Case Series

Insights into subarachnoid hemorrhage: A comprehensive case series analysis

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ABSTRACT

Subarachnoid hemorrhage (SAH) is a critical neurological emergency characterized by bleeding into the subarachnoid space, typically resulting from the rupture of intracranial aneurysms. SAH is associated with high morbidity and mortality rates, making it a subject of significant clinical interest and concern. This case series aims to explore a series of patients with SAH, shedding light on the diverse clinical presentations, management strategies, and outcomes, with the ultimate goal of improving our understanding of this complex condition and enhancing patient care.

Key words: Cerebral aneurysm, External ventricular drain, Headache, Subarachnoid hemorrhage, Vasospasm

Subarachnoid hemorrhage (SAH) represents a formidable challenge in the field of neurosurgery and critical care medicine. The overall global incidence of aneurysmal SAH is 6.1/100000 person years. This life-threatening condition occurs when blood escapes from the vessels into the subarachnoid space, the region between the arachnoid membrane, and the pia mater surrounding the brain [1]. The most common cause of SAH is the rupture of intracranial aneurysms, although other etiologies such as arteriovenous malformations (AVM) and trauma can also lead to this devastating event. SAH is associated with a staggering mortality rate, and survivors often grapple with significant neurological deficits, making early recognition and intervention paramount. The abrupt onset of severe headache, often described as the "worst headache of my life" is a classic presentation. Other clinical manifestations may include nausea, vomiting, altered mental status, and focal neurological deficits [2]. Despite advances in diagnostic techniques and treatment modalities, the management of SAH remains multifaceted and challenging. Prompt diagnosis, neuroimaging, and angiography are pivotal in identifying the source of bleeding and planning interventions [3]. Treatment options encompass surgical clipping or endovascular coiling of aneurysms, along with supportive care to manage complications and mitigate secondary brain injury.

This case series endeavors to elucidate the intricate landscape of SAH by presenting a cohort of patients with diverse clinical scenarios. Through a comprehensive analysis of their presentation, management approaches, and outcomes, we aim to contribute to the evolving body of knowledge surrounding SAH and, ultimately, enhance the care provided to these critically ill patients.

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CASE SERIES

Case 1

A 64-year-old female patient was admitted with complaints of severe headache, 12–13 episodes of vomiting, aphasia, and weakness of limbs on both sides. She had a known history of hypertension for 15 years and was taking tab cilnidipine 10 mg BD. On admission, her blood pressure (BP) level was 170/80 mmHg. Her laboratory findings showed elevated levels of white blood cells (WBC) (14330 cells/cumm), random blood sugar (RBS) (205 mg/dL), and C-reactive protein (CRP) (44.1 mg/L). There was no evidence of stenosis, occlusion, or aneurysms other than a mild developing hydrocephalus in the computed tomography (CT) angio-cerebral scan. The CT brain revealed the presence of SAH in the bilateral brain parenchyma, sylvian fissures, and basal cisterns. It also showed extension into lateral 3rd and 4th ventricles with dilated lateral and 3rd ventricles (Fig. 1). Two small calcified granulomas were also present on the right side of the fronto-parietal lobes. Hence, the case was diagnosed as SAH.

She was treated with injection brivaracetam 50 mg TDS, injection ondansetron 4 mg TDS, injection acetaminophen 1 g, injection furosemide 20 mg BD, tablet cilnidipine 10 mg BD, tablet nimodipine 60 mg 1-1-1-1, and injection edaravone 1.5 mg BD. Placement of an external ventricular drain (EVD) was done to manage hydrocephalus and elevated intracranial pressure

