Nephrogenic diabetes insipidus (NDI) presents an uncommon but formidable clinical challenge in the surgical patient. Two recent cases of NDI with differing aetiology are presented. These cases and a review of the literature illustrate well the diagnosis, fluid and electrolyte management, and outcome of NDI in the surgical patient, with particular attention to the high dependency unit (HDU).

Nephrogenic diabetes insipidus presents a formidable clinical problem in the peri-operative period, if not recognised and treated actively. The inappropriate excretion of large volumes of dilute urine will place the patient at risk of dehydration and hypernatraemia, with increasing coma, organ hypoperfusion, and death. NDI is characterised by the inability of the kidneys to concentrate urine in response to vasopressin (ADH), as shown by 24-hour urine volumes of >3 litres and serum osmolality of <280 mOsmol/l. NDI is thought possible.

Diagnostic of NDI is reliant on careful peri-operative monitoring of fluid balance and electrolyte measurements as well as a high index of clinical suspicion. In the surgical patient, the commonly cited causes of NDI are those related to the surgical injury, i.e., trauma, sepsis, and intercurrent illness. The other causes include malignancy, infection, or metabolic derangements. The diagnosis of NDI is often delayed or missed due to its clinical presentation in the post-operative period.

The various causes of NDI are presented in Table 1 (cranial diabetes insipidus is a distinct entity and may be caused by cranial trauma, neoplasia or infections). Nephrogenic diabetes insipidus is characterised by resistance of the kidney to vasopressin, and vasopressin analogues have no role in the treatment of NDI. Congenital NDI is thought to be caused by mutations in the genes coding for AQP2 and all known acquired mechanisms of NDI are thought possible.

Lithium-induced NDI (LID) is a common cause of NDI which is often under-diagnosed. Lithium is a mood-stabilising agent used in the treatment of psychiatric disorders, particularly bipolar affective disorders. The mechanism of lithium-induced NDI is thought to be related to the accumulation of lithium in the renal collecting duct epithelial cell, which inhibits the movement of water and electrolytes.

In Case 1, a 47-year-old man was admitted to the HDU following emergency laparotomy for perforated sigmoid diverticulitis. He had a life-long history of severe schizophrenia with depression. His past medical history was notable only for schizophrenia with depression. He had been treated with lithium carbonate for many years and was readmitted to hospital with high uncontrolled serum lithium levels.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 310 mOsmol/l. A formal water deprivation test was performed to exclude cranial diabetes insipidus. There was no response to vasopressin, with urine volumes of 11 litres in 24 hours and serum and urine osmolarities of 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the first post-operative day urine output was 5.4 litres, and a serum sodium of 157.4 mmol/l and a chloride of 124.0 mmol/l was recorded. Over the next few days, urine volumes peaked at 11 litres and the serum sodium at 161.0 mmol/l, whilst serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was thought likely and whilst pre-operative lithium levels were within the therapeutic range, no other possible etiology was identified.

In Case 2, a 9-year-old woman was admitted to the HDU following an anterior section of a locally advanced (TN) rectal adenocarcinoma. She had a medical history of hypertension and was under treatment with the blood pressure medication. Her past medical history was notable only for hypertension and was under treatment with the blood pressure medication.

In the immediate post-operative period it was noted that the plasma sodium concentration was 159.8 mmol/l and a serum osmolarity of 320 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

Lithium-induced NDI is thought possible, as the hazards of lithium-related NDI, including the development of nephrogenic diabetes insipidus, are well described in the literature. Lithium is a commonly used mood-stabilising agent used in the treatment of psychiatric disorders, particularly bipolar affective disorders. The mechanisms of lithium-induced NDI are thought to be related to the accumulation of lithium in the renal collecting duct epithelial cell, which inhibits the movement of water and electrolytes.

In Case 2, the patient was given intravenous sodium chloride (0.9%) solution to correct the hypernatraemia. However, the patient continued to pass large volumes of urine, with polyuria and hypernatraemia persisting despite stopping lithium. As is well described in the literature, pharmacological measures have been described such as giving thiazide diuretics to correct the hypernatraemia. However, in this case, thiazide diuretics were ineffective, with large volumes of urine continuing to be passed in the next few days.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.

In the immediate post-operative period it was noted that the plasma sodium concentration was 156.9 mmol/l and a serum osmolarity of 330 mOsmol/l. Serum and urine osmolarities were 338 and 202 mOsmol/l, respectively. Nephrogenic diabetes insipidus secondary to the effects of lithium was diagnosed with polyuria and hypernatraemia.