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Subarchnoid Hemorrhage Following Severe Anemia: A Rare Case Reports

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ABSTRACT

Introduction: Anemia occurs in 40–50% of patients with subarachnoid hemorrhage (SAH), while transfusion for severe anemia may increase the risk of intracranial hemorrhage. Approximately 85% of non-traumatic SAH results from ruptured intracranial aneurysms. We report a rare case of SAH following multiple transfusions in a patient with severe chronic anemia. **Case Presentation**: A 30-year-old woman developed status epilepticus after receiving eight units of packed red blood cells (PRBCs) for severe anemia. Before admission, she experienced a thunderclap headache, photophobia, scotoma, and nausea. Her blood pressure was 145/89 mmHg. Non-contrast head CT revealed occipital SAH, while electroencephalography showed generalized intermittent slowing. SAH was classified as Fisher grade II, Hunt and Hess grade III, and World Federation of Neurological Surgeons grade I. Hemoglobin increased from 3.1 g/dL before transfusion to 8.7 g/dL at discharge, while blood pressure increased from 83/57 mmHg to 147/86 mmHg. She required no further transfusions or antihypertensive therapy and was discharged in stable condition. **Conclusion:** Rapid correction of severe anemia may cause acute hemodynamic changes that potentially increase intracranial hemorrhage risk. Careful transfusion and close blood pressure monitoring are warranted in patients with severe chronic anemia.

Keyword: Anemia, Blood Transfusion, Subarachnoid Hemorrhage

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a life-threatening neurological emergency. Commonly caused by the rupture of an intracranial aneurysm. There are approximately 85% of non-traumatic cases (Macdonald & Schweizer, 2016). Anemia is reported in 40–50% of patients with SAH and is associated with poor neurological outcomes and increased mortality (Said et al., 2022). Conversely, blood transfusion often administered to correct severe anemia has been linked to elevated risks of intracerebral or subarachnoid hemorrhage due to sudden hemodynamic and rheological changes (Schmitt et al., 2022; English et al., 2018).These findings highlight the delicate balance between treating anemia and avoiding transfusion-related complications.

Patients with chronic anemia such as sickle cell disease and β-thalassemia major are persistently low blood viscosity (Keller et al., 2021; Sanju et al., 2019). A sudden increase in viscosity following transfusion may strain the cerebral vasculature, leading to endothelial injury, hypertension, or even hemorrhagic stroke (Schmitt et al., 2022). The inability of vessel walls to adapt to altered hemodynamic forces may precipitate intracranial bleeding, particularly in those with compromised vascular integrity or pre-existing microangiopathy (Grubb et al., 2016). Although several studies have examined ischemic complications in these populations, hemorrhagic events remain underreported and poorly understood (Said et al., 2022).

This report aims to describe a rare case of subarachnoid hemorrhage following blood transfusion in a severely anemic patient and to discuss the possible pathophysiological mechanisms linking transfusion, increased blood viscosity, and vascular rupture.

CASE PRESENTATION

The patient came to the emergency room with 2 times seizures at home at 12.00 noon, the seizures lasted approximately 1-2 minutes, between the two seizures the patient was loss of consciousness. The distance between the first and second seizures is about 15 minutes. During a seizure, the patient's entire body moves with fast rythm and her eyes glancing upwards. Before the seizure, the patient complained of severe headache she ever had. She complained her both eyes were pain when she tried to see light from lamp and there

was a black spot in the middle of the both eyes. At 3 pm in the emergency room the patient had another seizure for approximately 1 minute, convulsing all over her body with eyes glancing upwards. After the seizure the patient decrease of consciousness and cannot communicate well. 4 days ago the patient had just finished being treated in hospital caused by pansitopenia. The patient was hospitalized for 11 days and given 8 blood transfusions. It was the first time she had a blood transfusion. She had low activity than her friends since toddler and yellow skin. But she didn't complain anything else. She doesn't consume oral contraception.

