Case Report: Obstetrics

Seizures in the Early Postpartum Period: A Rare Case of Spontaneous Cryptogenic Subarachnoid Hemorrhage Posing Diagnostic Dilemma

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Abstract

Background: Seizures in the early postpartum period can have several differentials including eclampsia and cerebral venous thrombosis. Rare causes of seizures like spontaneous subarachnoid hemorrhage can create diagnostic dilemma in some cases. Though these cases are rare, yet those constitute the highly morbid complications of pregnancy and a detailed investigation is mandatory in all cases of postpartum seizures to come to a definitive diagnosis. In this article we present the case of a young primigravida who presented on the 10th puerperal day with headache and episodes of generalized seizures. Radiological evaluation revealed acute subarachnoid hemorrhage involving the right sylvian fissure and basal cisterns extending into the anterior interhemispheric fissure. The absence of any aneurysms or vascular malformations on catheter angiogram along with negative coagulation screen led us to the diagnosis of cryptogenic subarachnoid hemorrhage as the cause of seizure. The lady responded to conservative management and is now on follow up without any recurrence of symptoms

Keywords: Seizure, postpartum, subarachnoid hemorrhage, cryptogenic.

Introduction:

Though eclampsia remains the most common cause of seizures in the puerperal period, there can be other causes of postpartum seizures as well. Different cerebrovascular incidents and intracranial infections can give rise to similar conditions. As there is significant overlapping of the clinical features of the varied causes of postpartum seizures, a meticulous approach is often warranted for proper management of these conditions. We hereby present a case of spontaneous non aneurysmal subarachnoid hemorrhage (SAH), which is a rare cause of postpartum seizures.

Case Report:

A 33-year-old lady presented on the 10th day of puerperium with complaints of severe headache for last two days and two episodes of generalized tonic clonic seizures (GTCS). Patient had an elective caesarean

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section done at 39 weeks of gestation for maternal request under spinal anesthesia. The intraoperative and post-operative period was uneventful. She was discharged from the hospital on fourth postpartum day in satisfactory condition. This lady was a booked case in her first pregnancy. Antenatal period was uneventful. There were no complaints of headache, unconsciousness or visual disturbances during the antenatal period. There was no history of seizures in the past. Patient was normotensive throughout the antenatal period. On the 8th postpartum day, she started having severe headache which was persistent for the next two days. On the 10th postpartum day, she had one episode of GTCS which started insidiously, without any warning signs. She was unconscious during the event and could not recall the incident later. There was a brief period of post-ictal confusion. Following this event, she was brought to the hospital, where she had another similar event of GTCS. On examination, she was afebrile, pulse rate was 92 beats/minute and blood pressure (BP) 180/110 mm Hg (mercury). She was conscious, alert and obeying commands. There was no speech or visual problems or weakness or numbness in any parts of the body. Deep tendon reflexes were maintained and plantar response was bilaterally down going. Pupillary response was normal and fundoscopic evaluation was unremarkable. There was no neck pain or neck rigidity. Initial Computed Tomography (CT scan) of brain revealed acute SAH in the right sylvian fissure extending to the basal cisterns as well as the anterior interhemispheric fissure (Fig. 1). There were some evidences of convexity SAH also. There was no ventriculomegaly or blood inside the ventricle. The brain parenchyma was normal. Urine examination did not reveal any proteinuria. Following this she was started on nimodipine and levetiracetam. Complete hemogram, liver and renal function tests were normal. A formal Digital Subtraction Angiography (DSA) of the cerebral vessels was done. There was no evidence of any aneurysm or arterio-venous malformations (AVM) (Fig. 2). Coagulation studies including protein C, protein S, antithrombin III and Anticardiolipin antibody IgG, IgM were normal. No Lupus anticoagulant was detected. There were no further episodes of seizure during the hospital stay. The patient was closely monitored and discharged on day 6 of admission following substantial improvement of symptoms. On follow up at two weeks of admission, there was complete subsidence of headache with no further episodes of seizures. Follow up CT scan of brain done after three weeks showed complete resorption of SAH and no increase in the ventricular size. Repeat angiogram was advised but the patient denied any further investigations. The patient is now been followed up for two years without any further related complaints.

