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Research Article

DURAL VENOUS SINUS THROMBOSIS IN TROPICAL REGION FOLLOWING HEAD INJURY

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A R T I C L E I N F O

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A B S T R A C T

Dural Venous Sinus Thrombosis (DVST) is rarely reported as a sequel following traumatic brain injury (TBI). In emergency, if CT Brain shows bleed, it is difficult to differentiate that has the intracranial bleed preceded or followed that trauma. We studied five patients with history of road traffic accident with large bleed on CT scan brain but not having any external injury who presented to us at rural hospital, in Vidharbha. MRV was done which showed cortical venous thrombosis in all five patients. Anticoagulation therapy is the first choice in such cases although it remains controversial in traumatic brain injury, in view increase risk of bleeding. Central India have hot and dry climate and dehydration is thus more common. In tropical region, dehydration may be the aggravating factor for DVST following TBI. The discrepancy in external injury and internal bleed size can help as one of the clue for clinical suspicion for DVST.

INTRODUCTION

Dural venous sinus thrombosis (DVST) is rarely reported as a sequel following traumatic brain injury (TBI). [1] DVST usually follows hypercoagulable states such as pregnancy, puerperium, and use of oral contraceptives. It also occurs following skull fracture, intracranial infections, coagulopathies and severe dehydration [2,3] Central India has a tropical semi-arid climate where the rate of evaporation of water is higher than the rate of moisture received through precipitation. The temperature of Vidarbha region ranges from a minimum of 12-25°C to a maximum of 30-48°C, with relative humidity varying from 10-15% to 60-95%. [4] Few cases of TBI presenting with atypical contusions or large bleed with no external trauma in such high temperature regions may have DVST post TBI which might be due to dehydration.

Case 1: 61-year male, known case of seizure disorder, no other co-morbidities, presented with alleged H/o RTA (road traffic accident), while crossing road was hit by four-wheeler. He had 1 episode of seizures and was brought to hospital in post ictal state. He had no external injury. [Figure 1a] Computed tomography (CT) scan showed large bifrontal bleed, basal cisterns well seen, no fractures.[Figure 1b]

MRI (Magnetic resonance imaging) brain with Magnetic resonance venography (MRV) showed Dural venous sinus thrombosis. [Figure 1c, 1d] He was treated as per trauma protocol with hydration, anticonvulsants, antiedema measures and other supportive measures. Low molecular weight heparin (LMWH) was given for 5 days, then oral anticoagulant was continued with regular monitoring of prothrombin time. He improved well and was discharged. On follow up after 1 year he was doing well, repeat CT showed gliotic changes and is being managed with anticonvulsant and antiplatelet drugs.

Case 2: 26-year-old male, no co-morbidities presented with H/o RTA due to skid of his 2-wheeler but had no external injury except abrasion on forearm. [Figure 2a] His CT brain showed right frontal bleed, no fractures and MRI brain with MRV showed occlusion of anterior superior sagittal sinus. He was managed with similarly with hydration, anticonvulsants and LMWH for 3 days. Repeat CT brain showed resolving bleed and was discharged on anticonvulsants only. On follow-up after 5 months he is doing well and on anticonvulsants only.

Case 3: 37-year male, with no co-morbidities presented with H/o RTA while travelling on two-wheeler was hit by another two-wheeler. H/o vomiting present, but no external injury. [Figure 2b] His CT scan showed right temporal bleed. MRI Brain with MRV was suggestive of DVST. He was also treated with hydration, anticonvulsants and LMWH for 3 days and discharged in stable condition. He presented with altered sensorium and was found to have hyponatremia which was corrected. On follow-up after 5 months he is well oriented, serum sodium normal. Repeat CT showed resolution of bleed.