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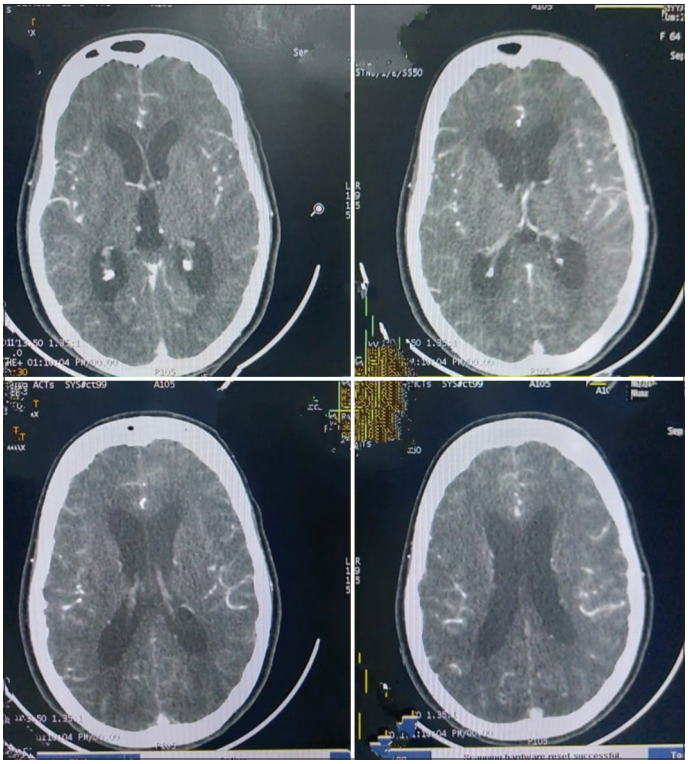

Figure 1: Computed tomography brain showing subarachnoid hemorrhage (case 1)

which occurred as a result of SAH. She was clinically stable with a controlled BP (140/80 mmHg) and discharged. On follow-up, she was stable and a repeat CT scan showed normal findings.

Case 2

A 60-year-old female patient was admitted with one episode of syncope, loss of consciousness, and one episode of vomiting since that day. She had a history of trauma (a fall from a bike). She was a known patient of Type 2 diabetes mellitus and was on oral hypoglycemic agents. On admission, her BP level was 160/90 mmHg. There was an elevated level of WBC (16590 cells/cumm). Plain CT-brain showed thin subdural hemorrhage (SDH) involving the right frontoparietal region and thin SAH involving the left sylvian fissure. An undisplaced longitudinally oriented fracture line in the right mastoid and petrous temporal bone with minimal adjacent pneumocephalus, hemotympanum, and hemomastoid were seen. The case was thus diagnosed as Thin SAH. She was treated with injection amoxicillin–clavulanate 1.2 g BD, injection of brivaracetam 50 mg TDS, injection of ondansetron 4 mg TDS, injection of pantoprazole 40 mg BD, tablet of clonidine 0.1 mg BD, and tablet cilnidipine 10 mg BD. She became clinically stable and was discharged.

Case 3

A 52-year-old female patient was admitted to the neurosurgery department with chief complaints of headache, vomiting, and episodes of seizures for 3 days. She denied having any medical history. On admission, she underwent all routine laboratory investigations such as blood tests and CT scans. Laboratory investigations showed increased levels of WBC (18790 cells/cumm), BP (180/100 mmHg), and decreased sodium levels (124 mmol/L). CT scan showed SAH in the right frontoparietal temporal region and basal cisterns. There was also SDH along the falx and tentorium cerebelli. Diffuse periventricular white matter ischemic changes were also seen. She has managed with injection of brivaracetam 50 mg, injection of pantoprazole 40 mg, tablet of nimodipine 60 mg 1-1-1-1, injection of ondansetron 4 mg, syrup zincovit 2tsp, and tablet of clonidine 0.1 mg BD. The patient was able to tolerate all the medications and she became clinically stable, hence discharged.

DISCUSSION

Hemorrhagic strokes (13% of strokes) include SAH and intracranial hemorrhage. SAH may result from trauma or rupture of an intracranial aneurysm or AVM. Understanding the range of symptoms from the classic thunderclap headache to more subtle signs such as nausea, vomiting, altered mental status, and focal neurological deficits, plays an important role in the management of SAH [4].