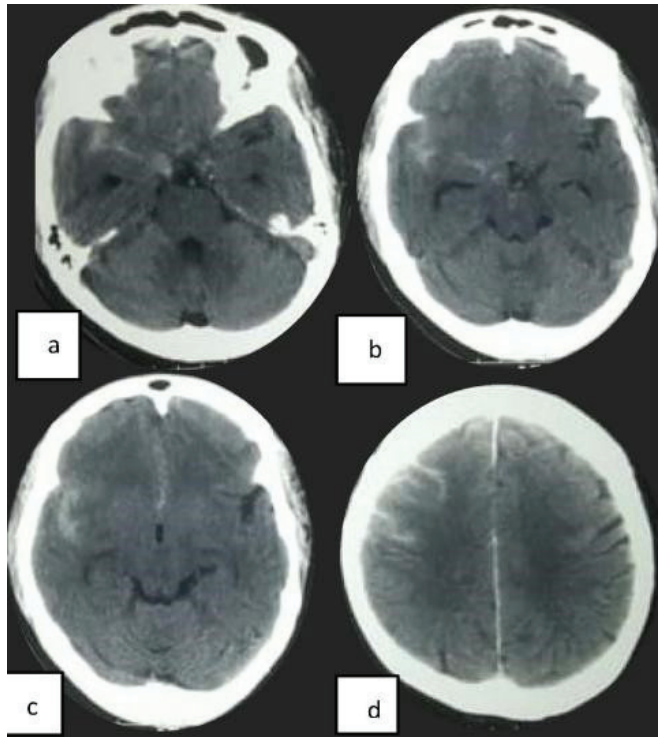

Fig. 1 CT scan of brain done on the day of admission. a. acute subarachnoid hemorrhage involving the right sylvian fissure. b. extension of acute subarachnoid hemorrhage in the basal cistern. c. extension of acute subarachnoid hemorrhage in the anterior inter hemispheric fissure. d. acute subarachnoid hemorrhage involving the convexity of right frontal lobe.

Discussion

Episodes of seizures in the early postpartum period raise the possibility of eclampsia in the first instance. Eclampsia is defined as a condition in which episodes of convulsions and/or coma occurs during pregnancy or early postpartum period in patients known to have signs or symptoms of preeclampsia. There are no clinical symptoms or laboratory tests to predict or diagnose eclamptic seizures, except for early detection of preeclampsia. However, patients presenting with eclampsia can have a spectrum of presentation starting from severe hypertension and proteinuria to mild or absent hypertension with no proteinuria.[1] It is also not unusual for seizures to precede overt hypertension or proteinuria in patients of eclampsia.

