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A Case Report of Concomitant Cerebral Venous Sinus Thrombosis and Intracerebral Hematoma in a Young Male Patient: Diagnosis and Management Particular Challenges

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Abstract

The diagnosis of cerebral venous thrombosis (CVT), a rare kind of stroke, may be difficult due to its complicated clinical manifestations, which can delay the timely start of anticoagulation. The therapeutic care of hemorrhagic transformations is more complicated. We reported a 32 year old male patient who had cerebral venous thrombosis. He was admitted to MZH in August 2024. At various phases of the illness, the case represented major challenges in the treatment, diagnosis, or causal evaluation. The possible long-term implications for the patient include epileptic seizures, depressive symptoms, and other behavioral issues, which are late complications. This case is a scenario of a profound cerebral vein thrombosis, which required critical care, and led to focal epileptic fits and deterioration in consciousness.

Keywords: Cerebral Venous Thrombosis; Seizures; Intracerebral Hemorrhage

Abbreviations

CVT: Cerebral Venous Thrombosis; ICP: Intracranial Pressure.

Introduction

Although cerebral vein and dural sinus thrombosis (CVT) are uncommon forms of stroke, with a median age of thirty-seven years old, they rank among the leading causes of stroke in young individuals [1].

According to epidemiological data, CVT has a yearly incidence of 1.16 to 2.02 instances per 100,000 people and is more common in women than in men (3:1) since it is linked to the administration of contraceptive pills, pregnancy, and the postpartum phase. Other prothrombotic causes include general or local infections, cancer, and blood clotting disorders [1,2].

Intracranial pressure (ICP) rises in cases of thrombosis of the veins or venous sinuses due to changes in the blood brain barrier and decreased absorption of CSF. This may be the only sign of the disease in twenty-six percent of cases, which could lead to a false diagnosis [3]. In seventy to ninety percent of patients, headache is the main presenting symptom in CVST, and frequently occurs in the absence of concomitant neurological signs or impairments. Headaches can occur suddenly, with an abrupt onset similar to subarachnoid hemorrhages. Although they are less common, additional symptoms include altered consciousness, papilledema, seizures, and localized neurological signs. Physicians should

retain a high suspicion of CVST due to the nonspecific aspect of such symptoms, particularly in situations including a headache that is new and of progressive course [4,5]. The superficial venous sinuses are the most typical site, and many sinuses are usually affected. The main goals of treating acute CVT are to stop the thrombus from getting more pronounced, restore venous circulatory function, and avoid thrombotic recurrence [4]. Due to its numerous clinical symptoms and complex radiological diagnosis, CVT needs to be cautiously handled, especially when relating to the course of treatment. The outcome for CVT is generally good [6]. On the other hand, a number of uncommon coexisting complications, such as focal neurological impairments or altered levels of consciousness, as well as long-term epileptic attacks or mental illnesses, may occasionally exacerbate it [7].

Case Report

History of Present Illness

Thirty two years old male patient presented with headache, slurred speech, one attack of epilepsy and altered consciousness on admission. Not known to have medical disease or history of trauma. The headache is generalized, located bilaterally, frontal area, temporal area and parietal area. The headache is described as aching. The severity of the headache is severe. The headache is constant. The headache has lasted for 2 days before admission. Exacerbating factors consist of noisy environment. Relieving factors consist of medication and rest. Associated symptoms consist of. Past history old left lower limb weakness -wasting due to old polio myelitis. Additional pertinent history: occasional caffeine use, tobacco use none and no alcohol use. By neurological examination: altered consciousness, GCS was 14, slurred speech, intact motor and sensory systems, intact cranial nerves, no meningeal irritation signs or neck rigidity, stable vital signs and normal general examination.

CT brain done showed: evidence of left temporoparietal subcortical and deep sub cortical blood density of intraparenchymal haemorrhage measuring about 30 to by 30 x 36 mm. Mild mass effect and midline shift to the contralateral side seen Figure 1 (A-H).

