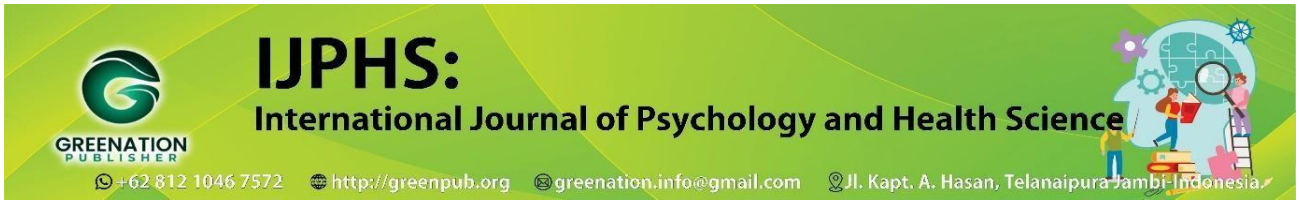

DOI: https://doi.org/10.38035/ijphs.v3i1
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A Patient Chronic Kidney Disease with Complication Subdural Hematome during Hemodialisis Programe

I Putu Eka Dharma Putra[1]
[1]Kertha Usadha hospital Singaraja, Bali, Indonesia, vios333vaa@gmail.com

Corresponding Author: vios333vaa@gmail.com[1]

Abstract: Subdural hematoma (SDH) is a critical neurosurgical condition often linked to chronic kidney disease (CKD) patients undergoing hemodialysis (HD). This case report presents a 56-year-old male with CKD stage V and SDH as a complication during HD therapy. The patient experienced a sudden decrease in consciousness following routine dialysis. Diagnostic evaluations revealed chronic subdural hematoma with midline shift, accompanied by chronic nephritis and anemia. Management included burr hole surgery, heparin-free dialysis, and supportive medical therapy. The patient showed significant recovery, highlighting the importance of timely intervention and a multidisciplinary approach to manage SDH in CKD patients undergoing HD. Case report: A 56-year-old male CKD stage V patient on routine HD presented with acute headache and decreased consciousness post-dialysis. CT imaging confirmed chronic SDH with a midline shift. Treatment involved burr hole drainage and heparin-free dialysis. The patient recovered well after four days of hospitalization and resumed dialysis without further complications. Discussion: The pathophysiology of SDH in HD patients involves changes in intracranial pressure and impaired hemostasis due to CKD and dialysis-related factors. The use of anticoagulants further exacerbates bleeding risks. This case underlines the need for careful monitoring, individualized HD protocols, and timely surgical intervention for SDH in CKD patients. Conclusion: This case emphasizes the critical risk of SDH in HD patients, advocating for preventive strategies, including modified dialysis protocols and multidisciplinary care, to mitigate complications and improve patient outcomes.

Keywords: Subdural hematoma, chronic kidney disease, hemodialysis, intracranial bleeding, burr hole surgery

INTRODUCTION

A subdural hematoma (SDH) is a frequently encountered neurosurgical condition, marked by ongoing and repeated bleeding due to the traumatic rupture of blood vessels. The clinical presentation of SDH varies depending on where it occurs within the skull. SDH can be categorized into acute, subacute, and chronic stages based on the time of its progression. Several risk factors contribute to

the likelihood of developing SDH, such as advanced age, head trauma, use of anticoagulants or antiplatelet medications, reduced intracranial pressure, and undergoing hemodialysis (HD). (1)

Chronic kidney disease (CKD) refers to a long-term structural or functional impairment of the kidneys, caused by various factors, and has become a significant global public health issue. Over time, it advances to end-stage kidney disease (ESKD), which is typically irreversible. In recent years, both the incidence and mortality rates of CKD have risen sharply. The main treatment options for CKD include kidney replacement therapies (KRT), such as hemodialysis (HD), peritoneal dialysis (PD), or kidney transplantation. CKD patients often experience a build-up of toxins in the body, leading to multiple complications. (2)

