

Case Report

Postpartum cerebral vein thrombosis

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ABSTRACT

Pregnancy increases the risk of cerebrovascular accidents to approximately 13 times the expected rate. Most of the cerebrovascular accidents are due to cerebral venous thrombosis. Headache, seizures and focal neurological deficits are some of the symptoms of cerebral venous thrombosis. Headache can also be due to postural puncture, pre eclampsia and eclampsia in the postpartum period. Neuroimaging with MRI with contrast helps in the diagnosis of cerebral venous thrombosis and giving a definite diagnosis, which aids in the treatment and prognosis. The cases presented in this report highlight the importance of investigations such as MRI or CT in the diagnosis of cerebral venous thrombosis in pregnancy and puerperium.

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Convulsions in a pregnant woman can be due to many causes. Eclampsia is a clinical diagnosis if there is hypertension and proteinuria. If the classic features of eclampsia are not present an attempt must be made to diagnose the etiology of the convulsions, preferably by imaging. An MRI or CT will help in the diagnosis and treatment of the present pregnancy/puerperium and also future pregnancies. The cases presented in this report highlight the importance of investigations such as MRI or CT in the diagnosis of cerebral venous thrombosis in pregnancy and puerperium.

Case Report. Patient One. A 27-year-old primigravida at 39+ weeks gestation was admitted to the delivery ward of Sultan Qaboos University Hospital, Sultanate of Oman with premature rupture of membranes of 12 hours duration. She was diagnosed to have breech presentation with a small pelvis (by pelvimetry). She had an emergency cesarean section under general anesthesia after a failed attempt at spinal anesthesia. A baby boy weighing 2800 grams was delivered as breech with an apgar score of 5 at one minute and 9 at 5 minutes. Estimated blood loss during the cesarean

was 500 ml. There were no intra-operative surgical or anesthesia problems. During this pregnancy, she was booked for antenatal care at 10 weeks of gestation. Her blood pressure (BP) at booking was 100/70 mm Hg and weight was 58 kg. She had regular antenatal visits with normal BP, weight gain and normal blood sugar. Her weight gain throughout the pregnancy was 11 kg and the BP on admission was 132/77 mm Hg. Her menstrual cycles were regular and there was no significant past medical or surgical illness. There was no family history of hypertension. She was a schoolteacher. At approximately 48 hours after surgery she started complaining of headache, mainly in the occipital area and when sitting up. This was thought to be spinal headache and was treated with regular analgesics. Four days after surgery she developed severe headache; BP increased to 160/90 mm Hg and she had a tonic clonic seizure lasting for 2 minutes. After the seizure, the patient became aggressive, frightened and screaming, which was controlled with an injection of Diazepam. She had normal reflexes, no protein in urine and investigations showed normal liver function, renal function, serum urate, coagulation screen and

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platelet count. As the blood pressure was rising to 180/105 mm Hg she was started on magnesium sulphate infusion. Epidural blood patch was considered after stabilizing the patient. A repeat neurological examination revealed mild hyper-reflexia, terminal neck stiffness and flexor plantar reflexes. An MRI and magnetic resonance venogram confirmed postpartum cerebral cortical vein thrombosis (**Figure 1**). An EEG showed normal response. She was started on low molecular weight heparin and oral phenytoin sodium. As she remained hypertensive, lisinopril 5mg (ACE inhibitor) was added. Her headache subsided gradually, and a thrombophilia screen was performed, which showed a normal antinuclear antibody (ANA), and anticardiolipin antibody (ACA). Lupus anticoagulant was negative, and Factor VIII C level was normal. On follow up 8 weeks after surgery she was clinically well, lactating normally and off all medications.

Patient 2. A 21-year-old primigravida who delivered a baby girl of 1860 grams spontaneously at 35 weeks gestation in a nearby hospital. Her antenatal period was uneventful except for mild thrombocytopenia noted in pregnancy, but she did not receive any treatment for this. She was treated for postpartum fever soon after delivery with antibiotics for presumed urinary tract infection but there was no proven bacterial culture and was discharged home. She was referred 5 days after discharge with history of loss of consciousness and generalized tonic clonic fits 17 days after delivery. There was no past history of epilepsy. Clinical examination at the time of presentation with fits revealed a disoriented woman, irritable with neck stiffness. The temperature was 38°C. There were no cerebellar signs, no focal neurological signs, and no papilledema. Deep tendon reflexes were elicitable.

Plantar reflexes were normal. Examination of the abdomen and pelvis was normal. A CT of the brain was carried out, and results showed a hyperdense area in the left temporal region. She was referred to this hospital for an MRI study, which confirmed left sigmoid sinus thrombosis (**Figure 2**). Further blood investigations revealed normal liver and renal function tests, no bacterial growth in the blood and no malarial parasite in the peripheral smear. Antinuclear antibody, anti DNA antibody and rheumatoid factor were negative. Her B2 glycoprotein immunoglobulin G (IgG) 1 was 0.6 U/ml, B2 glycoprotein 1 IgM 0.8 U/ml, ACA IgG 1.0 U/ml and ACA IgM 21.7 U/ml (normal <10 U/ml). Lupus anticoagulant was not carried out. Protein C and protein S levels and factor V Leiden mutation were not estimated. Antiphospholipid syndrome was diagnosed, and she was advised low molecular weight heparin followed by warfarin. She was followed up one year later and remains symptom free on aspirin and warfarin.

