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# A case report on acute severe hyponatraemia following parathyroid surgery for primary hyperparathyroidism—A rare but life threatening complication

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## ABSTRACT

**INTRODUCTION:** Parathyroidectomy is a common operation, which is well tolerated and associated with low morbidity. Patients are usually discharged within 24 hours of surgery. Severe postoperative hyponatraemia is a rare complication which can cause significant morbidity including seizure, coma, respiratory arrest and even death.

**PRESENTATION OF CASE:** We present two patients with clinically significant hyponatremia resulting in seizures and collapse within 24 hours after parathyroidectomy, an unreported complication following surgery for primary hyperparathyroidism. One patient required support on the High Dependency Unit and both were treated with fluid restriction which resulted in correction of their electrolyte balance.

**DISCUSSION:** We believe this was caused by the relative inability to secrete a water load after surgery and non-psychogenic polydipsia. Preoperatively, neither patient was prescribed any routine medications nor did they have any risk factors for hyponatremia. Both had normal preoperative sodium levels.

It is usual practice is to advise patients to increase oral water intake when they are hypercalcaemic. The aim of parathyroidectomy is to treat hypercalcaemia by stopping excess PTH secretion from abnormal parathyroid glands. These patients continued to follow this advice after surgery when they were eucalcaemic after their operation and because they were thirsty. The patients drank several litres of water in 12–24 hours after surgery. We believe that this may have contributed to this complication.

**CONCLUSION:** Healthcare professionals need to be aware of this complication and patients should be advised to restrict intake of free water after surgery.

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## 1. Introduction

Parathyroidectomy is a common operation performed in patients with primary hyperparathyroidism (PHPT); the aim is to achieve effective control of high calcium levels. It is associated with minimal morbidity when carried out by a competent and experienced surgeon [1]. Potential complications include failure to cure hypercalcaemia, hypocalcaemia, injury to recurrent laryngeal nerve, wound infection, bleeding and hypertrophic scarring. Seizures due to hypocalcaemia are exceedingly rare [2,3].

Hyponatremia is an unusual event after a 'low risk' operation in healthy patients. A combination of factors can precipitate hyponatremia [4] in postoperative patients. Predisposing factors for developing hospital-acquired hyponatraemia include old age, diabetes mellitus, chronic kidney disease, pulmonary infection, diuretic therapy, administration of antibiotics and opioid analgesia and the use of hypotonic intravenous solutions [4]. Although gender is not a predisposing factor, menstruating women have been reported to have significantly higher morbidity and mortality from hyponatraemia when compared to men or post-menopausal women [5]. This can occasionally be severe enough to cause serious neurological symptoms [5].

We observed clinically significant hyponatraemia after surgery for primary hyperparathyroidism in two patients. An unusual combination of events led to a rare, but serious complication. A PubMed search did not find any reported cases describing this phenomenon.

Abbreviations: ADH, anti-diuretic hormone; PTH, parathyroid hormone.

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## 2. Presentation of cases

The first patient was a 57-year-old Caucasian woman working in the Mediterranean region who was detected to have incidental hypercalcaemia while being investigated for facial pain. Further tests confirmed the diagnosis of primary hyperparathyroidism (PHPT). Her past medical history included previously treated hypothyroidism. The facial pain was attributed to trigeminal neuralgia treated with gabapentin and amitriptyline. Parathyroid localisation (neck ultrasound and sestamibi scan) was inconclusive. At the time of surgery, she was not on any routine medications.

A bilateral neck exploration and excision of three parathyroid glands was performed. The surgery was straightforward and the patient was transferred to the ward following a short, uneventful stay in recovery. Approximately 10 h later, she was feeling unwell and shaky, a serum calcium level was requested. The patient was witnessed to have generalized tonic-clonic seizures and was unresponsive for a few minutes. The arrest team started intravenous calcium gluconate followed by intravenous fluids. Arterial blood gases and glucose levels were normal but serum sodium was significantly low at 118 mEq/L (cf a preoperative level of 140 mEq/L) and corrected calcium level was 1.9 mmol/L (cf a preoperative level of 2.75 mmol/L). Electrocardiogram was normal. After regaining consciousness, she demonstrated expressive dysphasia, neck stiffness and nystagmus. A CT scan of the brain showed no evidence of acute infarction, focal lesions or mass effects. Following detection of hyponatremia, fluid restriction was implemented and the patient was transferred to the high dependency unit for close observation. The patient became awake and responsive within 1–2 h.

There was no recurrence of seizures and she was returned to the ward the next day. Her fluid intake was restricted to 1500 mL per day. The sodium level rose slowly and reached 139 mEq/L 48 h later. Her adjusted calcium level was 2.25 mmol/L. She was discharged 3 days after surgery.

The second patient was a 64-year-old Caucasian female referred for an incidental finding of hypercalcaemia with an associated raised PTH level. The referral letter by the General Practitioner makes reference to “strict instructions regarding staying hydrated” given to the patient. Biochemical investigations confirmed a diagnosis of PHPT with significant hypercalcaemia ( $\text{Ca}^{2+}$  3.10 mmol/L). Localisation studies using ultrasound and sestamibi scans found an abnormal left inferior parathyroid gland. She was referred for surgery and advised to continue good hydration.

