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Unusual Presentation of Cerebral Venous Sinus Thrombosis with Non-Aneurysmal Subarachnoid Hemorrhage, Intracerebral Hemorrhage and Pulmonary Thromboembolism in a 33-Year-Old Woman

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ABSTRACT

Cerebral venous sinus thrombosis (CVST) is a rare but important cause of stroke in young adults. Although hemorrhagic venous infarction is well recognized, subarachnoid hemorrhage (SAH) as a presentation of CVST is uncommon and can easily mimic aneurysmal rupture.

We describe a 33-year-old woman who presented with acute severe headache. Non-contrast CT revealed both subarachnoid and intracerebral hemorrhage. CT angiography was normal, with no evidence of aneurysm or arteriovenous malformation. CT venography demonstrated thrombosis in the distal part of the left internal cerebral vein, confirming CVST. During hospitalization, pulmonary CT angiography performed for new-onset dyspnea also revealed pulmonary thromboembolism (PTE). The patient was treated with anticoagulation and achieved clinical stability without progression of hemorrhage.

SAH due to CVST is rare, accounting for fewer than 3% of cases. Convexity-localized SAH and parenchymal hemorrhage should raise suspicion for venous origin, particularly when angiography is normal. Literature review demonstrates that CVST-associated SAH has often been misdiagnosed as aneurysmal hemorrhage. This case further highlights the potential for multisite venous thrombosis, as demonstrated by concomitant PTE.

CVST should be considered in young patients with SAH and normal CTA findings. Venous imaging is essential for accurate diagnosis, and clinicians should remain vigilant for systemic thromboembolism.

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Introduction

Cerebral venous sinus thrombosis (CVST) is an uncommon but potentially devastating cause of stroke, accounting for 0.5–1% of all strokes worldwide [1]. Unlike arterial ischemic stroke, CVST frequently affects younger individuals and disproportionately women, often in the setting of hormonal, infectious, or prothrombotic states [2,3]. The condition is notoriously variable in presentation, with symptoms ranging from isolated headache to seizures, focal neurological deficits, coma, or hemorrhage.

Intracranial hemorrhage is a recognized feature of CVST, typically resulting from venous infarction or venous hypertension. Between 30% and 40% of CVST patients develop parenchymal hemorrhage during their illness [4]. Subarachnoid hemorrhage (SAH), however, is exceedingly rare in CVST, reported in only 3% of cases [5].

The overwhelming majority of subarachnoid hemorrhage (SAH) cases are aneurysmal in origin; therefore, when a patient presents with sudden headache and subarachnoid blood on CT, the immediate clinical priority is exclusion of aneurysm by angiography. Yet, as several reports have highlighted, cerebral venous sinus thrombosis (CVST) may masquerade as aneurysmal SAH. In such cases, CT angiography (CTA) appears normal, delaying diagnosis unless venous imaging is pursued [6]. Awareness of this diagnostic pitfall is crucial.

Here we report the case of a young woman who presented with both SAH and intracerebral hemorrhage (ICH), whose CTA was normal but CT venography (CTV) revealed venous sinus thrombosis. During her hospital course, pulmonary CT angiography performed for new-onset dyspnea also demonstrated pulmonary thromboembolism (PTE), emphasizing the systemic nature of thrombotic disease.

Case Presentation

A 33-year-old previously healthy woman presented to the emergency department with sudden-onset severe headache, described as the "worst headache of her life." The headache was diffuse, most pronounced occipitally, and accompanied by nausea and vomiting. She denied trauma, fever, or recent illness. There was no past medical history of clotting disorders, no current oral contraceptive use, and no known family history of thrombosis.

On admission, vital signs were stable: blood pressure 122/78 mmHg, heart rate 84 bpm, oxygen saturation 98% on room air, and temperature 36.8°C. Neurological examination revealed no focal deficits. The Glasgow Coma Scale score was 15/15. Mild nuchal rigidity was present, but there was no papilledema. Cardiovascular and systemic examinations were unremarkable.

Imaging Findings

- **Non-contrast CT:** acute subarachnoid hemorrhage in the parietal convexity sulci bilaterally and a focal intraparenchymal hemorrhage in the left frontal lobe (Figure 1).

