Case Report

Acute Kidney Injury in a Patient with Aneurysmatic Subarachnoid Haemorrhage: A Case Report

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Abstract

Aneurysmatic SAH (aSAH) is a life-threatening condition, with an incidence of 6–10 cases per 100,000 patients annually. It can present with an intense headache described as "the worst headache of your life", nausea and vomiting, epileptic seizures, and an altered level of consciousness. Sympathetic activation due to complex pathophysiological processes may lead to extracerebral organ injury, including cardiac pathology, neurogenic pulmonary oedema, and acute kidney injury (AKI). The incidence of AKI in aSAH patients ranges from 16.3% to 25%. SAH patients who develop AKI have a poorer outcome and higher mortality. Uremic encephalopathy is a cerebral dysfunction caused by acute kidney injury and the accumulation of uremic toxins, resulting in an impaired level of consciousness and uremic coma. Therefore, AKI may mimic or worsen patients' neurological status. The diagnosis of uremic encephalopathy or coma is established after neurological improvement following dialysis.

Keywords: acute kidney injury (AKI); aneurysmatic subarachnoid haemorrhage; uremic coma; renal replacement therapy

1 Introduction

Subarachnoid haemorrhage (SAH) may occur due to traumatic brain injury or the rupture of an intracranial aneurysm, causing bleeding in the subarachnoid space. Aneurysmatic SAH (aSAH) is a life-threatening condition, with an incidence of 6–10 cases per 100,000 patients annually (1,

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2), manifesting as an intense headache described as "the worst headache of your life", nausea and vomiting, epileptic seizures, and an altered level of consciousness. Treatment of aSAH is either by neurosurgical clipping of the aneurysm or endovascular treatment (1). Among the most severe complications of aSAH are early rebleeding, severe vasospasm followed by delayed cerebral ischemia, and formation of hydrocephalus or cerebral oedema. However, sympathetic activation due to complex pathophysiological processes may lead to extracerebral organ injury (3, 4) in the form of ECG changes, Takotsubo syndrome, NSTEMI and STEMI, or cardiac arrest, neurogenic pulmonary oedema, and acute kidney injury (AKI). Sympathetic activation of the renin-angiotensin-aldosterone system induces renal vasoconstriction and a decrease in renal blood flow and glomerular filtration (3). Kidney function may also be impaired due to lower heart minute volume or the use of vasopressors and other necessary nephrotoxic drugs.

Mechanisms of developing AKI in neurocritical care are related to advanced age (>70 years old), previous renal impairment due to arterial hypertension or diabetes mellitus, severity of SAH, presence of intraventricular haemorrhage, delayed cerebral ischemia, use of antibiotics and vasopressors, and electrolyte imbalance (5, 6). The iodine contrast commonly used in diagnostic procedures may cause nephropathy, especially in pre-existing renal impairment. Sepsis, which develops in 75% of brain injury patients according to some studies (6), may also be a risk factor for AKI.

2 Case Report

A female patient aged 71 was admitted to the hospital due to a rupture of the anterior communicating artery (ACoA) with SAH and blood in the ventricles. The patient also suffered from arterial hypertension and diabetes mellitus type II with renal complications in the form of chronic renal insufficiency (GF 30 ml/min/1.73 m^2).

Upon admission to the hospital, the patient was somnolent, assessed as 2/4/5 on the Glasgow Coma Scale (GCS), Hunt Hess grade 3, Fischer 4 and with isocoric pupils. Due to the worsening of her neurological condition, she was intubated prior to performing digital subtraction angiography (DSA) of the brain. Two wide-neck aneurysms were found on the ACoA. As the patient was not suitable for embolization, an urgent neurosurgical procedure was performed. By the time the patient came to the operating room, she was anisocoric with the left pupil dilated in comparison to the right. Somatosensory and motor-evoked potentials (EP) were measured; motor EP were not present from the beginning of surgery. After craniotomy, both aneurysms were successfully clipped and a Codman sensor for intracerebral pressure (ICP) measurement was placed, which showed normal ICP levels. Postoperatively, the patient was placed in an induced coma using sufentanil, midazolam, and rocuronium. The patient also received antibiotics (vancomycin and meropenem), as well as the antiepileptic levetiracetam.

A control native CT scan, CT angiography, and CT perfusion were performed according to our post-surgery protocol. Native CT scans were then repeated on several occasions during the patient's ICU stay, showing regression of SAH and intraventricular haemorrhage.

After sedation and relaxation drugs were discontinued, neurologic testing showed that the patient remained at a GCS of 1/1/1. EEG ruled out nonconvulsive status epilepticus, repeated EP were unchanged, and brain MRI was unchanged with a few small areas of ischemia in the right frontal lobe.

Laboratory parameters exhibited metabolic acidosis (pH ranging from 7.14 to 7.43), high glucose levels (10–18 mmol/L, treated with continuous insulin infusion), variable electrolyte values tending towards hypernatremia, and potassium levels within the normal range. Acute exacerbation of kidney injury presented with high urea and creatinine levels and high serum osmolarity (300–350 mOsmol/kg) (Figure 1), with maintained diuresis (2,000 to 2,300 mL daily), occasionally stimulated with bolus doses of furosemide. Antibiotics were given empirically and then according to culture and sensitivity results (vancomycin and meropenem, then cefepime, followed by ceftazidime/avibactam). A nephrologist was consulted due to kidney injury, with urea levels of 45 mmol/L, creatinine 220 µmol/L, and high serum osmolality of 350 mOsmol/kg; it was recommended to continue volume replacement with 5% glucose and 0.9% NaCl. At this point, dialysis was not advised. In the following days, urea levels increased to 62.5 mmol/L, creatinine was 163 µmol/L, and diuresis was maintained (130 mL/h). On the 26th day of the ICU stay, dialysis (CVVHD) was initiated to exclude uremic coma. After 48 hours of CVVHD, urea and creatinine levels were lower (urea was 17 mmol/L, creatinine 48 µmol/L, and serum osmolarity 313 mOsmol/kg). Further dialysis was not indicated. Despite the improvement in kidney function, there was no improvement in neurological status; the patient reached a maximum GCS of 2/1/1. With no need for further neurosurgical treatment, the patient was later transferred to a regional hospital for further care.

