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Deep cerebral vein thrombosis due to anaemia in a child

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ABSTRACT

An eighteen-month-old child presented with vomiting, fever and altered sensorium of two days duration. He had anaemia and computed tomography of head revealed hyper-dense internal cerebral veins, vein of Galen and inferior sagittal sinus and bilateral thalamic hypo-density. The child improved with anti-coagulants and packed cell transfusion.

Keywords
anaemia,
computed tomography,
deep cerebral vein
thrombosis.

INTRODUCTION

Deep cerebral veins include the internal cerebral vein, vein of Galen, basal vein of Rosenthal and the straight sinus. Approximately 10% of the cases of cerebral venous thrombosis are contributed by deep cerebral venous system, the remainder being that due to superficial system [(1)]. Symptoms of Deep Cerebral Venous Thrombosis (DCVT) are confusing and non-specific. Hence the diagnosis is often delayed. A high index of suspicion can save valuable lives if proper treatment is initiated at an early stage. Recently we came across a case of DCVT with anemia which is unusual and hence is being reported.

CASE REPORT

An 18-month-old child presented with 3-4 episodes of vomiting since 2 days, fever and tonic posturing of all four limbs since one day. On examination, the child had fever, tachycardia of 150/min, respiratory rate of 44/min. He was in altered sensorium. Fundus was normal and the deep tendon reflexes were brisk. The plantar were extensor. Investigations revealed Hb of 6.34 g/dL, RBC Count of 4.43* 10^6/mm^3, WBC 17.6*10^3/mm^3, with a differential count of NE 64.4%, L28.2%, Mo. 7.1%, EO 0.3%; Hct 23.7%, MCV 53.4fL, MCH 14.2pg, MCHC 26.6g/dL, Platelet count 1236*10^3/microlitre, MPV 6.4, Procalictonin 0.797. Peripheral smear showed anisocytosis, hypochromia, microcytosis, poikilocytosis, and target cells. Serum electrolytes, LFTs, KFTs were normal. CSF was colorless, clear and on microscopy, contained 22 cells/microlitre with 98% polymorphs, 2% lymphocytes with RBC+ve, Gram staining was negative and culture was sterile after 72 hours.

Computed tomography revealed hyperdensity in the internal cerebral vein of Galen and the straight sinus (Fig. 1). There were bilateral

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thalamic hypodensities and hydrocephalus. Radiologists were sure of thrombosis of deep cerebral venous system and advised a follow up MRI in the follow up period.

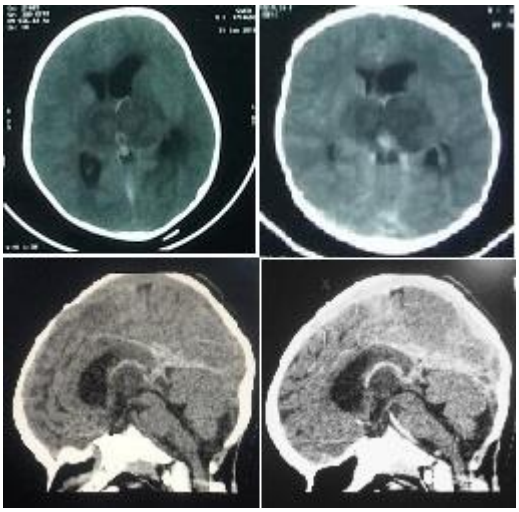

Figure 1. (Top row) Non-contrast (left) and contrast (right) axial computed tomography scan showing bilateral thalamic hypo-density and hyper-dense deep cerebral venous system (on plain scan).
(Bottom row) Non contrast (left) and contrast(right) sagittal CT showing hyper dense deep cerebral venous system on non-contrast scan which remains as such with contrast.

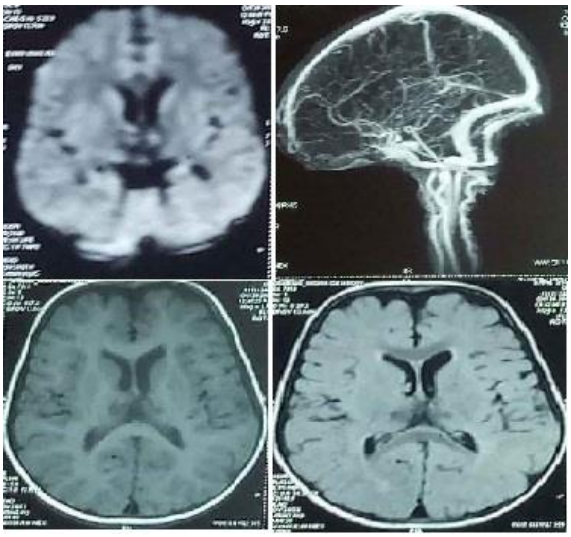

Figure 2. (Top row) Follow up MR showing very small area of infarct (DWI on left) surrounded by a ring of edema and patency of deep venous system demonstrated in MRV (right).
(Bottom row) Follow up MRI demonstrating small hypo-intense areas on T(left) and FLAIR (right) in bilateral thalami.

