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Hemorrhagic Infarction secondary to Cerebral Sinus Venous Thrombosis - a case report.

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Abstract:
We describe a 24-year-old patient who presented to the emergency department after a generalized tonic-clonic seizure episode. He reported focal repetitive jerks of right face and right upper limb for the past 3 days which then evolved into generalized tonic-clonic seizure. He also complained of an on and off headache for the past one month and palpitations for the past three months. He was a chronic alcoholic. He had no co-morbidities and reported no history of previous seizure episodes. On examination, he was conscious, oriented, and afebrile. His vital signs were normal and there were no signs of meningeal irritation. However, neurological examination showed decreased power in the right upper limb and right lower limb. Routine investigations were performed and the results were normal. The patient was suspected to have epilepsis partialis continuum with right todd's palsy. However, the MRI scan showed minimal diffuse restriction in the bilateral frontal region and T2/FLAIR hypersensitivity along with the left pre and paracentral gyrus. Considering his age, alcoholic history, and the insidious onset of headache, Cerebral Sinus Venous Thrombosis(CSVT) was suspected. MR Venogram showed absence of flow in frontoparietal part of superior sagittal sinus, left transverse and left sigmoid sinus and superior cortical veins on the left side showed which further confirmed the diagnosis. A diagnosis of hemorrhagic infarct in the left frontoparietal region with dural and cortical venous thrombosis was made. Further investigations were done to evaluate the cause for CSVT but the results were normal. The likely cause of CSVT could be due to dehydration from chronic alcoholism. The treatment was started immediately with heparin, mannitol, anticonvulsants, and antibiotics. The patient's condition started to improve. Unlike arterial strokes, CSVT has varied clinical presentations, thus posing a diagnostic challenge. Therefore, CSVT must be considered in young patients without vascular risk factors who complain of new-onset progressive headaches. In this way, the cause of CSVT can be evaluated earlier and the condition can be treated even before the development of neurological deficits.

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I. Introduction

With the introduction of advanced diagnostic imaging techniques, cases of CSVT have been increasingly reported. However, the clinical picture of CSVT is ambiguous and we often miss the diagnosis. Many a time, the presentation mimics epilepsy or hemorrhagic stroke and our focus gets diverted from CSVT towards other conditions. Here is one such case where the patient was initially diagnosed as epiplepsis partialis continuum with right todd's palsy. However, further investigations proved it to be wrong. Patients with CSVT need prompt treatment to prevent future neurological complications. Moreover, it has an excellent prognosis if treated early. Therefore, a high clinical and radiological suspicion is necessary for the early detection and treatment of CSVT.

II. Case Report

A 24-year-old male was brought to the emergency department after a seizure episode. He had generalized tonic-clonic seizures of the right upper limb and both lower limbs for 10 mins with uprolling of eyes, drooling of saliva, urinary incontinence, and clenching of teeth. He reported focal repetitive jerks of right face and right upper limb for the past 3 days without loss of consciousness which then evolved into generalized tonic-clonic seizure. The patient also had a history of headaches on and off for 1 month, palpitations for 3 months, with loss of appetite. He was a chronic alcoholic. The patient had no co-morbidities. The patient had no history of seizure disorder or tuberculosis.

On examination, the patient was conscious, oriented, and afebrile. His vitals include blood pressure – 110/60 mm Hg, pulse rate – 88/min, respiratory rate – 16/min. There were no signs of meningeal irritation.
CNS examination showed reduced power in the right upper limb and right lower limb.

- right upper limb – 3/5
- left upper limb – 5/5
- right lower limb – 4/5
- left lower limb – 5/5

Routine investigations were performed and the findings are listed below:

Table no 1: Complete blood count

Hemoglobin	12.3 g/dl
RBC	4.1 million/mm3
HCT	40%
MCV	85 fL
MCHC	34.4 g/dl
WBC	7,000 cells/mm3
Neutrophils	60%
Lymphocytes	5.7%
Platelet count	2.5 lakh/mm3

Table no 2: Renal function test

Random blood sugar	75 mg/dl
Urea	18 mg/dl
Creatinine	0.8 mg/dl
Sodium	140 mEq/L
Potassium	3.5 mEq/L

Table no 3: Liver function test

Serum bilirubin Direct Indirect	1.4 mg/dl 0.4 mg/dl 1.0 mg/dl
SGOT	36 U/L
SGPT	127 U/L
Serum ALP	77 U/L
Total protein	5.5 g/dl
Albumin	3.0 g/dl
Globulin	2.5 g/dl

Table no 4: Other investigations

HIV	Non-reactive
Serum cholesterol	71 mg/dl
Serum triglycerides	82 mg/dl

The results showed no abnormal findings. The patient was suspected to have epilepsis partialis continuum with right todd's palsy and was subsequently treated for it. However, on MRI, minimal diffuse restriction in the bilateral frontal region, T2-FLAIR hypersensitivity along the left pre and paracentral gyrus and internal T2 hypointensity was noted. On MR Venogram, the frontoparietal part of the superior sagittal sinus left transverse and left sigmoid sinus and superior cortical veins on the left side showed absence of flow, which was suggestive of venous thrombosis. A diagnosis of hemorrhagic infarct in the left frontoparietal region with dural and cortical venous thrombosis was made.