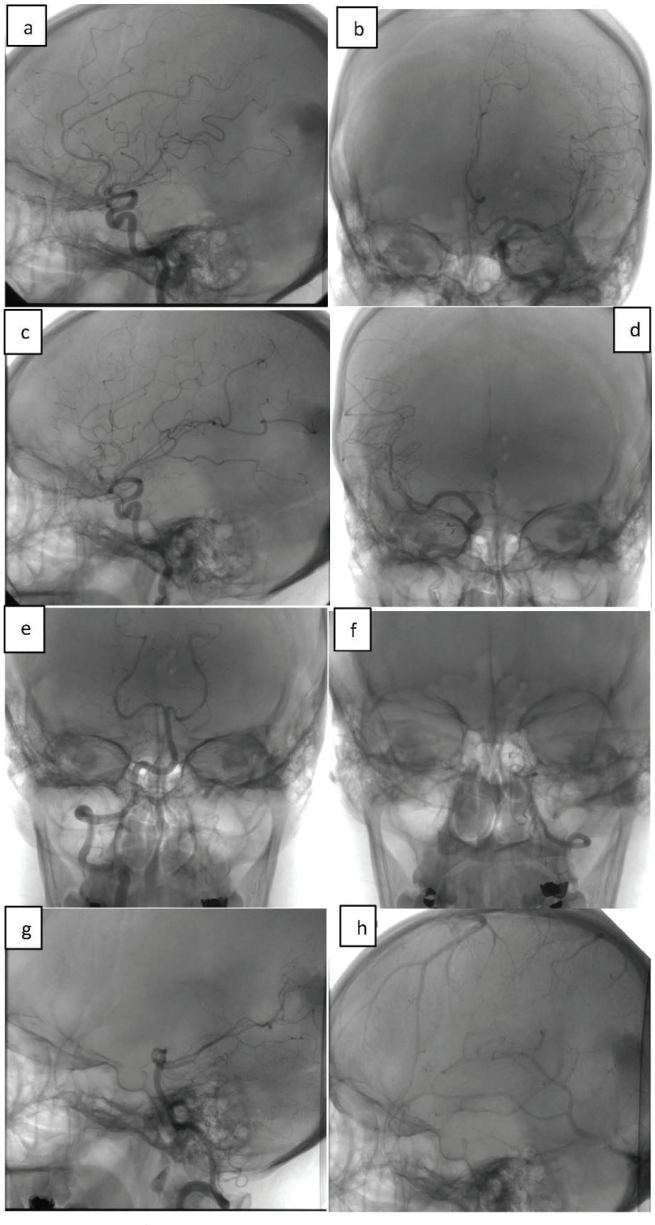

Fig. 2 DSA of cerebral vessels reveals normal vasculature pattern. a and b. Left internal carotid artery, anterior communicating artery, left anterior and middle cerebral artery shows normal course, caliber and branching pattern. c and d. Right internal carotid artery, right anterior and middle cerebral artery shows normal course, caliber and branching pattern. e. Right vertebral artery is dominant. f. Left vertebral artery is non dominant. g. Vertebro basilar arterial circulation shows usual branching pattern, basilar tip is clear. h. Venous phase shows normal drainage pattern

So, in patients presenting with postpartum seizures in the absence of proteinuria or history or persistent hypertension antenatally or in the postpartum period, consideration of eclampsia can not be ruled out definitively. Cerebral imaging findings in eclampsia are similar to those found in patients with hypertensive encephalopathy and includes edema or infarction in the subcortical white matter and adjacent grey matter, mainly in the parieto-occipital region as well as occasional intraparenchymal hemorrhages.[2]

There is significant overlapping of symptoms with some of the close differential diagnoses which poses diagnostic dilemma in such cases. Headache and convulsions in the postpartum period raise the suspicion of cerebral venous thrombosis (CVT) which is a common cause of maternal stroke. In some recent studies, the incidence of maternal strokes has been estimated to be approximately 30 in 100,000 pregnancies and 7.4% of maternal morbidity has been accounted to this condition.[3,4] The incidence of CVT in Indian population is higher as compared to literatures from developed countries.[5] Clinical features of CVT are related to either raised intracranial pressure due to impaired venous drainage or focal brain injury from venous infarction or hemorrhage. Most patients present with new onset stroke like symptoms including headache, cranial nerve involvements, seizures, altered sensorium and focal neurological deficits.[6] Radiological findings of CVT are very similar to that of eclampsia and include intraparenchymal hemorrhage and SAH as well. Only finding of thrombus on venogram gives a direct clue to the diagnosis, though small thrombi are difficult to pick up on conventional imaging.[5]

