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Case Report

A case of cerebral venous sinus thrombosis presented with SAH and isolated headache ☆

Nali Ahmed, MBChB[a,*], *Mohamed Ibrahim, MBChB, FRCR, MD/PhD*[a], *Dmitriy Starostin, MBChB, FRCR*[b]

[a] *Rotherham NHS Foundation Trust, Rotherham, UK*
[b] *Sheffield Teaching Hospitals NHS Trust, Sheffield, UK*

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ABSTRACT

Cerebral venous sinus thrombosis is a rare and challenging type of stroke. Coexistence of subarachnoid hemorrhage adds complexity to the diagnostic process leading to a missed or delayed diagnosis. Isolated headaches can be the only presentation and urgent neuroimaging using CT or MR venogram plays a pivotal role in the workup of these cases. We report a rare case of 64-years-old patient with subarachnoid hemorrhage and underlying cerebral venous sinus thrombosis who was presented with isolated headache where the management is different from arterial subarachnoid hemorrhage.

Introduction

The presence of subarachnoid hemorrhage (SAH) alongside cerebral venous sinus thrombosis (CVST) adds complexity to the diagnostic process due to overlapping symptoms potentially leading to a missed or delayed diagnosis [1]. Urgent neuroimaging plays a pivotal role in the workup of these cases [2]. The management of SAH secondary to CVST is quite different from that of arterial SAH. The usual treatment of sinus thrombosis is anticoagulation or local thrombolysis. Systemic anticoagulation is the first line treatment for CVST because of its efficacy, safety, and feasibility [3].

Case report

A previously healthy 64-year-old man presented to the Urgent and Emergency Care Center (UECC) with the chief complaint of an excruciating headache that abruptly awakened him from sleep in the early morning. He described it as the worst pain he had experienced in his life. The headache had gradually developed over the past 2 days and persisted. No associated symptoms were reported, and the physical and neurological examinations revealed normal findings with no evidence of meningism or focal neurological deficits. The patient had an unremarkable medical history, except for a previous

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* Corresponding author.
E-mail address: nali.ahmed@nhs.net (N. Ahmed).

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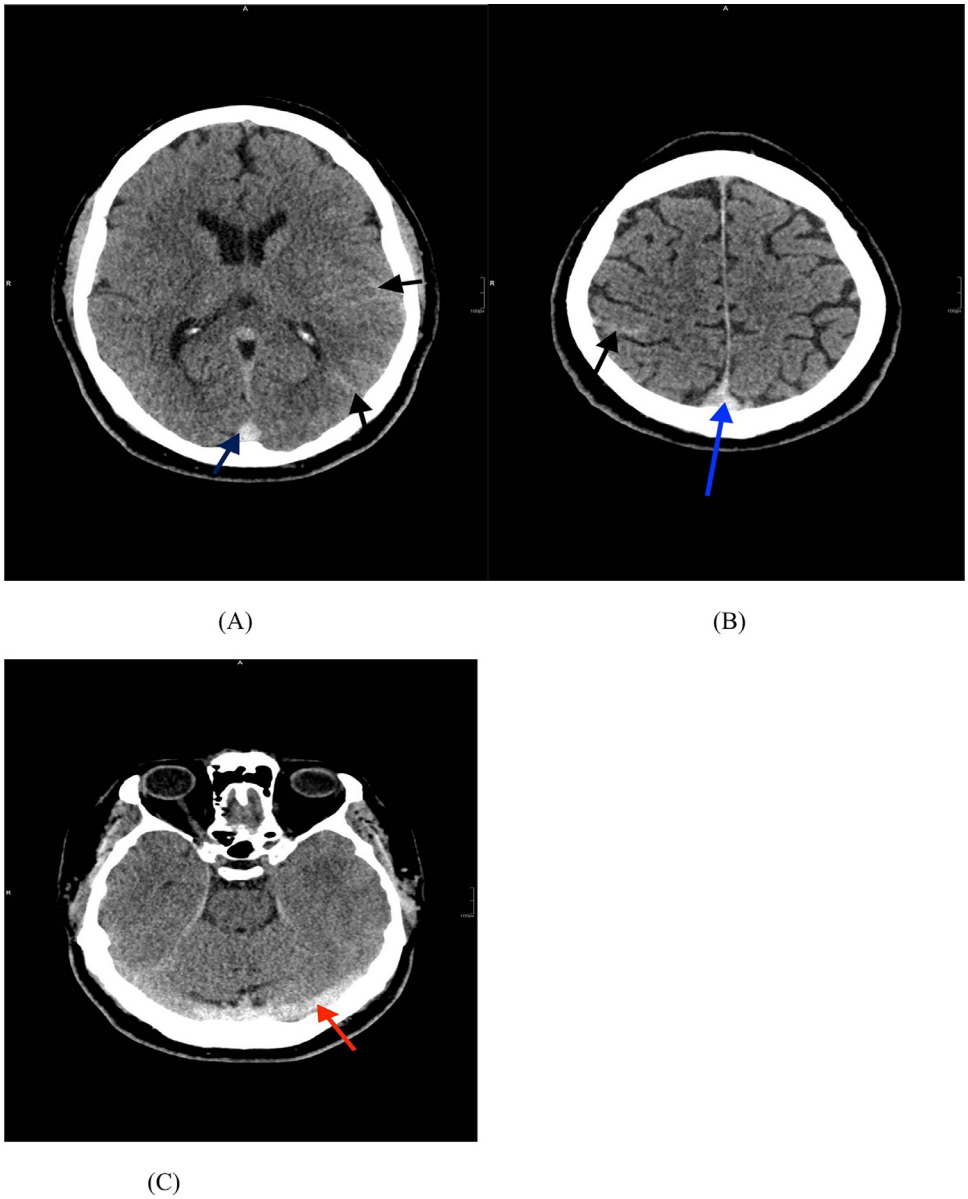

