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Case Reports

Hyperintense ipsilateral cortical sulci on FLAIR imaging in carotid stenosis: ivy sign equivalent from enlarged leptomeningeal collaterals

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ABSTRACT

Fluid-attenuated inversion recovery (FLAIR) imaging provides high contrast between hyperintense lesions and normal tissue. Hyperintense structures in convexity sulci are commonly linked to abnormal cerebrospinal fluid composition, whether blood, protein, or infection. A patient with hemispheric transient ischemic attacks from severe carotid stenosis had hyperintense convexity sulci on FLAIR magnetic resonance imaging, interpreted as possible prior hemorrhage, making the patient ineligible for carotid stent reconstruction. Retrospective analysis revealed that hyperintense sulci were dilated leptomeningeal collaterals. In severe arterial disease causing cerebral hypoperfusion, dilated leptomeningeal vessels should be considered a cause for serpiginous hyperintense structures on FLAIR imaging, similar to the "ivy sign" described in moya-moya patients.

1. Introduction

Fluid-attenuated inversion recovery (FLAIR) is routinely used in magnetic resonance imaging (MRI) of the brain because of its high lesion-to-tissue contrast from T2 prolongation with nulling of normal cerebrospinal fluid (CSF) background. It is widely recognized to be particularly sensitive for disease states that affect the CSF in the subarachnoid space, which then appears hyperintense on those images, including subarachnoid hemorrhage, meningitis, meningeal carcinomatosis, leptomeningeal metastasis, subdural hematoma, adjacent neoplasms, dural venous thrombosis, and status epilepticus [1–6,8,9]. It has also been described as a diagnostic pitfall in patients undergoing MRI on propofol [10] or supplemental oxygen, [11] and in case of prior administration of iodinated contrast material [14]. However, and although recognized in the moya-moya population as the "ivy sign" [17–19], the appearance of enlarged leptomeningeal collaterals presenting as serpiginous hyperintense structures on FLAIR imaging is not appreciated. We report on a patient in whom treatment planning was affected by the lack of proper understanding of such finding.

2. Case report

A 69-year-old woman presented to the emergency department with complaints of intermittent right hand weakness and numbness suggestive of transient ischemic attacks (TIAs). The patient was treated for hypertension and hypercholesterolemia, and was not on aspirin. MRI of the brain did not show restricted diffusion consistent with strokes, but showed diffuse serpentine hyperintensities within the left hemispheric convexity on FLAIR images, interpreted as possible subarachnoid hemorrhage (SAH) or meningitis (Fig. 1). The patient denied headaches and declined a spinal tap. A computed tomographic angiogram of the brain was obtained, which demonstrated a severe left carotid stenosis, the most likely source of the patient's TIAs, and a small aneurysm of the contralateral middle cerebral artery (Fig. 2). As there was concern for possible aneurysmal SAH, a cerebral angiogram was obtained, which confirmed a small, incidental right anterior temporal origin aneurysm, and showed relatively poor collateral circulation to the left hemisphere, with dilated, late-filling convexity collaterals (Fig. 3). Following multidisciplinary assessment, it was elected to offer carotid endarterectomy to the patient, as there was a remaining doubt as to the FLAIR MRI findings representing prior, clinically silent SAH which could interfere with the administration of dual antiplatelet therapy.

Despite minor chest pains without electrocardiographic or enzyme changes, the patient fared well after carotid endarterectomy and was discharged on aspirin with no recurrence of TIAs. A few weeks later, a follow-up MRI was obtained, showing complete resolution of the left convexity sulcal serpentine hyperintensities (Fig. 4). The source

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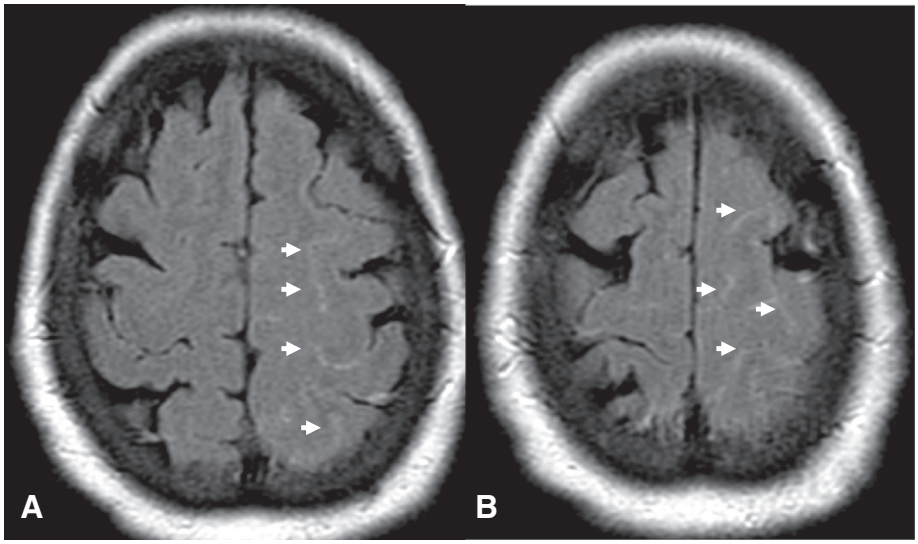

