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CASE REPORT

Cerebral venous sinus thrombosis with concomitant subdural hemorrhage and subarachnoid hemorrhages involving cerebral convexity and perimesenchephalic regions: A case report

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Abstract

We should suspect cerebral venous sinus thrombosis in patients with subacute onset progressive headaches with brain imaging evidence of localized cerebral edema with superimposed parenchymal, convexity subarachnoid as well as subdural hemorrhages.

KEYWORDS

cerebral venous sinus thrombosis, subarachnoid hemorrhage, subdural hemorrhage

1 | INTRODUCTION

Cerebral venous sinus thrombosis causing convexity and perimesencephalic subarachnoid as well as subdural hemorrhages simultaneously is very rare in medical literature. A high index of suspicion is required to timely, accurately diagnoses, and manages this challenging disease, as the clinical and imaging features may be very subtle.

Cerebral venous sinus thrombosis (CVST) is a very uncommon but an important cause of stroke, especially in young adults.[1] Its annual incidence rate ranges from 0.22 to 1.57 per 100,000 and is more common in women than men.[2,3] It is a difficult diagnosis to make because of its highly variable clinical presentation and subtle radiographic picture.[4,5] Even though rupture of aneurysms and arteriovenous malformations are the leading causes of subarachnoid hemorrhage (SAH), CVST can also be rarely associated with SAH.[6] Subdural hemorrhage (SDH) as a complication of CVST is even rarer and can easily be overlooked.[7] The diagnosis of this is even more difficult, yet can be very rewarding if done in a timely fashion.[8] In this article, we present a middle-aged woman whose presentation was with a worsening headache and a generalized tonic clonic seizure. She was initially presumed to have as aneurysmal SAH based on the computerized tomography (CT) scan of the head. However, upon further close review, she was found to have a subtle left transverse sinus thrombosis.

2 | CASE PRESENTATION

A 58-year-old right-handed postmenopausal woman presented to the emergency with progressively increasing holocephalic headache for 3 days. She did not have fever, nausea, and vomiting. The headache gradually worsened in severity and on the day of presentation, she developed a generalized tonic clonic seizure. Apart from the history of

well-controlled diabetes mellitus, she did not have significant past medical, personal, and family history. She had not received any vaccines in the recent past. On examination, she was ill-looking, but her vital signs were stable. She had Wernicke's aphasia. She did not have disc edema; cranial nerve examination was normal, and she did not have any focal deficits.

An urgent CT scan of the head without contrast showed left temporal hematoma with surrounding vasogenic edema (Figure 1). Hemorrhagic picture was noted in the insular cistern (Figure 1), left fronto-temporal subdural space (Figure 1), the perimesencephalic, ambient and suprasellar cisterns (Figures 2 and 3), and the sulci of the left temporo-parietal lobes (Figures 2 and 3). There was a subtle dense clot sign in the left transverse sinus (Figure 3). She was diagnosed as having left transverse sinus thrombosis leading to left temporal hematoma, SAH and SDH. She was treated with low molecular weight heparin, levetiracetam, and 3% hypertonic saline. The magnetic resonance venography (MRV) done the following day confirmed the presence of left transverse sinus thrombosis (Figure 4). CT angiography of the brain did not reveal any vascular malformations. Her complete blood counts, coagulation profiles, routine blood chemistry, and C-reactive protein were within normal limits. Antinuclear antibody and SARS-CoV-2 RNA polymerase chain reaction tests were negative. She underwent CT scan of neck, chest, abdomen and pelvis, mammogram, stool occult blood, carcinoembryonic antigen, CA-125, and Ca 19–9 tests, all of which were non-contributory to any occult malignancy. Thrombophilia screening was not done at this acute stage. We decided to get those done after the completion of anticoagulant treatment. She showed good clinical improvement over the course of the next few days. A repeat CT scan of the head done on day seven, revealed decreasing edema and stable hematoma volume with no new bleeding. The cause of her CVST was deemed undermined at this stage and she was discharged home on dabigatran 150 mg twice daily, as she could not get PT/INR done reliably due to the current lockdown imposed by the government. She has been doing well on follow up.

