

Acute ischaemic larger artery stroke presenting as a subcortical lesion with grey matter sparing: a case report

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Abstract

Large artery infarctions classically involve both white matter and grey matter in the distribution of the occluded artery. Rarely, large artery infarctions can present as isolated white matter lesions. We describe a 38-year-old, right-hand dominant woman, who presented with sudden onset right face-arm-leg weakness together with expressive aphasia. Neuroimaging demonstrated changes restricted to the white matter in the distribution of the left middle cerebral artery territory. Vascular imaging of her head and neck vessels showed irregular narrowing of the intracranial portions of the left internal carotid artery. Vasculitis was suspected due to these findings on imaging, and she was initially treated with steroids. However, due to the negative vasculitis work-up and an inadequate response, the steroids were discontinued, and she was treated with anti-platelets and statins. Early stroke rehabilitation commenced while inward.

Large artery ischaemic strokes can rarely present as white matter lesions when there is adequate collateral circulation, which leads to the sparing of the grey matter.

Key words: ischaemic stroke, larger artery stroke, large artery occlusion, subcortical stroke, MCA territory stroke

Introduction

Cerebral infarctions which occur as a result of large artery occlusions classically involve both the grey and white matter, leading to a parenchymal

hypodensity with indistinct grey matter-white matter borders and cortical sulcal effacement, in the distribution of the culprit artery.¹ The presence of collateral circulation may influence the distribution of the infarction. We describe a case of a large artery infarction which was presented as an isolated white matter lesion.

Case presentation


A 38-year-old, right-hand dominant woman, presented with sudden onset right face, arm and leg weakness. She was transferred to our unit 48 hours after the onset of the weakness. This patient was previously healthy apart from a long-standing non-specific headache.

Her neurological examination revealed a right-side upper motor neuron type facial nerve palsy and right hemiparesis of Medical Research Council (MRC) power 0/5, together with hemianaesthesia on the same side. She was also noted to have expressive dysphasia. Her general examination was normal and there was no evidence of vasculitic rashes. Her pulse rate was 92 bpm, regular and blood pressure was 160/90 mmHg in both arms. Distal pulses were of good volume and easily palpable. She did not have audible murmurs or carotid bruits. Examination of the abdomen was normal.

Non-contrast CT brain showed a hypodensity involving the left frontoparietal white matter without evidence of haemorrhage. Her MRI-Brain revealed diffusion restriction in the left middle cerebral artery (MCA) territory with sparing of the grey matter. She also had imaging of the intracranial arterial and venous

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systems. The magnetic resonance angiogram (MRA) showed an irregular narrowing of the petrous and cavernous segments of the left internal carotid artery (ICA). The rest of the MRA was normal. The magnetic resonance venogram (MRV) was normal.

The CT-angiogram (CTA) showed diffuse narrowing of the left ICA from the distal part of the cervical segment, involving the petrous, lacerum, cavernous, supra-clinoid and distal segments. The right ICA and posterior circulation were normal.

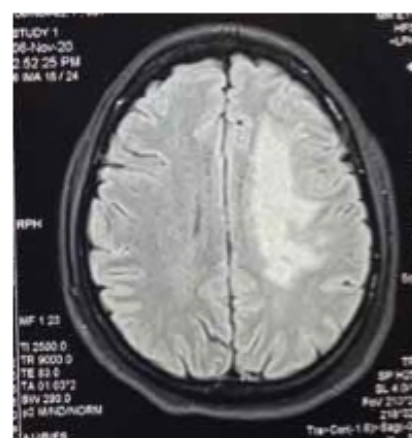


Image 1. Non-contrast CT-brain – axial view (48 hours after onset of the weakness) – white arrow showing the area of white matter hypodensity.

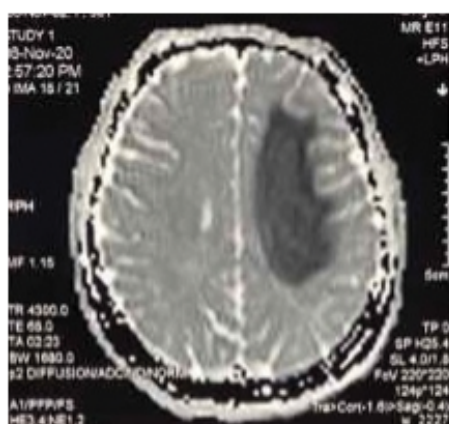
Image 2. MRI-Brain – axial views



a) Diffusion-weighted imaging (DWI) image



c) FLAIR image



b) Apparent diffusion coefficient (ADC) image

Further evaluation for an underlying aetiology was performed. Her cardiac investigations were normal, including a 12-lead ECG and transthoracic echocardiogram. A trans-oesophageal echocardiogram could not be performed due to technical reasons. The full blood count, fasting blood sugar and lipid profile were normal. The erythrocyte sedimentation rate was 9 mm/1st hour and CRP was 32 mg/L. Evaluation for infectious aetiologies such as syphilis and human immunodeficiency virus (HIV) were negative. Anti-nuclear antibodies (ANA), double-stranded DNA (ds-DNA) antibodies, anti-nuclear cytoplasmic antibodies (ANCA) and the beta-2 glycoprotein-1 antibodies (IgM and IgG) were negative.