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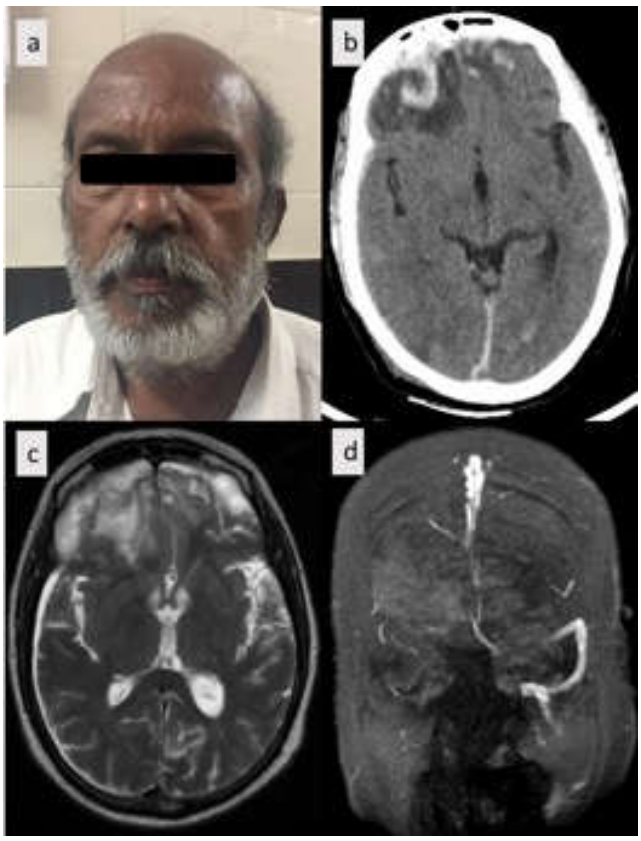

Figure 1 Clinical photograph, CT brain, MRI Brain & MRV of Case 1

Case 4: 52-year-old female, presented with H/o RTA due to fall from bike as a pillion rider. H/o seizures and vomiting present, no external injury. [Figure 2c] CT Brain showed bifrontal contusion and MRI brain with MRV was suggestive of occlusion of anterior superior sagittal sinus. She was also treated with hydration, anticonvulsants and LMWH for 3 days and discharged in stable condition. On follow-up after 1 months, CT brain showed resolving bleed with hypodensity. Patient is stable, oriented.

Case 5: 48-year male, with k/c/o hypertension presented with H/o RTA while crossing road hit by two-wheeler. Had h/o seizures and vomiting. On examination: drowsy, irritable, GCS E3V4M6, vitals stable, no external injury. [Figure 2d] His CT Brain showed left frontal contusion with perilesional oedema. MRI brain with MRV was done showed thrombosis of superior sagittal sinus. He was also treated with hydration, anticonvulsants and LMWH for 3 days. Repeat CT scan showed resolving bleed and was discharged in stable condition after 10 days on anticonvulsant. On follow-up after 1 month he was stable, oriented.

All the 5 patients had no external injury on head and on CT showed bleed and MRI Brain with MRV showed DVST. All of them had signs of dehydration like dry tongue and decreased skin turgor with increased blood urea to creatinine ratio. In our small case series of 5 patients, who were treated with LMWH for 3 to 5 days and on repeat imaging we could not find any fresh bleed whereas there was resolution of bleed and cerebral oedema with symptomatic improvement in headache. Fortunately, none of them required surgical procedure, but strict monitoring of patient's condition, imaging to be done when required, and surgery plan was always kept at stand by.