Vital sign revealed a hypertension with blood pressure 145/83mmHg, respiration rate 20x/minute, heart rate 70x/minute regular, temperature 36,2C. On physical examination showed facies cooley, anemic conjunctiva (+), nuchal rigidity (+), mild left facial parese UMN type, splenomegaly scuffner IV, photophobia, scotoma, lingual deviation to left, and jaundice. Laboratory investigation revealed anemia (8.9 g/dL), trombositopenia (128.000 sel/ul), leukopenia (3.41), coagulation factor increase not significant prolonged, increase of reticulocyte 5,5 %, increased of direct (2.70) and total bilirubin (6.32). Head ct scan without contras showed subarachnoid hemorrhage on left occipital area (figure 1). Peripheral blood smear revealed pancytopenia with a suspicion of hemoglobinopathy, differential diagnosis of thalassemia. Chest X-ray demonstrated cardiomegaly with a cardiothoracic ratio (CTR) of 68% (figure 2). The EEG indicated general intermittent slow activity which indicates the occurrence of a true seizure (Figure 3).

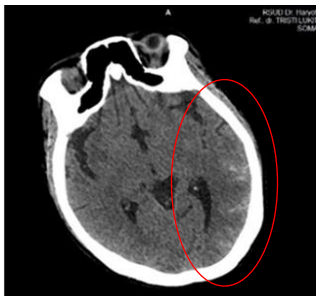

Figure 1. Non-contrast head CT scan demonstrated subarachnoid hemorrhage on left occipital area

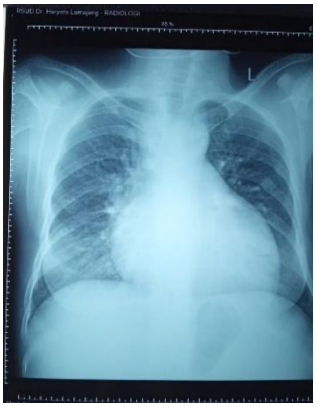

Figure 2. Thorax X ray showed cardiomegaly with a cardiothoracic ratio (CTR) of 68%

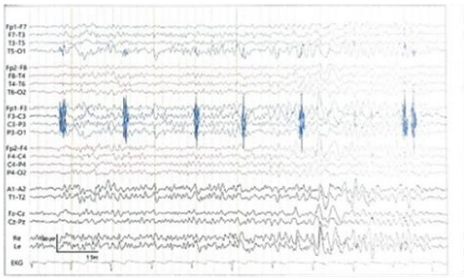

Figure 3. The EEG indicated general intermittent slow activity which indicates the occurrence of a true seizure

DISCUSSION

Subarachnoid hemorrhages (SAH) are life-threatening disease which cause accumulation of blood between the arachnoid and pia mater. Subarachnoid space is described as a space between the arachnoid membrane and the pia mater. It consists of the cerebrospinal fluid and the blood vessels that supply different areas of the brain. The incidence of subarachnoid hemorrhage in the United States is between 10 to 14 out of 100,000 individuals per year. The etiology of SAH can be either nontraumatic (about 85% are secondary to aneurysm rupture) or traumatic in nature. Most aSAH occur between 40 and 60 years of age. Most nontraumatic causes of aSAH (~85%) are caused by the rupture of an intracranial aneurysm. Hypertension, cigarette smoking, and family history are the most consistently observed risk factors with aneurysm formation (Ziu et al., 2023).

We report rare case aSAH in a young female, approximately 30 years old with the hemorrhage located in the occipital area. There was no history of hypertension, diabetes mellitus, chronic headache, and significant recent weight loss. Further CTA is needed to confirm the presence of an aneurysm. The novelty of this report lies in the occurrence of a hemorrhagic event after blood transfusion in a chronically anemic patient. It

suggesting a possible link between rapid viscosity changes and vascular rupture.