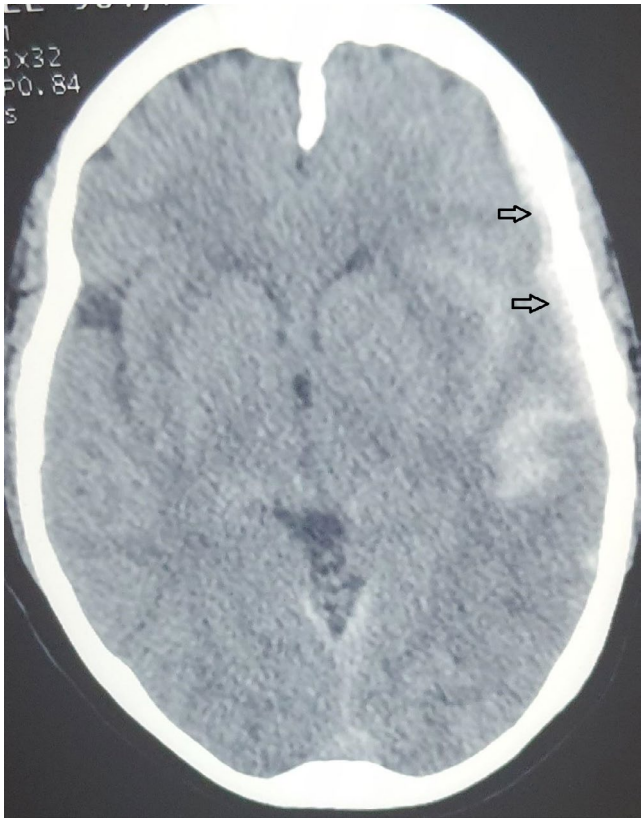

FIGURE 1 Plain CT head of the patient showing left temporal hematoma with surrounding edema, SDH in the frontotemporal areas (black arrows) and subarachnoid hemorrhage in the insular cistern

3 | DISCUSSION

CVST is well reported in medical literature to cause various types of intracranial hemorrhages (including intraparenchymal, subarachnoid and SDHs). Up to 40% of patients with CVST can have intracranial hemorrhages.[9] When the venous sinuses get occluded, the resulting stasis causes pressure build up and breaching of blood brain barrier causing vasogenic edema and venous infarcts.[10] Excessive venous hypertension can cause rupture of blood vessels leading to intracranial bleeding.[10] SAH occurs in CVST due to the rupture of thin cortical veins or rupture of parenchymal hematoma into the subarachnoid space.[11] SDH can also occur through similar pathophysiology: the venous hypertension and pressure build up in the fragile and valveless bridging veins leading to their rupture.[12]

The diagnosis of CSVT with various types of intracranial bleedings can be very challenging. The clinical presentation is often non-specific and imaging features on brain scan are often very subtle. Management requires a very careful use of anticoagulants in the setting of intracranial bleeding. The mode of presentation of aneurysmal SAH in the majority of patients is a sudden severe headache or thunderclap headache and this presentation of headache should always be assumed to be due to rupture of intracranial aneurysm unless proved otherwise.[13] Even though the headaches of CVST commonly have a gradual onset and progressive course with features of raised intracranial pressure, it can present with features of thunderclap headache in 2%–15% of the patients.[14,15] The subacute and gradually progressive

