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RESEARCH ARTICLE

BILATERAL DECOMPRESSIVE CRANIECTOMY AS A LIFE-SAVING PROCEDURE FOR A PATIENT WITH RECURRENT CEREBRAL VENOUS THROMBOSIS WITH GOOD FUNCTIONAL RECOVERY

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ABSTRACT

Cerebral venous sinus thrombosis (CVT) is a potentially curable cause of stroke in young. Though primary pathology is related to tissue damage resulting from the obliteration of venous outflow from functioning brain tissue, secondary damage resulting from mass effect and midline shift is a life-threatening complication wherein Decompressive craniectomy (DC) can be lifesaving. We aim to highlight the indication, appropriate timing and outcome of DC in CVT. Our patient, a middle-aged woman, had recurrent CVT, after an interval of three years, the only cause evident was anaemia secondary to abnormal uterine bleeding (AUB). She needed combined neurological and neurosurgical care during both the episodes and she recovered significantly even after having bilateral lesions and after undergoing decompressive craniectomy twice on separate occasions which is first of its kind in the literature to our knowledge.

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INTRODUCTION

Cerebral venous thrombosis (CVT) is an uncommon cause of stroke seen in young. The majority of patients improve with medical management. Some patients develop mass effect due to haemorrhagic infarct and cerebral edema where decompressive craniectomy (DC) is recommended as a life-saving procedure.[1] The percentage of patients requiring decompression varies from 1.2% to 7.4% and was associated with a good outcome.[1],[2],[3] Recurrent CVT is rare and is documented in around 2.2% to 6% in different studies.[3],[4] Polycythemia, genetic thrombophilia and male sex are the known risk factors for recurrence.[5] According to AHA/ASA 2011 guidelines, the indications for DC in CVT include neurological deterioration due to hemorrhagic infarct causing mass effect or intractable intracranial hypertension or coma in spite of best medical therapy.[6]We report a patient who developed recurrent CVT due to abnormal uterine bleeding (AUB) leading to severe anemia requiring DC on both occasions after an interval of three years due to recurrence of CVT with mass effect. A literature survey revealed no similar published case. Our main aim is to emphasize importance of timely decompression, and pursue an aggressive management strategy, as in the presented case. Patient must be extensively investigated to find out the underlying cause of CVT to avoid recurrence.

CASE REPORT

A 46-year-old female developed insidious onset, continuous throbbing frontal headache for one month in 2017; twenty days after the onset of which, she developed one episode of left focal seizure with secondary generalization. She had history of heavy menstrual bleeding and endometrial thickening sonographically, suggestive of AUB without any other risk factors of CVT. Magnetic Resonance Imaging (MRI) of brain showed right frontal haemorrhagic infarct with thrombosis of anterior and mid portions of the superior sagittal sinus (Figure 1). During admission, she showed deterioration of sensorium and imaging showed worsening of mass effect for which left frontal craniectomy was done. She presented to our hospital following the surgery. A detailed evaluation revealed anaemia (Hemoglobin-9.9 g/dL) with peripheral smear showing microcytic hypochromic picture. Vitamin B12 and folate levels were normal. Evaluation for Homocysteine /Anti Nuclear Anti-body (ANA) profile/protein C/protein S/Rheumatoid Arthritis (RA) Factor/Factor V Leiden mutational analysis was negative. The patient was managed with packed cell transfusions, supportive therapy, anticoagulants, and antiepileptics. She improved clinically and she scored zero on the modified Rankin Scale (mRS) after three months. Oral anticoagulation was stopped after one year as her anemia was corrected (hemoglobin-13.1g/dl). After two years she was lost to follow up.

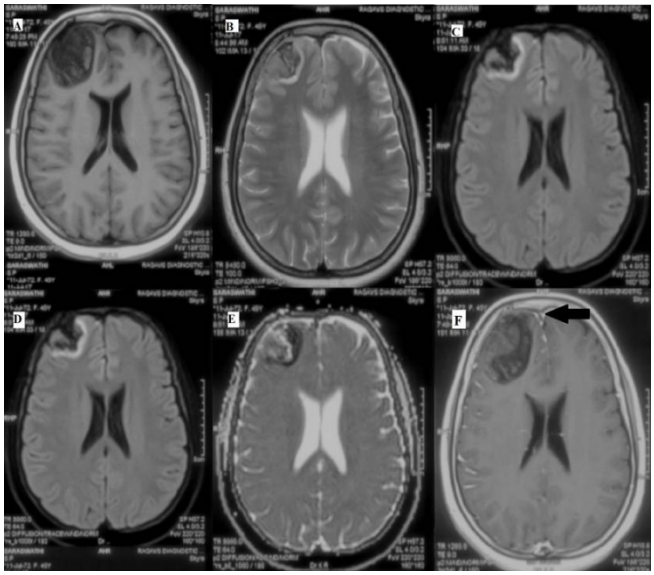

Figure 1: A-T1 weighted image showing mixed intensity lesion with predominant hyperintensity with specs of hypo-intensity, B&C- T2& flair showing mixed intensity lesion with hyperintense rim, D&E–Diffusion weighted image showing diffusion restriction & Apparent diffusion co-efficient showing corresponding hypo-intensity, F – contrast enhanced MRI showing filling defect in anterior part of superior sagittal sinus.

