18 year old female with Focal Cerebral Arteriopathy related to chronic methylphenidate treatment

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Case report

A 18 year old female with a personal history of hyperactivity disorder was admitted to our emergency department with a left M1 acute occlusion. She underwent endovascular treatment achieving complete recanalization despite a difficult procedure due to middle cerebral artery (MCA) irregularity.

The angiographic study revealed marked stenosis of the distal internal carotid (ICA), proximal middle cerebral and anterior cerebral (ACA) arteries, with contrast enhancement of the blood vessel on the Magnetic Resonance Angiography (MRA).

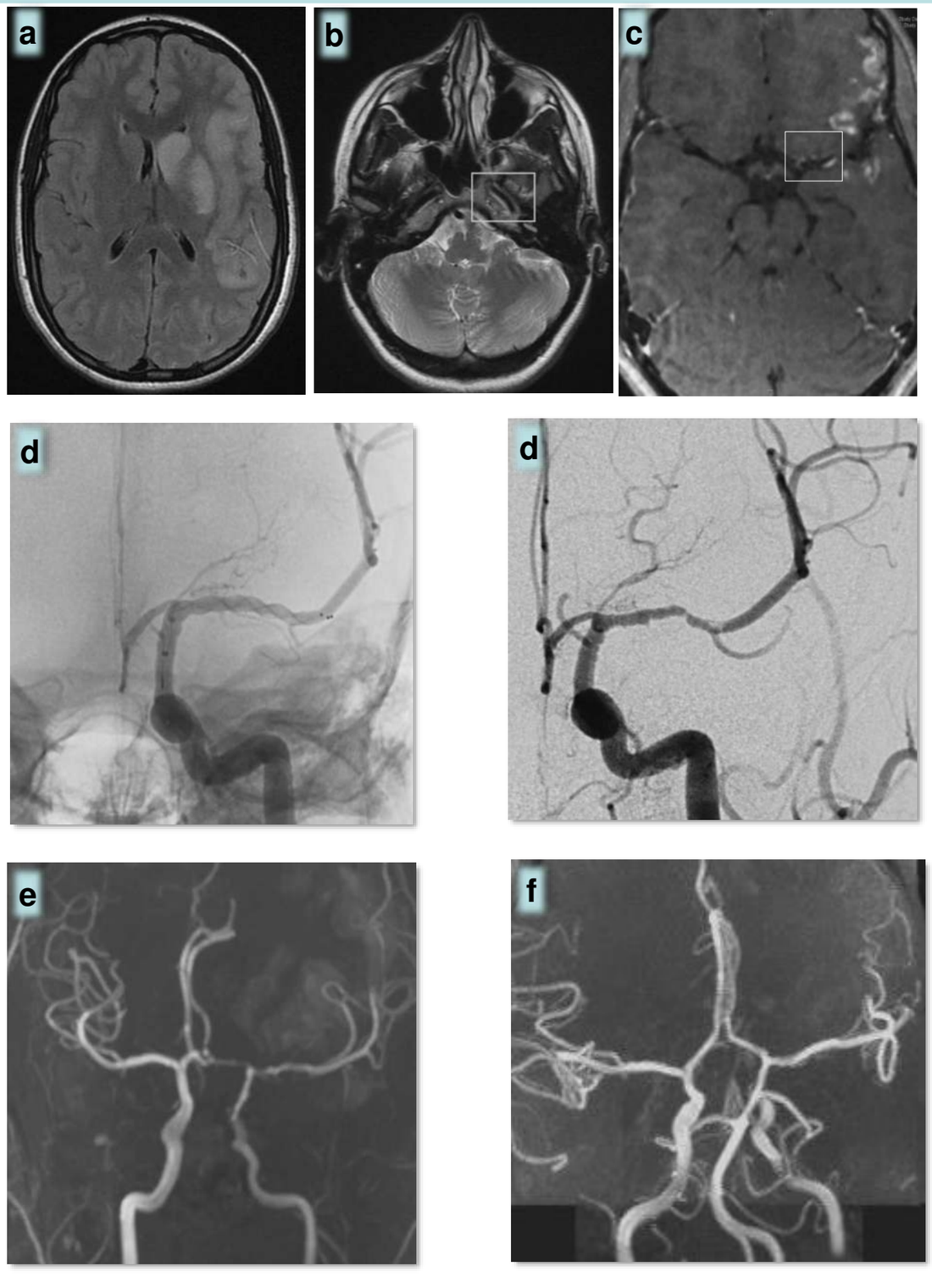
A thorough examination was conducted, including VVZ serology and CSF analysis.

The patient was under treatment with metilphenidate combined with paliperidone and hormonal contraceptive therapy and six months before she has developed a mild but persistent headache in relation to an increase on the dose from 36 to 54 mg. In the absence of other pathological findings this remains the most likely cause of the focal vasculitic process.

At discharge the patient presented persistent aphasia with a total NIHSS score of 9.

She received a combined antithrombotic and corticosteroid treatment with oral prednisone.

Follow-up MRA 6 months later showed partial improvement of the arterial narrowing without contrast enhancement of the blood vessel.



a) T2-FLAIR sequence showing acute infarct in left MCA territory. **b)** Left internal carotid hyperintensity on T2-weighted MRA due to inflammatory changes on the vessel wall. **c)** Gyriform enhancement at the left frontal lobe and left MCA wall enhancement in postcontrast T1-weighted sequence. **d)** MCA irregularity during acute mechanical recanalization. **e)** Baseline angio-MRA showing vessel irregularity and stenosis involving left ICA, MCA and initial segment of ACA.
f) Follow-up MRA at 6 months shows increase in arterial caliber.

Discussion

Focal cerebral artheriopathy describes a unilateral stenosis or vessel irregularity involving the terminal segment of the ICA, MCA and ACA. It accounts for up to 35% of all cases of childhood arterial ischemic stroke.

The differential diagnosis includes a wide spectrum of infectious/ inflammatory diseases despite the common angiographic pattern. Progressive arteriopathy is seen in the context of moyamoya disease, childhood primary vasculitis of the CNS and some extracraneal dissections while non-progression (or regression) of arterial lesions on follow-up imaging 6 months after initial stroke defines **transient cerebral arteriopathy (TCA).** Post-varicella arteriopathy is one of the main causes of TCA, followed by toxic (eg, cocaine) and thrombotic factors.

The pathophysiological mechanism of **methylphenidate-induced vasculitis** remains unknown. Vasoespasm, necrotizing angiitis and thrombosis of the cerebral artheries are seen in stroke after amphetamine abuse. Similar effects may be expected considering the structural and functional analogies.

Conclusion

Vasculitis related to metilphenidate treatment is an infrequent condition that should be taken into account in the etiological study of pediatric and young adults stroke in the absence of other pathological findings.

1. Focal Cerebral Arteriopathy: The Face With Many Names Tolani, Alisha T. et al. Pediatric Neurology , Volume 53 , Issue 3 , 247 – 252 **2. Transient cerebral arteriopathy in childhood** Sébire, Guillaume, The Lancet , Volume 368 , Issue 9529 , 8 – 10; **3. Cerebral Arteritis Following Methylphenidate Use,** Angeles Schteinschnaider et al., Journal of Child Neurology , Vol 15, Issue 4, pp. 265 – 267, **4. Transient Cerebral Arteriopathy, Postvaricella Arteriopathy, and Focal Cerebral Arteriopathy or the Unique Susceptibility of the M1 Segment in Children With Stroke,** Stéphane Chabrier, Guillaume Sébire and Joel Fluss, Stroke. 2016;47:2439-2441