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Case Report

Massive traumatic subarachnoid hemorrhage mimicking aneurysmal subarachnoid hemorrhage

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ABSTRACT

Introduction: Massive traumatic subarachnoid hemorrhage (tSAH) is a rare but potentially life-threatening condition that can mimic the clinical presentation of aneurysmal subarachnoid hemorrhage (aSAH). The accurate differentiation between these two entities is crucial, as their management and prognoses significantly differ.

Case presentation: We present a case of a 64-year-old male patient who presented to our emergency department after being involved in a motor vehicle collision. His radiological findings on a computed tomography (CT) scan were suggestive of aSAH based on its location, which showed massive SAH in bilateral sylvian fissures and the basal cisterns. There was no evidence of vasospasm. The patient later developed a stroke despite the use of Nimodipine.

Conclusion: While traumatic subarachnoid hemorrhage mimicking aneurysmal subarachnoid hemorrhage is a recognized phenomenon, it is relatively uncommon. We present a case of massive tSAH complicated by a stroke with no evidence of aneurysm on cerebral angiogram, shedding light on the diagnostic challenges in differentiating tSAH from aSAH and emphasizing the importance of accurate diagnosis for appropriate management, in addition, we aim to remind the readers that trauma may be a cause for massive SAH and should prompt a medical SAH management plan.

Introduction

Traumatic brain injury (TBI) is the most common cause of mortality and disability in patients under 40 years of age, and the incidence is increasing [1]. Traumatic subarachnoid hemorrhage (tSAH) is an important manifestation of TBI and its incidence varies from 26 % to 53 %. The most common cause of blood in the subarachnoid space is trauma [2]. The amount of blood in the subarachnoid space is often minimal and associated with other brain injury. Isolated large-volume subarachnoid hemorrhage in the basal cistern and Sylvian fissure is considered atypical in trauma and further investigations are needed to rule out vascular causes like aneurysms [3]. Aneurysmal subarachnoid hemorrhage (aSAH) can be associated with a decreased level of consciousness and may be a precipitating factor of trauma. It is important to exclude a berry aneurysm or other vascular malformations in cases of massive SAH because the management principles are different [4]. Many studies have addressed aSAH management and treatment modalities,

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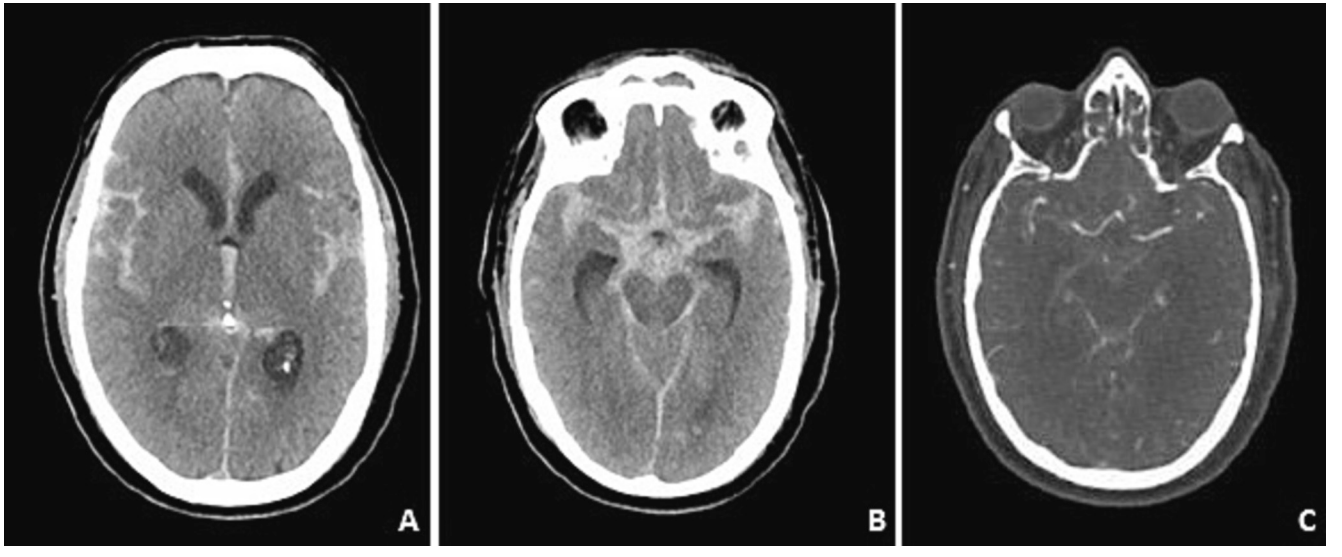

Fig. 1. A. Plain CT scan of the head showing bilateral subarachnoid hemorrhage with third ventricular extension.
B. Plain CT scan of the head showing subarachnoid hemorrhage in basal cisterns and bilateral sylvian fissure.
C. CT angiography of the head at the level of sylvian fissure showed no vascular malformation.