Patients with chronic kidney disease (CKD) face a 3 to 10 times higher risk of stroke compared to the general population. Due to common brain atrophy in CKD patients, the increased length of the pontine vein, which is more susceptible to tearing, raises the incidence of subdural hematoma (SDH). Those on long-term hemodialysis (HD) are at an even greater risk, with a 10-fold increase in the likelihood of developing SDH compared to the general population. This elevated risk is likely associated with HD-induced changes in intracranial pressure, cerebral blood flow, and subdural pressure. Although nontraumatic SDH (NSDH) in patients with end-stage kidney disease (ESKD) undergoing HD is infrequently documented, most of the cases have been reported in case studies or small case series. To better understand the pathophysiology and risk factors involved in NSDH development during HD, we carried out a systematic review and comprehensive analysis of the available literature. (3)

METHOD

A 56-year-old man of Balinese Hindu ethnicity, married, working as a farmer, came to Kertha Usadha General Hospital with a complaint of decreased consciousness that occurred a few hours before being admitted to the hospital. Initially, the patient complained of a headache, which did not improve with rest. The family later found the patient's consciousness worsening, and he was eventually brought to the nearest emergency unit. The patient experienced these acute symptoms after undergoing routine dialysis at the hospital the previous day.

The patient had no previous history of similar complaints. He has been diagnosed with stage 5 chronic kidney disease and undergoes routine dialysis at Kertha Usadha General Hospital twice a week.

The patient has never previously complained of similar symptoms. In terms of family history, most family members, especially the patient's parents, suffer from hypertension. However, the patient is the only one in the family undergoing routine dialysis. Additionally, there is a history of diabetes and stroke among elderly family members.

On physical examination, the patient appeared to be in moderate pain, with a somnolent level of consciousness. Blood pressure was 150/90 mmHg, pulse rate 88 beats per minute, respiratory rate 24 breaths per minute, and axillary temperature 36.7°C. The pain was rated at 6 out of 10 on the Visual Analogue Scale (VAS) due to the headache. The patient weighed 60 kg. On general physical examination, cardiomegaly was noted in the heart, while the lungs and abdomen were found to be within normal limits. Additionally, tenderness was observed in the symphysis region.

On laboratory examination, the complete blood count showed hemoglobin levels of 8.72 g/dL, platelet count of 254 x 10^3/µL, and leukocytes at 9.24 x 10^3/µL. The HBsAg and Anti-HCV tests were non-reactive. The lipid profile revealed total cholesterol, HDL cholesterol of 33 mg/dL, direct LDL of 168 mg/dL, and triglycerides of 88 mg/dL. Uric acid level was 8.5 mg/dL, serum iron was 133.9 µg/dL, and TIBC was 191 µg/dL. Urinalysis showed a specific gravity of 1.015, pH 5, leukocytes at 500 (+3), nitrite negative, protein at 75 (+2), glucose negative, ketones negative, urobilin negative, bilirubin negative, with many leukocyte and erythrocyte sediments. Blood

chemistry results were as follows: INR 1.1, APTT 33.7, GOT 17, GPT 13, total bilirubin 0.3 mg/dL, direct bilirubin 0.1 mg/dL, total protein 8.5 g/dL, albumin 4.0 g/dL, fasting blood sugar 80 mg/dL, BUN 91 mg/dL, serum creatinine 8.4 mg/dL, uric acid 8.6 mg/dL, sodium 140 mEq/L, and potassium 4.6 mEq/L.

The contrast-enhanced CT scan showed a disc-shaped density in the right frontal region with a maximal thickness of 1.8 cm, suggesting a differential diagnosis of 1) Epidural hematoma. There was also a crescent-shaped hemorrhage in the left frontotemporal region with a maximal thickness of 0.8 cm, with a differential diagnosis of 1) Subdural empyema or 2) Chronic subdural hematoma (SDH). A midline shift of 3.2 mm to the left was also observed. The ultrasound examination showed chronic nephritis in both the right and left kidneys.

Based on the anamnesis, physical examination, and supporting tests, the patient was diagnosed with stage V chronic kidney disease due to chronic pyelonephritis, mild normochromic normocytic anemia associated with chronic kidney disease, controlled hypertension, and subdural hematoma.