The severity of headache and absence of any focal neurological signs or cranial nerve involvement in our case was not congruent with the common features of CVT or cerebral infarcts. This led us to arrange for an early CT scan of brain. The CT scan findings of SAH involving the sylvian fissure, basal cisterns and the anterior interhemispheric fissure are more in favor of bleeding from large vessels i.e., aneurysms or AVM. Incidence of SAH is documented to be five times higher during pregnancy as compared to the nonpregnant state in some previous studies.[7] The most common cause of nontraumatic SAH in pregnancy and puerperium remains to be ruptured aneurysm.[8] Recent studies do not show any increased incidence of bleeding from unruptured cerebral aneurysm but contradictory evidence exists regarding bleeding risks of cerebral AVMs.[9,10,11] In our patient no obvious vascular abnormality was noted on DSA. It also excluded the diagnosis of CVT in this case. Although hemorrhagic complications are not uncommon in CVT with some studies reporting about one third of patients presenting with intracerebral hemorrhage

or hemorrhagic venous infarcts, isolated SAH is rare and reported to be 0.8% in one international series.[12] Patients of PIH can also present with SAH, but in most of the cases the bleeding is restricted to the cerebral convexities. There is one case series of SAH in PIH where the convexity bleed has extended to the sylvian fissure or interhemispheric fissure in one of the three cases described.[13]

For further evaluation of the cause of SAH, a detailed coagulation screening was done which also came out to be negative. Absence of any features of infection including fever or neck rigidity did not prompted us to do further investigations to rule out meningitis as a cause of seizures. Following this we came to the conclusion that the cause of seizures in our patient was cryptogenic SAH, which is a rare cause of seizures in pregnancy or postpartum period. In about 15% of patients undergoing DSA for spontaneous SAH in general population, no obvious vascular abnormalities were detected in different case series and are described as cryptogenic subarachnoid hemorrhage.[14] On detailed analysis of the pattern of SAH on CT scan images, two main subgroups have been described. van Gijn et all. described a subset of patients of spontaneous cryptogenic SAH who were in good clinical condition and fulfilled the radiological criteria of epicenter of the hemorrhage immediately anterior to the mesencephalon without extension of bleed to the anterior aspect of the interhemispheric fissure or lateral aspect of the sylvian fissures and absence of intraparenchymal bleed or intraventricular clot. Small volume of blood is accepted in dependent portions of the occipital horns.[15] This subgroup was described as perimesencephalic SAH and consisted of majority of the cryptogenic SAH patients. Those patients were found to have a benign course with good prognosis and low rebleeding and vasospasm rates as compared to aneurysmal SAH.[16] Based on the low rebleeding rates and given the risk associated with further angiographic studies being more as compared to finding aneurysms as the cause of bleeding, no further investigations were warranted in this subset of patients in most of the studies.[16,17] In contrast, the subset of patients of cryptogenic SAH not fulfilling the above criteria were designated as nonperimesencephalic SAH and were found to have a clinical outcome, rebleeding and vasospasm rates similar to aneurysmal SAH. The source of SAH in perimesencephalic SAH subset of patients was depicted to be venous in origin in majority of the cases.[15] Venous hypertension has been postulated to be the cause of bleeding in several case studies. It has been reported in some cases after physical exertion and the pathophysiology has been described as increased intrathoracic pressure leading to impaired jugular venous return and elevated intracranial venous pressure leading to venous bleeding.[18] Two cases of perimesencephalic SAH has been reported till date during pregnancy and only one case in the postpartum period, all presenting with severe headache, nausea, vomiting or photophobia.[19,20] To the best of our knowledge, no cases of cryptogenic SAH have been reported in the pregnancy or postpartum period presenting with seizures. We hereby report the only case of postpartum seizure due to cryptogenic SAH with a follow up of two years without recurrence of any symptoms including seizures.

Conclusion:

The differential diagnoses of seizures in pregnancy and postpartum period are varied and present with closely overlapping symptoms. Rare causes of seizures like non aneurysmal SAH can create diagnostic dilemma in some cases. Though these cases are rare, yet those constitute the highly morbid complications of pregnancy. Neurosurgical intervention may be warranted frequently in some of the cases including SAH due to ruptured cerebral aneurysm or AVM and a multidisciplinary approach should always be adopted for proper management of such cases.

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