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"Unravelling Sigmoid Sinus Thrombosis: A Clinical Case Study"

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KEYWORDS

Thrombosis, Sigmoid sinus, Serum electrolytes, Seizure, Alcohol dependency, Alcoholics

ABSTRACT:

Sigmoid sinus thrombosis (SST) is a rare but serious complication that can arise from chronic alcoholism, primarily due to its effects on coagulation and vascular integrity. Chronic alcohol consumption is associated with a hypercoagulable state, increasing the risk of venous thrombosis. In Sigmoid sinus thrombosis, thrombus formation in the sigmoid sinus can lead to increased intracranial pressure, neurological deficits, and even life-threatening conditions like cerebral venous sinus thrombosis. Symptoms often include headache, visual disturbances, and neurological deficits, which can mimic other conditions, complicating diagnosis. The management of Sigmoid sinus thrombosis typically involves anticoagulation therapy, addressing the underlying alcohol dependency, and monitoring for potential complications. This abstract highlights the need for increased awareness of Sigmoid sinus thrombosis in patients with chronic alcoholism and underscores the importance of early recognition and intervention to improve outcomes. Understanding the interplay between alcohol use and thrombotic events is essential for effective management and prevention strategies in this vulnerable population. A 29-year old male presented to the medicine department with seizure activity with MRI showing Left transverse and sigmoid sinus appear hypoplastic and thrombosed. The clinical presentation, examination findings and management have been discussed here.

INTRODUCTION:

Sigmoid sinus thrombosis is a rare but serious condition characterized by the formation of a blood clot within the sigmoid sinus, a major venous structure located at the base of the skull. This condition can lead to increased intracranial pressure and various neurological complications due to impaired venous drainage from the brain. Pathophysiology of the sigmoid sinus is that it is a part of venous drainage system of the brain, receiving blood from the internal jugular vein and the transverse sinus. Thrombosis in this area disrupts normal blood flow, potentially leading to venous hypertension, cerebral edema, and, in severe cases, ischemic brain injury(1). Its incidence is a very rare condition with an incidence of 1 to 5 cases per 1,000,000 people annually.

The most common cause of sigmoid sinus thrombosis is infection, particularly related to otitis media (middle ear infections) and mastoiditis. These infections can lead to inflammation and thrombosis in the venous sinuses due to direct invasion or increased pressure. Other factors, such as head trauma , hypercoagulable states such as Genetic conditions (e.g., Factor V Leiden) and Acquired conditions (e.g., pregnancy, certain cancers), Use of hormonal contraceptives increases the risk in female population. Out of all cases of cerebral and dural venous sinus thrombosis, malignancy as a cause accounts for 7.4% which shows that the risk of sigmoid sinus thrombosis in malignancy is increased by 5-fold. Alcoholism is also one of the contributing factors. The sigmoid sinus is located in a part of the dural venous sinuses, which are channels between the layers of the

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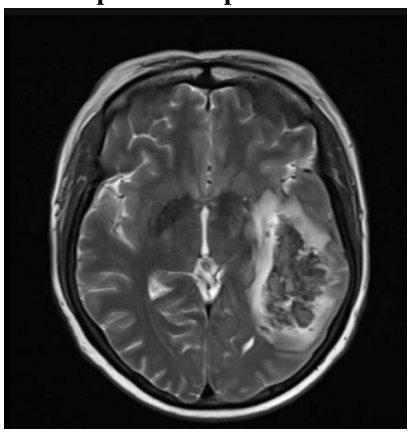
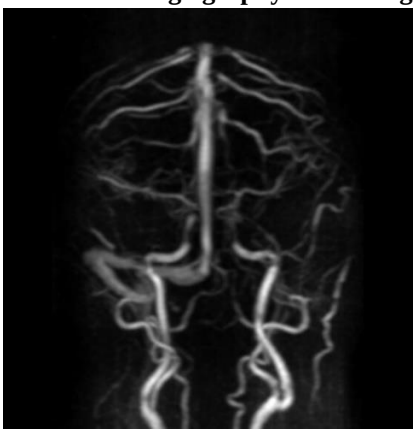
dura mater (the outermost membrane surrounding the brain and spinal cord). It is located in the posterior cranial fossa, near the base of the skull, and is a continuation of the transverse sinus. The course of sigmoid sinus curves downward in an S-shape (hence the name "sigmoid") and runs along the inner surface of the temporal and occipital bones .Its function is to collect venous blood from the transverse sinus and other surrounding venous channels, then drains into the internal jugular vein, which carries blood back to the heart. In Thrombosis of the Sigmoid Sinus a thrombus forms in the sigmoid sinus, it can block the normal drainage of venous blood from the brain, leading to increased intracranial pressure, reduced cerebral perfusion, and potential complications like brain swelling, stroke, or hemorrhage.

