Letter by Martínez-Martínez et al Regarding Article, “Variable Presentations of Postpartum Angiopathy”

To the Editor:

Stroke occurring during pregnancy and postpartum is a diagnostic challenge because there are several specific causes to be considered in its management and differential diagnosis, including postpartum angiopathy (PPA).1 Thus, we read with great interest the recent article by Fugate et al2 reviewing clinical features, patient characteristics, and neuroimaging in PPA. Although the evidence of multifocal narrowing of cerebral arteries in cerebral angiography is a requisite for its diagnosis, up to 39% of casual angiograms of patients with PPA may be normal as Fugate et al found in this multicenter series. However, the possible role of transcranial Doppler (TCD) in helping achieve early diagnosis as well as better monitoring of response to treatment has not been mentioned in this review. TCD is a valid noninvasive and easily available tool that can be performed as many times as necessary to identify/quantify the severity of cerebral arterial vasoconstriction and monitor the response to treatment in follow-up.3

We describe a case of a 41-year-old patient (gravida 5, para 1) with no complications in her current pregnancy. She was treated with norepinephrine for severe hypotension after uterine arterial disruption and cabergoline to prevent lactation after delivery. From Day 7 postpartum, she developed intense headaches and positive visual symptoms with right homonymous hemianopsia on examination. Cranial CT revealed a left occipital intracranial hemorrhage. CT angiography ruled out the presence of an underlying aneurism or arteriovenous malformation and no signs of cerebral arterial vasoconstriction were found. MRI venography ruled out a cerebral venous thrombosis. Twenty-four-hour urine analysis revealed mild proteinuria (439 mg/L). She presented several episodes of transient positive visual symptoms and on Day 7 after admission she developed cortical blindness. TCD revealed signs of mild to severe vasoconstriction in both left and right middle cerebral arteries measured by the Lindegaard index. A new diffusion-weighted MRI showed an ischemic lesion on the right occipital lobe and a left occipital lobe hemorrhage. MR angiography demonstrated multiple arterial narrowings involving proximal arteries on the circle of Willis and the basilar artery, which were confirmed by digital subtraction angiography.

Treatment with 60 mg nimodipine every 4 hours and 4 mg dexamethasone every 6 hours was started with no new neurological events. Progressive improvement of arterial vasoconstriction was demonstrated on follow-up TCD at 16, 18, 19, and 21 days after symptom onset. There was significant clinical improvement with right inferior quadrantopsia as the only deficit at discharge. Complete recovery of vasoconstriction was assessed at the 2-month follow-up with MR angiography.

Our case of PPA presented as headache and cerebral hemorrhage. Classic presentations include seizures and ischemic stroke, and so we find the current report of a high percentage of hemorrhages (39%),2 which had been less commonly reported,4 very interesting.

An important point on which we agree with the authors is that PPA diagnosis is often delayed because cerebral vasospasm cannot be seen in the first neuroimaging procedure, like in our case. Although the gold standard for diagnosis of PPA is digital subtraction angiography, TCD is a valid, noninvasive, and easily available tool that can be used as many times as necessary to identify/quantify the severity of the vasoconstriction as well as monitor the response to treatment on follow-up.3 We used this method with the Lindegaard index to assess improvements and response to treatment.

The diagnostic of PPA in our patient is based on an uneventful pregnancy, the relationship with the drugs prescribed (ergot derivatives and sympathomimetic drugs), and angiographic findings. It was also based on the reversibility of the disease, although delayed postpartum eclampsia cannot be completely ruled out because proteinuria was observed on admission. This was despite having our patient closely monitored during pregnancy, in which neither proteinuria nor hypertension was reported. In this sense, we agree that both eclampsia and PPA may belong to the same spectrum of disorders.1

In conclusion, TCD monitoring is feasible and may be recommendable for patients with stroke in the postpartum period, even with negative first angiogram neuroimaging, because it could be useful for the early detection of vasoconstriction and for monitoring the treatment response in PPA.

Disclosures

None.

Marta Martínez-Martínez, MD
Blanca Fuentes, MD, PhD
Exuperio Díez-Tejedor, MD, PhD, FAHA, FESO
Stroke Centre
Department of Neurology and Neuroscience Research
IdiPAZ Health Research Institute
La Paz University Hospital
Universidad Autónoma de Madrid
Madrid, Spain

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Marta Martínez-Martínez, Blanca Fuentes and Exuperio Díez-Tejedor

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