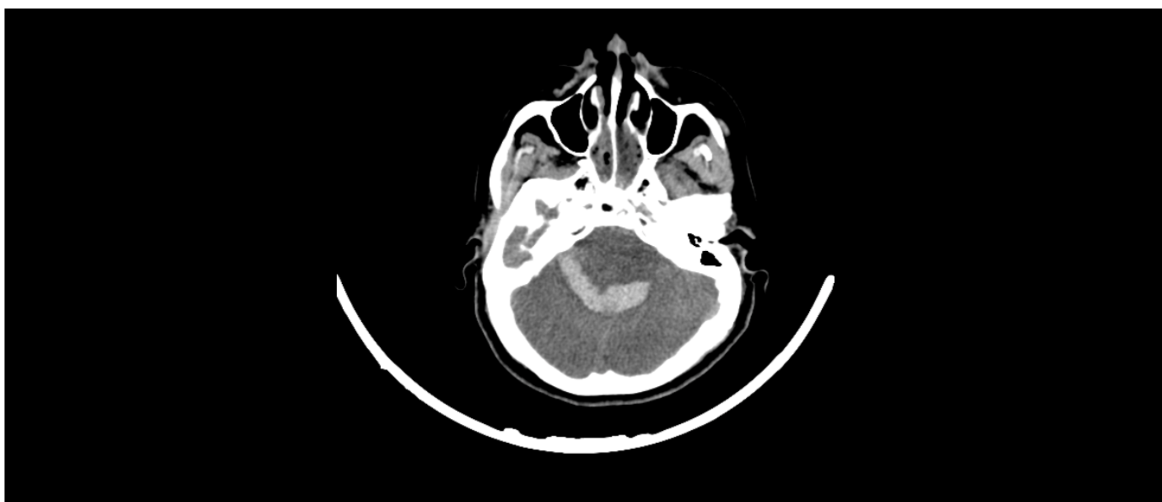


Fig. 1. Axial non-contrast brain CT scan reveals SAH in quadrigeminal and ambient cistern and also interpeduncular cistern in anterior aspect of the midbrain. Also, evidence of extension of the hemorrhage into the posterior flax cerebri is seen. Intraparenchymal hemorraghe is seen in both cerebral hemisphere and vermis

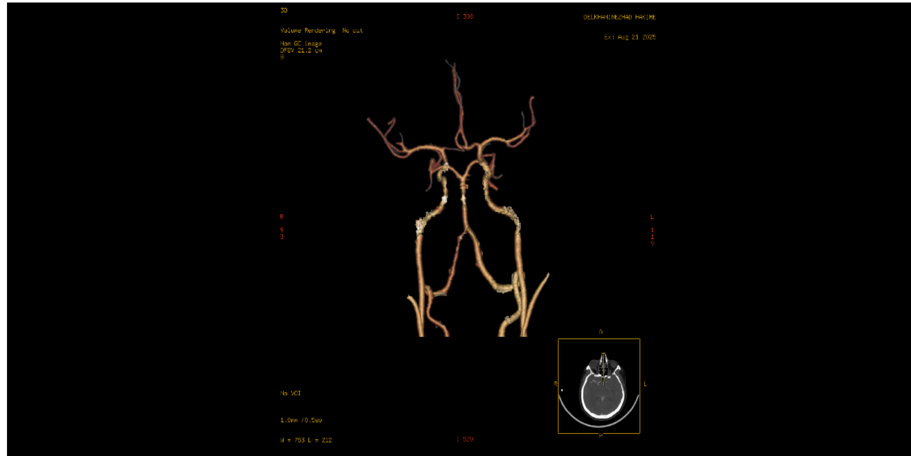


Fig. 2. Spiral CT Angiography of the brain which appears to be normal without any sign of aneurysm.