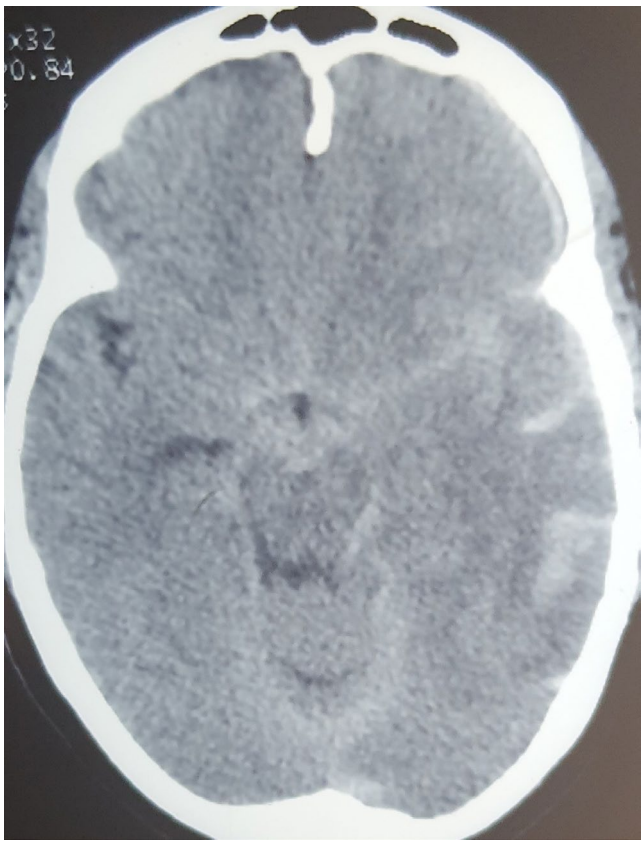

FIGURE 2 Plain CT head of the patient showing bleedings in the temporal sulci and perimesencephalic and suprasellar cisterns

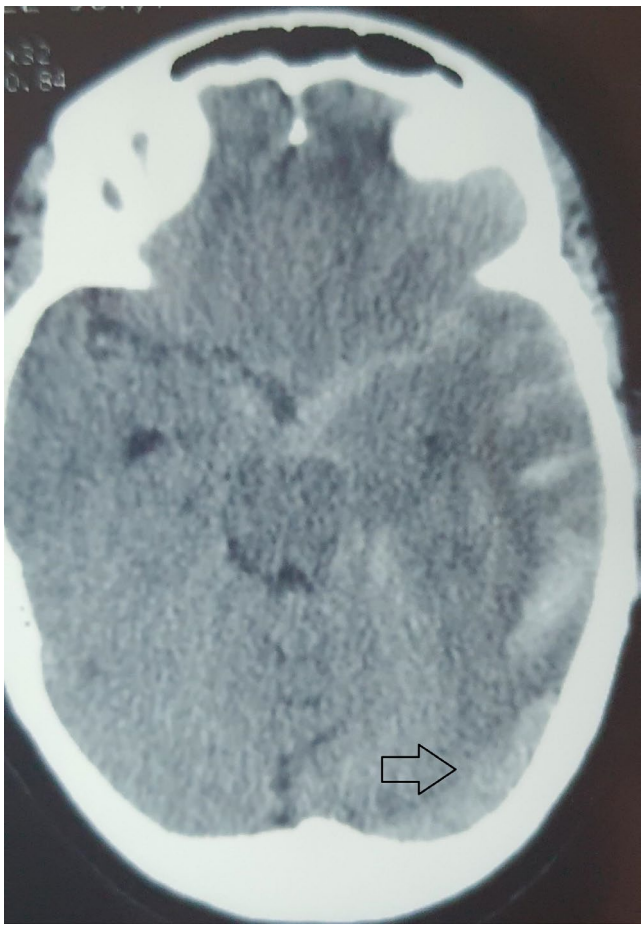

FIGURE 3 Plain CT head of the patient showing bleedings in the temporal sulci, ambient and suprasellar cisterns, and subtle dense cord sign in the left transverse sinus (dark arrow)

holocranial headache culminating in a generalized tonic-clonic seizure in our patient is typical of CVST and alerted us to look carefully at the scans for its imaging characteristics.

Concomitant SDH is very rare in CVST. The literature until recently was restricted to isolated case reports and case series.[7] In a retrospective multicenter cohort study of 260 patients with confirmed CVST over a period of 15 years in Mexico and Europe, the incidence of such SDH was reported to be 11%.[12] SDH can be isolated or may occur together with other types of intracranial hemorrhages and there are no clinical features which are specific to SDH.[7,12]

