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SUCCESSFUL OUTCOME OF ANTEPARTUM CEREBRAL VENOUS THROMBOSIS FOLLOWING DECOMPRESSIVE CRANIECTOMY

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INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is uncommon as compared to arterial strokes. Risk factors for CVST include trauma, infections, inherited and acquired prothrombotic states, hormonal preparations, neoplasms, dehydration, caesarian section, pregnancy and postpartum period. Pregnancy related CVST is reported in up to 17% of CVST[1] accounting for 2% of pregnancy related strokes.[2] Home delivery, poor peripartum care, dehydration, anemia and infections are additional risk factors in resource-limited countries. Physiological changes during pregnancy including raised coagulation factors, reduced levels of protein S, acquired protein C resistance, increased oestrogen and progesterone levels predispose for CVST.[3]

CVST patients present with features of raised intracranial tension, seizures, focal neurological deficits and altered consciousness.[4] Large haemorrhagic infarctions may occur due to venous obstruction, raised intradural and venous pressure resulting in rupture of venules. Such 'malignant' infarctions result in brain herniation, increased morbidity and mortality and benefit from timely emergency decompressive craniectomy.[5]

CASE REPORT

A 31-year primigravida with 8 weeks gestation presented in February 2024 with progressively increasing holocranial headache of two weeks and vomiting for three days. Headache was intermittent initially and had become continuous for a week. For two days before admission, she had rapidly evolving slurring of speech with mild weakness of right-side limbs. On the day of admission, she developed drooping of left upper eyelid, became dull and could not speak. She had no seizures, diplopia, ataxia or behavioural changes. She had attained menarche at 13 years, normal menstrual cycles and married one year ago. No past history of hypertension, arthritis, rash, systemic symptoms, stroke, venous thrombosis or limb ischemia.

Blood pressure was 120/70mmHg with regular pulse at 80/minute. General physical, cardia, chest and abdomen examinations were normal. She was awake with Glasgow coma score of 11 (E4, V2, M5). Speech was dysarthric with nonfluent aphasia and markedly impaired verbal comprehension. She had bilateral grade 2 papilledema and left partial third nerve palsy with pupillary involvement. Moderate right upper motor neuron facial weakness was present with right hemiparesis (3/5 proximal, 2/5 distal). Plantar response was extensorbilaterally and muscle stretch reflexes were brisk on both sides. Hemogram, biochemistry including homocysteine level and serology were normal.

In view of clinical features, possibility of antepartum cerebral venous thrombosis versus left supratentorial structural lesion with transtentorial herniation was considered. Emergency cranial magnetic resonance imaging revealed large left temporoparietal haemorrhagic infarction (volume 185ml), features of transtentorial herniation with medial temporal lobe compressing, shifting and distorting the midbrain. (Figure 1) MR venography and angiography showed thrombosis of left transverse sinus, sigmoid sinus and internal jugular vein without arterial occlusions confirming the diagnosis of antepartum CSVT with haemorrhagic infarction.(Figure 2)

Urgent neurosurgery consultation was obtained for decompressive craniectomy for the 'malignant' venous infarction with transtentorial herniation as lifesaving procedure. The emergency decompressive craniectomy was completed within 3.5 hours of admission.

Postoperatively, she was treated with antibiotics, parenteral antiedema therapy and prophylactic antiseizure medication. Cranial imaging two days after surgery showed mild increase in infarction size (200ml) without increase in haemorrhage following which she was started on subcutaneous enoxaparin (40mg twice a day) for the CVST. She was weaned off from mechanical ventilation and was extubated seven days after surgery. She had worsening of the right hemiplegia (0/5 upper limb, 2/5 lower limb) in the first two weeks after surgery and subsequently there was gradual improvement in the hemiplegia and aphasia. Obstetrician regularly monitored the patient and fetal status from the admission time till discharge. As the patient's relatives wanted to continue pregnancy, enoxaparin injections were continued.

Thrombotic profile including anticardiolipin antibodies were normal. At discharge on 25th day of hospitalization she was awake, alert with partial comprehension, minimal word output, dysarthria, right hemiplegia (upper limb 3/5, lower limb 4) with modified Rankin score of 4.

