Is cervicocephalic dissection a part “postpartum vasculopathy” spectrum? A case of postpartum posterior reversible encephalopathy syndrome, bilateral petrous carotid artery dissections, and stroke

Sir,
The postpartum dissection of large and medium sized arteries is a rare entity, being increasingly recognized due to the widespread availability of non-invasive imaging. Coronary arteries and cervicocephalic arteries (carotid and vertebral arteries) are commonly involved. Although single vessel dissection is reported, involvement of two or more arteries is not uncommon. Involvement of both the coronary and cervicocephalic arteries in the same patient at the same time possibly represents a unique predisposition shared between these two arteries. The association of postpartum posterior reversible encephalopathy syndrome (PRES) with cervicocephalic dissection is rare. We report the fourth case of cervicocephalic artery dissection associated with PRES.

A 29-year-old female patient had severe pregnancy-induced hypertension with albuminuria underwent preterm lower segment caesarean section (LSCS) at 31 weeks of pregnancy. She developed multiple seizures during the immediate postpartum period. She underwent magnetic resonance imaging (MRI) at a local hospital, which showed multiple ill-defined T2 hyperintensities in bilateral gangliocapsular regions (L>R) and in bilateral cerebral hemisphere white matter [Figure 1]. The MRI pattern was suggestive of PRES, for which she was treated conservatively in the local hospital. During the 14th postpartum day, she developed sudden onset left hemiplegia with reduced consciousness, suggestive of right middle cerebral artery (MCA) territory stroke. She was referred to our hospital, and was evaluated with a repeat MRI. MRI revealed a right MCA territory large, acute infarct [Figures 2 a-e]. There was minimal residual fluid-attenuated inversion recovery hyperintensity in the left caudate nucleus [Figure 2a arrow], corresponding to the previous findings of PRES, and complete resolution of rest of the gangliocapsular region and white matter hyperintensities. Time-of-flight (TOF) MR angiogram (MRA) showed occlusion of the right internal carotid artery (ICA) and MCA[Figure 2f]. Mild short segment stenosis (<50%) was seen at the origin of the left anterosuperior division of MCA. Double lumen was noted in the left distal petrous ICA [Figure 2f, arrow] with a dissection flap in-between. Axial T1-weighted image showed a hyperintensity within the false lumen of the dissected segment of left petrous ICA [Figure 2g, arrow] with reduced calibre of normal flow void. Time of flight (TOF) MRA source images [Figure 2h] showed a T1 hyperintense signal in the right petrous ICA wall (long arrow).

Her vasculitis workup (antinuclear antibody, Anti-double stranded DNA antibody, phospholipid antibody-IgG and IgM) was negative. Serum electrolytes, liver function tests, renal function tests, random glucose test, bleeding parameters, and the platelet count were normal. Her total count was elevated, with predominant neutrophilia. Her erythrocyte sedimentation rate (ESR) was 77 mm/h. Human immunodeficiency virus, hepatitis B surface antigen, and venereal disease research laboratory tests were negative.

The postpartum vasculopathy represents a group of disorders consisting of pre-eclampsia/eclampsia, PRES, and reversible cerebral vasoconstriction syndrome (RCVS). Postpartum coronary and cervicocephalic dissection also appear to be a part of the spectrum of postpartum angiopathy/vasculopathy.[1] The coexistence of PRES/RCVS with cervicocephalic dissections favors this hypothesis. While multiple gene mutations are known to predispose to large artery (aorta) dissections,[2] apart from collagen vascular disorders such as Ehlers–Danlos and Marfan syndrome, no gene mutations predisposing to coronary cervicocephalic artery dissections are identified. As
possible genetic predisposition is more likely in postpartum vasculopathy, genetic studies in such cases are required for better understanding of the pathophysiology and treatment. A single case report of a four vessel cervicocephalic artery dissection in association with haemolysis with elevated liver enzymes and low platelet count (HELLP; hemolysis, elevated liver enzymes, low platelet count) syndrome has been described,[3] where a possible inadequate immune response was hypothesised as the possible predisposing cause. However, this theory was not supported by other case reports or case series. The head and neck manipulation as an etiology for the occurrence of arterial dissection is unlikely in our case because the dissections were in the petrous ICA segment, which is the most immovable portion of ICA.

There is no predisposed segment of vertebrobasilar and carotid arteries that undergoes frequent dissections. In our case, bilateral petrous ICA segment was involved, whereas in the literature, involvement of different segments of the intra- and extracranial portions have been described. A few cases associated with reversible cerebral vasoconstriction syndrome have also been reported.[4,5] An association between PRES and cervicocephalic artery dissection is extremely rare, with only three cases being reported in the literature till date.[4,6] We report the fourth case of cervicocephalic artery dissection following PRES.

In our case, the patient had eclampsia prior to delivery, developed PRES during the immediate postpartum period, as well as bilateral carotid artery dissections. The carotid wall intramural haemorrhage (dissection) was showing T1/

Figure 2: MRI after stroke onset. (a, b) Axial FLAIR and (c) Axial T2-weighted MRI showing residual left caudate nucleus hyperintensity (arrow) and resolution of rest of the previously noted abnormalities. (d) DWI and (e) ADC images showing a large right MCA territory acute infarct. (f) TOF MR angiogram showing occlusion of the right ICA and MCA, and the dissection flap in the left petrous ICA (arrow). (g) Axial T1-weighted image showing a hyperintense blood signal in the left petrous ICA wall (arrow). (h) Source images of TOF MRA showing the hyperintense intramural blood in the right petrous ICA wall (long arrow), and a double lumen in the left petrous ICA (short arrow)

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Meningitis retention syndrome: An unusual complication of viral meningitis

Sir,

Meningitis retention syndrome (MRS) is a very rare syndrome mainly reported to be associated with herpes simplex virus type 2 (HSV-2) meningitis. The development of urinary retention in the context of meningitis and cerebrospinal fluid (CSF) pleocytosis, without any lumbosacral radiculomyelitis is known as meningitis retention syndrome (MRS).[1] Only a few reports of MRS have been reported in literature.[1‑6] A few cases have, however, been seen with meningitis caused by West Nile and HSV-1 virus meningitis, as well as with listeria and tuberculous meningitis. We report a case of MRS associated with HSV-1 meningitis.

A 20-year-old, unmarried male presented to the emergency department with complaints of severe throbbing headache, neck stiffness, and vomiting for 6 days. He had taken multiple analgesics for his headache with no relief. On examination, he was febrile with neck stiffness. Kernig's sign was positive, but mentation was normal. Fundus examination and non-contrast computed tomography head was normal. CSF analysis showed a white blood cell count of 180 cells per high power field, predominantly lymphocytes, with the glucose level being 44 mg/dl and the protein level being 75 mg/dl. The Gram's stain was negative. He was managed symptomatically for viral meningitis.

On the 4th day of admission, he developed urinary retention and was catheterized. Detailed examination revealed normal power and sensation in the lower extremities. Deep tendon reflexes were normal. Also, perianal sensation and anal tone were normal. He did not have genital lesions or a history of sexual contact. The bladder scan revealed a postvoid urine volume of 450 ml. Brain contrast magnetic resonance imaging (MRI) showed mild meningeal enhancement [Figure 1], and spinal contrast MRI did not reveal any features suggestive of myelitis or radiculitis.

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References


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