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Case Report

Anaesthesia Section

Perioperative Management of Hypernatraemia: An Anaesthetic Perspective

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ABSTRACT

Preoperative hypernatraemia remains a rare yet formidable challenge in anaesthetic practice, especially in emergency surgical situations where time constraints often limit full preoperative optimisation. Severe hypernatraemia is associated with increased perioperative morbidity and the risk of significant neurological complications if corrected too rapidly. Present case is a unique case of a 68-year-old male with type 2 diabetes mellitus who presented for emergency hepatic jejunostomy due to suspected bowel obstruction. The patient had a one-month history of recurrent vomiting, jaundice, and generalised weakness. Preoperative investigations revealed marked hypernatraemia, with a serum sodium level of 158 mEq/L, raising serious concerns about intraoperative and postoperative management. Given the urgency of the procedure, gradual preoperative correction of sodium was initiated with hypotonic saline while proceeding to surgery. Anaesthetic management involved rapid sequence induction with close airway protection, maintenance with sevoflurane and remifentanil, and intraoperative haemodynamic monitoring. The patient experienced transient intraoperative hypotension, requiring norepinephrine support. A major focus of care was the controlled correction of hypernatraemia, which was achieved over 72 hours postoperatively, avoiding rapid osmotic shifts. The patient was extubated on postoperative day 2, transferred to the surgical ward on day 4, and discharged home on day 10 without neurological sequelae. This case underscores the importance of vigilant perioperative care, multidisciplinary coordination, and a tailored anaesthetic approach in managing patients with severe hypernatraemia. It also adds to the limited body of literature addressing anaesthetic considerations in such high-risk, time-sensitive cases, where both fluid and electrolyte management play a pivotal role in patient outcomes.

Keywords: Acute kidney injury, Blood pressure monitoring, Critical care management, Fluid therapy protocols, Intraoperative monitoring, Postoperative care

CASE REPORT

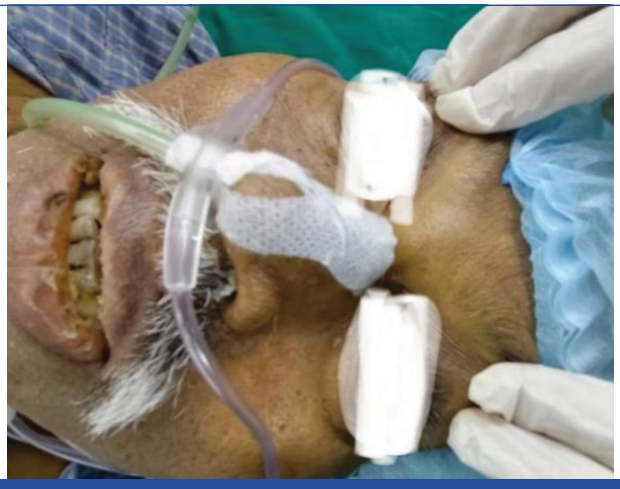
A 68-year-old male patient with an American Society of Anesthesiologists Physical Status classification of 3 was admitted for emergency triple bypass surgery (gastrojejunostomy, jejunojejunostomy, hepaticojejunostomy) due to bowel obstruction, most probably caused by malignancy. He was a known case of type 2 diabetes mellitus and had been on injection human actrapid on a sliding scale for the last year; however, his blood sugar was not well controlled, with an HbA1c of 7.7. He presented with yellowish discoloration of the sclera, generalised weakness, and recurrent vomiting for the past month.

The patient was conscious but not oriented, appearing toxic and tachypneic (22/min), requiring oxygen support at four liters with a saturation of 98%. He exhibited severe tachycardia (130 bpm) and had a blood pressure of 100/70 mmHg with low pulse volume. The patient was febrile (38° Celsius) with a leukocyte count of 15,000 cells/microliter. His laboratory parameters were depicted in [Table/Fig-1]. The patient's general appearance, with marked icterus, is shown in [Table/Fig-2].

