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CEREBRAL VENOUS THROMBOSIS -A CASE SERIES OF ATYPICAL PRESENTATION

General Medicine

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ABSTRACT

Cerebral Venous Sinus Thrombosis is blood clot in draining veins and venous sinuses of brain, causing hindrance in the blood drainage system in brain, disturbing the internal homeostasis of brain, resulting in local oedema, ischemia, venous haemorrhage, damage to brain parenchyma and blood brain barrier. In our case series we discussed 3 cases i.e 37yr/female came with complaints of headache, vomiting and gtcs, NCCT brain was suggestive of SAH without parenchymal involvement, which is a rare finding. Another case 45 year old female presented with complaints of headache and weakness of left upper and lower limb & gtcs, she is a known case of nephrotic syndrome, which is a risk factor for thrombosis. The last case was of 32 years old female, primi-gravida with 36 weeks of gestation came with complaints of headache, blurring of vision, 2-3 episodes of vomiting & gtcs, MRI brain was suggestive of CVT and thus CVT should also be considered as one of the diagnosis apart from eclampsia, meningitis & PRES in pregnancy. An early diagnosis can be very fruitful as it might prevent long term disability and reduce mortality significantly.

KEYWORDS

Cerebral Venous Sinus Thrombosis, SAH, Nephrotic Syndrome.

INTRODUCTION

CVT is an important cause of stroke in young adults caused by complete or partial occlusion of deep cerebral venous sinuses or cortical veins[1]. It accounts for 0.5-1% stroke admissions[2]. It is three times more common in females than in males due to associated pregnancy, puerperium and use of OCP[3]. Due to wide spectrum of clinical presentation it is commonly misdiagnosed. Here we report three cases atypical presentation of CVT.

Case 1

Here we report a case of 37 year old female, who was brought to emergency room with history of headache of 8 days duration which was sudden in onset, severe, diffuse, progressive, also complaints of vomiting since 8 days & 1 episode of GTCS, no history of OC Pill consumption, at admission patients vitals were normal. Conscious oriented to time ,place ,person with no focal neurological deficit. NCCT Brain showed Intracranial bleed with SAH secondary to ?AV malformation and treated with Inj mannitol 20% TID, Tab Nimodipine 60mg 6thhrly Inj Levetiracetam 500mg Bd .

MRI Brain with venogram was done for further evaluation which changed the diagnosis to CVT with atypical bleed. Following which Inj LMWH 40mg bridged with Tab Acitrom .On follow-up CT scan vasogenic edema increased and was started with Inj 3% Nacl infusion and Inj Dexamethasone. Edema was reduced after 2 days and Patient was symptomatically better & discharged later.

NCCT BRAIN: showed moderate left subarachnoid hemorrhage along tentorium cerebelli with left posterior parietal intra-parenchymal haematoma possibly due to ruptured aneurysm

MRI Brain (plain and contrast): Atypical Intra-parencymal Bleed in left parieto-temporal region with surrounding edema causing mass effect in the form of midline shift towards right side, subdural bleed in left fronto-parietal region. Above features possibly secondary to Venous trombosis

MRV – Evidence of linear filling defect noted in distal part of left transverse and sigmoid sinuses.

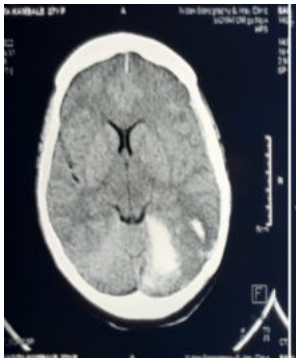

Fig 1 (CT Brain Plain)

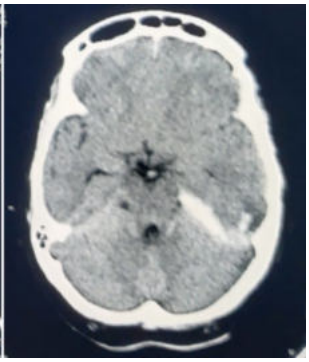

Fig 2 (CT Brain Plain)

