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## RESEARCH ARTICLE

### SPONTANEOUS NON TRAUMATIC SUBARACHNOID HEMORRHAGE IN A HEMODIALYSIS PATIENT

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#### ABSTRACT

Non –traumatic subarachnoid hemorrhage (SAH) is an uncommon occurrence in hemodialysis patients and has been infrequently reported in medical literature so far. We report a case of spontaneous, non-aneurysmal SAH in a young maintenance hemodialysis patient with a poor outcome. The dialysis prescription following bleeding needs to be modified in these patients in order to prevent further worsening of cerebral oedema and progression of hemorrhage. Switch over to peritoneal dialysis or continuous forms of renal replacement therapies with regional anti-coagulation is the best option for such patients till neurologic stabilization is achieved.

## INTRODUCTION

Non traumatic SAH is usually attributable to rupture of an intracranial aneurysm and attributable to rupture of an intracranial aneurysm and accounts for 3 % of all strokes in the United States (Lloyd-Jones, 2010). The mortality associated with SAH may have decreased during the past few decades, but still stands at >25% (Qureshi, 2005). Patients with ESRD on maintenance hemodialysis have been shown to be a higher risk of stroke including SAH (Seliger *et al.*, 2003). Patients on maintenance hemodialysis are at 6-8 times higher risk for mortality compared to the general population (Mortality, 2011) and cardiovascular disease is the leading cause of mortality in patients of maintenance dialysis with stroke being one of the third most common cause of cardiovascular mortality (Bethesda, 2007). The overall incidence of stroke in dialysis population has been variously reported to be 17.2-33/1000 patient years with ischemic stroke being the most common type (Iseki, 2000). Polycystic kidney disease, a risk factor for ESRD is also associated with intracranial aneurysms.

However the occurrence of non traumatic SAH in the absence of cerebral aneurysms is rarely encountered in dialysis and seems to be underreported. We could find only one previous case report (Jayasurya, 2015) from India. We report a case of spontaneous non traumatic non aneurysmal SAH in a young patient on maintenance hemodialysis.

**Case Report:** A 19 years old female, previously diagnosed to have Type 1 diabetes mellitus since the age of 8 years with diabetic nephropathy, systemic hypertension and developmental mental retardation was diagnosed to have ESRD 4 years earlier and was on regular thrice a week hemodialysis. She had high blood sugar and blood pressure managed by antihypertensives and insulin with regular optimization. She had undergone an uneventful regular hemodialysis on 18/02/19 at our dialysis center and the dialysis session had been uneventful following which she had returned home in the evening. She presented to the emergency unit with sudden onset of breathlessness and reduced responsiveness to emergency department at 2am on 19/02/19.

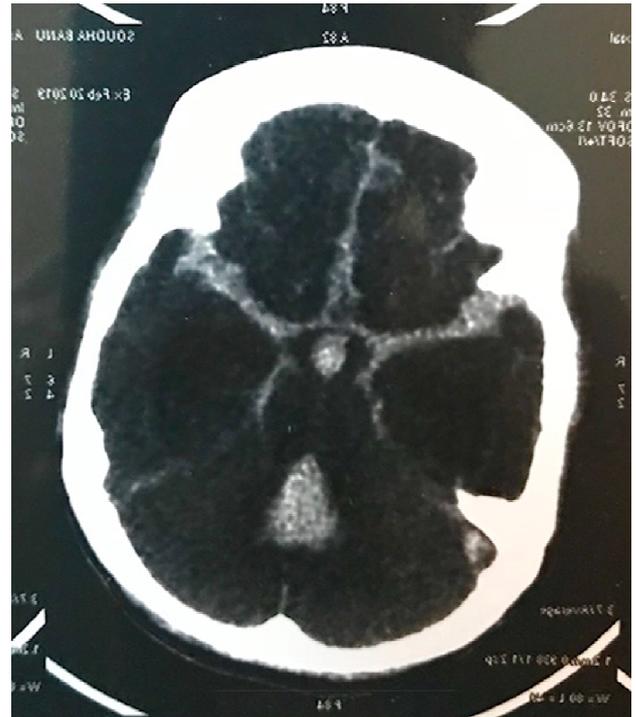
On examination her Glasgow coma scale was E2V2M3, blood pressure was 180/100 mmHg with a heart rate of 106 beats per minute. She was not maintaining saturation at room air, and hence she was intubated in emergency department and shifted to the ICU. Contrast enhanced CT (Fig 1) brain done revealed acute subarachnoid hemorrhage with intraventricular extension and diffuse cerebral edema. There was no history of fall or trauma to the head, no history of bleeding disorder or intracranial bleed in the past. CT angiography did not reveal any AVM or intracranial aneurysm. Patient was started on injection mannitol after neurosurgery consultation. Patient had fall in blood pressure during the course of hospitalization that was managed with nor adrenaline and vasopressin infusions. She was hemodynamically very unstable for dialysis and it was deferred and she was managed conservatively. Patient had sudden bradycardia and desaturation on 3<sup>rd</sup> hospital day and could not be resuscitated and died due to her illness.