On the electrocardiogram, the heart rate was 130 beats per minute, indicating sinus tachycardia. The chest X-ray suggested increased bronchovascular markings and perihilar opacity. A two-dimensional echocardiography showed an ejection fraction of 60%, mild concentric left ventricular hypertrophy, degenerative changes in the aortic and mitral valves, and Grade 1 diastolic dysfunction. Ultrasonography revealed hepatomegaly with Grade I fatty liver, an overdistended gallbladder with sludge, a mildly bulky pancreas, and prostatomegaly. The ultrasound findings were confirmed with a Computed Tomography (CT) scan and Magnetic Resonance Cholangiopancreatography (MRCP), which suggested periampullary carcinoma of the duodenal region with metastasis in the liver.

Parameter	Value	Normal range
Leukocyte count (/µL)	15,000	4000-11,000
Sodium (meq/L)	160	135-145
Blood urea (mg/dL)	59	15-40
Fasting blood sugar (mg/dL)	179	70-110
Hba1c (%)	7.7	<5.6
Total bilirubin (mg/dL)	14.03	0.2-1.2
Direct bilirubin (mg/dL)	12.57	0-0.3
Indirect bilirubin (mg/dL)	1.46	0.2-0.9
Total proteins (g/dL)	6.50	6.0-8.0
Serum albumin (g/dL)	3.0	3.5-5.0

[Table/Fig-1]: Laboratory parameters of the patient.



[Table/Fig-2]: Showing general appearance of the patient having Icterus++.

Preoperatively, the patient was started on hypotonic saline (0.45% NaCl) and 5% Dextrose (300 mL) with blood sugar level monitoring. Sodium correction targeted up to 10-12 mEq/L over 12 hours in the ICU, but sodium levels remained at 158 mEq/L.

IVC screening was done, revealing that the IVC was collapsible, so passive leg raising was performed, and 200 mL of 0.45% NaCl was administered to improve preload. An eighteen-gauge intravenous catheter was placed in the left upper limb, and anaesthesia was induced with propofol (1.5 mg/kg) and rocuronium (1.2 mg/kg) with rapid sequence intubation to minimise the risk of aspiration. Anaesthesia was maintained using a combination of sevoflurane (1-1.5 MAC) and O_2: Air. The patient was switched to volume control mode with a tidal volume of 6 mL/kg, using a PEEP of 5 cm H_2O and FiO_2 of 0.4. A left radial arterial line was secured for invasive haemodynamic monitoring and frequent blood sampling. The internal jugular vein was cannulated for measuring Central Venous Pressure (CVP), which helped guide fluid therapy and assess volume status.

Fluid management focused on gradually correcting the patient's hypernatraemia while maintaining adequate intravascular volume. This involved continuing the hypotonic saline (0.45% NaCl and 5% Dextrose) and replacing ongoing losses with balanced crystalloid solutions. The correction of serum sodium was approached gradually, targeting a rate of 0.5 mEq/L/hr to prevent neurological complications associated with rapid changes in serum osmolality. Hourly arterial blood gas analysis and electrolyte monitoring were performed to assess acid-base status and electrolyte levels, which were crucial for managing the hypernatraemia. Urine output was maintained at >0.5 mL/kg/hr to ensure adequate renal perfusion. Throughout the procedure, adequate tissue perfusion was maintained using sufficient fluid and vasopressors. Hypotension was managed with norepinephrine infusion (0.05-0.1 mcg/kg/min) and vasopressin (2 mcg/hr), while atropine was used to correct bradycardia. The central venous pressure was measured at 9 cm H_2O.

The patient was shifted intubated to the Intensive Care Unit (ICU) for fluid and electrolyte management with gradual correction of hypernatraemia and pain management. The patient's serum sodium levels normalised over the next 72 hours. His mental status improved, and he was successfully extubated on postoperative day 2. He was transferred to the surgical ward on postoperative day 4 and discharged home on day 10 with no neurological sequelae.