The most recent therapy provided included NaCl 0.9% at 8 drops/minute, a diet of 35 kcal/kg body weight/day and protein intake of 0.8 grams/kg body weight/day plus additional protein to compensate for losses. Oral therapy included candesartan 8 mg once daily, ranitidine 1 ampule twice daily, folic acid 1 tablet twice daily, and osteocal 500 mg three times daily. A surgical procedure (burr hole operation) was performed to treat the subdural hematoma (SDH). The patient was hospitalized for 4 days, during which wound care and dialysis were performed as scheduled. Tranexamic acid injections were administered throughout the hospitalization, and dialysis was carried out with heparin-free treatment. On the 5th day, the patient was discharged. On the 10th day, the patient returned for a follow-up at the outpatient clinic. The patient reported no complaints or deterioration and continued follow-ups with the surgery clinic and regular dialysis according to the schedule.

RESULT AND DISCUSSION

Hemodialysis (HD) could elevate the risk of developing acute subdural hematoma (SDH), a condition that carries a high mortality rate, though the full impact of this disease remains uncertain. Studies have highlighted the significant occurrence and death rates associated with SDH in patients undergoing long-term dialysis, especially among the elderly. Various factors are believed to contribute to this increased risk.(4)

The risk of bleeding increases when Fragmin is used during dialysis, especially when combined with antiplatelet drugs for treating cerebrovascular disease. This heightened risk can also be attributed to uremia, a condition that impairs platelet function, primarily due to severely reduced kidney function. Anemia further contributes to this bleeding tendency. Hemodialysis patients, as well as those with other bleeding disorders, are more susceptible to spontaneous or traumatic subdural and intracerebral hematomas. (5)

Rossier et al. also discovered that elderly dialysis patients face a higher risk compared to the general population. This increased vulnerability is attributed to factors such as advanced age, comorbid conditions, cognitive decline, vitamin D deficiency, protein malnutrition, and post-dialysis hypotension. Cognitive dysfunction may stem from uraemia, and neuropsychiatric side effects from commonly prescribed medications are more likely in patients with ESRD, particularly those undergoing dialysis.8 Haemodialysis has been linked to greater fluctuations in intracranial pressure, changes in cerebral blood flow, and reduced pressure in the subdural space.9 Therefore, extra caution is needed regarding the elevated risk of subdural hematoma in patients receiving anticoagulant treatment. (4)

Mehrotra et al. reported that the patient showed progress with adherence to the treatment plan, though some limitations were noted. First, there was a lack of data on lifestyle factors, Glasgow

Coma Scale scores, frailty, and certain laboratory results. Second, it was not possible to distinguish between acute and chronic SDH due to the absence of neuroimaging records in the reports. Lastly, the accuracy of SDH and comorbidity diagnoses in the claims data could not be confirmed. (6)

The rising incidence of subdural hematoma is closely linked to the increased use of anticoagulants in long-term haemodialysis patients. Therefore, careful management is essential to prevent this serious condition. The routine use of heparin during HD sessions may exacerbate bleeding tendencies, contributing to the higher occurrence of subdural hematoma in these patients. Providing appropriate care is vital to avoid this debilitating complication.(7)

This case report highlights a significant presence of underlying comorbidities in patients who develop SDH and face a high risk of immediate mortality. Therefore, the benefits of long-term anticoagulation therapy should be carefully assessed and monitored against the risk of central nervous system bleeding in maintenance haemodialysis. Emphasis on early detection and proactive management of chronic kidney disease, in accordance with KDIGO foundation guidelines, is essential to address the condition at earlier stages.

This report confirms that patients undergoing haemodialysis (HD) are at a higher risk of developing subdural hematoma (SDH). This elevated risk is linked to the increased mortality rate seen in dialysis patients affected by SDH. As a result, it is crucial to introduce preventive strategies to reduce the incidence of SDH in individuals with end-stage renal disease (ESRD), particularly those receiving HD treatment. Immediate steps must be taken to address this critical issue.