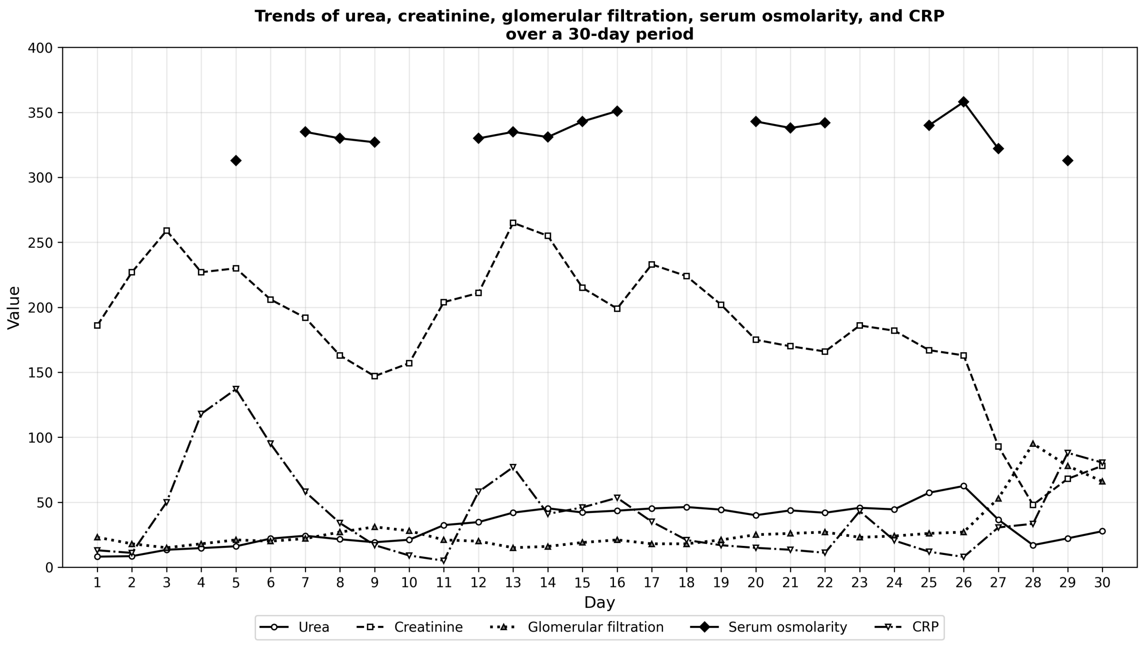

Figure 1. Trends of urea, creatinine, glomerular filtration, serum osmolarity, and CRP over a 30-day period.

3 Discussion

The incidence of AKI in aSAH patients varies among different studies from 16.3% to 25% (3, 5). SAH patients who develop AKI have poorer outcomes and higher mortality (7). Our patient's kidney function may have worsened due to a combination of multiple risk factors, such as sympathetic activation, intraventricular haemorrhage, contrast administration, pre-existing renal impairment due to diabetes mellitus, sepsis, and antibiotic nephrotoxicity. As a Codman ICP sensor had been placed, vancomycin was introduced from the first day after admission to the ICU according to our protocol, although in modified doses. Uremic encephalopathy is a cerebral dysfunction caused by acute kidney injury with a glomerular filtration under 15 mL/min (8), and the accumulation of uremic toxins, resulting in an impaired level of consciousness and uremic coma. Consequently, one should consider that AKI may mimic or worsen patients' neurological status.

According to studies (8, 9), uremic toxins such as urea, indoxyl sulfate, guanidine compounds, indolic acid, phenols, and carnitine may act as neurotoxins, causing astrocyte activation and neuronal death, cerebral endothelial dysfunction, and inflammation. There are no specific laboratory tests to diagnose uremic encephalopathy, nor are there specific radiological methods (8). EEG is also an inadequate diagnostic method, as the loss of alpha waves and bursts of delta and theta waves are not specific to uremic encephalopathy (8). Electrolyte imbalance should also be excluded as the cause of encephalopathy (8). Therefore, the diagnosis of uremic encephalopathy is confirmed after neurological improvement that comes soon after dialysis (8). However, dialysis may have a detrimental influence on intracranial pressure, cerebral blood flow, and brain tissue oxygenation (10). Dialysis disequilibrium syndrome (DDS) is a life-threatening complication of dialysis that presents in the form of neurological impairment and potentially fatal cerebral oedema (10). Therefore, optimal timing for renal replacement therapy in aSAH patients can be challenging as the risks may outweigh any potential benefits. DDS may develop due to a sudden change in electrolyte levels and the development of acidosis in brain tissue upon the initiation of dialysis, causing increased brain osmolality (11, 12). Continuous HD is more suitable than intermittent HD for patients with SAH, as it is linked to better haemodynamic stability and a lower incidence of DDS (6, 10).

The treatment of neurosurgical patients after aSAH can be a challenging and lengthy process with an uncertain outcome. Therefore, a multidisciplinary approach may help improve outcomes.

Conflict of Interest

The authors declare no conflict of interest.

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