Case Report

Cerebral Sinus Venous Thrombosis (CSVT): A Case Report

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ABSTRACT

CSVT means cerebral sinus venous thrombosis here cerebral refers to the brain and sinovenous refers to the large vein that drain the brain. So, it is called as venous sinus. The system of vein found between the layers of dura matter the tough outer layer if your brain that lies directly under the skull. CSVT mainly occurs when a blood clot forms in the brain draining towards your heart .so, that the blood cells may break and leak the blood into the brain tissues forming a hemorrhage and also increasing the risk to cause of childhood and neonatal stroke. It is difficult to diagnosis with variable etiology and also it is multi conditional or multifactorial and prognosis, that require fine medical skills and a high suspicious index. It is a rare form of stroke the risk occurs mainly in new born, and also in the womb, teens, children up to 18 years of the age there is a risk for new mothers is especially high during the first few weeks after delivery. The symptoms appear in relation increase intracranial pressure and imitating a pesuditumor cerebri.

Keywords: Brain, CSVT, children, stroke.

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INTRODUCTION

Cerebral sinus venous thrombosis (CSVT) is a rare type of venous thromboembolism (VTE).[1] CSVT accounts for almost 0.5% -3% of all the strokes,[2] affecting primarily younger people,[3] with an estimated incidence of 3-4 per million in adults and 7 per million in children.[4] When a blood clot forms in the venous sinuses of the brain, it is known as cerebral venous sinus thrombosis (CVST). The clot prevents blood from leaving the brain. As a result, the blood arteries become clogged with pressure. This can cause brain enlargement and bleed (hemorrhage).[5] This sequence of events is part of a stroke, which can affect both adults and children. Even newborns and kids in the womb are susceptible. A stroke can damage the brain and central nervous system. A stroke is a dangerous condition that needs immediate medical intervention.[6] Hereditary prothrombotic disorders, antiphospholipid antibody syndrome (APLS), cancer, pregnancy, autoimmune diseases, and infections are all common causes of CVST.[7] It can lead to increased intracranial pressure, encephalopathy, strokes, cranial nerve palsies, seizures, and headaches.[8] Increased intracranial pressure, encephalopathy, strokes, cranial nerve palsies, seizures, and headaches are all symptoms.[9] The cause of headache is most likely nerve compression within veins ,[10] increased intracranial pressure, or, in rare cases, venous infarction. [11,12] CVST-induced headaches are difficult to distinguish from other main headache conditions. Most CVST headaches have a subacute onset and generate generalized throbbing pain. Neurologic impairments, photophobia, or symptoms of elevated intracranial pressure are some other neurological abnormalities.[13-16] CVST can sometimes cause damage to the brain parenchyma.[17] If there is a high suspicion following an unenhanced head CT, CT venography and MRI can be employed. Depending on the cause of CVST, the European Academy of Neurology suggests intravenous heparin or subcutaneous low molecular weight heparin first, followed by oral anticoagulation for at least 3 to 12 months. [18]

Demographic Details:

Name: Sridhar

Age:16yrs

Gender: male

Weight: 70 kg

A 16-year-old male was admitted with chief complaints of headache with 4-5 episodes since last 1 yr. For 6 months, the headache lasted for 2-3hours in the left frontal region and subsided spontaneously. Headache in the holocrine region, dull, achy continuous, headache aggravated in the last 5 days–15 minutes over the frontal region and also left eye was blurred vision since morning but subsided. History of giddiness' since 1 yr. On and off 4-5 episodes in a month after getting up from supine and sitting position, lasting for 30 sec -1minute each time. Vomiting with nausea multiple episodes in one day. The patient has decreased appetite