SAH due to aneurysm rupture is often associated with delayed cerebral ischemia in the 2 weeks after the bleeding episode [5,6]. Vasospasm of the cerebral vasculature is thought to be responsible for the delayed ischemia and occurs between 4 and 21 days after bleed. The calcium channel blocker nimodipine 60 mg every 4 h–21 days, along with maintenance of intravascular volume with pressor therapy, is recommended to reduce the incidence and severity of neurologic deficits resulting from delayed ischemia which had been used as a treatment option in these cases. Nimodipine has a definite neuroprotective effect, such as an antioxidant effect, which improves the cerebral metabolic rate of oxygen and reduces brain injuries due to calcium overload during cerebral blood flow reperfusion. Its neuroprotective effect can also relieve brain edema and glial cell swelling after SAH [7-9].

Severe hydrocephalus may require urgent placement of a ventricular catheter for external Cerebrospinal fluid (CSF) drainage. EVD was done in one of the cases to manage hydrocephalus [10,11]. Seizure prophylaxis should also be carried out in SAH cases as in these cases brivaracetam was used as the antiepileptic drug. Further management aims to maintain controlled BP, reducing possible edema by the use of diuretics, and a repeat CT scan to evaluate the ventricular size.

Based on the cases and literature, it is important to consider aspects such as optimal timing for intervention (surgical or endovascular) in SAH cases. The use of nimodipine is also practiced widely in SAH which is based on standard treatment guidelines. Factors influencing the choice between coiling and clipping, and strategies for post-treatment care and monitoring are also important in this condition [12]. Promptly assess and diagnose SAH through imaging studies, such as non-contrast CT scans and angiography to identify the source of bleeding. Consider early neurosurgical intervention for securing ruptured aneurysms. Options include surgical clipping or endovascular coiling, with

the choice guided by factors such as aneurysm location, size, and patient-specific characteristics. Coiling is a less invasive procedure where tiny coils are placed inside the aneurysm to promote blood clotting and prevent rupture. Clipping, on the other hand, involves placing a small metal clip around the neck of the aneurysm to stop blood flow. Maintain adequate BP control to prevent rebleeding while ensuring sufficient cerebral perfusion. Individualize BP targets based on the patient's clinical condition and the presence of other comorbidities [13]. Administer nimodipine to prevent and manage cerebral vasospasm, a common complication of SAH, to improve neurological outcomes. Consider prophylactic antiepileptic medications to prevent seizures, which can occur as a complication of SAH. Monitor intracranial pressure in cases where cerebral edema or other factors may contribute to increase increased intracranial pressure (ICP). Implement strategies to manage elevated ICP promptly. Address hydrocephalus promptly, which can occur due to blood accumulation or impaired CSF circulation, through the placement of the EVD. Utilize multimodal monitoring in critical cases, incorporating tools such as cerebral oxygen monitoring and continuous electroencephalogram to assess brain tissue oxygenation and detect potential ischemia or secondary insults. Initiate early rehabilitation and establish a comprehensive follow-up plan to address physical, cognitive, and psychological sequelae of SAH. Implement measures to prevent and manage complications such as infections, deep vein thrombosis, and stress ulcer prophylaxis.

CONCLUSION

This case series offers a comprehensive exploration of SAH, highlighting the diverse clinical presentations, diagnostic challenges, and therapeutic interventions encountered. The variability in symptoms underscores the importance of maintaining a high index of suspicion, particularly in cases with atypical presentations. The therapeutic journey revealed the complexities in managing SAH, with neurosurgical interventions, BP control, and nimodipine administration emerging as key pillars in the approach. Complications including vasospasm and hydrocephalus were effectively managed through a multimodal strategy, incorporating advanced monitoring techniques. Ultimately, this case series aims to enrich the existing knowledge base on SAH, providing clinicians with practical insights into the nuanced management of this critical condition. Further, collaborative efforts and ongoing research remain imperative to improve outcomes and enhance the quality of care for individuals affected by SAH.

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