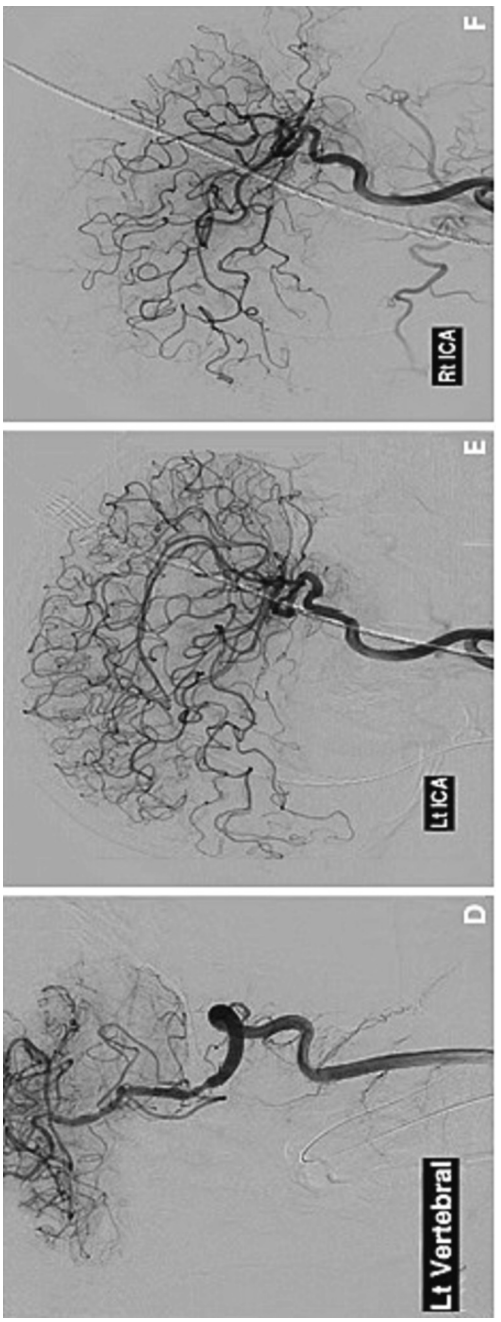

Fig. 2. D, E, F. Digital subtraction angiography showed no clear vascular malformation such as aneurysm, but vasospasm in the left vertebrobasilar trunk and bilateral internal carotid artery.

including the management of vasospasm, which is common in aneurysmal bleeding [5]. In the setting of tSAH, vasospasm is rare [3]. Isolated studies, using the Marshal scale for tSAH, have reported a linear correlation between the increase in the extent of hemorrhage and worsened neurological outcomes [6]. This case illustrates that massive tSAH can be associated with stroke even in the absence of vasospasm.

Case presentation

A 66-year-old male of Asian descent was brought by an ambulance to the emergency department (ED) after being involved in a motor vehicle collision as an unrestrained passenger. The patient had a medical history of diabetes and hypertension. The initial Glasgow Coma Score (GCS) at the scene was E3V3M2. He had normal vital signs and bilaterally equally reactive pupils. On arrival at the ED, a CT scan of the brain showed massive SAH in the bilateral Sylvian fissures and the basal cisterns, associated with hemorrhage in the third ventricles, but there was no convexity of tSAH (Fig. 1a, b). Sub-galeae hematoma was noted in the frontal region with a small component of soft tissue injury and surgical emphysema. CT scan of the thorax showed posterior basal segmental consolidation on both sides, likely representing aspiration pneumonia. CT angiography of the head was done and showed no vascular malformation (Fig. 1c). The patient was shifted to the operating theater for a lifesaving extra-ventricular drain (EVD) insertion and intracranial pressure monitoring. Clear cerebrospinal fluid came with an initial pressure of 7 cm of water. He was shifted post-operatively to the trauma intensive care unit (TICU). Nimodipine was started. On the next day, digital subtraction angiography was done, and it showed no aneurysm or arteriovenous malformation, but multiple stenoses were noted in the left vertebrobasilar trunk and bilateral ICA (cavernous and supraglenoid segments) which were likely atherosclerotic (Fig. 2e, d, f). He was started on Dalteparin 5000 IU daily as there was no evidence of ongoing bleeding. In his first week in the TICU, the patient had ventilator-associated pneumonia and he was treated with antibiotics. On day 6, CT of the head was done and showed significant improvement of the previously seen SAH. On day 8, sedation was stopped, and the patient was extubated, and his GCS was E4V4M3, the fluid balance was neutral. On day 12, the patient had weakness of the left upper limb, and the CT of the head was repeated which showed hypodensity in the right parieto-occipital region, with matched perfusion defect. There was no evidence of vasospasm on transcranial doppler ultrasound (TCD). CT angiogram of the head was also done, and it showed irregular narrowing of the intracranial vertebral and basilar arteries – the same finding as the initial angiogram which was done on day one - which was likely atherosclerotic rather than vasospastic (Fig. 3a, b). MRI head was done after 2 weeks, and it showed a stroke in the right parieto-occipital area (Fig. 4a, b, c). The stroke team was consulted, and they ordered an echocardiography of the heart and a carotid ultrasound, which were normal. It was considered an ischemic stroke and Aspirin was started. EVD was removed after 2 weeks from admission. Gradually, the patient's verbalization improved, and he was transferred to the neurosurgery high-dependency unit on day 24. The patient then underwent two months of intensive physiotherapy at the regional rehabilitation center. The patient's GCS was E5V5M5 at follow-up after 4 months, with remnant weakness in the left upper limb (3/5) and lower limb (4/5). The patient was ambulating with minimal assistance and a spastic gait.