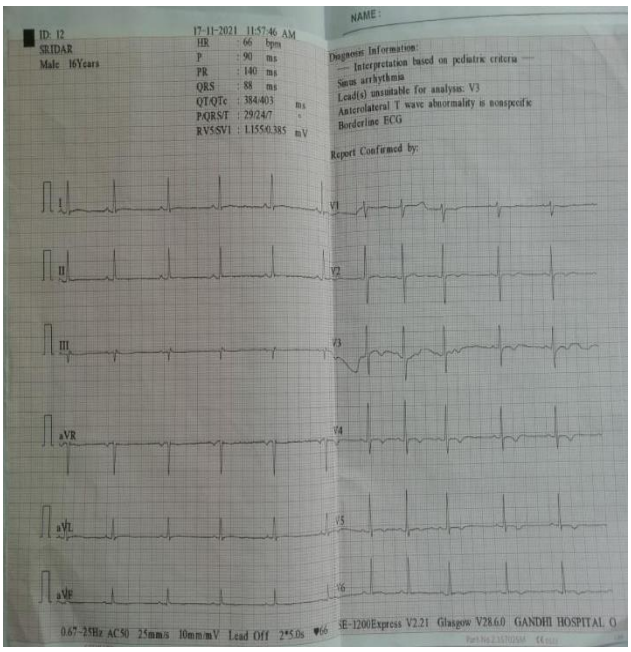
and sleep. The patient has no history of seizures, rhinorrhea, or lacrimation.

The patient was examined, and the temperature was afebrile, the pulse rate was: 69 bpm, spo_2:100%, central nervous system: no abnormality detected, Blood Pressure: 90/6mmhg.

Socio-economic history

The patient belongs to a middle–class family. They live in a rented house with all the facilities. They maintained proper hygiene around the surroundings.