CASE PRESENTATION - A 29-year-old male patient, arrived at the medical department experiencing seizures at late night. He was casting for one minute, involuntary movements in the upper and lower limbs, salivary spitting, post-ictal disorientation, and a history of 1 episode of vomiting. He had another episode of seizure at casualty in an hour. He was casting for 10 seconds, He had involuntary motions of both Upper Limb and Lower Limb; eye rolling with frothing of saliva was noted. He is a chronic alcoholic who has been drinking every day for the previous 5 years. He last binged was the day before, and he also smokes. No history of tongue biting or incontinence during the seizure was noted. There is no history of fever and no history of fall. The patient had no pertinent past illness history. The patient had no pertinent personal or family history**.** On general examination, the patient was conscious, oriented of his surroundings, There was no pallor, pedal oedema, clubbing, cyanosis or lymphadenopathy or peripheral lymphadenopathy and no other significant abnormality was detected. On clinical examination , his vitals were BP -160/100 mmHg, PR – 107 pm, SPO_2 - 93 %, CBG - 275 mg /dl, temp - 101^{O} F. On neurological examination higher mental functions were normal, speech was fluent, there was no focal weakness, coordination was normal, no sensory deficit, reflexes were intact and cranial nerves were normal. Chest, cardiovascular and abdominal examination revealed no abnormality as Chest x -ray, ECG, Echocardiogram were normal. In Central Nervous System examination , it revealed right and left are normal in tone , power is 4/5 both. planter is extended in both sides. On admission he was primarily treated with heparin 6 unit s/c as anticoagulant. Antiepileptic injection levipil and injection lorazepam were given as anxiolytics. Cephalosporin group of Antibiotics Injection Xone were started after taking sample for culture. His serum electrolytes were Sodium - 123.8 mEq /L , Potassium - 2.81 mEq /L , Chloride - 86.1 mEq /L for which hydration and electrolyte corrections were done with injection 3% Nacl over 4-5 hours and injection Kcl 40 mg in 500ml NS over 4-5 hoursfor which his values got corrected to Sodium-130.8 mEq /L, Potassium -3.96 mEq /L, Chloride -101.5 mEq /L. Other Investigations had no significant changes. Thiamine injection was given to prevent complications of alcohol. Psychiatric counselling was also given to the patient.(2)

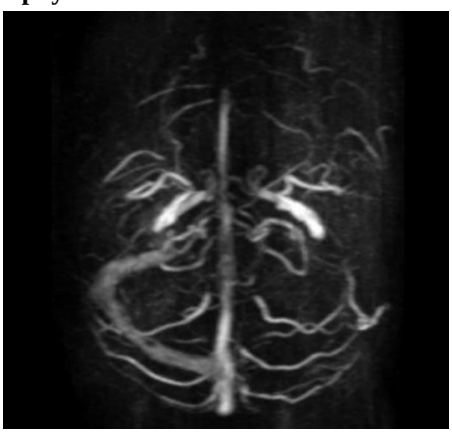
MRI Report of the patient is discussed. 3T MR Angiography and Venography are taken.



T2 SEQUENCE AXIAL



MRV-CORONAL PLANE



MRV-AXIAL PLANE

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SECTION OF MRI BRAIN

MRI brain with MRV gave the impression of venous sinus thrombosis involving right transverse, sigmoid sinus and proximal straight sinus.

IMPRESSION : Acute venous hemorrhagic infarct in the left posterior temporal and occipital lobes with adjacent thrombosed left vein of Labbe **.** Left transverse and sigmoid sinus appear hypoplastic and thrombosed. Mild edema seen in posterior aspect of left thalamus. Mild diffuse prominence of sulcal spaces.

DISCUSSION: Sigmoid sinus thrombosis is a rare but serious condition involving the formation of a blood clot (thrombus) in the sigmoid sinus, a venous channel located within the brain. This condition can impede blood drainage from the brain, leading to increased intracranial pressure, stroke, and other severe neurological complications. The sigmoid sinus is a part of the larger venous system of the brain, which helps drain blood from the brain into the jugular vein. The common differential diagnosis of sigmoid sinus thrombosis is bacterial meningitis as both the conditions may present with headache and altered mental status as Meningitis is more likely to present with fever, photophobia, and a positive Brudzinski or Kernig sign. Lumbar puncture shows elevated white cells, proteins, and decreased glucose (4). Other main challenge we face in this case is misinterrupting the diagnosis as cerebral venous sinus thrombosis which has a similar case presentation but can be differentiated from sigmoid sinus thrombosis by MRV CT Venography , ischaemic stroke which can also be differentiated with CT angiography or MRI as it will show arterial blockage rather than venous thrombosis(3), sometimes even Mastoiditis and otitis media can cause local infections that can spread to the sigmoid sinus and result in similar symptoms, including headache, fever, and ear pain; distinguishing feature in this case scenario will be Presence of ear pain, discharge, and mastoid tenderness with imaging (CT or MRI) showing localized infection rather than primary thrombus in sigmoid sinus. In certain hospital setups the unavailability of MRI scan with MRV features also hinders quick diagnosis for which they have to be referred to higher centres for treatment wherein the treatment gets delayed. In this case 29 year old young male with chronic alcoholic history was found to have Sigmoid sinus thrombosis which is the cause for seizure activity and other neurological manifestations. The main aim of treatment in sigmoid sinus thrombosis should be with heparin treatment to prevent further thrombosis by reducing high blood viscosity and in this case we did electrolyte and hydration status correction along with it and prevent further neurological manifestations.

CONCLUSION -In conclusion, Sigmoid sinus thrombosis can frequently result as a rare complication to chronic alcoholics, which is usually misdiagnosed or overlooked in clinical practice. Clinicians should investigate the possible cause for patients with postural headache, focal neurological impairments, or imaging signs of venous sinus thrombosis in MRI Brain in MRV , and do a lumbar puncture and cerebral venous sinus angiography. Patients whose clinical symptoms and imaging improve with conservative care with rehydration and anticoagulation probably do not need epidural haemorrhage patching, but should be followed for cerebral haemorrhage.

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