The patient underwent successful targeted parathyroidectomy with intra-operative PTH measurements. The operation was uneventful and the patient was admitted for a routine overnight stay. The patient was reviewed the following morning and found to be well with a normal adjusted calcium level (2.47 mmol/L). Later that day she attended the ENT clinic for a routine postoperative laryngoscopy. The patient felt unwell and nauseated and the examination was deferred. Whilst waiting to return to the ward, the patient collapsed. She was found to be unresponsive. The arrest team resuscitated the patient quickly with oxygen delivered via a bag-valve mask. Arterial bloods gases revealed significantly low sodium of 121 mEq/L (cf a preoperative level of 133 mEq/L). An electrocardiogram did not show any acute changes. Formal laboratory blood tests repeated on the ward confirmed a hyponatraemia of 124 mEq/L and fluid restriction of 1.5 L was implemented. Serum sodium had recovered to 135 mEq/L the following morning and the patient was discharged home.

Both patients recovered completely without any neurological sequelae.

## 3. Discussion

Postoperative seizures or collapse are attributed to a variety of causes including metabolic and electrolyte disturbances, acid-base disturbance and hypoxic brain injuries. Cardiac dysrhythmias, head trauma, cerebrovascular accidents, drugs, alcohol withdrawal and infections can also precipitate seizures.

In the first patient, hypocalcaemia was initially suspected as the cause of seizures. However, mild hypocalcaemia often seen after parathyroid surgery is not severe enough to cause seizures. More significant hypocalcaemia can cause increased neuromuscular excitability manifested as perioral numbness, cardiac arrhythmias, laryngeal spasm and tetany [6].

Biochemical results in these patients showed an unexpected hyponatraemia, the most probable cause of seizures and loss of consciousness. Hyponatremia may be classified into three categories relating to total body sodium and extracellular fluid volume: hypervolemic, hypovolemic and euvolemic hyponatremia [4]. Postoperative hyponatremia can result in serious and devastating neurological effects [7] or prolong hospital stay and patient recovery [8].

Postoperative hyponatremia may result from pharmacological agents, syndrome of inappropriate ADH secretion (SIADH), over infusion of hypotonic fluids, altered renal function and hypothyroidism [4,9]. Although 2–10% of patients develop hyponatremia [4], neurological sequelae does not occur in the majority of cases. In severe cases patients develop cerebral oedema and tentorial herniation manifesting as altered mental status and seizures [4].

Of the above causes, ADH secretion after surgery is most likely. Approximately one-third of all cases of hyponatremia is attributed to SIADH [10]. However, the incidence is only an estimate as most studies are retrospective where classical diagnostic criteria have not been applied accurately [11]. The elevation of ADH levels can last for 2–5 days after major surgery [12]. Seizures are associated with severe cases of hyponatraemia or rapid decline in serum sodium [4]. However, they are more likely to occur in young premenopausal females, as female sex hormones inhibit Na/K adenosine triphosphate activity in the brain [5].

The two patients consumed several litres of water after surgery. Non-psychogenic polydipsia is a rare cause of hyponatremia [4]. Non-psychogenic polydipsia may occur in healthy patients due to excessive iatrogenic hypotonic fluid infusion or over ingestion of free water. Documented cases of voluntary water intoxication include patients undergoing vigorous exercise, drinking alcohol, or instructed to “drink freely” as preparation for pelvic ultrasound or uroflowmetry [13,14]. Symptomatic hyponatraemia due to non-psychogenic polydipsia is uncommon as renal capacity for water excretion normally accommodates 28 L per day [15]. It is not well understood why some present with symptomatic hyponatremia. It is believed other contributing factors restricting water excretion could possibly add to the severity of hyponatremia [16]. Hobson and English suggested that water intoxication is a result of defective hypothalamic control on ADH secretion [17]. Hariprasad et al. reported peculiar results in some hyponatraemic patients who had fluid deprivation tests. The urine became concentrated but the serum was hypo-osmolar. The mechanism for this is unclear [18]. Another study highlighted the possible role of secondary SIADH in the development of severe symptomatic hyponatremia as brain oedema induced by hyponatremia can stimulate ADH release [19].

Manifestations of water intoxication were described by Rowentree in 1923. The early symptoms are dizziness, malaise, blurred vision, headache, nausea and vomiting. Signs are seizures, psychosis, cramps, anhydrosis and hyperpyrexia. Severe cases may be complicated by brain and lung oedema and end by coma and death [4].

These two cases highlight the occurrence of severe hyponatremia in an unusual setting. The first patient lived in a tropical region and was acclimatised to drinking 4–5L a day. She continued drinking at this rate in the immediate postoperative period resulting in hyponatremia which we believe was precipitated by excessive ADH secretion usually seen following surgery [9,20]. Although hypothyroidism can contribute to hyponatremia, her preoperative TSH level was 0.13 mIU/L. In the second patient, similar mechanisms appear to have been responsible. In addition, clinician advice to increase water intake was followed well into the postoperative period after the correction of hypocalcaemia.

#### 4. Conclusion

We believe that the severe hyponatraemia in these patients was due to an excess of free water ingestion and post-operative inability to excrete water by mechanisms such as ADH release as part of the stress response. As patients with primary hyperparathyroidism are often advised to increase fluid intake, it is important for both clinical and nursing staff to be aware of this complication. The risk of this complication can be reduced by limiting this advice to the preoperative period, careful fluid monitoring in the early postoperative period and encouraging the intake of isotonic fluids instead of water.

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N/A.

#### Consent

Written consent obtained from patients.

#### Author contribution

S. Hillary: data collection, writing the paper.  
H. Heamed: data collection.  
M. Berthoud: contributor.  
S. Balasubramanian: editing, contributor, supervisor.

#### Guarantor

S. Balasubramanian.

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