The presentations may vary, the characteristic presenting symptom is the thunderclap headache, which patients may describe as the 'worst headache of my life (Ziu et al., 2023). Headache is one of the most common reasons for presentation seen in up to 2% of patients. Symptoms that increase the likelihood of a subarachnoid bleed as the cause of headache include exertional onset, syncope, vomiting, neck pain, and seizures. Focal neurologic deficits, meningismus, and/or retinal hemorrhage may be present, but up to 50% of SAH patients have a normal neurologic exam (Kumar, 2019). Photophobia is defined as a painful psychosomatic discomfort triggered by intense light flow through the pupils to the brain, but the exact mechanism through which photophobia is induced by subarachnoid hemorrhage (SAH) is not well understood (Li et al., 2019). In our case, the chief complaint of the patient was seizures, in addition to severe headache, photophobia and nausea.

The Ottawa SAH rule and the 6-hour-CT rule are highly sensitive and can be used routinely when SAH is considered in patients with headache. The components of the Ottawa are age >= 40 years old, neck pain or stiffnes, witnessloss of consciousness, onset during exertion, thunderclap headache which peak intensity immediately, and limited neck flexion on exam. Patient require investigation if one or more finding present (Hoh et al., 2023). In this case, patient 3 requires evaluation of 6 symptom, which need further investigation. Many tools are available to assess for SAH including non-contrast CT, LP, CTA, and MRI (Kumar, 2019). The CT scan revealed a subarachnoid hemorrhage (SAH) in the bilateral occipital region and cerebral atrophy. Due to the seizure complaints, an EEG was needed to determine the etiology of the seizures. The EEG revealed abnormal findings in both wake and sleep stages, including intermittent slow activity (ISA), indicating a mild degree of cerebral cortical dysfunction with potential for seizures.

In another case, an intracerebral hemorrhage occurred in a child with major thalassemia following three packed red cell transfusions. The transfusions increased the hemoglobin concentration from 4.2 mg/dl to 9.4 mg/dl over seven days. On the ninth day, the patient experienced two episodes of projectile vomiting followed by two episodes of generalized tonic-clonic convulsions. The Glasgow Coma Scale (GCS) was 8/15 indicating signs of raised intracranial pressure. The heart rate was 68 beats per minute, and blood pressure was 120/80 mmHg (99th percentile) in the right upper arm in a supine position. The cause of the parenchymal hemorrhage (without coagulopathy) in this patient appears to be the inability of the blood vessel walls to manage a sudden increase in blood viscosity after repeated transfusions (Sanju et al., 2019).

Similar to previous reports, our patient developed intracranial bleeding shortly after multiple blood transfusions. However, unlike earlier cases that occurred in children with thalassemia, our patient was an adult with no known hemoglobinopathy. In this case, the patient was previously hospitalized due to severe anemia and received eight packed red cell transfusions over eleven days. Post-transfusion hemoglobin increased to 8.7, and the patient was discharged. Four days later, the patient returned to the emergency room with complaints of seizures. The patient reported severe headache, photophobia, and nausea.

Recently, research found intracerebral hemorrhage and aneurysm according to prior established criteria for MRI and MRA on sickle cell anemia. The saccular aneurysm prevalence of 9% in a relatively young adult +- 30 years with sickle cell anemia may have a higher prevalence of cerebral aneurysms on internal carotid interna (Kumar, 2019; Strouse et al., 2016).

The case demonstrates a possible link between rapid increases in blood viscosity after transfusion and cerebral hemorrhage in a non-hemoglobinopathy patient. The mechanism rarely discussed in the literature. New onset seizure on thalassemia may be caused by intracranial infection, ICH, metabolic derangements, posterior reversible encephalopathy syndrome, and cerebral infarct. The precise cause of ICH in patients with thalassemia remains unknown. However, many hypotheses have been put to explain this catastrophic event (Li et al., 2019).