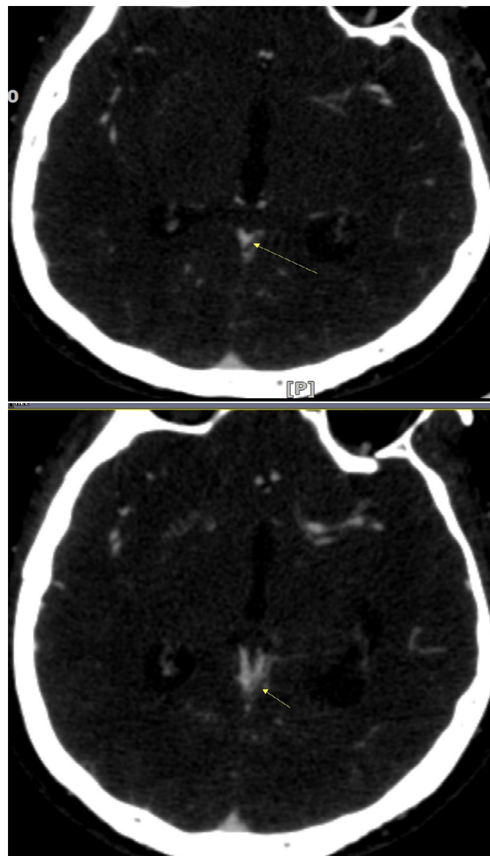


Fig. 3 and 4. Axial images through the brain (ct venography) injection which reveals: Evidence of internal thrombosis with length of 10 mm and diameter of 2.5 mm is seen in distal part of the LT side internal cerebral vein (short arrow) and extension to vein of galen (long arrow) suggestive for sinus vein thrombosis with about 70-90% stenosis in distal part of the vein of galen.

- **CT Angiography (CTA):** normal arterial circulation without aneurysm, arteriovenous malformation, or stenosis (Figure 2).

- **CT Venography (CTV):** thrombosis in the distal part of the left internal cerebral vein, confirming CVST (Figures 3-5).

- **Pulmonary CT Angiography:** performed due to new-onset mild dyspnea, demonstrated filling defects in segmental pulmonary arteries consistent with acute PTE (Figures 6&7).

Laboratory findings showed blood investigations, including complete blood count, electrolytes, liver

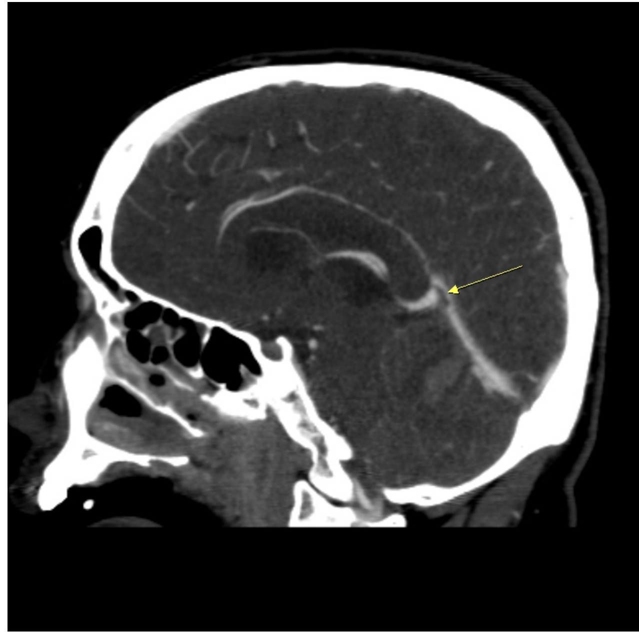
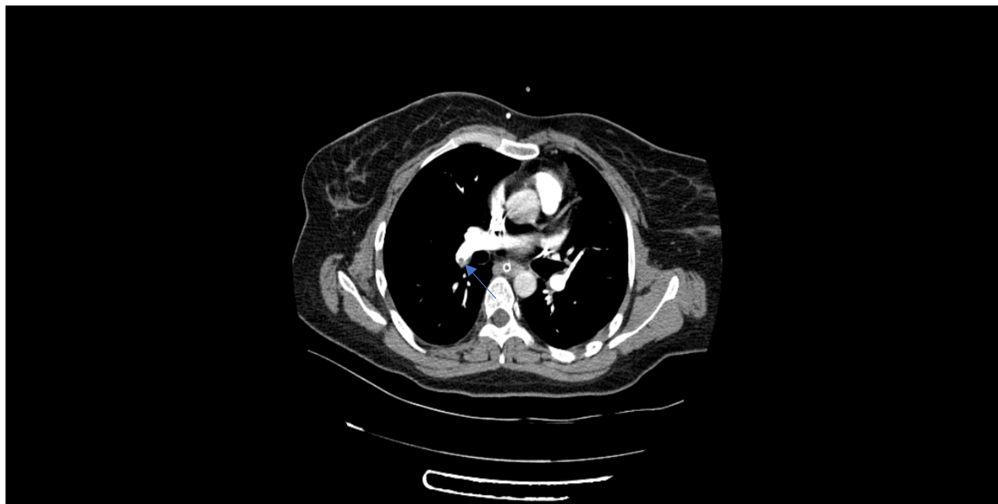


Fig. 5. Sagittal reformatted image through the brain (ct venography) injection which reveals the filling defect(arrow) in the left internal cerebral vein which has extended into the vein of galen.