Fig. 1 – Noncontrast CT head (A and B) revealed: bilateral subarachnoid hemorrhage predominantly in the left temporoparietal region and, to a lesser extent, in the right high parietal lobes (black arrows) and bilateral subarachnoid hemorrhage predominantly in the left temporoparietal region and, to a lesser extent, in the right parietal lobe. Hyperdense superior sagittal sinus raising concerns of underlying venous thrombosis (blue arrows). (C) Left transverse sinus appeared hyperdense denoting thrombosis (red arrow).

right-sided anterior circulation transient ischemic attack (TIA) in 2013 and no personal or family history of venous thromboembolism (VTE). Urgent noncontrast CT of the head was done (Fig. 1).

A CT venogram was requested shortly after the noncontrast CT (Fig. 2).

The patient underwent a comprehensive hemocoagulative study, which yielded unremarkable results except for borderline Protein S levels (0.65 IU/mL).

Based on the radiological findings simultaneous SAH and CVST and following interdisciplinary consultations involving the hematology and neurology teams, the decision was taken to anticoagulate the patient thus he was commenced on therapeutic Tinzaparin to treat the cerebral venous thrombosis, with careful neurological observation to note any fall in GCS. He was subsequently bridged to warfarin. Additionally, the patient was also given painkillers to provide pain relief. The patient's condition subsequently improved, and he was discharged on warfarin with a scheduled follow-up appointment at the hematology clinic to consider transitioning to a direct oral anticoagulant (DOAC).

The stroke team followed up on this patient as an outpatient 6 weeks postdischarge. He reported issues with short-term memory. He is due for a repeat CT venogram to assess