DISCUSSION

Hypernatraemia, defined as a serum sodium concentration exceeding 145 mEq/L, is a common electrolyte disorder that poses significant challenges in perioperative management. The prevalence of hypernatraemia among hospitalised patients has been reported to be between 1% and 4% [1]. Preoperative hypernatraemia, when severe (>155 mg/dL), is associated with increased morbidity and mortality and presents unique anaesthetic considerations [2]. It affects multiple physiological systems, including neurological function, cardiovascular stability, and fluid homeostasis—all of which are critical concerns during anaesthesia and surgery [3]. The incidence of preoperative hypernatraemia in surgical patients ranges from 1.9-9.1% [4]. Risk factors include old age, chronic kidney disease, diabetes insipidus, and excessive fluid loss, often compounded by inadequate water intake [5]. In emergency surgical scenarios, such as bowel obstruction, the risk is further elevated due to fluid sequestration and vomiting [6].

The goal of management involves the identification of hypernatraemia and the correction of volume disturbances and hypertonicity. This case report describes the anaesthetic challenges in severe preoperative hypernatraemia (serum sodium 160 mEq/L) undergoing emergency triple bypass surgery. In this patient, rapid correction of hypernatraemia was not opted, as it can cause a rapid shift of water into the brain, leading to cerebral oedema and seizures.

Hypernatraemia is classified based on the patient's fluid status and sodium content into three types:

- Hypovolemic hypernatraemia (water deficit > sodium deficit): occurs due to extrarenal losses (diarrhoea, vomiting, significant burns).
- Hypervolemic hypernatraemia : occurs when there is a greater gain of sodium than water. Causes include the administration of hypertonic saline, sodium bicarbonate, accidental salt ingestion, and mineralocorticoid excess (such as in Cushing syndrome).
- Euvolemic hypernatraemia : is typically associated with extrarenal losses, such as increased insensible water loss (e.g., hyperventilation) or renal losses of free water [7].

Preoperative hypernatraemia presents unique anaesthetic considerations [8], including electrolyte imbalances and multiple system involvement like neurological dysfunction, cardiovascular instability, and fluid homeostasis, which increase the risk for anaesthesia and surgery [8]. In emergency surgical scenarios, such as bowel obstruction, the risk is further elevated due to fluid sequestration and vomiting [9].

Hypernatraemia is a hyperosmolar state resulting from a decrease in total body water relative to sodium levels. It typically occurs due to a reduced sense of thirst and limited water consumption, frequently worsened by conditions that increase fluid loss. The management aims to identify hypernatraemia and address both volume imbalances and elevated sodium concentrations.

Signs and symptoms of hypernatraemia are predominantly related to disturbances in the central nervous system due to brain cell shrinkage and are especially prominent when the increase in sodium concentration is large or occurs rapidly [10]. Symptoms can range from thirst, weakness, and increased neuromuscular excitability (such as hyperreflexia) to more severe manifestations like lethargy, confusion, seizures, or coma [1].

Acute hypernatraemia (developing within 48 hours) can lead to sudden shrinkage of brain cells, resulting in vascular complications such as rupture of blood vessels, cerebral bleeding, subarachnoid haemorrhage, and in some cases, death—especially observed in paediatric and neonatal patients. The level of consciousness is associated with the severity of hypernatraemia [11].

Patients with chronic hypernatraemia (lasting more than 48 hours or of unknown onset) are less likely to develop severe neurologic symptoms due to adaptive responses that generate osmolytes [12]. Chronic hypernatraemia can trigger adaptive changes, and if rehydration occurs too quickly (with a plasma sodium increase of ≥12 mmol/L or ≥0.5 mmol/L per hour), it may result in cerebral edema and seizures, particularly in infants [8].

The assessment of low volume status includes several clinical, haemodynamic, and biochemical indicators. Clinically, patients may exhibit increased thirst, a dry tongue, decreased skin turgor, and orthostatic hypotension. In severe cases, patients may show signs of anorexia, lethargy, restlessness, seizures, confusion, respiratory failure, weakness, or even coma.

From a haemodynamic perspective, findings typically demonstrate low Central Venous Pressure (CVP), arterial pressure variation, and elevated arterial pressure during passive leg raising (PLR). Biochemical indicators include elevated haematocrit and albumin levels, high serum uric acid, increased urine osmolarity, and low urine sodium levels. In patients with hypernatraemia, urinary sodium osmolality and serum sodium osmolality are always increased. Rapid correction of hypernatraemia can lead to swift water movement within the brain, resulting in cerebral oedema and seizures [1].

