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■ Case Report ■

Selection of Treatment for Large Non-Traumatic Subdural Hematoma Developed during Hemodialysis

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A 49-year-old man with end-stage renal disease was admitted to the hospital with a severe headache and vomiting. On neurological examination the Glasgow Coma Scale (GCS) score was 15 and his brain CT showed acute subdural hematoma over the right cerebral convexity with approximately 11-mm thickness and 9-mm midline shift. We chose a conservative treatment of scheduled neurological examination, anticonvulsant medication, serial brain CT scanning, and scheduled hemodialysis (three times per week) without using heparin. Ten days after admission, he complained of severe headache and a brain CT showed an increased amount of hemorrhage and midline shift. Emergency burr hole trephination and removal of the hematoma were performed, after which symptoms improved. However, nine days after the operation a sudden onset of general tonic-clonic seizure developed and a brain CT demonstrated an increased amount of subdural hematoma. Under the impression of persistent increased intracranial pressure, the patient was transferred to the intensive care unit (ICU) in order to control intracranial pressure. Management at the ICU consisted of regular intravenous mannitol infusion assisted with continuous renal replacement therapy. He stayed in the ICU for four days. Twenty days after the operation he was discharged without specific neurological deficits.

Key Words: acute subdural hematoma; end stage renal disease; hemodialysis; renal replacement therapy.

Traumatic acute subdural hematoma (SDH) has been actively addressed in many studies, and treatment guidelines for traumatic acute subdural hemorrhage (TASDH) specifically recommends surgical evacuaiton if the thickness is greater than 10 mm, and the midline shift is greater than 5 mm on computed tomographic (CT), regardless of initial Gasgrow Coma Scale (GSC) assessment.[1] However, the guidelines may not apply to non-traumatic acute SDH developed in patients with severe systemic disease conditions like end-stage renal failure because it would be quite different from traumatic SDHs. In fact, there is no treatment guidelines for non-traumatic SDHs associated with a serious underlying condition. We experienced a case of a large non-traumatic acute SDH developed in a patient undergoing hemodialysis and report clinical characteristics and outcomes demonstrated during the process of care and treatment options we used with the aim of providing useful guidelines for treatment of patients in similar conditions.

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CASE REPORT

A 49-year-old man was admitted to the emergency room of our hospital with acute severe headache and vomiting. The patient had been undergoing hemodialysis three times a week for 10 years under the diagnosis of renal disease complicated by hypertension. He developed severe headache and vomiting during hemodialysis at another hospital and was taken to our hospital on the same day. On admission, the patient was responsive and alert with GSC score of 15 in neurological examination without abnormal findings. He just complained of a mild headache. CT scans received from the previous hospital re-

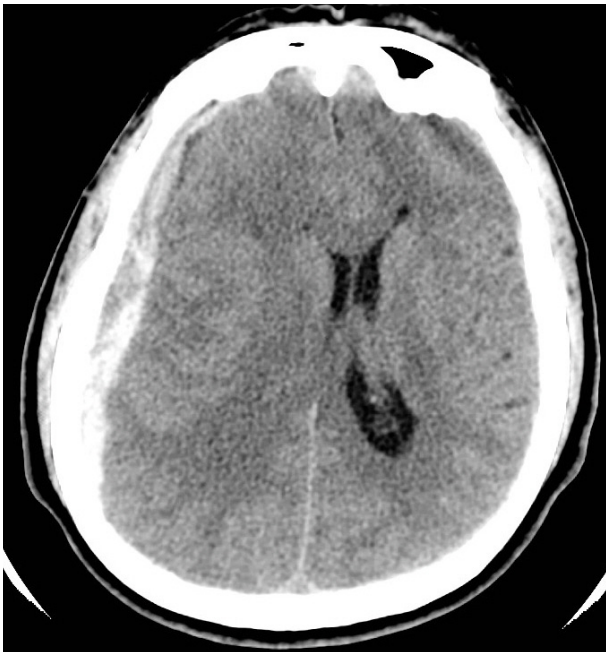

Fig. 1. Initial brain CT image showing acute subdural hemorrhage over the right cerebral convexity with around 11 mm thickness and 9 mm midline shift.