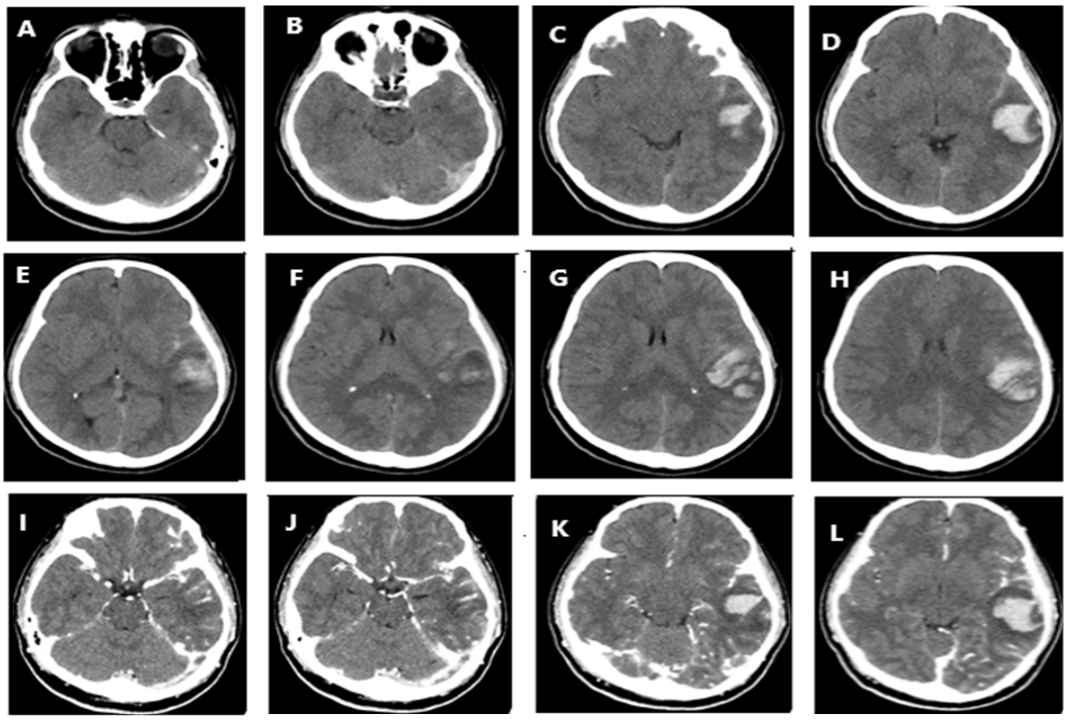

Figure 1: CT brain venography with contrast injection_showed that the left transverse and sigmoid dural venous sinuses appear of increased attenuated density in the non-contrast study and nonopacified in the contrasted study which signifying venous thrombosis. The left internal jugular vein is also seen non opacified at the level of the the jugular foramen Figure 1 (I-L).

Initial impression and management as a case of extensive CVT including left IJV with ICH after ICU admission was administration of enoxaparin therapeutic dose 70 mg sc bid and loading dose 2 g levetiracetam then 1000 mg iv bid. Neurosurgeon opinion advice to continue conservative management with close observation of consciousness and any deterioration of neurological condition (Figure 2).

