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Case Report

Status Epilepticus following a Laparoscopic Surgery in a Patient of Chronic Kidney Disease – A Case Report

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Abstract

Seizures may occur in close relation to surgical procedures or with the use of anesthetic agents in several situations. The causative factors include an interruption of treatment with antiepileptic drugs (AEDs) or inadequate blood concentrations resulting from impaired gastrointestinal absorption. In operative procedures not involving the brain, transient seizures can arise from metabolic derangements or drug neurotoxicity. Other causes include hypoxia, hypotension, and embolic infarction. Seizures occurring shortly after injection of moderate to large amounts of local anesthetic should raise the suspicion of inadvertent intravenous injection. Seizures may also indicate withdrawal from unsuspected chronic use of excessive amounts of alcohol, sedative medications, mood-stabilizing agents, or AEDs. Rarely, sleep deprivation and drugs like flumazenil may precipitate seizures. We discuss the management of status epilepticus following laparoscopic urology procedure in a patient of chronic kidney disease with a history of the previous craniotomy.

Key words: Chronic kidney disease, Laparoscopic urological procedure, Metabolic derangements, Previous craniotomy, Seizures

INTRODUCTION

Peri-operative seizures, though rare, are a recognized complication of anesthetic agents. If prolonged, they can cause brain damage due to hypoxia, apnea, prolonged post-operative mechanical ventilation, and delayed awakening from anesthesia. Management includes maintaining a patent airway with adequate ventilation and protecting the patient from injuries resulting from seizures.

Laparoscopic interventions, if prolonged, in compromised patients, might lead to an increase in the intra-abdominal pressure (IAP) secondary to pneumoperitoneum. This may be detrimental and lead to various metabolic derangements as a result of decreased renal blood flow (RBF).[1,2]

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CASE REPORT AND RESULTS

A 49-year-old, 46 kg, male patient was posted for laparoscopic right ureterolithotomy and a laparoscopic left nephrolithotomy. His previous history included a craniotomy for the posterior cerebellar bleed 2 years back. At that time, he was also detected to have uncontrolled hypertension. Ultrasonography had shown right ureteric calculus and left renal multiple calculi. He had a history of DJ stent placement thrice in the past. His serum creatinine had been around 4.5–5 mg/dl.

A thorough pre-operative evaluation was done and in consultation with a nephrologist, a heparin free dialysis was planned preoperatively. Post-dialysis, hemoglobin of 10 g/dl, urea 48 mg/dl, and creatinine 4.9 mg/dl were reported. The rest of the investigations, including the electrolytes, chest X-ray, and electrocardiograph was within normal limits.

An informed high-risk consent was taken in view of chronic kidney disease (CKD), prolonged laparoscopic surgery, possible hypothermia, and a need for post-operative hemodialysis.

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The patient was induced with propofol 60mg, fentanyl 80 mcg, and atracurium 40 mg with ETT 8 mm. Laparoscopic surgery for the right ureteric stone was undertaken and the patient was positioned with a wedge under right flank for 150 min. Then, the position was changed and a wedge was placed under the left flank for the left renal surgery for about 130 min. IAP was maintained to 10–12 mm Hg, with an end-tidal carbon dioxide ($ETCO_2$) 38–42 mm Hg. Hemodynamic parameters were maintained. Prolonged laparoscopy surgery prompted us to get an arterial blood gas (ABG) after about 180 min into the procedure.

Findings-ABG-pH 7.14, pCO_2 45 mmHg, pO_2 151 mmHg, HCO_3 16.5 mmol/l, Lac 3.2 mmol/l, and glucose 360 mg/dl.

Random blood sugar by glucometer – 326 mg/dl (though the patient was not a diabetic).

Insulin infusion was started. At this stage, the surgeon was informed about the need to cut the procedure short. ABG after further 60 min showed pH 7.124, pCO_2 46.4 mmHg, $P0_2$ 176 mmHg, HCO_3 17.5 mmol/l, Lac 6.4 mmol/l, and glucose 318 mg/dl. In view of metabolic acidosis, we planned to shift him to intensive care units (ICU) on the ventilator and also considered renal replacement therapy (RRT). The patient was turned supine. There were good respiratory efforts and he opened his eyes on verbal commands but seizure activity was observed. Injection midazolam 2 mg followed by injection Levacetam 500 mg was given. The event was documented and the attendants were informed about the same. ICU team took charge of this patient. As the patient was being shifted to ICU, seizure activity was observed again. Injection midazolam 2 mg was repeated. Thereafter, seizure activity increased for which injection thiopentone sodium 200 mg was given. Neurological consultation was sought and electroencephalography (EEG) monitoring was started, which showed continuous seizure activity. Propofol infusion was started (2–10 mg/kg/h, titrated to hemodynamics) and immediate hemodialysis was planned.

ABG after hemodialysis – pH 7.23, P $C0_2$ 44 mmHg, PO_2 193 mmHg, HCO_3 19.4 mmol/l, and lactate 2.2 mmol/l.

However, the patient was kept intubated and was sedated on midazolam-fentanyl infusion in view of continuous seizure activity. He was started on injection Levacetam 500 mg intravenous twice daily, injection phenytoin 100 mg intravenous 8 hourly, and injection carbamazepine 500 mg intravenous twice a day. The seizure activity was controlled over a period of 72 h.

