis. A report of 50 cases. Brain 1997;120(7):1105–13.
[10] Burke MJ, Lamb MJ, Hohol M, Lay C. Unique CT perfusion imaging in a case of HaNDL: new insight into HaNDL pathophysiology and vasomotor principles of cortical spreading depression. Headache J Head Face Pain 2017;57(1):129–34.
[11] Pettersen JA, Aviv RI, Black SE, Fox AJ, Lim A, Murray BJ. Global hemispheric CT hypoperfusion may differentiate headache with associated neurological deficits and lymphocytosis from acute stroke. Stroke 2008;39(2):492–3.
[12] Rodríguez-López C, Garzo Caldas N, Uriarte Pérez de Urabayen D, Sánchez Tornero M, Hilario Barrio A, Saiz Díaz R, et al. A new MR radiological sign in HaNDL syndrome. A case report. J Clin Neurosci 2019;61:274–6.
[13] Floery D, Vosko MR, Fellner FA, Fellner C, Ginthoer C, Gruber F, et al. Acute-onset migrainous aura mimicking acute stroke: MR perfusion imaging features. Am J Neuroradiol 2012;33(8):1546–52.

[14] Miller C, Goldberg MF. Susceptibility-weighted imaging and computed tomography perfusion abnormalities in diagnosis of classic migraine. Emerg Radiol 2012;19(6):565–9.
[15] Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018;38(1):1–211.
[16] Gelfand JM, Wintermark M, Josephson SA. Cerebral perfusion-CT patterns following seizure: cerebral perfusion-CT patterns following seizure. Eur J Neurol 2010;17(4):594–601.
[17] Rocha EA, Topcuoglu MA, Silva GS, Singhal AB. RCVS2 score and diagnostic approach for reversible cerebral vasoconstriction syndrome. Neurology 2019;92(7):e639–47.
[18] Miller TR, Shivashankar R, Mossa-Basha M, Gandhi D. Reversible cerebral vasoconstriction syndrome, part 2: diagnostic work-up, imaging evaluation, and differential diagnosis. Am J Neuroradiol 2015;36(9):1580–8.
[19] Abdel Razek AAK, Alvarez H, Bagg S, Refaat S, Castillo M. Imaging spectrum of CNS vasculitis. RadioGraphics 2014;34(4):873–94.