Low Molecular Weight Heparins Can Lead To Hyperkalaemia

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Citation

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Abstract

Low molecular weight heparins (LMWH) have an important role in the management of many clinical conditions, either prophylactically or therapeutically.1,2,3 They are known to cause side-effects such as haemorrhage, thrombocytopenia, hypersensitivity reactions, skin necrosis and osteoporosis.1 Hyperkalaemia is a recognised side-effect of heparin and LMWH use, but this may be easily overlooked in a clinician's day-to-day practise.1,2,3,4,5 A case of Clexane (enoxaparin) induced hyperkalaemia in an elderly patient on a surgical ward is described.

CASE REPORT

A 79-year-old man was admitted to a surgical ward in May 2004 for management of an abdominal viscus rupture as suspected from clinical examination and radiological investigations. He had a past medical history of hypertension treated with bendroflumethiazide. At this time, his urea was 20.4mmol/Litre (normal range 2.5–6.7mmol/L), creatinine 128mmol/L (normal 70–120mmol/L), sodium 138mmol/L (normal 135-145mmol/L) and potassium 4.2mmol/L (normal 3.5-5.0mmol/L). At laparotomy, a perforated caecum was found necessitating a right hemicolectomy and loop ileostomy. The rupture had followed caecal distension from a large bowel obstruction. Histology later returned non-neoplastic.

Postoperatively, he was commenced on Clexane 20mg daily and completed a five day course of intravenous cefuroxime and metronidazole. Fluid resuscitation intravenously in the peri-operative period did not include potassium supplementation. Five days post-operatively, his serum urea was 9.0mmol/L, creatinine 100mmol/L, sodium 130mmol/L and potassium 4.9mmol/L.

His recovery was slow and he was left on the prophylactic dose of clexane to reduce risks of venous thromboembolism.

Thirteen days post-operatively, his potassium had risen to 5.8mmol/L. The potassium remained elevated above 5.0mmol/L on serial evaluations without significant change to the other indices. The highest recorded potassium level of 6.0mmol/L was noted twenty days after surgery and he was given an intravenous insulin-dextrose infusion. After this treatment, the potassium level reduced to 5.5mmol/L but gradually rose to 5.8mmol/L three days later.

As the hyperkalaemia persisted without obvious cause, a medical review was requested and the possibility of clexane-induced hyperkalaemia was entertained. A review of his medication charts revealed that other than the clexane, he was not on medications that predispose to hyperkalaemia or renal dysfunction. He was not consuming potassium enriched food or drink. He appeared asymptomatic to the hyperkalaemia and a 12 lead ECG showed no features of hyperkalaemia. His calculated creatinine clearance was 39ml/min using the Cockcroft-Gault equation, representing a mild degree of renal impairment.[6]

The clexane was discontinued twenty eight days after surgery. Over the subsequent seven days and without further intervention, his potassium level normalised to 4.8mmol/L. His pre-discharge urea was 6.8mmol/L, creatinine 118mmol/L and sodium 135mmol/L.

DISCUSSION

Unfractionated and LMWH are commonly used in the management of patients in medical, surgical and rehabilitation settings.[2] Outpatient usage of LMWH is also not uncommon.[2] The heparins are thought to predispose to hyperkalaemia by inhibition of aldosterone secretion.[1,4] Aldosterone, is an adrenal hormone that functions at the renal level to cause sodium retention and potassium excretion.

The risk of heparin-induced hyperkalaemia is higher in patients with chronic renal failure, diabetes mellitus, acidosis, and those on potassium-sparing/retaining drugs. In the United Kingdom, the Committee on Safety of Medicines (CSM) has recommended that plasma potassium should be measured in at-risk patients, both before and after initiating heparin therapy. The advice is particularly important if patients are likely to continue the heparin for more than seven days as the risk appears to increase with the duration of administration.[1] However, hyperkalaemia can occur even when patients receive heparin for a shorter duration.[7]

This case illustrates the need to be aware of, and the importance of recognising hyperkalaemia as a potential side-effect of heparin and LMWH therapy. This is especially the case in at-risk patients, as hyperkalaemia could represent an additional co-morbid factor with a potential for serious and sometimes life-threatening complications.

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