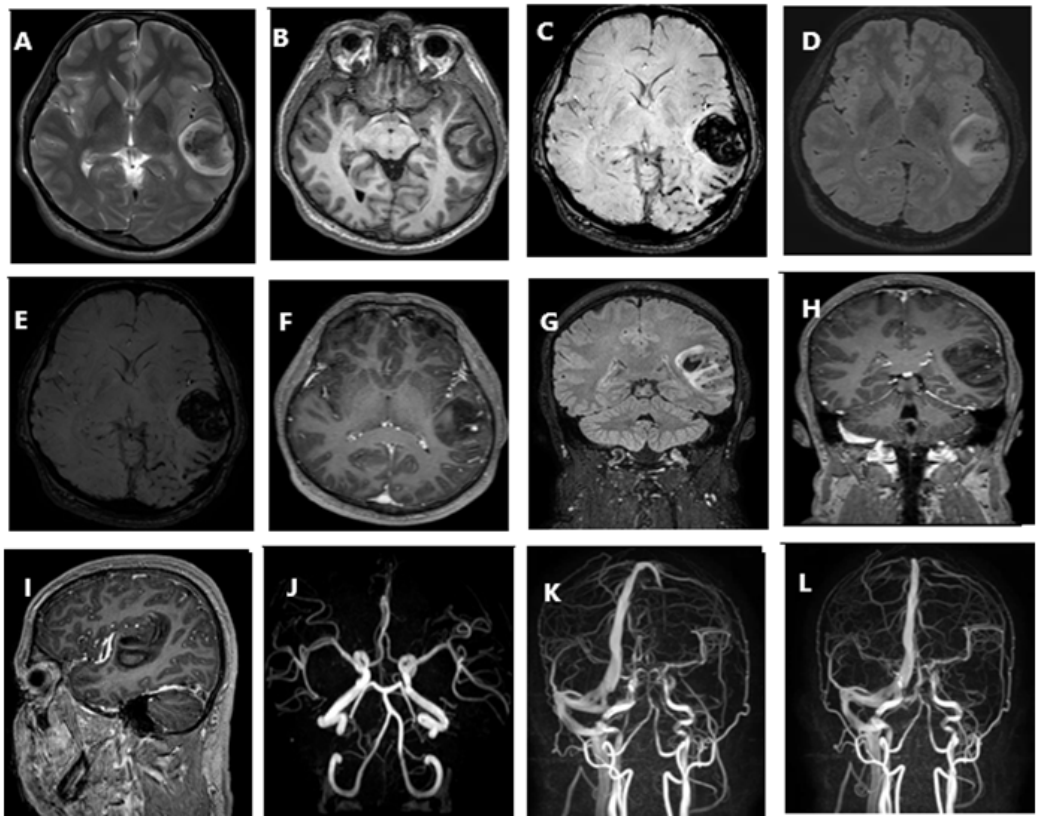

Figure 2: Multiplanar MRI of the brain with MRA MRV reconstruction with contrast injection was done and showed evidence of left temporoparietal cortical and deep sub cortical altered signal intensity lesion of acute/subacute blood intensity measuring about 31 x 56 x 40 mm, the parenchymal brain haematoma is surrounding by mild soft tissue oedema with mild mass effect to the related structure with minimal pressure effect at the brain ventricular system with no significant midline shift, no definite underlying pathological lesion could be seen, picture highly suggestive of lobar venous parenchyma brain haemorrhage Figure 2 (A-H). Nonvisualization of the left sigmoid and transverse sinus due to thrombosis , other dural venous sinuses including the superior sagittal, straight and right transverse sinus appear normal Figure 2 (I-L).

Two days after admission, patient complain of sudden deterioration of consciousness, severe headache and vomiting. Urgent follow up CT brain showed increase midline shift and uncal herniation compared with previous scan. IV administration of manitol 20% for 2 days and dexamethasone were rapidly administrated and neurosurgeon opinion advice to continue conservative management with close observation of ICP for probable intervention by decompressive craniotomy. Patient was intubated ventilated and on ICP monitor for 7 days.

Patient consciousness improved and manifestations of increased ICP subsided, ventilation and sedation removed and ICP monitoring were removed after 7 days. Patient regained consciousness completely with no neurological deficit. Follow up CT scan of brain without contrast showed some improvement with decrease of ICH size and surrounding brain edema. Patient was discharged from the hospital functionally independent and able to return to home country.

Discussion

In clinical practice, identifying concomitant intracerebral hemorrhages with CVST constitutes a significant obstacle. There are multiple reasons for this complexity. At first, CVST symptoms might be vague and frequently coexist with those of other neurological disorders. Since headaches are a defining symptom of many neurological illnesses, it is difficult to diagnose CVST based only on clinical presentation. Moreover, the simultaneous occurrence of intracerebral bleeding increases the diagnostic problem. There is a chance that hemorrhagic events will obscure the usual symptoms of CVST or be mistakenly linked to bleeding alone, delaying diagnosis [8].

It is necessary to describe the distinct pathophysiology of cerebral venous sinus thrombosis which is based on two interrelated events: elevated intracranial pressure (ICP) from an obstructed venous system, which can cause

papilloedema and isolated intracranial hypertension, as well as global symptoms caused by venous infarct, such as hemiparesis. The wide range of pathophysiology associated with CVST facilitates comprehension of the variety of clinical manifestations [9,10].

Patients with CVST had a greater tendency to experience seizures than those with arterial stroke [11]. This may be related to the build-up of waste products that arise from venous obstruction. About 25% of patients with CVST have focal convulsions, while another 25% start out with focal convulsions that eventually become generalized. Generalized seizures were present in the remaining 50% from the beginning [12].