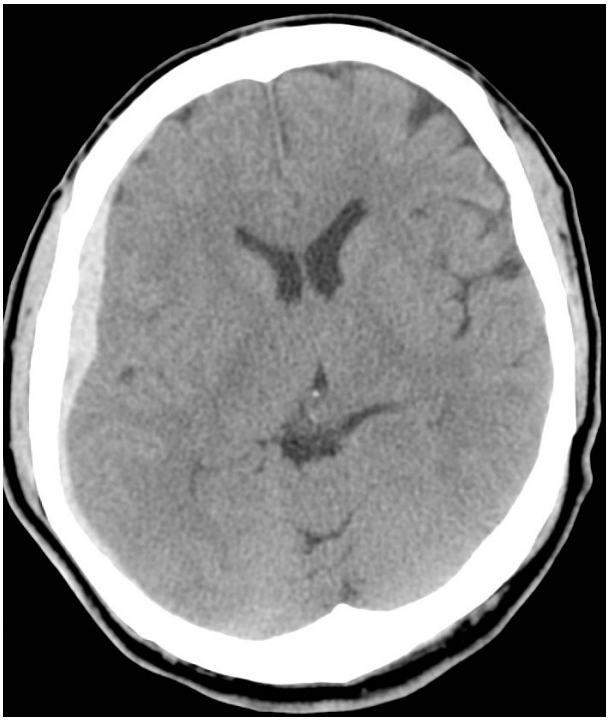

Fig. 2. Brain CT image at admission 3 days showing decreased amount of subdural hemorrhage and improved midline shift.

vealed a high-density acute SDH with a thickness of nearly 11 mm and a midline shift of nearly 9 mm in the right cerebral hemisphere (Fig. 1). In hematology test, BUN was 66.5 mg/dl, creatinine was 12.0 mg/dl, hemoglobin was 12.0 g/dl, platelet level was 198,000/μl, PT was 12.9 sec (INR 0.97) and aPTT was 36.9 sec. The patient complained only about mild headache despite the presence of a large acute SDH. His clear consciousness without neurological abnormalities, severe condition of end-stage renal disease and unidentified cause of hemorrhage in hematology test led us to perform conservative treatment after consulting with the patient and his family. We performed neurological examination every day on a regular basis, hemodialysis three times a week and administered antiepileptic drugs and topiramate (50 mg per day) and mannitol before hemodialysis. The patient's severe headache continued after admission, but CT scans revealed neither an increase in hematoma volume nor aggravating midline shift. On day 3 after admission, CT scan revealed a decrease in SDH volume and a significant improvement in the midline shift (Fig. 2), and we decided to continue conservative treatment. On day 9 after admission, the patient was undergoing hemodialysis but developed severe headache with fiercely fluctuating blood pressure, forcing us to discontinue hemodialysis and bring him back to the ward. On day 10, the patient still suffered from severe headache, global pain and frequent vomiting despite continued treatment. A repeat CT scan revealed a larger amount of low-density subacute SDH and slightly aggravated midline shift (Fig. 3A). We carried out trepanation and an emergency evacuation procedure under general anesthesia. After removing large amounts of liquid blood and small amounts of clotted blood, we still confirmed the existence of considerable amount of blood clots in the subdural space and inserted a catheter to suck the remaining hematoma. Once the patient recovered from anesthesia, he received an emergency hemodialysis without any problems. We therefore decided he could resume the routine hemodialysis three times a week. On day 2 after surgery, head CT scan revealed a small amount of blood clots but significant improvement in midline shift. We removed the catheter (Fig. 3B). While the patient remained under close observation in the general ward until he developed post-seizure despite antiepileptic drug therapy on day 9 after surgery. Head CT scan revealed a larger SDH, compared with postoperative residual hematoma, with a trivial postoperative midline shift. We decided to double the dose of antiepileptic drug (topiramate 100 mg/day) and to add mannitol to standard therapy as part of aggressive approach. And then we performed continuous renal replacement therapy (CRRT) after taking the patient to the intensive care units (ICU). Since there was residual SDH in the patient, heparin was not infused during CRRT, and this therapy was needed for 4 days. While CRRT was