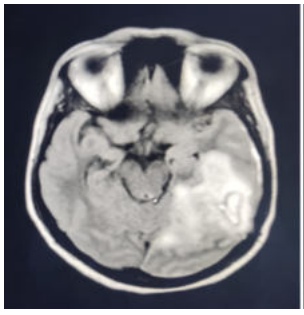

Fig 3 (MRI BRAIN FLAIR)

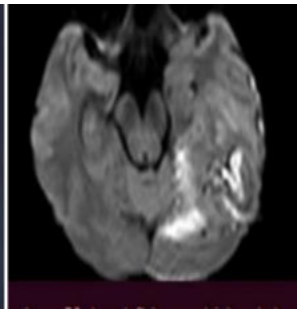

Fig 4 (MRI BRAIN -DWI)

Fig 5 (MR Angiography)

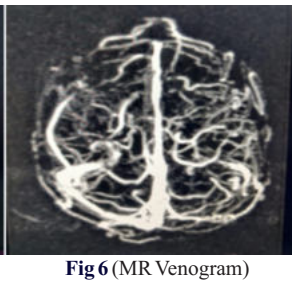

Fig 6 (MR Venogram)

Case 2

A 45 year old female patient presented with complaints of headache since 4 days and weakness of left upper and lower limb since 1 day and 1 episode of GTCS. Patient was a known case of nephrotic syndrome since 2 years on oral Prednisolone 20mg/day, had left medications since 7 months after which she had relapse. On examination Bp-160/90mmhg and PR- 86bpm. Patient was conscious and irritable. Power in the left upper limb and lower limb was 1/5. Tone was increased in both the left limbs. Plantar reflex was extensor on left side. Patient was subjected to MRI Brain with MRV.

Blood investigations- Serum creatinine- 0.5, Total protien- 4.5, Serum Albumin-1.8g/dl, Urine albumin- 2+, total cholesterol- 293mg/dl, TGL- 165mg/dl, HDL-75mg/dl, LDL- 185mg/d.

MRI Brain- Acute infarct in right peri-rolandic region and Cortical and subcortical white matter of right posterior temporal region sencondary to cerebral venous thrombosis.

MRV- Cerebral venous thrombosis in superior sagittal sinus and adjacent draining cortical veins, straight sinus, right transverse sinus and sigmoid sinus.

MRA- Mild atherosclerotic changes in vessels of circle of willis.

Renal biopsy showed Minimal change disease.

She was started with Enoxaparin 60mg s/c twice daily for 5days, IV Mannitol 100ml thrice daily, IV Levetiracetam 1gm in two divided doses. After which the power improved from 1/5 to 4/5 in both left upper and lower limb. Patient was started with Oral Acitrom 3mg/day on day 4, Oral Prednisolone 20mg/day and was discharged with same medications in a stable condition. Now patient is in regular follow up.

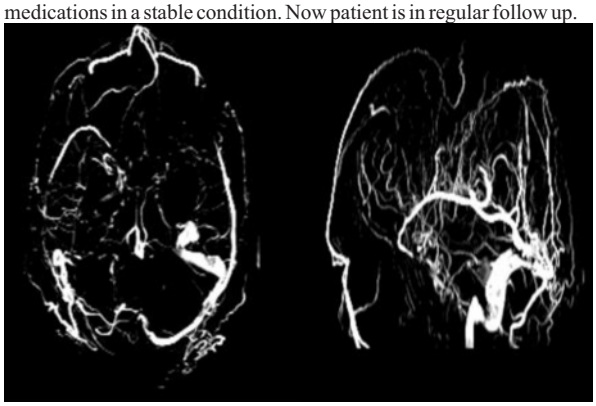

Fig 7 (Shows straight sinus thrombosis)

Fig 8 (Shows superior sagittal Sinus thrombosis)

Case 3

Here we report a case of 32 years old female, primigravida with 36 weeks of gestation came with complaints of headache since 1 day, radiating to cervical region associated with blurring of vision, also complaints of 2-3 episodes of vomiting & involuntary movements of all 4 limbs. When the patient attended our hospital, she was conscious, oriented to time, place & person. BP-200/110mmhg, PR-88bpm, RR-18cpm. GCS-15/15.