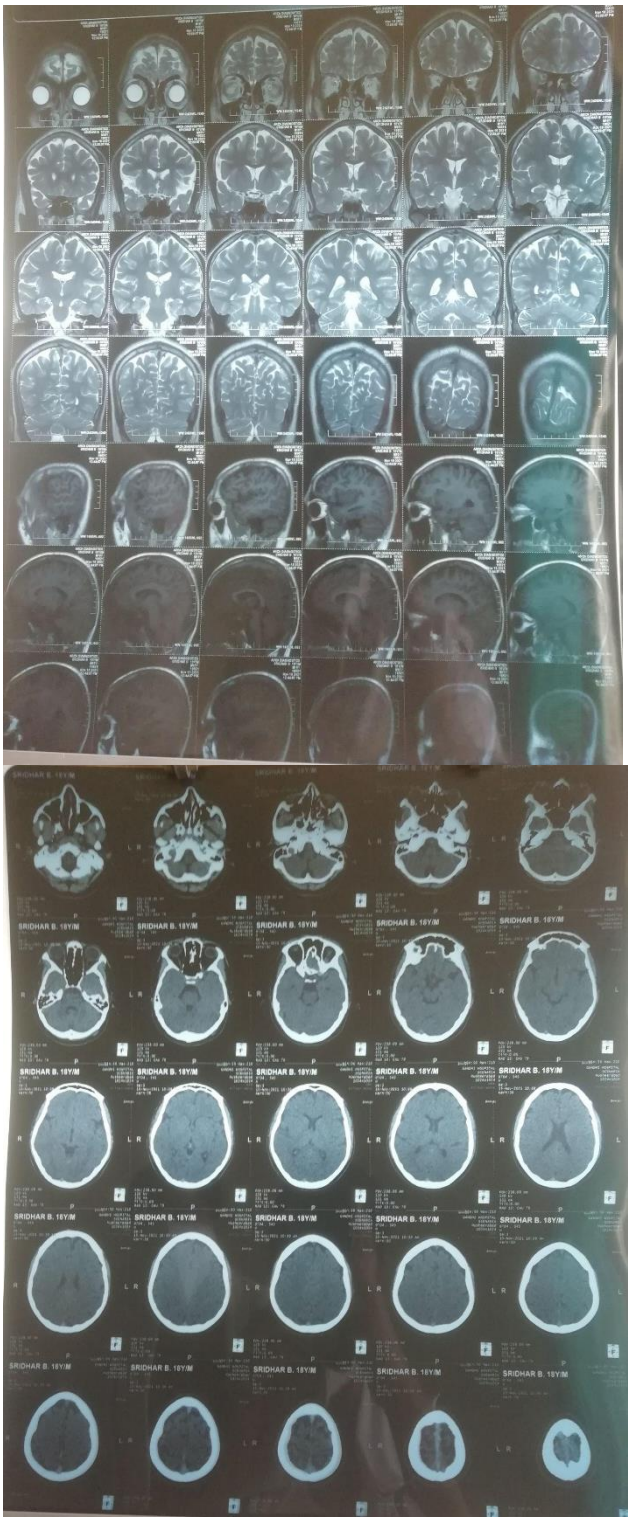
Laboratory Findings



ECG: the impression was T-wave is decreased v3-v5 flattering of T-wave

MRI with MRA with MRV:

1. Small focal area of blooming in SW images, with hypersensitivity in-phase images in left parietal lobe - calcified granuloma.
2. Absent flow related signals in superior sagittal sinus with linear filling defect in right transverse lines– acute to sub-acute Dural venous sinus thrombosis.
3. Hypo plastic left transverse and sigmoid sinus.
4. All major intracranial arteries show the normal caliber.



Treatment:

We started the patient with the treatment on admission

1. Inj. Mannitol 100ml/Iv/TID for 5 days
2. Inj. Low molecular weight heparin 60 mg/ SC for 5 days,
3. Inj. Pantoprazole 40 mg/iv /OD,
4. Inj. Ondansetron 4 mg iv BD,
5. Tab. Naproxen 250 mg BD Day 3

6. Syp.laculose was added on day 3 PO/TID 5ml

Day 4 head ached was on and off, so they added

7. Tab.warfarin 4mg was OD was given

After giving warfarin and heparin they advised to take PT INR After 24 hours.

On the day 7 patient with no complaints was treatment was given

They added oral glycerol 5ml.

DISCUSSION

In 80% of cases, predisposing risk factors are discovered. Pro-thrombotic disorders, head injuries, inflammatory diseases, dehydration, and malignancy are examples of these.

Magnetic Resonance Venography is the preferred and most sensitive diagnostic test, as it permits the venous blockage to be identified together with any consequences, such as cerebral edema and areas of venous infarction, as was the situation in our instance.

Stabilization, anticoagulants, medication, and/or repair of the underlying disorders are all part of the management of a patient with proven CVST. Despite the risk of hemorrhage in venous infarcts, anticoagulation is the mainstay of treatment, even if a hemorrhagic venous infarct already exists, thus we usually give heparin after the diagnosis is confirmed. Following introducing heparin, our patient showed modest clinical improvement. [19]

During the treatment, by using the following combination of drugs, the patient may have a risk of:

1. Warfarin and heparin

The oral anticoagulants may prolong the activated partial thrombosis time because patient is receiving heparin, while heparin may prolong the PT INR in patient is receiving warfarin.

2. Warfarin and sodium valproate

Sodium valproate transiently potentiates the hypo prothrombic effect of warfarin and other oral anti coagulants.

3. Naproxen with Warfarin

NSAID with oral anticoagulants potentiate the risk of bleeding. [20]

CONCLUSION

I have taken this case from the general medicine department. I have dealt with history collection, physical examination, investigations, and drug studies. I observed the patient for 7 days and gave health education to the patient and to the parents. The patient and his parents were following the given instructions.

The primary aim of this disorder is to educate parents, better knowledge of cerebral sinus venous thrombosis (CSVT), better parental counseling about its complications, prevention of further prognosis and recurrence. CSVT is a rare type of venous thromboembolism, affecting primarily younger people.

Abbreviations

CVST- cerebral venous sinus thrombosis

Consent

The written informed consent was taken from the patient for publication of this case report.

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