Figure no 1

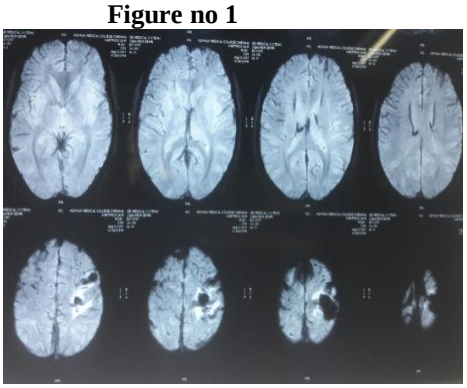


Figure no 2

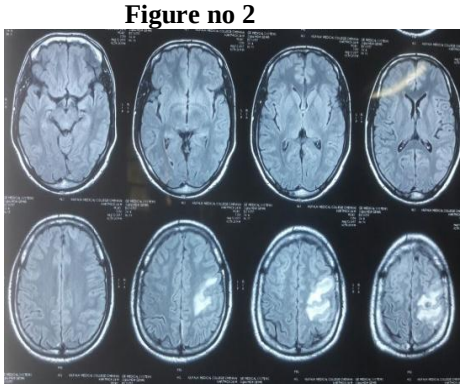


Figure no 3

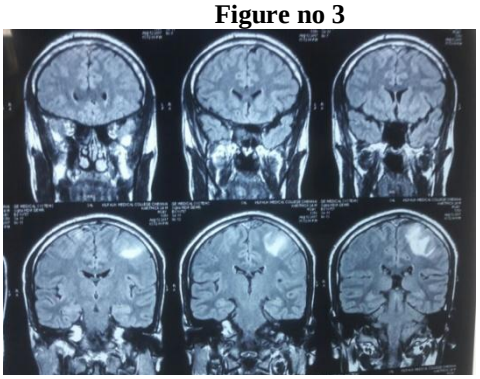
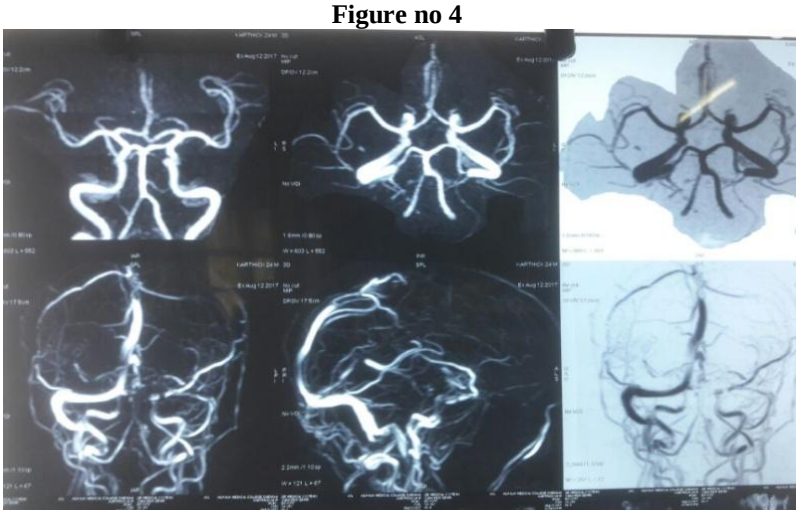


Figure no 4



Fundoscopy showed no evidence of papilloedema. A neurologist was consulted and the following investigations were done to evaluate the cause of CSVT in this patient. The findings are listed below:

Table no 5:

ESR	15 mm/h
PT INR apTT	13.2 seconds, 0.94 35 seconds
Antinuclear antibodies	Negative
Serum homocysteine	10 µml/L
Protein C/S, antithrombin levels	normal

The results were unremarkable. The cause could be attributed to dehydration from chronic alcoholism. The patient was treated with heparin, mannitol, anticonvulsants, and antibiotics. Upon review 5 weeks later, the patient was symptomatically better and reported no recurrence of seizures.

III. Discussion

Cerebral Sinus Venous Thrombosis (CSVT) refers to the occlusion of venous channels in the cranial cavity, including Dural venous thrombosis, cortical vein thrombosis, and deep cerebral vein thrombosis [1]. CSVT represents almost 0.5% -3% of all types of stroke, affecting predominantly younger people [2]. CSVT is rare but can cause serious neurological complications including death.

CSVT is a condition with variable aetiologies, presentation, and prognosis. The aetiology is often gender-specific and varies between developed and underdeveloped countries. Some of the risk factors include thrombophilia e.g. factor V Leiden, deficiency of protein C, protein S or antithrombin deficiency, Pregnancy, OCPs, particular blood disorders especially polycythemia vera and paroxysmal nocturnal hemoglobinuria, septic foci, trauma, sickle cell anemia, SLE, APLA, dehydration, homocysteinemia, and COVID-19 [3]. In 20% of patients, no specific cause can be found. In this patient, the etiological workup was unremarkable. However, one possible explanation for CSVT in this patient could be dehydration resulting from chronic alcoholism. It is important not to test for prothrombotic conditions after starting the patient with anticoagulant therapy.