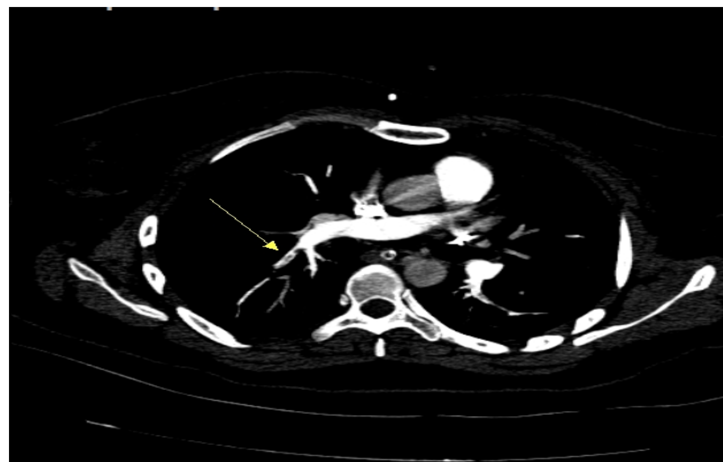


Fig. 6 and 7. Evidence of multiple filling defect are seen in lobar, segmental and subsegmental branches of the RT lower lobe and also segmental and subsegmental braches of the RT upper lobe pulmonary artery suggestive for PTE.

and renal function, and coagulation profile, were within normal ranges. Thrombophilia screening was planned as an outpatient investigation.

Following multidisciplinary discussion, anticoagulation with therapeutic low-molecular-weight heparin (LMWH) was initiated despite the presence of intracranial hemorrhage, consistent with international guideline recommendations [7]. The concomitant diagnosis of pulmonary embolism further strengthened the indication for systemic anticoagulation. The patient was monitored in the neurology ward with supportive measures, including hydration and analgesia.

The patient's symptoms gradually improved over the next several days. Repeat CT on day 5 showed stable hemorrhage with no expansion. No seizures or new neurological deficits occurred. Respiratory symptoms also improved under anticoagulation. She was transitioned to oral anticoagulation and discharged with outpatient follow-up arranged.

Discussion

CVST is rare, with an annual incidence of 1.3–1.6 per 100,000 (1). Risk factors include oral contraceptives, pregnancy, puerperium, hereditary thrombophilia, infection, and systemic inflammatory conditions [2,3]. However, up to 20–25% of cases may occur without identifiable risk factors, as in our patient. Intracranial hemorrhage occurs in up to 40% of CVST cases but typically manifests as venous infarction rather than SAH [4]. Non-aneurysmal SAH due to CVST is rare, estimated at 3% [5]. Unlike aneurysmal SAH, which often involves the basal cisterns, CVST-associated SAH tends to localize to cortical convexity sulci. This distinction can guide diagnostic suspicion. Our patient's combined SAH and ICH presentation highlights the hemorrhagic spectrum of CVST. Similar mixed hemorrhages have been reported in prior case studies [8–10].

Non-contrast CT is the initial imaging modality. While it may reveal direct signs such as hyperdense dural sinuses, sensitivity is limited [11,12]. CTA is routinely performed in suspected SAH, but its value for venous thrombosis is poor. Abbasi et al. demonstrated that CTA alone cannot reliably detect CVST, underscoring the need for venous imaging [6]. CTV and MRV remain the diagnostic gold standards. In our patient, CTA was normal but CTV revealed thrombosis, confirming that reliance on arterial imaging alone risks misdiagnosis.

The coexistence of CVST and pulmonary embolism in this patient underscores the systemic nature of venous thromboembolism. In the ISCVT registry, extracranial venous thrombosis (deep vein thrombosis or pulmonary embolism) was reported in approximately 7–10% of patients with CVST [13,14]. Recognition of such multisite involvement is crucial, as it has therapeutic implications and mandates systemic anticoagulation.

Conclusion

This case emphasizes the importance of considering CVST in young patients presenting with SAH and normal CTA findings. Venous imaging ensures accurate diagnosis and early initiation of life-saving therapy. The additional finding of PTE in our patient illustrates the systemic thrombotic potential of CVST, highlighting the importance of vigilance for extracranial venous thrombosis.

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Ethical Considerations

Ethical statement

We confirm that this work was conducted in accordance with the Declaration of Helsinki, and that all participants provided informed consent.

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Conflict of Interests

The authors declare that they have no competing interests.

Data availability statement

Data are available from the corresponding author upon reasonable request via email.

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