Discussion. Thrombosis of the cerebral veins and venous sinuses is an uncommon condition. The estimated risk is 11.6 cases of peripartum intracranial venous thrombosis per 100,000 deliveries in the United States of America (USA).¹ It is a potentially serious disorder, in which there is severe intracranial hypertension, which may be fatal or cause serious neurological sequelae. Diagnosis is made, during life, on the clinical condition and neuro-imaging features. Sanchette et al² described clinical and computed tomography features of 25 patients with peripartum cerebral venous thrombosis. Most of the patients presented in the postpartum period and did not receive proper antenatal care. Headache (92%), altered sensorium (80%), seizures (76%), papilledema (80%) and

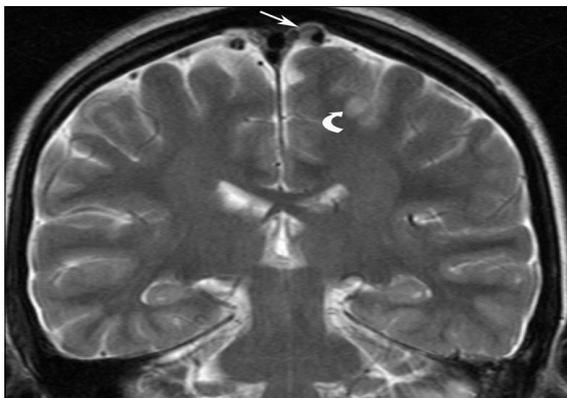


Figure 1 - Magnetic resonance imaging of the brain, coronal T2W image (TR/TE 4500/105 ms) showing thrombosis of a draining cortical vein (arrow) proximal to its junction with the superior sagittal sinus. Edema of the superior parietal gyrus in the drainage area of the thrombosed vein is shown (curved arrow).

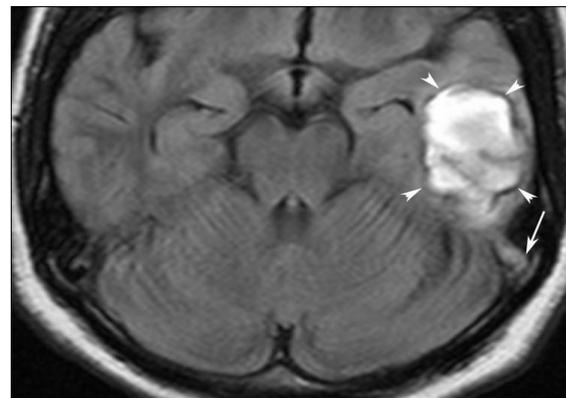


Figure 2 - Magnetic resonance imaging of brain, axial FLAIR image (TR/TE/IR 9000/105/2500 ms) showing a large subacute hematoma in the left temporal lobe (arrowheads). Left sigmoid sinus is thrombosed (arrow).

hemiplegia (52%) were the common modes of presentation. Lanska and Kryscio¹ studied the risk factors for peripartum stroke and intracranial venous thrombosis in 17 states in the USA in 1993-1994, and concluded that pregnancy related hypertension and cesarean delivery are important risk factors for both stroke and intracranial venous thrombosis. In a prospective study, 64 cases of cerebral venous thrombosis in pregnancy and puerperium were evaluated by Panagariya and Maru.³ Most were below 25 years of age and 79.6% occurred in the first 2 weeks after delivery. The authors excluded patients with pre eclampsia and eclampsia but included patients with CT scan suggestive of venous thrombosis. Headache in the postpartum period can be a manifestation of a variety of diseases including post dural puncture headache, cerebral venous thrombosis, eclampsia, and so forth. In 2 different case reports, headache, apparently thought to be due to epidural analgesia, were later proven to be due to cerebral venous thrombosis.^{4,5}

A female patient with antiphospholipid syndrome may present for the first time in her life with cerebral venous thrombosis in pregnancy and puerperium.⁶ Deischens et al⁷ studied coagulation factors, factor V Leiden and ACA in 40 cases of cerebral venous thrombosis, and found 15% congenital thrombophilia, and 8% had increased ACA.

Clinical suspicion along with neuroimaging is crucial in making a diagnosis of cerebral venous thrombosis.⁸ Andrade-Machado et al⁹ described 5 cases of non-infectious thrombosis of the cerebral venous sinuses and veins in adults, and puerperium was the most common risk factor with headache and neurological focal signs as the presenting complaints.⁹

The first case presented here has a combination of risk factors with postpartum hypertension, cesarean delivery, and a failed attempt at spinal analgesia. The headache was initially thought to be due to postdural puncture but then MRI revealed cortical

venous thrombosis, and she was treated with heparin. An MRI was crucial in making the diagnosis. She recovered completely a few days after starting heparin therapy. The second patient was diagnosed with antiphospholipid syndrome and started on aspirin and warfarin.

A thorough investigation of patients with postpartum headache and convulsions is indicated to diagnose the etiology and appropriate therapy for further prevention.

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