Case Report

Subarachnoid Haemorrhage as a Presentation of Cerebral Venous Sinus Thrombosis


Abstract

Cerebral venous sinus thrombosis can present with a variety of clinical symptoms ranging from isolated headache to deep coma. Subarachnoid haemorrhage is a rare presentation. In the report, we describe a case of non-puerperal venous thrombosis of many dural sinuses manifesting on imaging solely as localized subarachnoid haemorrhage without underlying parenchymal involvement.

INTRODUCTION

Cerebral venous thrombosis (CVT) involves thrombosis of dural venous sinuses and/or cortical and deep veins of the brain. It is a fairly common stroke syndrome in India in comparison to the Western hemisphere and is known to present with protean clinical features rendering recognition difficult. CVT is an established cause of parenchymal haemorrhage, however subarachnoid haemorrhage is extremely rare. There are only two case reports of subarachnoid haemorrhage as the presenting symptom of cerebral venous thrombosis. To the best of our knowledge this is probably the first report from India as well as the first case in which occlusion of many major dural sinuses presented with only subarachnoid haemorrhage with normal brain parenchyma.

CASE REPORT

A 40 years right handed man presented with sudden onset holocranial bursting headache of moderate intensity located in the frontal as well as occipital region associated with recurrent vomiting, photophobia and phonophobia. There was no relief in pain with analgesics. After about 48 hours, patients had an attack of right focal seizures with secondary generalization and it was associated with drowsiness for which he was admitted in the Neurology Department where he had another seizure followed by unconsciousness and was incontinent for urine and faeces. A right hemiparesis was noticed (power grade 2/5, MRC grading) when the patient regained consciousness. On examination, the patient was afebrile, vitals were normal, GCS score was E4 M4 V2, fundi were normal, and signs of meningeal irritation were present. There was also weakness of right upper and lower limb with a right UMN facial palsy plantar responses were flexor bilaterally. Systemic examination was normal. A non contrast CT scan of brain obtained within a few hours of onset of seizures demonstrated blood in the subarachnoid space in the region of left sylvian fissure (Fig. 1). Patient was put on IV mannitol, ranitidine and epsolin. A neurosurgical consultation was taken and a possibility of subarachnoid haemorrhage due to left MCA aneurysmal bleed was kept. Within the next 24 hours drowsiness and weakness of right side disappeared.

Routine haematological examination (Haemoglobin, total and differential leucocyte count) and blood/serum biochemistry (sugar, creatinine, liver function tests, sodium and potassium) were normal. Elisa test for HIV 1 and 2 was negative. Platelet count, prothrombin time,

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Fig. 1 : Plain CT scan brain showing blood in left sylvian fissure.
and concentration and APTT was normal. Digital subtraction angiography was done through percutaneous transfemoral route and selective catheterisation of bilateral internal carotid arteries and left vertebral artery was performed. There was a normal arterial, capillary and branching pattern. The venous phase revealed non-opacification of superior sagittal sinus, transverse sinuses and inferior sagittal sinus with evidence of multiple collaterals (Fig. 2A, B, C). CSF examination revealed that the colour was turbid red, total cells 100/cmm with 80% lymphocytes and 20% polymorphs, protein was 78 mg% and sugar 71 mg%. RBC count was 1,85,000/cmm and the wet smear showed fair number of RBCs of which 40% were crenated. Gram’s stain and AFB stain were negative.

MRI Brain was done on a 1.5 Tesla GE system. T1 and T2 weighted images showed that the brain parenchyma was normal and there was no evidence of a venous infarct. MR venogram done by 3D TOF technique confirmed thrombosis in various sinuses as already described (Fig. 3A, B). The protein C, protein S, antithrombin III, antiphospholipid and anticardiolipin antibody levels were normal. Patient was given anti-congestive treatment along with anticonvulsants. He was put on low molecular weight heparin for 7 days followed by oral anticoagulants. INR was maintained between 3 to 3.5. Patient was discharged after a month on warfarin 7.5 mg per day and was symptom-free.

**DISCUSSION**

The patient presented with a sudden onset of headache associated with recurrent vomiting followed by seizures, altered sensorium and transient hemiparesis. Neck rigidity was present. The diagnosis of subarachnoid haemorrhage was confirmed on head CT scan and CSF examination. A DSA and MRV showed occlusion of a large number of dural sinuses namely superior and inferior sagittal sinuses, transverse and sigmoid sinus on the right.

The prospective clinical diagnosis of cerebral venous thrombosis is difficult because of a wide spectrum of clinical manifestations and the diagnosis is typically made on the basis of imaging studies. Headache is the most common symptom in cases of CVT followed by convulsions which occur in 50 to 70% of patients. It has been shown that 38% of patients with CVT have subcortical haemorrhage on MRI, while on CT scan subcortical haemorrhage may be the only manifestations
There are only two reports of subarachnoid haemorrhage as the manifestations of CVT. In the first study a single patient has been reported with a localized subarachnoid haemorrhage in the right posterior fossa on CT scan. MRI coupled with MR venogram revealed thrombosis of right transverse and sigmoid sinus with haemorrhagic infarction of the right cerebellar hemisphere. In the second study there were three cases of isolated thrombosis of vein of Trolard that presented with unilateral localized subarachnoid haemorrhage without parenchymal involvement.

To the best of our knowledge, the present report is the first case in which occlusion of many major dural sinuses presented with only subarachnoid haemorrhage without parenchymal involvement.

Digital subtraction angiography is still the gold standard for the assessment of CVT. However, combined MRI and MRV have replaced carotid angiography in the diagnostic workup of patients with a suspicion of CVT because these techniques are noninvasive and reliable and also provide information about the brain parenchyma. The mechanism behind the development of subarachnoid haemorrhage in the present case is uncertain but could be due to rupture of a dilated collateral vein. The left focal seizure and hemiparesis are likely to be due to the irritative effect of blood in the sylvian fissure and central sulcus. Anticoagulants have been recommended as the treatment of choice, conventional or low molecular weight heparin initially followed by oral anticoagulants for a period of 3-4 months. Recanalisation occurs in about 50% of cases by 4 months. So there is no evidence of continuing anticoagulants longer than that period.

**References**