The diagnosis of CVST is clinically suggested by subacute onset progressive headaches with features of raised intracranial pressure. The diagnosis is confirmed by demonstrating the presence of blood clots in the dural venous sinuses or in the cortical or deep cerebral venous system. Plain non-contrasted CT scan of the brain can show imaging evidence of thrombosis in 70% of the cases.[4] These signs include the dense clot or triangle sign, empty delta sign, cord signs, and evidence of parenchymal ischemia and hemorrhage and other intracranial hemorrhages. Even though magnetic resonance imaging (MRI) is the most sensitive technique for demonstrating the thrombus, guidelines suggest either MR venography or CT venography can be used for confirmation.[16] The gold standard for diagnosis is, however, digital subtraction angiography.[4] Investigation for predisposing causes to thrombosis should be done in all patients. The causes range from use of oral contraceptives and pregnancy-related to rare vasculitis and inherited thrombophilias.[4] In 25% of patients above 55 years, thrombophilia related to an underlying malignancy have been reported. Such patients should undergo appropriate screening tests for occult malignancy based on their age and sex.[4]

The specific treatment of CVST is therapeutic anticoagulation in the acute stage, irrespective of presence of intracranial bleeding. The preferred agent is low molecular weight heparin.[16] Patients may also require treatment for seizures with standard antiepileptic drugs, keeping in mind drug interactions with warfarin, use of hyperosmolar agents, endovascular therapy and decompressive hemicraniectomy in the acute stage.[4] Current guidelines recommend using warfarin (or any oral vitamin K antagonists) at an international normalized ratio range of 2.0–3.0 for a duration between 3 and 12 months to prevent relapse of the thrombosis.[16] The direct oral anticoagulants are safe and effective alternatives

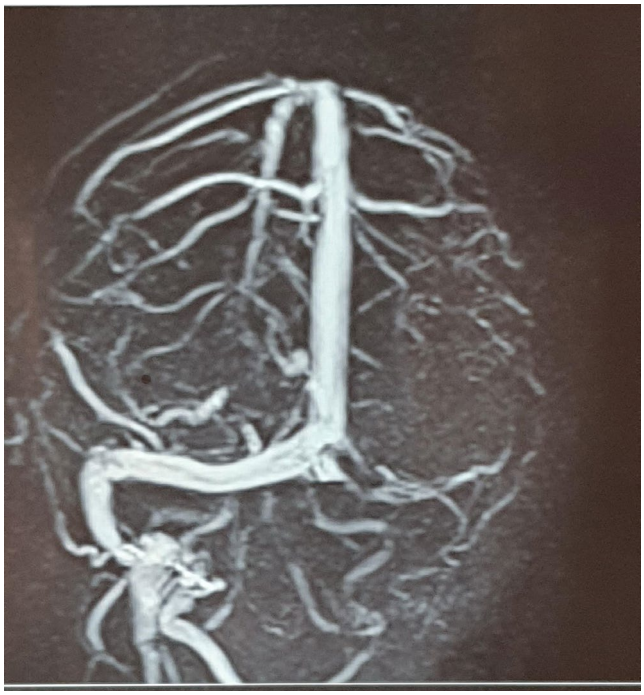

FIGURE 4 MRV showing thrombosis of the left transverse sinus

to vitamin-K antagonists, as in this particular case. However, current guidelines do not recommend using these pending results of ongoing clinical trials.[4] Almost 75% of the patients who develop CVST attain a complete functional recovery, about 15% of the patients die or have residual neurological deficits requiring long-term assisted care.[3] The risk of recurrence is reported to be 2%–7%.[3]

4 | CONCLUSION

This case illustrates that a high index of suspicion is required to diagnose CSVT as its clinical and imaging features may not be typical. Localized cerebral edema with superimposed parenchymal, convexity subarachnoid as well as SDHs in a patient with subacute onset progressive headache is highly suggestive of CVST. Non-contrasted CT, CT venography, MRI and MR venography can help confirm the diagnosis. Most patients respond very well to immediate anticoagulation irrespective of presence of various types of intracranial bleeding concomitantly.

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CONFLICTS OF INTEREST

The authors do not have any conflicts of interests.

AUTHOR CONTRIBUTIONS

All the authors have contributed equally to conception, design, manuscript preparation, critical revision, and finalization. All the authors agree to be accountable for all aspects of the work.

ETHICAL APPROVAL

Ethical approval was not required for the publication of this report.

CONSENT

The consent to publish all the relevant details as a case report was obtained prior to preparing this manuscript.

DATA AVAILABILITY STATEMENT

The details supporting the information in this manuscript can be made available from the corresponding author upon request.

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