

Review of Three Cases of Cortical Sinus Venous Thrombosis(CSVT).

KEYWORDS

Cerebral venous thrombosis, computered tomography, magnetic resonance.

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ABSTRACT Cortical venous thrombosis(CVT) is a rare cause of stroke in adults. Its varied presentations make the clinician to have strong degree of suspicion and investigate accordingly thus instituting early treatment. We present case series of CSVT with review on its clinical presentations, diagnostic investigations, treatment and prognosis.

INTRODUCTION:

Cerebral venous sinus thrombosis and cortical venous thrombosis leading to ischemia and infarction of the brain are being increasingly detected in recent times with the advent of greater awareness amongst the clinicians and greater availability of non invasive radio diagnostic modalities like magnetic resonance imaging and MR venography.patients often present with profound neurological manifestations in the form of focal neurological deficits, seizures, headache(1) and other features of raised intracranial tension, leading to misdiagnosis and delay in treatment. High index of suspicion is required in appropriate clinical settings to diagnose this entity in earliest.

CASE REPORTS-CASE1:

A 40 years old chronic alcoholic abuse and smoker, presented with complaints of acute onset three episodes of GTCS type of seizures in past few hours without any history of weakness of limbs, cranial nerve deficits, sensory symptoms, speech abnormality. there was no history of fever, vomiting, blurred vision, ear discharge and head injury. He denied past history of any major illness.

On examination patient was afebrile, puse rate 80/min, regular; blood pressure 130/90mm of Hg. There was no pallor, icterus, tremors, diaphoresis or signs hepatic dysfunction. Neurological examination revealed aconscious and coherent male with normal power, tone and reflexes with out any sensory defict, ataxia or signs of meningeal irritation. Fundus examination showed early papilloedema features. Examination of other systems was normal

Investigations revealed normal CBP,Renal function tests and blood sugars. Serum bilirubin slightly raised and liver enzymes was normal. Chest radiography and ultrasound abdomen revealed no abnormality. Prothrombin time and INR was with in normal limits. CT brain plain and contrast showed . MR venogram showed thrombosis of superior sagital, both transverse and sigmoid sinus thrombosis. Patient was started on injection. HEPARIN (low molecular weight) and tab. WARFARIN 2mg after achieving target INR between 2 & 3. Patient advised review after 3 months for coagulation profile study.

CASE2:

A 19 years old male presented with sudden onset of weakness of left lower limb and upper limb since 1 day preceded by head ache, vomiting.On examination patient had left hemiparesis with left plantar extensor,no sensory and cranial nerve deficit.MR venogram revealed thrombosis of both transverse and right sigmoid sinus.Patient was started on Injection LMWH and Tab.ACITRAM 2 mg OD simultaneously.Later only Tab. ACITRAM 2mg was continued.Patient recoverd well and discharged and adviced for follow up after 3 months for coagulation profile study.

CASE3

A 25 year old postpartum presented with history of loss of consciousness on $13^{\rm th}$ postpartum day for 1 hour with spontaneous recovery followed by irritability.No history of hypertension, diabetesmellitus.No focal neurological deficit on examination.CT brain showed dot sign,lambda sign. MR venogram showed posterior sagital sinus thrombosis. Patient was treated with anticoagulants initially with low molecular weight heparin for 5days with acitram 2mg daily. patient recovered well .

DISCUSSION:

Cortical venous sinus thrombosis an uncommon cause of stroke in our country. Puerperal CVST accounts for a majority of these cases. The main causes of non puerperal CVST are dehydration, antiphospholipid antbody syndrome, Hype rhomocysteneimia, factor 5 Leiden, Malignancies and certain drugs like oral contraceptives and L-arginase. In about 25% of patients, no predisposing risk factors can be discovered despite extensive investigations. In the 2 cases which have been presented, no definitive cause could be found on initial work except for alcoholism as risk factor in case 1.

CVST most commonly involves superior sagtital sinus thrombosis(72%) followed by lateral sinus(70%). In 30-40% cases more than one sinus is involved (2). The sinuses involved in our cases are, in case1 superior sagittal sinus, both transverse and right sigmoid sinus and in case 2 both transverse and right sigmoid sinus. Clinical manifestations seen in CVST are headache(75-90%) papilloedema, focal neurological deficit(75%) and seizures. Rarely patient may present as stroke in young as in case2 (3). Unusually , patient present with a thunderclap headache mimicking suba-

rachnoid hemorrhage.

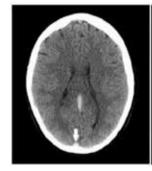
Neuroimaging modalities of choice in CVST are computerised tomography scan and MRI with venogram.CT scan may be normal in 15-30% cases but MRI with MRV is almost 100% diagnostic.(4)

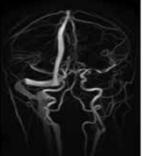
Workup for underlying primary thrombophilic state is ideally postponed to8-12 weeks after the acute episode as there may be falsely negative or positive in the acute state, and the results are also affected by the administration of anticoagulants(5). Anticoagulants must be withdrawn atleast 2weeks prior to testing for mere abnormalities.

Anticoagulation is the cornerstone of treatment in CVST. It is only withheld in patients with septic thrombophlebitis and in large hemorrhagic venous infarct with significant mass effect. Oral anticoagulants should be overlapped with heparin and the later discontinued once therapeutic INR of 2-3 is achieved. Oral anticoagulants should be continued for atleast 3 to 6 months if no underlying procoagulant state was found or life long if there is an irreversible procoagulant state. In case no.1 &2 anticogulants was administered as there is no hemorrhage and there was no mass effect. Thrombolysis of clot using local administration or systemic injection of urokinase or recombinant tissue plasminogen activator has been evaluated and has been found to be effective though it is caught with danger of hemorrhagic transformation of infarct(5).

CONCLUSION:

CVST is a great masquerade as it can present in various forms and confuse the clinician. It manifests as a stroke with seizures confusing it with arterial strokes. It may present with headache and papilloedema the clinical sine-quanon of ICSOL. Hence it requires high index of suspicion is needed for early diagnosis, ensure aggressive use of anticoagulants and lessen the morbidity and mortality in the cases.





REFERENCE (1) Menta SR, Swamy aj, Varadarajulu R GuptaA.A clinicial profile of cortical venous thrombosis.: A report of 20 cases. JAPI 2002, 50:76-7 (2) Brousser MG, BarnettHJM cerebral venous thrombosis. In Stroke: pathophysiology, diagnosis and management, 2nd end. (3) G. sansonik, stroke, aha journals.org. 2011. (4) Wang AM.MRA of venous sinus thrombosis Clin. Nruroxvi 1997; 4:158-64. (5) Deschiers MA, Conard J. horellu MH et al. Coagulation studies, Factor V leiden and anticardiolipin antibodies in 40 cases of cerebral venous thrombosis.stroke: 1996: 27:1724-30 (6) Freus Jt, MuroGJ, Mc Dougall CG, Dea32 Cerebral venous thrombosis, combined intra.