She was stabilized with IV labetalol, mannitol and Magnesium sulphate and undergone emergency LSCS under spinal anaesthesia on same day.

Following LSCS, her BP was 140/80mmhg, MRI brain was done and findings were Hemorrhagic infract in parafalcine left parietal region, Small intraparenchymal hemorrhage in right frontal cortex, Subarachnoid hemorrhage in left high parietal region & partial venous thrombus in right transverse sinus.

Inj LMWH 60mg S/C BD, later which was overlapped with Tab Acetrom, Inj Mannitol 100ml TID, Inj levetiracetam 500mg bd.

On POD 8, She was discharged in a stable condition and was continued on oral levetiracetam 1000mg/day in divided doses & Tab Acitrom 3mg at 6pm. On further follow up, she remained stable when her BP within the normal range.

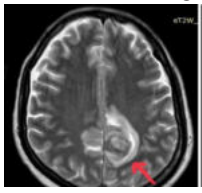

Fig 9

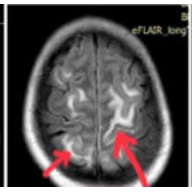

Fig 10

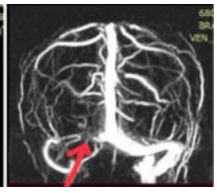

Fig 11

Fig 9, 10, & 11 shows hemorrhagic infract in parafalcine left parietal region, Subarachnoid hemorrhage in left high parietal region & partial venous thrombus in right transverse sinus.

DISCUSSION

CVT is rare but serious cerebro-vascular accident its diagnosis is challenging as the symptoms are non-specific and the outcome is severe if misdiagnosed. As the symptoms are non-specific its diagnosis is delayed on an average for seven days as it was delayed in case 1. The mean age of CVT is 39.1yrs in females[3].

Medical conditions like thrombophIlia, IBD, Pregnancy, dehydration, infection, medications like OCPs, substance abuse, SLE, Obesity, etc are risk factors for CVT. OCPs have been found in 53.4% of CVT patients[4].

Symptoms of CVT differ depending on the cerebral sinus and veins involved, Superior saggital sinus is most commonly involved (62%), which casuses headache, hemiparesis, hemisensory loss, hemi-anopia & seizure. Transverse sinus thrombosis causes temporoparietal haemorrhagic infarction with headache , aphasia, seizures. Sigmoid sinus thrombosis is rare in isolation can cause mastoid pain and, lower cranial palsies. Thrombosis of the deep veins like internal cerebral veins, basal veins of Rosenthal, vein of Galen, straight sinus causes oedema of the thalami, which leads to altered mental status ,coma, gaze palsy, etc. CVT most commonly leads to Intracranial hypertension which presents as headache, papilloedema and visual impairment which is seen in all three of our cases[2].

Patients with suspected CVT require urgent neuroimaging to confirm the diagnosis, using either CT or MR to visualize the thrombus directly. NCCT shows specific signs including venous sinus or deep vein hyperdensity, termed as dense triangle sign or the cord sign[5]. NCCT may be normal in up to 30% of patients[6] and thus, all patients with suspected CVT require further imaging like MR venography or CT venography for confirming the diagnosis[7]. MR venogram is the most sensitive technique for demonstrating the presence of the thrombus material.

The management includes anti-thrombotic, etiological, symptomatic treatment.

Anti coagulation includes intravenous heparin therapy followed by

oral anti-coagulation with Vit-K antagonists. American Heart Association/American Stroke Association (AHA/ASA) and the European Federation of Neurological Societies recommend to start the anticoagulation therapy even if a parenchyma hemorrhage is identified[8]

LMWH is a preferred anticoagulant. It is followed by long term anticoagulation with Vitamin K anticoagulants. INR is maintained between 2-3 for 12 months[7]. Duration of anticoagulation is dependent on underlying risk factors for bleeding and recurrence[9].