The patient was started on Acylovir and anticoagulants (enoxaparin). External ventricular drainage was instituted. He had recurrent seizures which were controlled with anti-epileptics. Sensorium improved over a period of time and the EVD was removed. He was discharged after 20 days of hospitalization in conscious state with power of 4/5 in all limbs with mild hypotonia.

Investigations for possible thrombophilia were normal. These included cardiolipin antibodies (6.03-Normal <12 MPL),Homocysteine(11.62micromol/L-Normal 5.46-16.2), Protein C (67-Normal 70-140 IU/dL),MHFTR mutation(+ve),Phospholipid IgG(1.52-Normal <10), Phospholipid IgM(2.42, Normal <10).On follow up, he was ambulant with mild dystonia and MRI done at 3 and 9 months revealed recanalization of the deep cerebral venous system (Fig. 2).

Discussion

Deep cerebral venous thrombosis is a rare cause of stroke in children and may be associated with poor prognosis [2]. The diagnosis is often delayed as the symptoms of venous occlusion are varied and non-specific. Children may present with encephalopathy as seen in our case or may have headache, seizures or raised intracranial pressure.

In our patient, all the biochemical parameters were normal. Hematological investigations revealed microcytic, hypochromic anemia and the diagnosis cerebral DVT was established with a non- contrast computed tomography (CT).CT may be normal in 30% of the cases [2, 3]. The thalami only or the basal ganglia may show edema or infarction. At times, the thalamic involvement may be unilateral even with bilateral DCVT [4]. Sidek et al in 2015 could collect 24 cases of isolated deep cerebral vein thrombosis and added one of their own. Out of 25 cases, 15 were bilateral infarctions [5].

Hyper-density in the deep venous system on CT is suggestive of the diagnosis. MRI can serve as a contributory tool by demonstrating hypo-intense lesion on T2WI and hyper-intense image on T1WI[6, 7]. Though MRI appears to be sensitive for detection of cerebral deep vein thrombosis, it could provide additional diagnostic benefit in only two cases of CDVT,where thrombosis could not be suspected on CT[8].

Hydrocephalus was due of thalami causing occlusion of the third ventricle. Recanalization can occur [9] and was seen in our patient as well. Our patient was treated with low molecular weight heparin,Vitamin K and packed cell transfusion.

Clinical outcome was favorable. Thrombosis resolved at 5 months.

Onset may be acute (less than 48 hours), subacute (48hrs to 1 month) and more chronic disease. Mortality was reduced from 48% to 13% with the use of heparin, increasing the importance of early diagnosis[10].The predisposing factors include dehydration, hypercoagulable state, states of infection and malignancy[2].

Acute thrombosis decreases the level of anti-thrombin, protein C, and protein S. Therefore the tests for thrombophilic states should be performed at least 6 weeks after an acute thrombotic event and INR should be maintained between 2-3. Investigations for hypercoagulability panel were negative in our patient except anemia.

Various mechanisms have been postulated to explain iron deficiency leading to CSVT.Serum iron prevents thrombopoiesis [11] and acts as an inhibitor of thrombocytosis.Therefore, iron deficiency leads to increased platelets and results in a hypercoagulable state .Increased erythropoietin activity during iron deficiency anemia stimulates megakaryocytosis. Microcytosis decreases cell deformability and increases viscosity leading to deranged flow pattern [12]. In the presence of infection, increased metabolic demand can create anemic hypoxia with predisposition to venous thrombosis [13].

Ourpatient, had anemia with occult infection as indicated by high TLC.This, in our opinion was responsible for the DCVT. He did not have any evidence of dehydration or thrombophilic state (Cardiolopin antibody was negative and homocysteine, Protein C, Phospolipid IgG and IgM levels werereported to be normal). Early diagnosis coupled with institution of low molecular weight heparin and blood transfusion resulted in good outcome.

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