Some of the preventive and health maintenance strategies I recommended under the guidance of my supervisor include the following: The patient should improve their nutritional intake, minimize anemia, and consume high-quality, high-protein, low-sodium, and vitamin-rich foods to prevent hypoproteinemia. I also advised the patient's family to strictly limit sodium and water intake during dialysis. Since the patient's weight is below the normal range for their age, I recommended a gradual weight increase of no more than 3-5% of their body weight, while avoiding rapid ultrafiltration. Additionally, I advised the patient not to eat during haemodialysis and to have a meal 1-2 hours before starting dialysis for optimal safety.(8)

At this stage, the removal of solutes and water through dialysis accounts for only about 40% of the expected target, resulting in minimal impact on the peripheral circulating blood volume and avoiding significant drops in blood pressure. Compared to other treatments, haemodialysis is more likely to cause emotional disturbances such as anxiety and fear, which increase the psychological burden and raise the likelihood of hypotension. I made efforts to communicate frequently with the patient, helping them express their negative emotions and maintain a positive psychological state. Additionally, by providing comfort and reassurance, the patient's overall satisfaction was high, which contributed to improved clinical outcomes. (9)

Intracranial bleeding in haemodialysis patients is a critical and potentially fatal condition. Compared to the general population, these patients experience a higher frequency of spontaneous or traumatic subdural and intracerebral haematomas. This increased risk is partly linked to haemostasis dysfunction, a common issue in end-stage renal disease and haemodialysis. Studies have reported an estimated 3.3% incidence of subdural haematomas among chronic haemodialysis patients, accompanied by a strikingly high mortality rate of up to 85%. However, recent data on the broader incidence of subdural and intracerebral haematomas in haemodialysis patients is lacking. It is believed that the current incidence may be lower than 3.3%, likely due to advancements in anticoagulation methods, such as the use of low-dose heparin instead of systemic heparinization, and the reduced necessity for oral anticoagulants to ensure the patency of modern arteriovenous access.(10)

Despite advancements, haemodialysis patients with intracranial haemorrhage continue to face a notably high mortality rate. The standard treatment for subdural or intracerebral haematomas

typically involves a combination of surgical drainage, where feasible, and conservative supportive measures such as heparin-free dialysis, strict bed rest, and anti-oedema therapy. However, addressing impaired haemostasis is often not a primary focus of these treatments.(11)

Patients undergoing haemodialysis, as well as those with bleeding tendencies, are susceptible to both spontaneous and traumatic subdural and intracerebral hematomas. Furthermore, surgical evacuation of blood from the subdural space frequently leads to the recurrence of hematomas. This phenomenon is linked to the well-documented haemostatic impairment observed in haemodialysis patients. Additionally, even in individuals without an obvious bleeding disorder, localized haemostatic deficiencies contribute to the reformation of subdural hematomas after surgical removal. (12)

Therefore, a pharmacological approach aimed at enhancing haemostatic function could potentially improve outcomes for haemodialysis patients suffering from subdural and/or intracerebral hematomas. Tranexamic acid, a strong antifibrinolytic agent, effectively tilts the balance between coagulation and fibrinolysis towards coagulation, thereby boosting the blood's haemostatic capacity. This agent has demonstrated success in managing various bleeding conditions. (13)

Following the surgical removal of a third hematoma, a fourth subdural hematoma developed, leading to an anticipated poor prognosis. At this point, tranexamic acid treatment was initiated. Remarkably, the patient experienced a rapid and complete clinical recovery, confirmed by CT scans showing the resolution of the hematomas. Such spontaneous recovery is exceptionally rare, especially in patients with a clear bleeding tendency. This suggests that tranexamic acid, by enhancing impaired haemostasis, effectively halted intracranial bleeding, prevented the hematomas from expanding further, and facilitated their swift resorption.(14)

CONCLUSION

In summary, we propose that tranexamic acid could be a valuable treatment option for haemodialysis patients with subdural and/or intracerebral hematomas. While conclusions cannot be definitively drawn from a single case, a trial use of tranexamic acid is reasonable given the typically poor prognosis of the condition and the drug's established safety profile. Furthermore, tranexamic acid may also benefit patients without a prior bleeding tendency who experience recurrent bleeding after surgical drainage of a subdural hematoma. However, its potential effectiveness, particularly when combined with surgical interventions and supportive care, warrants further investigation. Collaboration with surgical teams and careful management of heparin use during haemodialysis are crucial for optimizing patient outcomes.

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