Discussion

We present an unusual case of massive subarachnoid hemorrhage resulting from traumatic brain injury. Traumatic SAH is considered a benign consequence of traumatic brain injury and is managed with a focus on the reduction of cerebral oxygen requirement and maintenance of cerebral perfusion pressure. Our case serves as a reminder that massive SAH may indeed result from

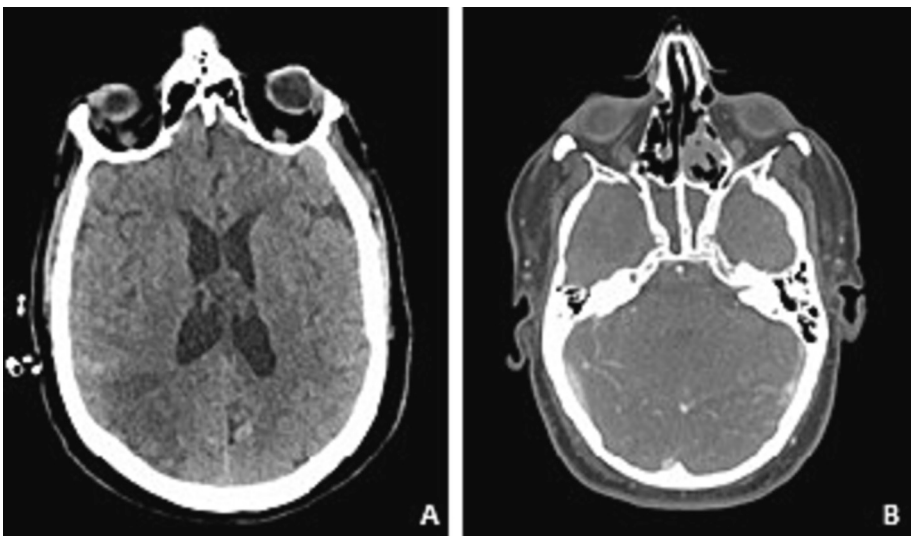

Fig. 3. A. CT scan of the head done at day 13 and showed Interval development of hypodensity in the right parieto-occipital region.
B. CT angiography of the head done at day 13 after the weakness showed no evidence of new vasospasm.

