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CEREBRAL VENOUS THROMBOSIS PRESENTING AS SUBARACHNOID HAEMORRHAGE

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ABSTRACT

We present a 31 years old lady who was 19 days postpartum who was admitted with fits and loss of consciousness. At presentation Glasgow Coma Scale was 8/15. No focal deficit. CT scan brain showed findings suggestive of subarachnoid haemorrhage (SAH) in left sylvian fissure along with few parenchymal haemorrhagic areas in frontal and parietal lobes. MRI brain and MRV confirmed superior sagittal sinus thrombosis, inferior sagittal, straight and right transverse and sigmoid sinus thrombosis. Patient was treated conservatively and was anticoagulated for 6 months and had a complete recovery. Further workup of thrombophilia was negative. Postpartum being the only risk factor for cerebral venous thrombosis (CVT) in her. SAH can be a rare presenting symptom of CVT and should not preclude patients from receiving anticoagulation.

KEY WORDS:

Cerebral venous thrombosis, Subarachnoid hemorrhage, Thrombophilia

INTRODUCTION:

CVT is responsible for 1% to 2% of all strokes in adults.¹ CVT presenting with an associated subarachnoid hemorrhage (SAH) has been rarely reported in literature.^{2,3}

We present a similar case of postpartum CVT who presented with a sylvian SAH and was treated with anticoagulation and fully recovered.

Case report

A 31-year old female resident of Islamabad presented with one-day history of headache, fits associated with loss of consciousness. She had a history of child birth about 19 days ago, which was normal delivery without any complications. On examination at presentation she had stable vital signs. Her Glasgow Coma Scale was however 8/15. Pupils were equal and reactive. There were no signs of meningeal irritation. There was no focal deficit. Her CT scan showed left sylvian fissure subarachnoid haemorrhage as well as findings suggestive of superior sagittal sinus and right

transverse sinus thrombosis as shown in figure 1 and 2. Next important question was whether to start her on anticoagulation or not in presence of subarachnoid haemorrhage. She was started on LMWH in therapeutic dose and bleeding was monitored.

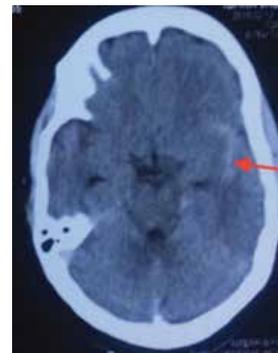


Figure 1. CT brain (plain) of the patient showing hyperdense signal in left sylvian fissure suggestive of subarachnoid haemorrhage.

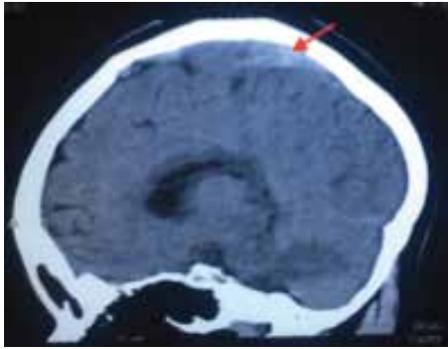


Figure2. CT brain (plain) sagittal view showing hyperdense superior sagittal sinus suggestive of superior sagittal thrombosis.

Her MRI and MRV brain confirmed superior sagittal sinus thrombosis, inferior sagittal, straight and right transverse and sigmoid sinus thrombosis. Her thrombophilia workup was also done and was found negative. Fortunately, she improved really quickly and had no worsening of haemorrhage. Her CT angiography brain was also done and ruled out any intracranial aneurysms. She was given anticoagulation for 6 months and had full resolution of symptoms.

Discussion

SAH can be a rare presentation of CVT. The distribution of SAH associated with CVT is usually different from that of SAH of arterial origin, which has a characteristic pattern. In fact, when SAH is localized at the cerebral convexity and spares the basal cisterns and skull base, CVT should be considered.⁴

The exact mechanism of cortical SAH caused by CVT is unknown. One possibility is the rupture of venous parenchymal hemorrhagic infarcts into the subarachnoid space.^{5,6} Another possible mechanism is venous hypertension and subsequent rupture of dilated, valveless, thin-walled, bridging subarachnoid cortical veins devoid of smooth muscle fibers.⁵

A third mechanism of SAH development could be a local inflammatory response caused by CVT, which would increase the vascular permeability allowing for extravasation of blood into the subarachnoid space⁶.

Management of SAH secondary to CVT is quite different from that of arterial SAH. The usual treatment of sinus thrombosis is anticoagulation. Systemic anticoagulation is the first line treatment for CVT

because of its efficacy, safety, and feasibility.⁷ In a further placebo-controlled trial, 60 patients were randomized to either lower molecular weight heparin followed by warfarin or placebo.⁸ The anticoagulated patients had better outcomes than controls, but the difference was not statistically significant. The investigators suggested that anticoagulation with LMWH was safe, even in patients with cerebral hemorrhage.⁸

CVT should be considered in the differential diagnosis of patients presenting with SAH without evidence of aneurysm and presence of SAH should not preclude patients from receiving anticoagulation.

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Farheen Niazi; concept, data collection, data analysis, manuscript writing, manuscript review