Diagnostic Steps for Hypernatraemia

1. Measure plasma protein and lipid levels.

2. Assess the extracellular volume status as hypovolemic, euvolemic, or hypervolemic based on the patient's history and physical examination findings.
3. Evaluate urine sodium levels: In cases of volume depletion, urine sodium excretion is typically low (<20 mmol/L). However, elevated urine sodium levels (>20 mmol/L) may be observed in conditions like osmotic diuresis, diuretic use, postobstructive nephropathy, or the recovery phase from acute tubular necrosis, despite the presence of hypovolemia.
4. Measure Urine Volume (UV) and urine osmolality.
5. Check for concomitant electrolyte disorders [5].

A positive fluid balance of 4.8 L is required to reduce serum sodium from 160 to 140 mEq/L, representing a decrease of 20 mEq. To achieve this, 5.1 L of free water should be administered over 48 hours at a rate of approximately 120 mL per hour. Additionally, insensible water loss of 30 mL per hour must be replaced, bringing the total infusion rate to 150 mL per hour for 40 hours. This fluid can be provided as 5% dextrose, 0.45% saline, or free water via nasogastric tube or orally. Caution must be exercised with large volumes of 5% dextrose due to the risk of hyperglycaemia, which may necessitate insulin administration to prevent glycosuria. If insulin is not provided, osmotic diuresis could worsen hypernatraemia. Initially, 5% dextrose should be administered at a rate of 3-5 mL/kg per hour, which should then be reduced to 1 mL/kg per hour once serum sodium levels return to normal [7].

Sodium and glucose levels should be monitored every 4-6 hours until sodium levels reach 146 mEq/L. It is important to identify specific causes of hypernatraemia, as management differs according to the underlying cause. The use of hypotonic saline allows for a gradual correction of the sodium imbalance, which is essential to avoid rapid fluid shifts. The rate of correction should be calculated based on the patient's current sodium levels and body weight, aiming for a decrease of no more than 8-10 mEq/L in 24 hours. This gradual approach helps prevent osmotic demyelination syndrome.

Frequent monitoring of serum electrolytes, including sodium levels, is critical during the initial management phase. This helps to adjust fluid management, ensuring the correct rate of sodium supplementation and addressing any other electrolyte imbalances, such as hypokalaemia and hypocalcaemia, which can be common in patients with liver disease. In the context of liver disease and potential hepatorenal syndrome, close monitoring of urine output can aid in the early detection of renal dysfunction [7,8].

Anaesthetising a patient with severe hypernatraemia, particularly in cases of liver cirrhosis and portal hypertension, is a challenging task. The anaesthetic management of patients with severe hypernatraemia requires a delicate balance between correcting the electrolyte abnormality and maintaining haemodynamic stability.

Rapid correction of hypernatraemia can lead to cerebral oedema and potentially fatal neurological complications, while inadequate correction may result in continued cellular dehydration and organ dysfunction [10]. Rapid sequence induction is essential to minimise the risk of aspiration. Careful titration of anaesthetic agents is crucial due to the altered pharmacokinetics and pharmacodynamics in patients with liver disease, and dosages may need to be reduced.

Invasive haemodynamic monitoring is often necessary, including arterial line and CVP monitoring. In some cases, more advanced monitoring, such as transoesophageal echocardiography or pulmonary artery catheterisation, might be indicated depending on the patient's cardiovascular status and the nature of the surgery. This multimodal approach to managing haemodynamic instability demonstrates the complexity of anaesthetic management in critically ill patients undergoing emergency surgery [10].

CONCLUSION(S)

This case highlights the importance of meticulous perioperative management in patients with preoperative hypernatraemia. Key factors in achieving a successful outcome include gradual correction of the electrolyte imbalance, careful titration of anaesthetic agents, and vigilant monitoring. This report contributes to the limited literature on the anaesthetic management of severe preoperative hypernatraemia in emergency surgical settings. Future research could focus on developing standardised protocols for managing such cases and investigating the long-term outcomes of patients with preoperative electrolyte imbalances.

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