Treatment of raised intracranial pressure includes medical and surgical Medical treatment includes mannitol, acetazolamide, hyperventilation, head end elevation.

CVT have a good prognosis after anticoagulation therapy; a minority of patients can have a malignant evolution with high intracranial hypertension and transtentorial herniation needs surgical intervention like decompressive craniectomy.

Indications for craniectomy include uncal herniation, midline shift (>5 mm) and herniation-induced ischemia in the territory of the posterior cerebral artery territory . A persistent intracranial pressure >20 cmH2O is also suggested as a criterion for surgery[10].

As seen in case 1 the NCCT brain revealed the rare possibility of evidence of blood in the subarachnoid space without any parenchymal bleed or direct signs of CVT, thus radiologically suggesting SAH. Subsequent MR angiographic & venographic evaluation confirmed the diagnosis of CVT.

The presentation of CVT as SAH in initial NCCT Brain has been rarely reported. The most common cause of non-traumatic SAH is rupture of an intracranial aneurysm (85%). Less common causes are clotting disorders, cerebral amyloid angiopathy, primary metastatic or meningeal neoplasms, vasculitis, drug abuse, cerebral venous occlusive disease.

The exact mechanism behind SAH associated with CVT is not known. Venous hemorrhagic infarction causing venous hypertension can be responsible for secondary rupture into the cortical veins over the cerebral surface. This would explain the site of SAH bleed seen in the brain convexities and the inter-hemispheric fissure as seen in this case . An important radiological clue for suscepting CVT in patients who presents with SAH on NCCT Brain could be the sparing of the basal cisterns , even in the absence of parenchymal involvement. This should prompt the clinician to ask for M R Angiogram and Venogram at the earliest.

Interestingly, the patient improved with anticoagulant therapy despite subarachnoid bleed being present radiologically. As noted in this case, there was no worsening of the clinical condition. Hence, SAH in this specific clinical setting is not a contradiction for anticoagulation.

As seen in case 2, which is a known case of nephrotic syndrome, severe hypoalbuminemia appears to be the most significant biochemical risk factor for thrombosis. CVT is a rare complication of nephrotic syndrome, with an overall incidence of 1.5 per 100,000 annually,it has increasingly been reported, probably reflecting under-diagnosis. Hyper-coagulation is due to an imbalance between the clotting activator system and the inhibitor system. The urinary leakage of coagulation regulatory proteins, such as anti-thrombin III and plasminogen, and an increased platelet activation and elevated fibrinogen and factors V and VIII also seem to have a role in thrombotic mechanism.

Prophylactic anticoagulation may be given if the serum albumin is <2g/dl, serum fibrinogen >6g/L or antithrombin III level is <70% of the normal.

In our case serum albumin was 1.8g/dl with hyperlipidemia which is an important risk factor for thrombotic complications. Serum albumin was 1.6g/dl at her first presentation of nephrotic syndrome which is also an indication of prophylactic anticoagulation.

As seen in case 3, which is a case of CVT in pregnancy, coagulation factors I, II, VII, VII, IX and XII increase during pregnancy. In addition , oestrogen contributes to venodilation and congestion. Venous stasis results in hypercoaguable state in which risk of thrombosis increases . Virchow's triad (hypercoagulation, venous congestion, and tissue damage) reduce the risk of bleeding during labour, but they increase the risk of thrombo-embolism in the puerperium.

Other risk factors associated with thrombosis include post-partum haemorrhage, varicose veins, C-section, obesity, preeclampsia, associated malignancy, a genetic defect of coagulation inhibitor, anaemia (< 9.9 g/ dL) and placental abruption.

CONCLUSION

CVT is a serious cerebrovascular disorder. It has good prognosis if diagnosed and treated early.It is one of the differential diagnosis in ICH, eclampsia, PRESS, Acute ischaemic stroke, meningitits. Females, pregnancy, Nephrotic syndrome are risk factors for CVT in our cases. MRI with MRV is the investigation choice. Early diagnosis and treatment with anti-coagulants is beneficial irrespective of bleed.

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