First, coagulation defect can be found quantitative or qualitative defect/s in platelets, deranged prothrombin time (PT), international normalized ratio (INR) and partial thromboplastin (aPTT) (Li et al., 2019). On laboratory test, the patient had abnormality on coagulation aspect. Those can be shown on the quantity of trombocyte 129.000 sel/uL; aPTT 60,9 second; D-Dimer 1,0 mg/dL; PT 14,5 second; and INR 1, 39. Abnormality of coagulation cascade, resulting in thrombotic and hemorrhagic complications due to intravascular fibrin deposition, microangiopathic

thrombosis, and depletion of platelets and coagulation factors (Costello et al., 2025).

Second, the inability of blood vessel walls in sickle cell anemia is due to their weakened state. Chronic exposure to low-viscosity blood means these vessels may not handle a sudden increase in blood viscosity after transfusion, which can lead to hemorrhagic stroke (Keller et al., 2021; Sanju et al., 2019). Third, reversible cerebral vasoconstriction syndrome (RCVS) characterized by sudden explosive and severe headaches that occur over days to weeks due to underlying widespread cerebral vasoconstriction. Although typically benign and self-limited, RCVS can lead to severe complications such as ischemic stroke, convexity subarachnoid or other intracerebral hemorrhage, and posterior reversible encephalopathy syndrome (PRES) (Nesheiwat & Al-Khoury 2024).

Once the diagnosis of SAH is established, the most important time-sensitive goals include confirmation of airway security and stabilization of hemodynamics. The next priorities are to reduce systolic blood pressure (BP) and reverse anticoagulation to mitigate the risk of aneurysm re-rupture. Specific BP goals are unclear and need to be weighed against the risk of ischemia or infarction with hypotension. Guidelines recommend targeting BP < 160 systolic, although many consider lower targets of 140–150. Nicardipine (5 milligrams per hour (mg/h) intravenous (IV), may increase by 2.5 mg/h maximum 15 mg/h) (Terrett et al., 2023). Patient blood pressure was 145/85 mmHg so we didnt gave anti hypertension drug for the patient. The patient blood pressure followed up a day later, was 110/79 mmHg.

Prevention of vasospasm by administering a calcium channel blocker, such as nimodipine. Nimodipine was given to avoid the vasospasm on subarachnoid hemorrhage. Nimodipine is typically given to patients with SAH caused by aneurysm rupture (Pickard et al., 1989). However, in this case, according to the anamnesis and imaging, the etiology could not be determined due to the rare risk factors associated with the aneurysm and cerebral venous thrombosis caused by coagulopathy in sickle cell anemia.

Anticoagulants were not administered because current evidence indicates that antifibrinolytic therapy is not indicated for the routine management of patients with aneurysmal subarachnoid hemorrhage (aSAH). On the other hand, we could not determine whether the etiology of the subarachnoid hemorrhage was due to aneurysm rupture or another cause. A CTA is needed to identify the origin of the hemorrhage. Management of status epilepticus, the patient was given a maintenance of phenytoin 100 mg TID and oral folic acid 1 mg BID. Phenytoin was administered until the patient was seizure-free for three days. Phenytoin was then switched to an oral form. Folic acid was provided due to phenytoin's side effect of causing folate depletion in some epileptic patients.

Clinicians should be aware that rapid correction of severe anemia through multiple transfusions can precipitate hemodynamic instability and cerebral bleeding, especially in chronically anemic patients. In summary, while anemia and transfusion are well-known clinical concerns, their association with hemorrhagic stroke remains underrecognized. This case adds to the limited literature emphasizing the importance of gradual transfusion and close monitoring of blood pressure and viscosity in high-risk patients.

CONCLUSION

The present case highlights a potential association between rapid correction of severe anemia and subarachnoid hemorrhage. Careful hemodynamic monitoring during transfusion is essential, particularly in patients with chronic anemia or impaired vascular integrity.

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