She was initially treated with aspirin, atorvastatin, and intravenous methylprednisolone due to the possible diagnosis of vasculitis. Thrombolysis was not performed due to the delayed presentation. Methylprednisolone was converted to oral prednisolone after 5 days. However, when the results of the inflammatory markers and vasculitis screen were available, a decision was taken to discontinue steroids. The antiplatelets and statins were continued. Stroke rehabilitation commenced, and the patient was eventually discharged with a long-term rehabilitation plan.

Discussion

Ischaemic strokes due to large artery occlusions normally result in infarctions involving both the grey and white matter.¹ However, if there is well-developed collateral circulation, the grey matter can be spared, and an isolated white matter lesion can remain. Such collaterals are known to occur between the ICA and the external carotid artery.² Additionally, collateral vessels can occur between the main cerebral arteries and the leptomeningeal vessels.³ This explains the isolated white matter infarction, despite our patient's extensive narrowing of the ICA.

The development of collateral circulation in most vascular beds is a chronic process. Long-standing ischaemia can trigger the release of vascular endothelial growth factor (VEGF) and subsequent neovascularization leading to the development of collateral circulation.³ In our patient, the diffuse narrowing of the left ICA is likely to have been a long-standing abnormality. This may have triggered the development of collateral circulation. Her long-standing headache may have been a manifestation of cerebral ischaemia.

Collateral vessel formation is well known to occur in vasculopathies such as Moyamoya disease.⁴ It is a cerebrovascular disease which is characterized by progressive stenosis of the terminal portion of the intracranial ICA and its proximal branches.⁴ However, these vasculopathies would typically cause multiple areas of narrowing, unlike the single large narrowing noted in our patient. However, fibromuscular dysplasia has been reported to lead to long tubular stenotic lesions in addition to the characteristic beaded lesions.⁵

Vasculitis involving the cerebral vessels typically cause multiple stenotic segments of varying degrees with intervening vascular dilatation, which leads to a "string of beads" appearance on vascular imaging,⁶ which was not seen in our patient.

The ICA is radiologically divided into 7 segments.¹ From origin to termination, these segments are as follows, cervical, petrous, lacerum, cavernous, clinoid, ophthalmic and communicating. The CTA clearly revealed the extent of the narrowing of the ICA in this patient.

Digital subtraction angiography (DSA) was not performed as it would not have revealed additional information to those from the MRA and CTA. Furthermore, the low probability of vasculitis and the invasive nature of DSA precluded its use in this clinical setting. However, a DSA may have been useful in demonstrating radiological features of certain vasculopathies such as Moyamoya disease.

The unusual nature of the lesion on neuroimaging and the absence of traditional vascular risk factors led us to suspect an inflammatory process and to treat her with steroids. When the ischaemic nature of the lesion became apparent, the steroids were discontinued.

Conclusion

Large artery infarctions can rarely present as isolated white matter lesions in the presence of adequate collateral circulation.

Author declarations

Consent for publication

Obtained from the patient and her family.

Competing interests

None to declare.

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None.

Authors' contributions

SP, NTW, PM, SB and AF all contributed to the clinical management of the patient. SP wrote the manuscript. All authors read and approved the final manuscript.

References

1. Osborn AG, Hedlund GL, Salzman KL. Osborn's Brain. 2nd Edition. 2017. ISBN - 9780323477765
2. Erickson KM, Cole DJ. Carotid artery disease: stenting vs endarterectomy. *Br J Anaesth.* 2010; **105** (S1): 34-49. DOI: 10.1093/bja/aeq319. PMID: 21148654.
3. Lucitti JL, Mackey JK, Morrison JC, et al. Formation of the collateral circulation is regulated by vascular endothelial growth factor-A and a disintegrin and metalloprotease family members 10 and 17. *Circ Res.* 2012; **111**(12): 1539-50. DOI: 10.1161/CIRCRESAHA.112.279109.
4. Liu Z, Han C, Zhao F, et al. Collateral circulation in Moyamoya Disease. *Stroke* 2019; **50**: 2708-15. DOI: 10.1161/STROKEAHA.119.024487.
5. Mettinger KL. Fibromuscular dysplasia and the brain II. Current concept of the disease. *Stroke.* 1982; **13**(1): 53-8. DOI: 10.1161/01.str.13.1.53. PMID: 7039003.
6. Abdel A, Abdel K, Alvarez H, Bagg S, Castillo M. Imaging spectrum of CNS vasculitis. *Radio Graphics* 2014; **34**(4): 873-94. DOI: 10.1148/rg.344135028. PMID: 25019429.