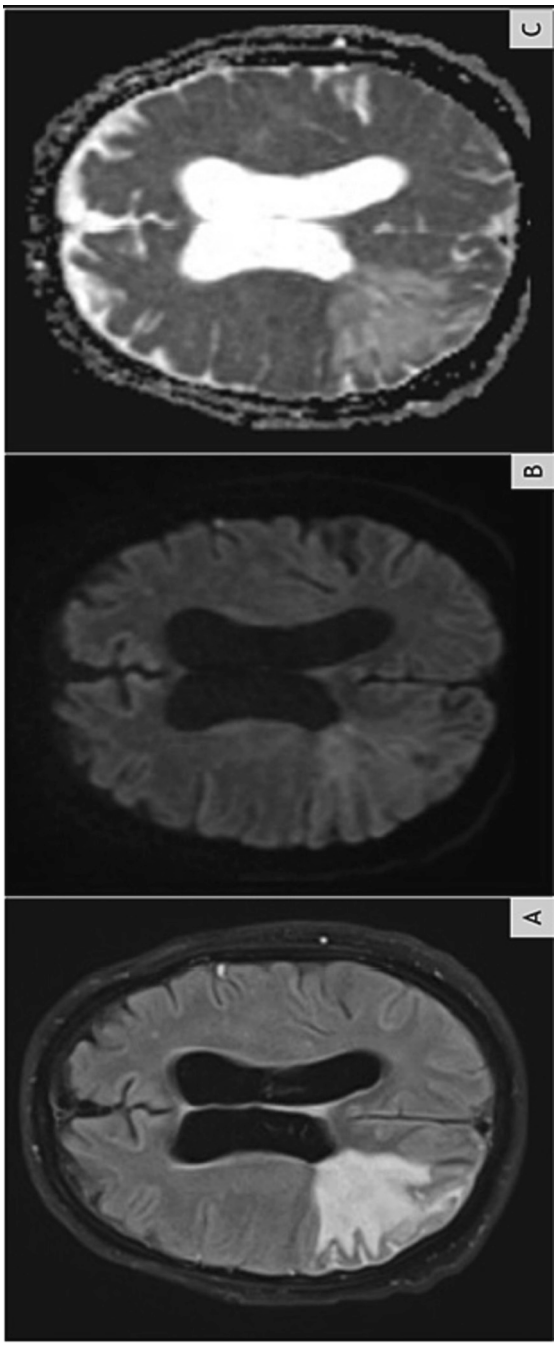

Fig. 4. A, B, C. MRI head 2 weeks later showing evidence of stroke in the right parieto-occipital area.

trauma and should prompt the consideration of medical treatments usually reserved for the management of spontaneous SAH. These should be considered even in the absence of evidence of a ruptured intracranial arterial aneurysm and should include monitoring for vasospasm and measures aimed at reducing this dreaded complication.

The most common cause of tSAH is motor vehicle collision in 73 %, followed by fall in 20 % of the cases [7]. TSAH is considered an independent factor of bad outcomes in severe TBI [6]. The exact mechanism is unknown, but there are some theories to explain it, such as vertebrobasilar stretch due to hyperextension or tearing of bridging veins or pial vessels. Usually, a plain CT head is the best initial step as it is rapidly performed and cost-effective [8]. The amount and the location of tSAH are important prognostic factors [6–8].

Massive tSAH is not common. In a study by Macciocchi et al., which included 1317 patients with traumatic brain injury, tSAH was identified in 3.4 % of cases. In cases of massive tSAH, it is preferred to do a CT angiogram to rule out the possibility of a vascular malformation as the preceding event to the trauma [4]. In our reported case, the patient had low initial GCS and there were no witnesses to the trauma, CT angiogram and DSA were done which were negative for a vascular malformation. Another CT angiogram was done for follow-up at day 13 which was also negative for an aneurysm and arteriovenous malformation.

Vasospasm, a known complication of aSAH, is characterized by the narrowing of cerebral blood vessels, potentially leading to cerebral ischemia and neurological deficits. The incidence of vasospasm in aSAH is approximately 30 % [9]. However, vasospasm in the context of tSAH is relatively rare compared to aSAH. The incidence and prevalence of vasospasm specifically in tSAH are not well-defined due to limited research on this specific population. One study on 90 tSAH patients showed the risk of vasospasm was around 35.6 %, and the risk was higher in more diffuse and massive tSAH [10]. Vasospasm in tSAH is usually less severe than in aSAH, and the duration is shorter. It can start as early as two days after the bleeding, contrary to aSAH, which usually occurs between 4 and 14 days [11]. It is important to note that the pathophysiology of vasospasm in TBI patients is not only related to tSAH, and there is around 10–30 % risk of developing radiological vasospasm in TBI patients without the presence of tSAH. One study showed that the risk of developing brain infarction secondary to vasospasm in cases of tSAH is around 17 % [11]. Another study showed that there were no cases of brain infarction in TBI without radiological evidence of vasospasm [12]. In our case, there was clinical evidence of stroke, which was confirmed by CT head and MRI head, but a CT angiogram of the head did not show any evidence of vasospasm. There was a diffuse irregular narrowing of the intracranial vessels since day one, which is most likely atherosclerotic rather than vasospastic, as vasospasm usually does not appear in day one [11].

Proper management of tSAH and measures for prevention and treatment of vasospasm are not clear in the literature, and there are some differences between aSAH and tSAH. One difference is that TBI could be associated with other injuries or bleeding, and the regular SAH protocol used in aSAH, especially permissive hypertension, could be harmful in polytrauma cases [13]. On the other hand, there are differences in the literature regarding the role of calcium channel blockers in tSAH. One study showed that there was a benefit for a small subgroup [14] and another systematic review showed no benefits of calcium channel blockers [15]. In our case, the use of nimodipine did not prevent the stroke.

Conclusion

This case report illustrates the diagnostic challenge posed by massive traumatic subarachnoid hemorrhage mimicking aneurysmal subarachnoid hemorrhage. Clinicians should maintain a high index of suspicion for tSAH in patients with a history of head trauma, even in the presence of radiological findings consistent with aSAH. Close monitoring, interdisciplinary collaboration, and adherence to evidence-based practices are essential in optimizing patient outcomes.

Ethics approval and consent to participate

This study was approved by the Institutional Ethical Review Board at Hamad Medical Corporation. All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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