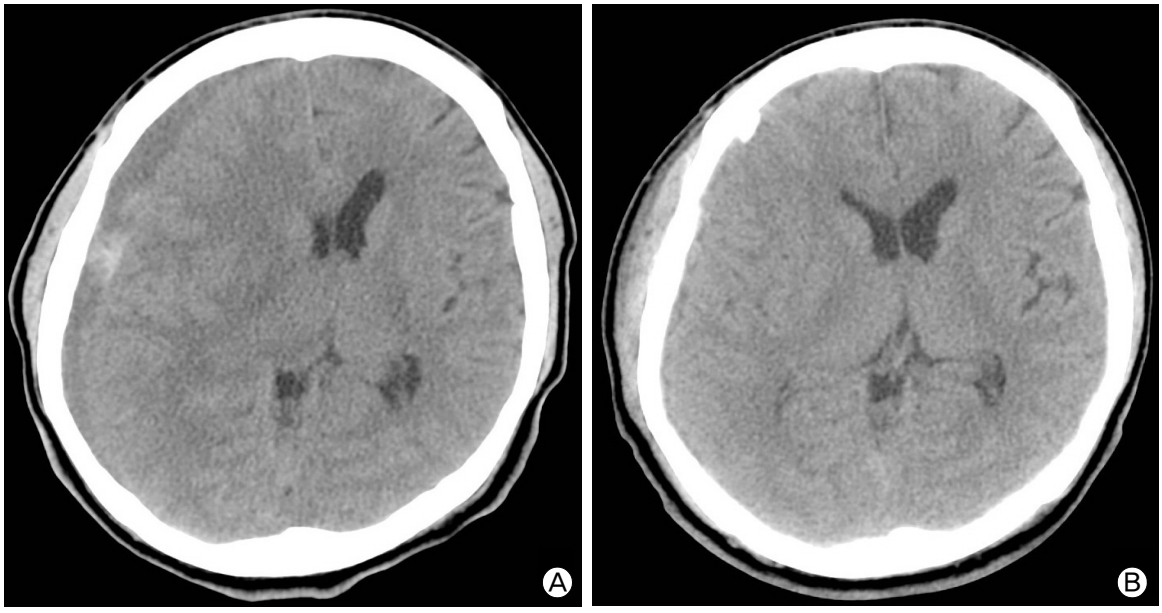

Fig. 3. (A) Preoperative brain CT image showing increased amount of subdural hemorrhage and midline shift. (B) Postoperative brain CT image showing markedly decreased amount of subdural hemorrhage and midline shift.

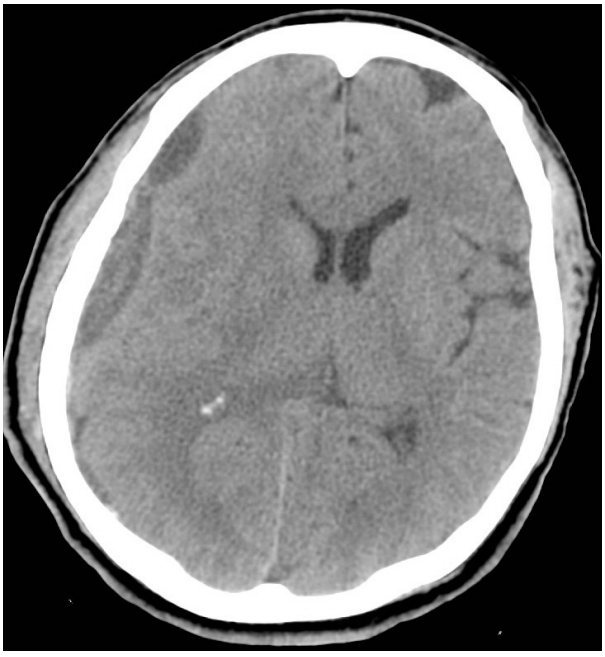

Fig. 4. Brain CT image showing only small amount of subdural hemorrhage.

being applied, liquid medications, including mannitol, could be infused on a regular basis, improving patient outcomes, including headache, and the patient did not develop post-seizure again during hospitalization. CT scan obtained on day 14 after surgery (Fig. 4) still revealed residual SDH causing no mass effect with a mild midline shift. On day 20 after surgery, the patient was discharged without specific neurological abnormalities and displayed no SDH on CT image until 1 year after discharge.

DISCUSSION

End-stage renal disease patients are vulnerable to bleeding because accompanying uremia impairs platelet function and alters interactions between platelets and endothelial cellsions, meaning that they have a much higher risk of cerebral hemorrhage, compared with normal people.[2-6] They particularly have a 10 to 20 times higher risk of developing non-traumatic acute SDH, compared with normal people.[4,6] Non-traumatic acute SDH is caused by chronic volume overload and impaired blood clotting function, meaning that chronic fluid overload in hemodialysis patients induce venous hypertension, causing the rupture of bridging vein that passes to dura mater. It is understood that this bleeding tendency can easily progress to SDH when platelet function is impaired by uremic toxins. In addition, heparin, which is commonly used to prevent blood clots in hemodialysis increases the risk of hemorrhage.

Bullock et al.[1] claimed that traumatic acute SDH require a surgical evacuation procedure if the thickness is greater than 10 mm, and the midline shift is greater than 5 mm on CT, regardless of GSC scores. However, no specific treatment guidelines are available for non-traumatic acute SDH. In particular, treatment methods for acute SDH developed in patients with severe systemic disease like end-stage renal failure have been rarely documented. Bronus and De Deyn[7] embraced a radiological

diagnosis of SDH developed in reneral disease patient in their review article investigating neurological sequela of SDH. They added those patients could be successfully treated with either peritoneum dialysis or hemodialysis without anticoagulantion although some patients require a neurosurgical procedure. Koo et al.[3] reported poor prognosis in 88% of end-stage renal disease patients who underwent surgery for spontaneous intracranial hemorrhage whereas good prognosis was observed in one episode. They attributed poor prognosis to massive hemorrhage resulted from chronic bleeding tendency, systemic vulnerability, accompanying medical conditions and dialysis-induced recurrent hemorrhage and high frequency of brain edema. Power et al.[6] reported 46% of total patients who developed acute SDH during hemodialysis died within 30 days, 58% of them died within one year. Most of patients died of brain damage directly caused by SDH and had a hematoma thickness of more than 20 mm and a midline shift of more than 10 mm. Sood et al.[4] also asserted SDH-induced brain damage was responsible for 40% of death in acute SDH patients within 30 days.