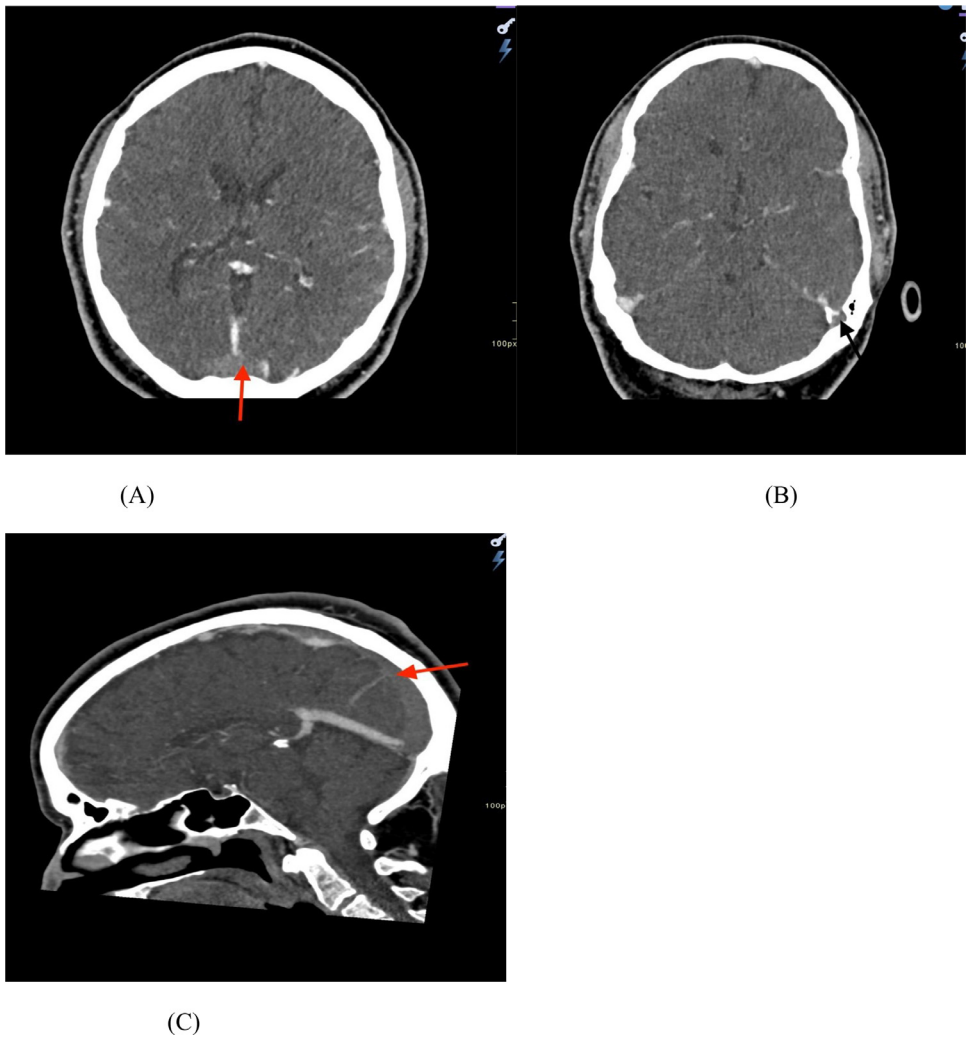

Fig. 2 – CT venogram of the head (A-C) demonstrated filling defects in the superior sagittal (red arrows) and left transverse sinuses (black arrow) confirming extensive cerebral venous sinus thrombosis (CVST).

for full recovery, complete recanalization, and stopping of anticoagulant therapy.

Discussion

The overall incidence of cerebral venous thrombosis is approximately 1.32 per 100000 [2,4,5] with a female-to-male ratio of 3:1 [6,7]. It typically affects younger patients with a median age of 37 [6]. However, the case presented here was atypical for age or gender. Ten of 233 patients with CVST had evidence of cortical SAH [8] and 3% of SAH was caused by CVST [9].

Multiple pathophysiological theories exist to explain how hemorrhage is caused by venous thrombosis. It is argued that it could be due to the rupture of venous parenchymal hemorrhagic infarcts into the subarachnoid space [10]. In our case, this was not found. However, it seemed more likely to have happened with another explanation that suggests the elevated venous pressure precipitates the rupture of fragile bridging subarachnoid cortical veins [11] as the SAH occurred in the neighboring areas of the thrombosed venous sinuses.

Common predisposing and risk factors linked to cerebral venous sinus thrombosis typically encompass contraceptive use, obesity, malignancy, pregnancy, and prothrombotic conditions [6,12,13]. However, none of these existed in the aforementioned case.

The transverse sinus is the most frequently found site of thrombosis, followed by the sigmoid and superior sagittal sinus [14]. This aligns with our case, which had thrombosis in all 3 mentioned sinuses.

CVST can be asymptomatic [15,16] or present most frequently with headache [6] consistent with the case presented. It can also present in focal neurologic deficits, seizures, or encephalopathy [17,18]. Additional manifestations include cavernous sinus syndrome, multiple cranial nerve palsies, or even more rarely with SAH, similar to our case [19,20].

Given the myriad of presentations and the fact that it is the first brain imaging in suspected stroke or acute headache, CT is often a practical first test [2].

In about one-third of cases, CT demonstrates direct signs of CVST, including (1) the dense triangle sign; a triangular area of high attenuation density in the sagittal sinus, or deep cerebral veins; (2) the cord sign; a high attenuation cord-like appearance in the transverse sinus; or (3) the empty delta sign on CT venogram; a triangular or delta-shaped region of contrast enhancement surrounded by a nonenhancing area [21,22].

More frequently, CT can detect indirect signs of CVST, which can be focal ischemic hypodense areas caused by vasogenic edema or venous infarction, usually not respecting the arterial boundaries. Additionally, the indirect signs include intracerebral hemorrhage, hemorrhagic infarcts, or rarely (<1%) subarachnoid hemorrhage. This is consistent with the findings of the case presented [20].

The distribution of SAH associated with CVST is usually different from that of SAH of arterial origin, which has a characteristic pattern. In fact, when SAH is localized at the cerebral convexity and spares the basal cisterns and skull base, CVST should b be considered [8].

The next best modality could be either MR venography or CT venography. However, due to ready availability, less time-consuming, and its high sensitivity (95%) and specificity (91%) [23], CT venography is more commonly utilized. It gives a good visualization of the major dural sinuses [24] and may demonstrate filling defects, sinus wall enhancement, and increased collateral venous drainage [24]. In our case, a CT venogram showed filling defects and confirmed the diagnosis.

Management of SAH secondary to CVST is quite different from that of arterial SAH. The usual treatment of sinus thrombosis is anticoagulation or local thrombolysis. Systemic anticoagulation is the first line treatment for CVST because of its efficacy, safety, and feasibility.

Despite concerns of increased bleeding risk and controversies, available evidence recommends starting with low-molecular-weight heparin even in the presence of hemorrhagic venous infarction, intracerebral hemorrhage, or isolated subarachnoid hemorrhage, as they are not contraindications for anticoagulant treatment in CVST [23,25,26]. The ultimate aim is to switch to warfarin or a direct oral anticoagulant after the acute phase [27,28].

Conclusion

The simultaneous occurrence of SAH and CVST is rare and the only clinical presentation can be an isolated headache of a variable character. The identification of CVST in cases with SAH is necessary as the management of SAH secondary to CVST is anticoagulation or local thrombolysis which is quite different from that of arterial SAH.

Patient consent

Written consent was obtained from the patient.

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