Computed tomography head showed cerebrocerebellar atrophic changes. Gliotic changes were seen in vermis and right cerebellar hemisphere. Cerebrospinal fluid (CSF) was sent for microscopic examination and it showed CSF proteins of 31.9 mg/dl. The glucose of 78 mg dl – inconclusive. The patient was managed on multiple antiepileptic drugs (AEDs) as per clinical response and EEG findings. MRI brain showed chronic right cerebellar hemorrhage with surrounding gliosis, post-operative changes in the right cerebellar hemisphere, and overlying subdural hygroma.

The patient was tracheostomised in view of prolonged ventilator support and a total of five hemodialysis were done during his stay in neuro-ICU. Neurological status was regularly monitored, antiepileptics were gradually tapered, and he was weaned off on the 7th post-operative day and decannulated on the 16th day. The neurological status was intact and he was discharged on the 20th day on two oral antiepileptics (phenytoin and carbamazepine).

DISCUSSION

Post-operative seizures, though rare, are a recognized complication of anesthetics.[3] Patients at risk include those with epilepsy, poor pre-operative control, and those undergoing brain surgeries. Rarely, seizures can occur as an isolated event in previously normal patient and this should prompt search for structural or chemical brain abnormality. Vigil needs to be exercised during post-operative period as numerous risk factors, can be potential cause of seizures.[1- 3] These include:

1. Pre-existing seizure disorder
2. Lengthy surgery
3. Pre-existing brain tumor, aneurysm, or scarring
4. Alcoholism and its withdrawal
5. Illicit drug use
6. Local anesthetic systemic toxicity
7. Drug interactions
8. Chemical and electrolyte abnormality.

In the event of persistent seizures, intravenous benzodiazepines should be used (lorazepam or diazepam). If the seizures persist, a second dose of benzodiazepine with phenytoin (20 mg/kg over 30 min) may be used. For refractory seizures, phenobarbital (1.5 mg/kg/min or 100 mg/70 kg/min or a max of 20 mg/kg/min), midazolam (0.1–0.3 mg/kg in 3–5 min followed by an infusion of 0.05–0.4 mg/kg/h), thiopental (5–10 mg/ kg in 10 min followed by infusion of 100–400 mg/h), lidocaine (1.5–2 mg/kg in 2–5 min followed by an infusion of 2–3 mg/kg/h for 12 h), isoflurane (0.5–1.5%), and

ketamine (50–100 mg followed by 50–100 ml/h) can be used.

Due to pneumoperitoneum in laparoscopic surgeries, absorption of CO_2 by the peritoneum leads to increase in the IAP which is usually limited to 12–14 mm Hg. The induced high IAP and its consequences in modifications of organ perfusion and stimulation of major hormonal systems are associated with functional alterations of various organs.[4] Studies have shown the effect of elevated pneumoperitoneum on RBF. Changes in renal function also appear under laparoscopy but are difficult to assess as no reliable markers exist to monitor rapid changes in glomerular filtration rate in clinical practice. Furthermore, the head-up position should be avoided to prevent impeding the venous return further. The duration and intensity of pneumoperitoneum correlate with the risk of acute kidney injury.

Patients with CKD are often afflicted with neurological complications,[5] which include stroke, cognitive dysfunction, encephalopathy, peripheral, and autonomic neuropathies. The encephalopathy is due to the altered brain function induced by an agent or the condition known as posterior reversible encephalopathy syndrome, which can lead to altered mental status and motor disturbances.[6] The direct effects of uremia may be due to metabolites, guanidino compounds, fluid, and electrolyte disturbances. Iatrogenic causes include erythropoietin induced hypertension, polypharmacy, and transplant rejection.

The uremic toxins are likely to contribute to CNS injury either directly or indirectly; these toxins have been investigated for a possible role in direct neurotoxicity in the context of CKD.[7] Indirect effects of uremic milieu include their contribution to systemic inflammation, endothelial dysfunction, and atherosclerosis. Alterations in mental status can be accompanied by motor disturbances, including tremors, fasciculations, asterixis, disorientation, delirium, hallucinations, seizures, and coma.[8]

Fluid and electrolyte disturbances are frequent in patients with CKD and have adverse effects on CNS function. Dialysis disequilibrium syndrome results from rapid changes in urea and other osmolites during dialysis.[9] The osmotic gradient between the blood and brain causes cerebral edema, the presentation of which includes headache, tremor, disturbed consciousness, and convulsions.

In severe acute kidney injury, RRT is the only therapeutic option, which includes intermittent hemodialysis, continuous and intermittent hemofiltration, and hemodiafiltration. Early versus late initiation of RRT needs to be individualized, depending on the associated risk factors, use of clinical scoring system, biochemical markers, and response to drug therapy.[10]

In patients undergoing craniotomy, the risk of seizures is 6% during the 1st post-operative week and 17% over 5 years. Although anecdotal experience suggests that AEDs may stop perioperative seizures, neither valproic acid nor phenytoin given intraoperatively and during the post-operative period has been shown to prevent the development of epilepsy months to years later.[3]

CONCLUSION

Neurological complications are highly prevalent in CKD and are a major cause of morbidity and mortality. Acute encephalopathies may be caused by a variety of metabolic and pharmacological exposures; there may be rapid escalation to seizures or coma, superimposed on chronic conditions such as stroke, cognitive impairment, and dementia. Identification and risk stratification are crucial for the perioperative management of patients with CKD. To improve the clinical outcome, a thorough pre-operative assessment, renal function optimization, maintenance of hemodynamic stability, and avoidance of nephrotoxic drugs are desirable. Information regarding the need of RRT needs to be explained in advance.

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