## DISCUSSION

The hospitalization rate from stroke is increased 5-10 fold in dialysis population compared to general population (Seliger, 2003), a large majority are ischemic in nature with hemorrhagic strokes accounting for only 15% of strokes. Various factors such as hypertension, altered cerebral hemodynamics during hemodialysis, inherent bleeding diathesis associated with renal failure and heparin usage may increase the risk of hemorrhagic stroke. In general population, about 85% of all SAH is caused due to a rupture of saccular aneurysms at the base of the brain. Non aneurysmal perimesencephalic hemorrhage accounts for 10% of SAH and other rarer causes account for the remaining 5%. Risk factors for SAH include non modifiable genetic factors such as autosomal dominant polycystic kidney disease (ADPKD) and rare causes such as Ehlers-Danlos disease 4, neurofibromatosis Type 1, Marfan's syndrome etc and modifiable risk factors such as smoking, excess alcohol and hypertension. Very little is known about the pathogenesis as well as the optimum management of SAH in those on regular dialysis. A PubMed search also revealed only six relevant reports and only three of them have specifically addressed non traumatic SAH in dialysis population. There seems to be a paucity of information on SAH in dialysis, despite the fact that SAH is distinct pathogenetically from other intracranial bleed like intracranial hemorrhage (ICH) and subdural hematoma.

Sakhuja *et al.* (2014) reported in 2014 the incidence and outcomes of non traumatic SAH in patients on maintenance hemodialysis in the United States in a retrospective cohort study using the National inpatient sample database over a 6 year period. Out of 149,091 hospitalizations with non traumatic SAH, 1631 (10.9%) were patients on maintenance dialysis. The incidence of hospitalizations due to SAH among patients on maintenance hemodialysis (73.5/1, 00000) population was significantly higher than in the general population (11.2/1, 00000). Unadjusted all-cause mortality was higher in the dialysis population compared to general population (34.8% vs 21.9%) and being on maintenance hemodialysis was found to be an independent predictor of mortality following SAH. A Canadian study by Sood *et al.* (2014) reported the three year incidence of major non traumatic hemorrhage in elderly (>65 yrs) incident dialysis patients. The incidence of SAH was 0.1% (0.33/1000 person years) signifying that it is a relatively rare event. It has been reported that hemorrhagic strokes occurred more frequently

than ischemic strokes in patients of South Asian ethnicity compared to those of Caucasian origin. The management of intracerebral bleed and subarachnoid hemorrhage in Japanese dialysis patients over a 4 year period was reported by Murakami *et al.* (2004). 36 patients with ICH and 5 patients with SAH were seen and all patients except 2 were managed by continuous hemofiltration after admission. 2 patients with SAH received peritoneal dialysis and they were switched over to hemofiltration thrice weekly after repeat CT scan revealed no evidence of cerebral edema. A favorable outcome was reported in only two of the five patients with SAH. However unlike the poor reporting of prevalence of SAH in dialysis patients, the incidence of subdural hematoma (SDH) has been well studied and the incidence is reported to be 10 times higher than in the



**Figure 1. CT Brain showing subarachnoid hemorrhage with intraventricular extension**

general population (Sood, 2007). The probable causative factors include volume overload leading to venous hypertension along with uremia induced platelet dysfunction along with the intradialytic use of anticoagulants. Compared to the previous case report (Jayasurya, 2015) by Jayasurya *et al.*, our patient did not have a positive outcome and died following complications in the hospital. It is likely that anticoagulation was not primarily responsible for the hemorrhage, given that this had happened at home after more than 10 hrs after dialysis. Most previous cases of SAH have been reported in middle aged patients; ours is the first to be reported in a very young dialysis patient with no other predisposing factors. Poorly controlled blood pressure control as well was not a causative factor for the SAH. It is necessary to investigate the cause of SAH in cases where the cause is not trauma related. CT angiography is the imaging modality of choice to detect arterio-venous malformations (AVM) and aneurysms that cause SAH.

**Management:** The management of SAH in non dialysis population includes general supportive measures including control of BP, electrolyte and fluid balance, prevention of deep venous thrombosis, prevention of delayed cerebral

ischemia with calcium channel blockers and adequate nutritional support. This is combined with definitive management of aneurysms with surgical clipping or coiling when necessary (Vangijn *et al.*, 2007). Case fatality rate in dialysis population is higher as reported by Sakhuja *et al.* (2014). The optimum management of SAH in dialysis patient is unclear and management is extrapolated from data from patients with intracerebral bleeds. Intermittent hemodialysis is associated with wide fluctuations in serum osmolality and BP and this may have adverse neurologic consequences. Peritoneal dialysis and continuous forms of renal replacement therapy may provide better cerebrovascular stability in patients with cerebral oedema (Davenport, 2008; Davenport, 2013). Such therapies produce less fluctuation in serum osmolality, cerebral perfusion pressure and intracranial pressure. Patients can be shifted over to conventional hemodialysis once they are achieve neurologic stabilization. The dialysis prescription needs to be altered to prevent rapid osmolar changes and hypotension by reducing dialysis time, increasing frequency, decreasing blood and dialysate flow rates and choosing small surface area dialysers. Since systemic anticoagulation should be avoided in hemorrhagic strokes, heparin free dialysis, use of regional anticoagulation with citrate or Nafomostat can be used where available. We could not find any precipitating event for SAH in our young patient. She was hemodynamically too unstable for dialysis despite being on high dose vasopressor support and did not survive her illness. Further research into the etiopathogenesis and management of SAH in dialysis population is therefore the need of the hour.

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