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Case report-3

A patient with young stroke-rare presentation of superior sagittal sinus thrombosis

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Abstracts:

A 22 years young lady, normotensive, non-diabetic patient persented clinically with right sided weakness, headache, visual blurring & repeated convulsion. She had history of vaginal delivery 2 weeks ago . Examination revealed patient to be mildly anaemic and moderately dehydrated. She was confused & disoriented, speech was slurred & incoherent. Pupil was moderately dilated bilaterally and reaction was sluggish , mild degree opthalmoparesis especially in abduction. On Fundoscopy , bilateral Papillloedima was persent. Muscle Power was reduced in right side of body, proximally grade 3 distally grade 2, right sided plantar was extensor. Investigation revealed neutrophilic leukocytosis, ESR-45 mm in 1st hour, all others metabolic profile was normal. ANA and anticardiolipin antibody was negative but D-dimer was positive. CT scan of brain showed multiple small hemorrhagic spot in parietal and temporal region and MRI of brain with contrast showed bifrontal venous infarct with hemorrhagic transformation with evidence of thrombosis of posterior aspect of superior sagittal sinus.Treatment was given with inj. Dexamethasone, Anticonvulsant, ,Enoxaperin and Acetazolamide. Patient vapidly improved and aspirin was added and discharged with advice. On subsequent pregnancy patient did not develop such complication after delivery.

Introduction:

Cerebral venous sinus thrombosis (CVST) is a rare but dangerous condition , occurring with an incidence of 3-4 case/million/year[1]. Cerebral venous thrombosis present a diagnostic challenge due to its varied presentation patterns. Women tend to be at an increased risk of particularly between the age of 20-35 years, mainly due to the use of oral contraceptive pill and in post partum period[2] .The clinical presentation of Cerebral venous sinus thrombosis is varied and include headache, vomiting and seizure. Thundrclap headache usually indicate sub-arachnoid hemorrhage; it may also be seen in sinus thrombosis and patient may present with recurrent seizure. Cranial nerve syndromes are seen with venous sinus thrombosis,this includes the following: vestibular neuropathy, pulsatile tinnitus,unilateral deafness,double vision, visual bluring[3]. Cerebral Venous Thrombosis may be septic and non-septic[4]. Septic causes include facial cellulits, otitis media , mastoiditis, meningitis and non septic causes include pregnancy, pureperium. OCP, dehydration, thrombophilia and Bachets syndrome. Focal neurological deficit is very common. Thrombosis to superior sagittal sinus may present with unilateral paralysis that may extend to the other side secondary to extension of clot into cerebral vein. On the basis of location of the sinus, this may present as unilateral lower limb weakness or paraperesis. Initially cytotoxic, later vasogenic oedema cause increase in ICP but venous sinus thrombosis associated with the idiopathic intracranial hypertension[3]. In this article we report a patient who developed a superior sagittal sinus thrombosis with no discemable underlying cause. It is hope that the presentation of this case willl draw attention to this rare and potentially devastating disease; helping clinicians to increase awareness of clinical characteristics, methods of diagnosis, management of CVST & outcome.

Case Report:

Mrs. Laboni, aged 22, hailing from Keranigonj was admitted into Dhaka National Medical Institute Hospital with the complaints of headache, blurring of vision and severe vertigo for 4 days, on her 12th day of post partum period. She also complained loss of consciousness in several episodes. After admission, patient developed

generalized convulsion for several times and became confused and disoriented.Next day patient developed right sided weakness and slurring of speech. Her headache was thunderclaps type which was associated with repeated vomiting and visual blurring. Patient was normotensive, non-diabetic, non smoker, housewife of a middle class family. She had no history of head injury, jaundice, fever or significant family history. Her pregnancy period was uneventful and menstruation was regular previously. On examination, patient was restless and anxious looking, non-cooperative, pulse-90/m, BP-135/90 mmHg, Temp-99^{0}F, moderately dehydrated and mildly anemic. On systemic examination, all the systems seemed to be normal except nervous system which revealed-patients was confused, disoriented, GCS-11,speech slurred and incoherent, memory could not be assessed. Primitive reflexes were absent. On cranial nerve examination- Patient's acquity of vision was reduced in both eye with pupil moderately dilated & sluggish to light reflex.