Feliciano and De Jesús[8] proposed conservative treatment for patients with traumatic acute SDH who have normal neurological status despite the presence of a large amount of hemorrhage (hematoma thickness > 10 mm), systemic disease and possible risk of anesthesia. Mathew et al.[9] reported the need of trapanation and evacuation for traumatic acute SDH patients who received conservative treatment treatment after their hematoma thickness of more than 10 mm was confirmed by initial CT. Of those patients, 6% to 26% developed chronic SDH, and they underwent evacuation at different time periods: within 11-20 days after injury and 3-7 months after injury. [10] In this article, the patient had a relatively large thickness of nearly 11 mm and a midline shift of nearly 9 mm on initial CT. However, his acute SDH was non-traumatic and neurological condition was clear and he suffered from renal failure. We therefore provided conservative treatment upon agreement of the patient and his family. We also contemplated evacuation and trepanation as a possible intervention because there was a high probability of developing subacute or chronic SDH in his conditions. Despite conservative treatment for 10 days after admission, the patient's severe headache did not subdue, and head CT revealed a larger subacute SDH, calling for an emergency surgery. The large amount of SDH, which is 11 mm thick on initial CT image, was somewhat reduced and the midline shift was improved on CT obtained on day 3 after admission. This finding is interpreted as hematoma expansion in which hematoma spreads through the surface of the right cerebral hemisphere, in patients with diffuse brain atrophy rather than a decrease in amounts of SDH. Diffuse brain atrophy was confirmed by CT obtained after discharge in this case. Our view on hematoma expansion was previously discussed in our previous study, and we also suggested the hematoma expansion raises the possibility of further progress to subacute or chronic SDH in the same study.[11] In this article, the patient underwent hemodialysis one day before surgery and had fiercely fluctuating blood pressure and intense headache during hemodialysis. This could be explained as compound effects of new brain edema formation during hemodialysis and aggravation of brain edema, which appeared to be induced previously by hemodialysis during the progression of SDH. However, we were not prudent in conducting hemodialysis for patients with intracranial lesions despite the potential risk of intracranial pressure that may result from increased fluid overload during hemodialysis, conducting hemodialysis for the patient three times after surgery. Fortunately, the patient reported a mild headache after surgery and recovered enough to walk. All of sudden, the patient developed post-seizure on day 9 after surgery. Head CT scan revealed a larger SDH, compared with postoperative amounts of hematoma and aggravated midline shift of around 6 mm. Even though the overall progression of SDH was not significant, intracranial hypertenion appeared to continue. We therefore concluded that the current hemodialysis program scheduled three times a week and the administration of mannitol for each hemodialysis would not be effective in managing overacting intracranial pressure and chose an alternative option, CRRT. CRRT is understood as the first option for acute renal disorder accompanied by hemodynamic instability, brain edema and extreme volume overload.[12] Its role in removing fluid, urea, creatinine and electrolyte steadily and gradually and maintaining cerebral perfusion pressure is coupled with its function in preventing intracranial hypertension, making it the procedure of choice for renal diseases.[13] However, the use of CRRT in end-stage renal disease patients with intracranial hypertenion has been rarely reported. Fletcher et al.[14] succeeded in stabilizing intracranial pressure in patients with intracranial hypertenion unresponsive to therapy using CRRT but did not experience a decrease in intracranial pressure. In this article, the patient was taken to the ICU from the general ward to perform CRRT using the jugular vein. Heparin was not used during CRRT. CRRT made it possible to infuse mannitol four times a day, which was more aggressive than the previous dose of intravenously injected mannitol once a day for hemodialysis. Antiepileptic drugs could be also used with no dose limitations. As a result of intensive therapy for four days, patient outcome improved without specific

neurological sequela and the patient was discharged.

We conclude that conservative treatment can be an effective management practice for a large acute SDH with thickness ≤ 10 mm and midline shift ≤ 5 mm in end-stage renal disease patients. It is however important to keep the patient and family informed of a possible need for a surgical intervention in the event of emergency including an increase of hemorrhage and aggravation of patient outcome. This article emphasizes the effective role of CRRT in the management of intracranial pressure because CRRT allowed aggressive therapy based on sufficient and steady administration of mannitol. Short-term CRRT can be a valuable option in addition to surgical treatment, depending on patient condition, when treating acute SDH developed in chronic renal disease patients. When proper treatment method is used on a timely manner, better prognosis can be expected.

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