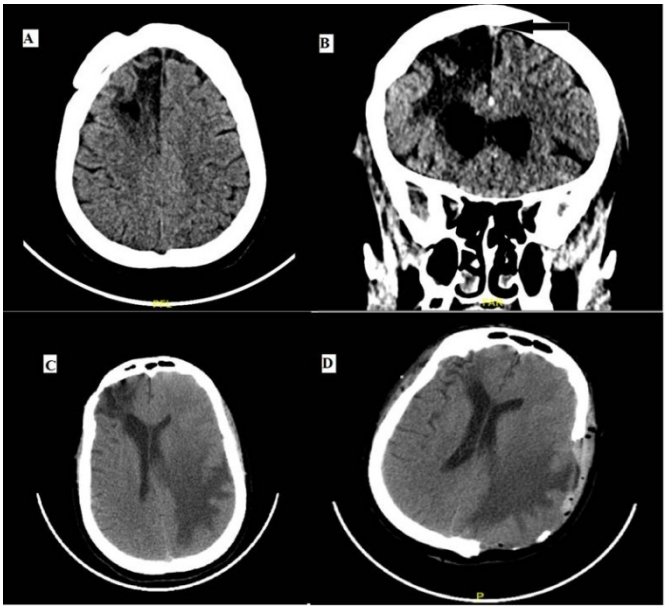

Figure 2. A- hypodensity on left frontal region; previous craniectomy defect and healed infarct seen on right side, B- filling defect in anterior part of superior sagittal sinus, C- progressive increase in size of hemorrhagic infarct causing midline shift, D- post decompressive craniectomy

The patient again presented to us in 2020 with recurrent right focal seizures with secondary generalization and mild right hemiparesis. She had not undergone gynecological evaluation following discharge from our hospital and had recurrent AUB for 3 months before presentation. Imaging showed recurrence of CVT with involvement of anterior superior sagittal sinus and new infarct involving left frontal lobe with anemia (Hemoglobin-7g/dl). Over next four days she developed headache and sensorium rapidly deteriorated in spite of medical management. Repeat imaging showed increase in the size of haemorrhagic infarct with midline shift (Figure 2). Patient underwent DC (contralateral to the initial site) with placement of bone flap in the abdomen.

Postoperatively she needed intensive care management for a prolonged duration with which she showed improvement. Her mRS score was two after three months of discharge.

DISCUSSION

We report a case of recurrent CVT (twice) over a span of three years involving the same segments of the superior sagittal sinus secondary to severe anemia. In spite of aggressive medical management in both instances, she deteriorated and had to undergo DC to relieve the mass effect. Of interest to note is that the CVT in both instances was in opposite hemispheres. However, in both cases, following DC, she recovered completely though with prolonged intensive care. The causes of CVT include hematological abnormalities, infections, puerperium, oral contraceptives, dehydration, genetic hypercoagulable states and systemic malignancies.[7]Anemia, especially iron-deficiency anemia, is a well-known cause through thrombocytosis associated with iron deficiency, decreasing pliability of microcytic cells causing hyper viscosity.[8] An interesting fact to note is the propensity of thrombosis to occur in the same segment of the venous sinus. In our patient, in spite of extensive workup, we were not able to find any etiologic cause other than anemia which was treated with packed cell transfusion and iron supplements. Further evaluation showed AUB as the cause. Since, it was not treated; our patient had a second episode of CVT. The experience from our patient thus signifies the importance of treating the underlying cause to prevent recurrence. CVT has a relatively good prognosis when compared with arterial stroke. The overall death rate is below 5%.[9] Medical management is reported to be successful in more than 80% of patients. [10] It includes parenteral heparin, antiedema measures including mannitol, oral glycerol and antiepileptics.[11] The underlying etiology has to be treated. In cases of severe CVT not responding to anticoagulants, endovascular thrombolysis or mechanical thrombectomy might be an option, although evidence to support this approach is currently lacking.

The signs for worsening in CVT include drop in Glasgow coma scale score, oculomotor nerve palsy and midline shift of more than 5mm. The main aim of surgical therapy is to relieve the herniation which is the major cause of death in CVT.[10]The types of DC available are sub-temporal decompression, circular decompression, fronto-parietal or temporoparietal DC, large fronto-temporoparietal DC, hemisphere craniectomy, and bifrontal DC.[12] In a retrospective analysis conducted at NIMHANS, the favourable outcome for patients undergoing DC is around 76-80% and GCS score was the significant predictor for prognosis.[2] Lechalnoine has reported a case of bilateral decompression in a patient who had failed medical and endovascular therapy.[10] Our patient differs in the point that she required decompression on two different occasions which to our best knowledge has not been reported in literature.

CONCLUSION

Optimal timing of DC in appropriate cases can be lifesaving even when done more than once and done in opposite hemispheres. Treating the underlying cause to prevent recurrence is as important as treating the acute phase of CVT.

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