Her colour perception was also impaired, Fundoscopy showed bilateral papilloedema with haemorrhagic exdudate. Extraoccular muscle examination showed abduction restricted bilaterally with partial ptosis of of right eyelid. Rests of the cranial nerves were normal. Muscle power was reduced in right side of body, proximally grade 3 and distally grade 2 in both upper and lower limbs. DTR were exaggerated in right side of body with extensor right planter reflex. On examination of left side of body, no neurological abnormalities were detected. Her sensation and cerebellar signs were normal. Patient did not show any sign of meningeal irritation. Her convulsion was partial seizure type with secondarily generalized. Relevant investigation was as follows-Hb%-10.8 gm/dl, ESR-45 mm in 1st hr, TC-13,000, PBF revealed neutrophillic leucocytosis, RBS-162 mg/dl, blood urea-43 mg/dl, S. creatinine-0.8 mg/dl, NA^+-137 meq/L, K^+-3.62 meq/L and Cl-105.4 meq/L, BT-3.3 minute and CT-4.1 minute. X-ray chest P/A view, LFT, lipid

profile, ANA, anticardiolipin antibody showed no abnormality and ultrasonography of abdomen were normal. S. Fibrinogen level 200 mg/dl and D-dimer 2 microgram/ml. CT scan of brain showed multiple small and tiny hemorrhagic foci in both upper parietal region, suggestive of traumatic axonal injury. MRI of the brain with contrast revealed bifrontal venous infarct along with evidence of hemorrhagic transformation of left frontal lobe with thrombosis in posterior aspect of superior sagittal sinus.patient did not agree to do MRV of brain and DSA.Finaly the case was diagnosed as superior sagittal sinus thrombosis with venous infarct. Patient was treated by Inj. Dexamethasone, I/V Ceftriaxone, Tab-Phenytoin and Lavetiracetam and Acetazolamide. She was also treated by Inj. Enoxaperin and Aspirin. Patient was clinically improved and was discharged with advice of subsequent follow-up.

Discussion:

Young stroke and its association with superior sagittal sinus thrombosis is very rare. In sagittal sinus thrombosis, intracranial hypertension with headache, vomiting and papilloedema constitute the entire syndrome[5].Causal factors are protean, onset is insidious and 20% of cases, no cause is found[6]. The pathogenesis include the infection, trauma, pregnancy and puerperium, inflammatory bowel disease and hypercoagulable state[6]. One reported study showed superior sagittal sinus is involved in 62 percent cases of all venous thrombosis[7].Of the 795 cases the mean age of the time of diagnosis was 27 years. The most common symptoms were severe headache (53%), followed by seizures (49%); hemiplegia, quadriplegia or paraperesis (48%); Coma (38%); visual disturbance (25%) and nuchal rigidity (18%). Common signs are elevated CSF pressure or increased ICP (46%), coma (38%) and papilloedema[8]. Our patient fulfill most of the criteria in above described study. A study shown that the postpartum state but not the pregnancy itself was associated with an increased risk of cerebral infarction and particularly , intracerebral hemorrhage[9]. Our patient presented at postpartum period and showed the hemorrhagic infarct. Clinically mimicking conditions are acute stroke, blood dyscrasia with storke, cavernous sinus thrombosis, head injury, pseudotumour-cerebri, meningitis and hyperviscosity syndrome secondary to hypercoagubility, CNS lupus. All mimicking condition are excluded by the proper clinical history and relevant investigation. The somewhat slower evolution of the clinical stroke syndrome, the presence of multiple cerebral lesion not in the typical arterial territories and greater epiliptogenic and haemorrhagic tendency favours venous over arterial thrombosis[5]. Our patient evolved over 4-5 days and multiple lesion in cerebral hemisphere and several bout of seizure favour venous sinus thrombosis. Investigations are used in this condition to confirm the diagnosis, and establish any predisposing causes. In 35% of the published cases. CT scan with contrast may be demonstrated and empty delta sign which is a useful radiological sign for the diagnosis of superior sagittal sinus thrombosis[10]. Our patient had done CT head without contrast before

admission that revealed multiple hemorrhagic spot. The most sensitive examination technique is MRI in combination with MRV[11]. T1 weighted and T2 weighted MRI will show the hyperintense
signal from the thrombosed sinus and hemorrhagic infarct. As our patient could not provide MRV; we had done MRI of brain with contrast that revealed the hyperintense thrombosis in posterior part of sagittal sinus and hemorrhagic infarct. The initial management of confirmed CVST should include stabilization and the prevention of cerebral herniation. This may involve the use of manitol or surgery[10]. Despite the risk of the hamorrahge in venous infarct, anticoagulation are the mainstay of treatment, even in the presence of hamorrhagic infarct[12].Our patient was treated by low molecular heparin and aspirin and was bestly improved and discharged.

Conclusion:

CVST is a challenging condition due to its wide range of clinical presentation. Clinician should have a high index of suspicion, even in the absence of predisposing conditions, in order to facilitate a prompt diagnosis through neuro-imaging. Expedited standard management for the CVST should be employed to ensure best possible outcome for patients.

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