Fig. 1. (A and B) Axial FLAIR images show linear hyperintense structures coursing through the subarachnoid spaces of the high frontal-parietal convexity (arrows).

images of the computed tomographic angiogram obtained upon admission were retrospectively reviewed (Fig. 5), showing no SAH, and markedly dilated enhancing vascular structures corresponding to the enlarged collaterals seen at angiography, which were thought to represent maximally dilated convexity arteries with slow flow within them.

3. Discussion

Hyperintensities in the subarachnoid space of cortical convexity sulci on FLAIR MRI have long been recognized as a radiological marker for a number of clinical conditions. This finding, which has been referred to as “dirty CSF”, “sulcal hyperintensity”, “hyperintense CSF,” “leptomeningeal hyperintensity,” or “hyperintensity within the subarachnoid space” was initially reported in a number of conditions which cause an abnormality in the CSF, such as subarachnoid hemorrhage [1–3], leptomeningeal carcinomatous disease [4], meningitis [5], meningeal chemical irritation [6]. All these conditions have in common abnormal CSF composition, in particular protein concentration, which has been shown experimentally to consistently result in bright FLAIR signal above a certain threshold [7] such that in chemical meningitis, inflammatory meningitis, dural sinus thrombosis, status epilepticus, and neoplastic infiltration of the subarachnoid space, sulcal hyperintensities on FLAIR imaging have been considered by some to have a diagnostic value superior to that of CT [8,9].

However, FLAIR sulcal hyperintensity was also shown to be present in a variety of situations for which there would be no reason for abnormal CSF, and which result in alterations of normal physiology, including propofol administration [10] and general anesthesia [11] leading to erroneous interpretation of MRI scans in anesthetized patients. Although the chemical composition of propofol and other anesthetic agents was initially thought to be the cause for changes in CSF signal at MRI, it was demonstrated that it was the

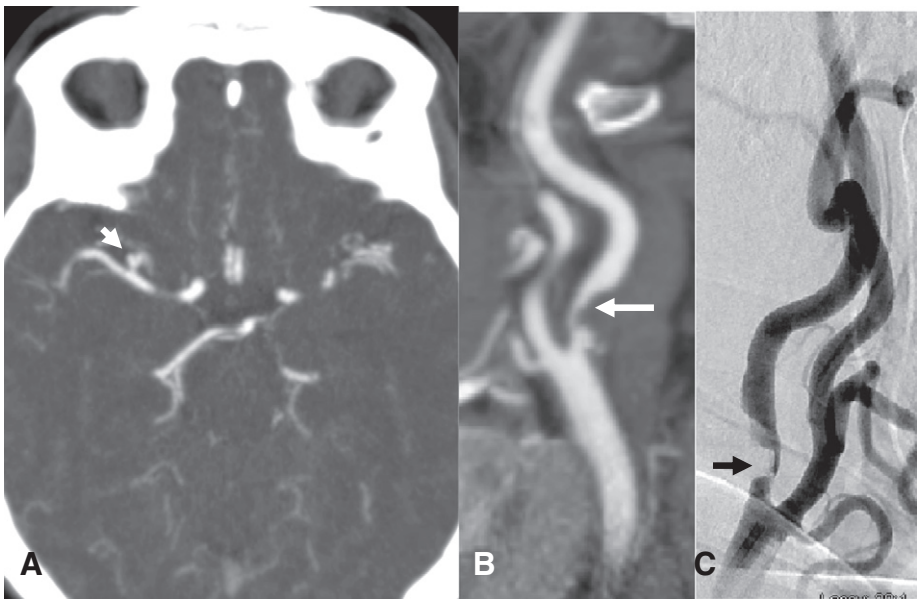

Fig. 2. CT angiographic and angiographic findings. (A) Axial image of CT angiogram shows a 3 mm aneurysm of the right middle cerebral artery at the origin of the anterior temporal branch. (B) Sagittal reconstruction shows a critical ulcerated stenosis of the post-bulbar left internal carotid artery. (C) Left common carotid artery angiogram, anterior oblique view confirms the presence of a severe (95%) stenosis at the origin of the left internal carotid artery.