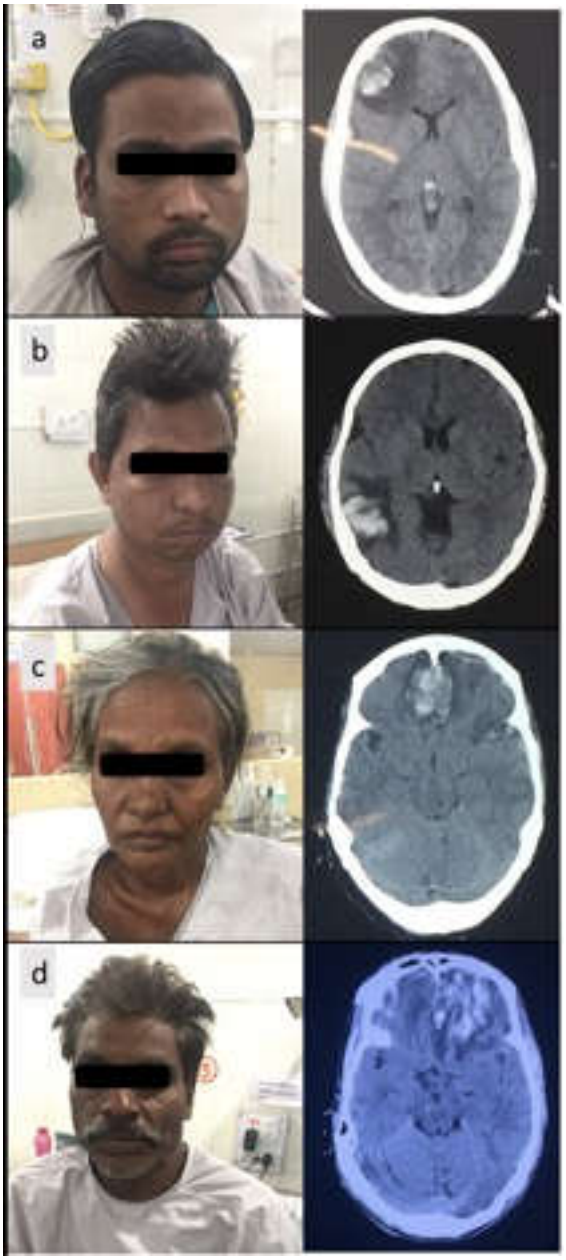

Figure 2 Clinical photographs with respective adjacent CT Brain images of Cases 2,3,4 & 5

DISCUSSION

Computed tomography (CT) scan is done in almost all cases of TBI, because of its availability even at remote cities, fear of litigation, risk missed diagnoses, and where there are inadequate facilities to monitor the patients. [5] In many instances, the CT brain may show large bleed which may be seen even in absence of external major injury. So, there is always a dilemma for the treating doctors; that, if the trauma caused the bleed, or vice versa? Angiography is usually advised in such cases for ruling out vascular causes.

DVST if diagnosed early and managed promptly may improve prognosis. Various imaging modalities can help in early diagnosis if there is clinical suspicion. The classic finding of sinus thrombosis CT images is a hyper attenuating thrombus in the occluded sinus; contrast-enhanced imaging shows the empty delta sign, a central intraluminal filling defect that represents a thrombus surrounded by contrast-enhanced dural collateral venous channels and cavernous spaces within the

dural envelope. On MR images, there is absence of a flow void and the presence of altered signal intensity in the sinus. [6]

Various hypothesises are available for occurrence of DVST in TBI patients. Carrie and Jaffe stated that abnormalities in the clotting mechanism, disturbances in blood flow or damage to the capillary endothelium may predispose and lead to thrombosis. [7] Pre-existing coagulopathy may be a factor in predisposing patients. [8] Case series by Delgado Almandoz JE *et al* showed strong co-relation between skull fracture and DVST, whereas in our series we could not find any.

Treatment includes maintaining good hydration, and the administration of anti-edema agents like dexamethasone and mannitol. Anticonvulsants are necessary to prevent seizures. [9] Anticoagulation therapy is the first choice although it remains controversial in traumatic brain injury, in view increase risk of bleeding. Stam J *et al* advised that LMWH, which has a longer life than unfractionated heparin, can also be administered since it has a more predictable response at standard doses and lower incidence of thrombocytopenia and hemorrhagic complications [10] However, monitoring of patients' condition and repeat imaging is essential for prevention of fatal complications. D'Alise MD *et al* reported successful management of DVST with heparin and urokinase infusion directly into the sinuses via a transfemoraltransvenous micro catheter. [11] In the present short series we have treated the patients with LMWH for short duration for 3 to 5 days, and one patient who had very large bleed was treated with oral anticoagulant with regular monitoring of prothrombin time.

Dehydration is strongly and independently associated with venous thromboembolism in all age group and types of patients. [12] In the present study we also found strong positive correlation of dehydration with DVST. Antiedema medications can also precipitate dehydration if fluids are not supplemented adequately. In the dry and hot region like Vidarbha, where dehydration is very common in people doing farm work and travelling, it could be one of the cause of DVST. In this small case series, we found discrepancy in external injury and internal bleed size in all patients. DVST needs a high index for suspicion for prompt diagnosis, and this discrepancy could be one of the clue for the diagnosis.

There are many limitations in this study, being a small case series the results may vary if compared to large sample size. We have included only those patients who had no external injury, maybe it is possible that the patients who have external injury and contusion, who had not undergone MRI with MRV may also have DVST. We have not investigated for the other causes of hypercoagulability like Factor V Leiden, deficiency of antithrombin, protein C and protein S, elevated homocysteine. Lastly there are case reports of spontaneous recanalization of DVST [3], so the role of LMWH as a treatment is controversial.

CONCLUSION

In tropical region, where the climate is hot and dry dehydration may be the aggravating factor for DVST following TBI. DVST needs a high index for suspicion for prompt diagnosis. The discrepancy in external injury and internal bleed size can serve as one of the clue for clinical suspicion.

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Declaration of patient consent: The author certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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