She was on regular antenatal follow-up with continuation of enoxaparin along with folic acid, iron and calcium supplementation. Serial cerebral imaging revealed gradual subsidence of the hemorrhagic infarction volume. (Figure 1) Obstetric ultrasound examination revealed normal growth, liquor quantity, doppler parameters without anomalies. She was admitted in August 2024 at 35th week of gestation and underwent emergency lower segment cesarian section (LSCS) under general anesthesia after stopping the enoxaparin injections for two days. Live female baby of 2.28kg was extracted and the baby criedimmediately with 1 minute APGAR score of 8/10 and 5 minutes score of 10/10. There was no significant blood loss and patient did not require blood transfusion. At discharge after LSCS, enoxaparin was switched over to warfarin with continuation of levetiracetam. During the last follow-up in March 2024, she was awake, alert with incomplete global aphasia, upper limb 4/5, lower limb 5/5 with mRS of 2. Warfarin was stopped for planned cranioplasty.

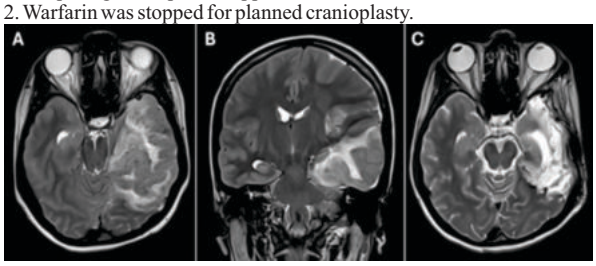

Figure 1 showing the T2 weighted magnetic resonance images of the patient. Axial (A) and coronal (B) were obtained at admission demonstrating the large left temporal and parietal haemorrhagic infarction with midline shift, medial temporal lobe herniating and compressing and markedly distorting the midbrain. Optic nerve is wavy tortuous and the perioptic cerebrospinal fluid space is enlarged.

Image C was obtained 4 months later during follow-up and reveals gliosis with resolution of haemorrhagic infarction. Midbrain morphology and position have become normal with opening up of peri-mesencephalic subarachnoid space.

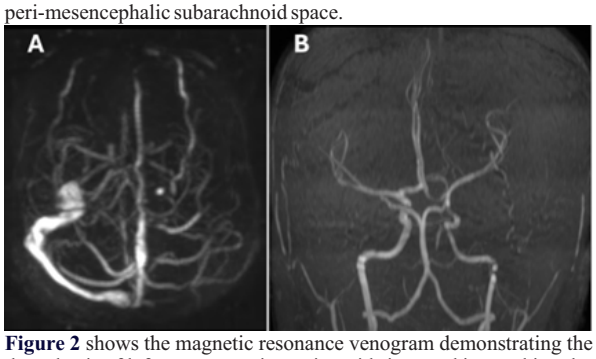

Figure 2 shows the magnetic resonance venogram demonstrating the thrombosis of left transverse sinus, sigmoid sinus and internal jugular vein. Time of flight magnetic resonance imaging reveals patent intracranial arteries. Left middle cerebral artery is elevated due to large temporal lobar infarction.

DISCUSSION

CVST occurring during pregnancy can be life-threatening for the mother and the fetus presenting a therapeutic challenge. Our patient presented with features of CVST during her first trimester without any premorbid known prothrombotic states. She developed large hemorrhagic venous infarction from left transverse and sigmoid sinus occlusion and required emergency decompressive craniectomy as a life-saving measure. DECOMPRESS 2, a study on 118 patients revealed good outcome in about 35.6% with a modified Rankin score of < 2. Mortality was 33.9%, combined death or severe disability was 39% and predictors of poor outcome include coma, evidence of coning in this study.[5]

Anticoagulation of choice during pregnancy is subcutaneous heparin (preferably low molecular weight heparin) which should be continued till delivery and six to eight weeks after delivery.[6],[7] There is a risk of vaginal bleeding with heparin and the incidence was found to be higher in normal pregnancies.[8] Our patient was closely monitored throughout the pregnancy and delivered a normal baby with caesarian section. Although caesarean section is a risk factor for CVST,[9] our patient had uneventful postpartum state for six months. Vitamin K antagonists including warfarin may be used during lactation and DOAC should be avoided.[10]

CVST during pregnancy mandates early diagnosis and prompt institution of parenteral anticoagulation. Decompressive craniectomy is a life-saving measuring for large venous infarctions to reduce mortality and achieve feto-maternal outcome.

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