# Severe hyperkalaemia in the setting of tumour lysis syndrome

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## DESCRIPTION

A 49-year-old man with a structurally normal heart presented to the emergency department with pyelonephritis secondary to extended spectrum beta lactamase *Escherichia coli*, resulting in both septic shock and an acute kidney injury. He had a background history of a renal transplant for adult polycystic kidney disease complicated by BK viraemia with a baseline creatinine of 141 $\mu$mol/L and metastatic ampullary adenocarcinoma requiring a Whipple's procedure with right-sided native nephrectomy. His regular medications were sodium polystyrene sulfonate, trimethoprim, tacrolimus, folic acid, pantoprazole and prednisolone. There had been long-standing difficulties with controlling hyperkalaemia but no previous episodes of a potassium level recorded >7.0 mmol/L. On admission, the patient had a blood pressure of 75/53 mm Hg and a heart rate of 130 beats/min. He was hypothermic with a temperature of 35.4°C. His initial laboratory findings demonstrated a creatinine level of 203 $\mu$mol/L, a potassium level of 5.7 mmol/L, a phosphate level of 1.32 mmol/L, a serum calcium level of 2.24 mmol/L and a lactate level of 2.4 mmol/L.

For resuscitation, the patient received 3 L of compound sodium lactate and intravenous meropenem. He also received a single intravenous dose of dexamethasone to cover for underlying adrenal insufficiency. He subsequently developed a broad complex tachycardia, caused by severe hyperkalaemia with a peak potassium level of 11.8 mmol/L. The ECG demonstrated a rate of 142 beats/min without evidence of P waves and with the QRS segments resembling a sine wave (figure 1A). Two boluses of 2.2 mmol/L calcium carbonate, eight units of intravenous actrarapid and 100 mL of 8.4% of sodium bicarbonate were administered. This treatment resulted in progressive resolution of his ECG changes, with a corresponding potassium level of 5.0 mmol/L (figure 1B,C).

The development of acute QRS widening and hypotension should prompt the consideration of life-threatening hyperkalaemia.[1 2] Owing to the loss of P waves and broadening of the QRS complex, a severe hyperkalaemia can mimic ventricular tachycardia. The underlying cause of hyperkalaemia-induced wide complex tachycardia is an increase in the resting membrane potential due to inactivation of sodium channels, resulting in prolonged membrane depolarisation and a shorter repolarisation time.[1 2] Therefore, treatment of hyperkalaemia-induced arrhythmias should be targeted at restoring the normal resting membrane gradient. Most commonly, either calcium gluconate or calcium chloride is used as these agents directly antagonise the effects of potassium on the cellular membrane.[3]

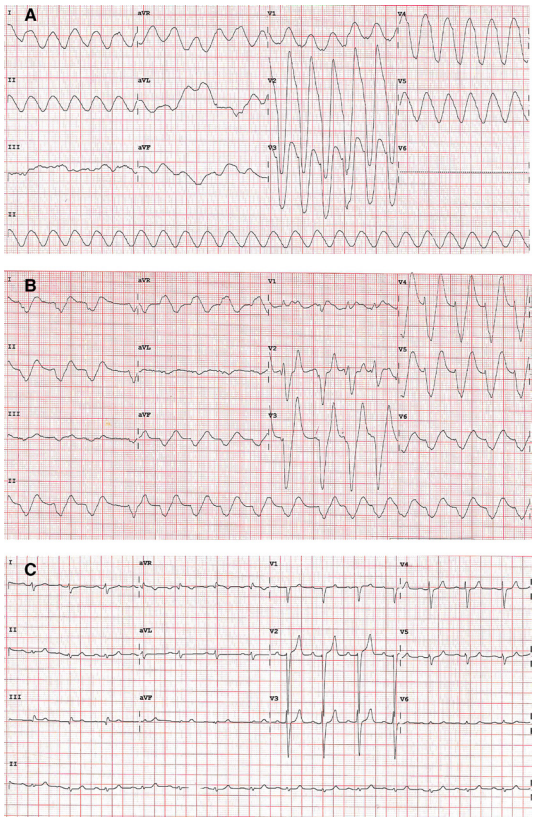


**Figure 1** Progression of severe hyperkalaemia with treatment. (A) Severe hyperkalaemia (K 11.8 mmol/L) with sine waves. (B) Broad complex QRS and peaked T waves. (C) Resolution of hyperkalaemia (K 5.0 mmol/L).

It is important to identify the cause of hyperkalaemia. The mechanism triggering the acute rise in potassium in this case is likely multifactorial and not entirely clear. In this case, our individual had a history of a kidney transplant with a single remaining kidney and was on both tacrolimus and trimethoprim therapy, which are known to cause hyperkalaemia. A final precipitating cause in this case was likely the administration of intravenous dexamethasone on admission to the emergency department, precipitating an episode of tumour lysis syndrome. Supporting this mechanism was the observation that 2 hours after the administration of dexamethasone, there was an increase in the serum phosphate level to 2.43 mmol/L and a fall in the serum calcium level to 1.86 mmol/L. Unfortunately, no lactate dehydrogenase level or uric acid levels were obtained during the initial resuscitation stage of management.



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Although tumour lysis syndrome is usually precipitated by the administration of chemotherapy, there are numerous reports of corticosteroid therapy triggering tumour lysis syndrome.[4 5] Although it is rarer for solid tumours such as pancreatic cancer to have tumour lysis syndrome, there are a number of risk factors for tumour lysis present in this case.[6 7] Renal insufficiency, a rapidly growing tumour with high tumour burden, the presence of active infection and use of other medications that increase potassium all can increase the risk of tumour lysis syndrome.[8]

## Learning points

- Consideration of hyperkalaemia as an important differential in the management of broad complex tachyarrhythmias.
- Identification of medical management that may precipitate the development of hyperkalaemia in certain patient populations, including those with malignancy and underlying kidney disease.

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