The major forms of clinical presentation include isolated intracranial hypertension syndrome, focal neurological deficits, and cavernous sinus syndrome [4]. However, headache is the most common presenting symptom in CSVT patients. The mechanisms of neurological symptoms in CVT include (1) Development of intracranial hypertension as the result of occlusion of the major venous sinuses and (2) localized oedema of the brain, venous infarction, and petechial haemorrhages [5]. Due to thrombosis, venous congestion and hemorrhagic infarction of brain parenchyma can occur which further result in cerebral oedema and mass effect. The neurological deficits depend on the area of the brain that is affected.

MRV findings include flow defects and the presence of collaterals at the site of occlusion. The thrombus signal diminishes in the third week [6]. So, early detection is crucial. In the case of chronic CSVT, angiography should be done to confirm the site of occlusion.

The degree of hemorrhagic venous infarction in CSVT is one of the important prognostic factors. Deep cerebral venous thrombosis also has a negative impact on prognosis due to the bilateral involvement of the thalami [7].

The mainstay of management of thrombosis of the dural sinus and cerebral veins (CVT) is systemic heparinization [8]. Heparin is considered safe even in the presence of intracranial hemorrhagic lesions [9]. In case of severe deterioration despite heparin therapy, thrombolysis or open thrombectomy has proven to be beneficial [10]. Decompressive surgery may be needed in case of herniation.

Due to the wide variability in the clinical and radiological picture of cerebral sinus venous thrombosis (CSVT), it is often overlooked. It is important to differentiate CSVT from epilepsy and hemorrhagic stroke so that timely heparinisation can be started. Accurate diagnosis and early treatment are life-saving. This patient's young age and insidious onset of headache made us suspect CSVT. MRI with contrast MRV further confirmed the diagnosis.

A non-contrast CT scan is a part of routine investigation performed in patients with stroke. Although certain signs in CT indicate CSVT, such signs occur in a small proportion of patients. Magnetic Resonance Venography with contrast is the investigation of choice to confirm CSVT. Therefore, a high clinical and radiological suspicion is necessary for the early detection and treatment of CSVT.

References:

[1]. Gaillard F, Bell D. Cerebral venous thrombosis. *Radiopaedia*.. 2008. https://doi.org/10.53347/rID-4449.
[2]. Ruiz-Sandoval JL, Chiquete E, Bañuelos-Becerra LJ, Torres-Anguiano C, González-Padilla C, Arauz A, León-Jiménez C, Murillo-Bonilla LM, Villarreal-Careaga J, Barinagarrementería F, Cantú-Brito C. Cerebral venous thrombosis. *J Stroke Cerebrovasc Dis.* 2012 Jul; 21(5):395-400.
[3]. Stam J. Thrombosis of the cerebral veins and sinuses. *N Engl J Med.* 2005;352:1791-8.
[4]. Idiculla PS, Gurala D, Palanisamy M, Vijayakumar R, Dhandapani S, Nagarajan E. Cerebral Venous Thrombosis: A Comprehensive Review. *Eur Neurol*. 2020; 83:369-379.

[5]. Saposnik, Gustavo, Barinagarrementeria,Fernando, Brown,Robert D, Bushnell,Cheryl D, Cucchiara,Brett, Cushman,Mary, deVeber,Gabrielle, Ferro,Jose M, Tsai,Fong Y. Diagnosis and Management of Cerebral Venous Thrombosis. *American Heart Association.* 2011; 42(4): 1158-1192.
[6]. Alvis-Miranda HR, Milena Castellar-Leones S, Alcala-Cerra G, Rafael Moscote-Salazar L. Cerebral sinus venous thrombosis. *J Neurosci Rural Pract*. 2013; 4(4):427-438.
[7]. Herrmann K, Sporer B, Yousry T. Thrombosis of the Internal Cerebral Vein Associated with Transient Unilateral Thalamic Edema: A Case Report and Review of the Literature. *AJNR Am J Neuroradiol*. 2004;25(8):1351-5.
[8]. Shaileshkumar S Garge, Virti D Shah, Nirmal Surya, Satish S Khadilkar, Pranav D Modi, Sharad B Ghatge. Role of local thrombolysis in cerebral hemorrhagic venous infarct. *Neurology India*. 2014; 62(5): 521-524.
[9]. Ferro JM, Canhao P. Acute treatment of cerebral venous and dural sinus thrombosis. *Curr Treat Options Neurol*. 2008;10:126-37.
[10]. Medel R, Monteith SJ, Crowley RW, Dumont AS. A review of therapeutic strategies for the management of cerebral venous sinus thrombosis. *Neurosurg Focus*. 2009 Nov. 27(5): E6.

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