Neuroimaging and laboratory testing are essential in the diagnosis of CVST. D-dimer levels are a useful marker for people who have a low risk of CVST. Even while a routine CT or MRI scan is helpful in the preliminary evaluation of suspected CVST, a negative result does not prove the illness is not present. Under these circumstances, a venographic examination either magnetic resonance or CT venography is considered essential to verify or delineate the extent of CVST. The American Heart Association's evidence-based guidelines suggest starting antiepileptic medication early for patients who have experienced a single seizure. The routine prescription of antiepileptic medication is not recommended in the absence of seizures. It is considered appropriate to try vitamin K antagonists after low molecular weight or unfractionated heparin for initial anticoagulation. When there is considerable cerebral bleeding or a mass effect that causes persistently high intracranial pressure, decompressive craniotomy is a reasonable therapy option [13].

Conclusions

This case study offers insightful information about how to handle a challenging situation involving a 29-year old male Pakistani who has been diagnosed with intraparenchymal hemorrhage and CVST. The planned course of treatment, consisting of anticoagulation and antiepileptic drugs, gave priority to regular medical care over decompressive surgery. The patient's family members participated in cooperative communicates that informed this choice.

The most significant conclusion is the value of a customized, interdisciplinary strategy in addressing the difficulties presented by CVST instances. This means keeping a high threshold for suspicion of CVST, particularly when nonspecific symptoms like headaches and seizures are present. Comprehensive diagnostic examinations, including laboratory testing and neuroimaging, are essential for enabling early detection. Eventually, physicians can rely on these experiences to deal with similarly complex clinical circumstances requiring CVST, taking into consideration numerous contributing aspects and participating in coordinated decision-making.

Declaration of interest

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Authors' Contributions

This work was carried out in collaboration among all authors. 'Author AE' designed the study, performed the statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. 'Author DM' and 'Author MS' managed the analyses of the study. 'Author HA' managed the literature searches. All authors read and approved the final manuscript.

References

1. Pleșa FC, Jijie A, Toma GS, Ranetti AE, Manole AM, et al. (2023) Challenges in Cerebral Venous Thrombosis Management-Case Reports and Short Literature Review. Life (Basel) 13(2): 334.

2. Durmuş B, Yperzeele L, Zuurbier SM (2020) Cerebral venous thrombosis in women of childbearing age: diagnosis, treatment, and prophylaxis during a future pregnancy. Ther Adv Neurol Disord 13: 1756286420945169.

3. Tuță S (2022) Cerebral Venous Outflow Implications in Idiopathic Intracranial Hypertension-From Physiopathology to Treatment. Life 12: 854.

4. Capecchi M, Abbattista M, Martinelli I (2018) Cerebral venous sinus thrombosis. J Thromb Haemost 16: 1918-1931.

5. Shabbir T, Hunsucker R, Martin D, Shabbir Z, Abou-El-Hassan H, et al. (2023) A 44-year-old male with cerebral venous sinus thrombosis. Cureus 15: 36974.

6. Luo Y, Tian X and Wang X (2018) Diagnosis and Treatment of Cerebral Venous Thrombosis: A Review. Front Aging Neurosci 10: 2.

7. Mehvari HJ, Saadatnia M, Tabrizi N (2018) Seizure in cerebral venous and sinus thrombosis. Epilepsia Open 3(3): 316-322.

8. Torere BE, Weigold J, Aiwuyo HO, Alugba G, Ntukidem O, et al. (2023) Diagnostic and therapeutic challenges of concurrent intracranial hemorrhage and cerebral venous thrombosis in a patient with acute lymphoblastic leukemia: a case report and literature review. Cureus 15: 37482.

9. Itrat A, Shoukat S, Kamal AK (2006) Pathophysiology of cerebral venous thrombosis--an overview. J Pak Med Assoc 56(11): 506-508.

10. Schaller B, Graf R (2004) Cerebral venous infarction: the pathophysiological concept. Cerebrovasc Dis 18: 179-188.

11. Davoudi V, Keyhanian K, Saadatnia M (2014) Risk factors for remote seizure development in patients with cerebral vein and dural sinus thrombosis. Seizure 23: 135-139.

12. Mu S, Li J, Lin K, Fang Y, Lin F, et al. (2022) Predictive Factors for Early-Onset Seizures in Patients With Cerebral Venous Sinus Thrombosis. Front Neurol 13: 842807.

13. Saposnik G, Barinagarrementeria F, Brown RD Jr, et al. (2011) Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 42: 1158-1192.