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HEPARIN INDUCED HYPERKALEMIA IN A HOSPITALIZED PATIENT: A CASE REPORT OF RARE ASSOCIATION

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ABSTRACT
Heparin induced hyperkalemia is a rare casue of hyperkalemia especially in the hospitalized patients with co-morbidities such as diabetes, chronic kidney disease etc. Heparin has the potential to induce hyperkalemia due to decreased aldosterone synthesis. Here we report two cases of elderly patients developing hyperkalemia due to heaprin use. A 73 year old male was admitted to the hospital from ED with altered mental status and found to have Acute on Chronic kidney injury complicated by uremia. Labs on admission were Na+ 132 mEq/L, K+ 5.6 mEq/L, Creatinine 3.19 mg/dL. Past medical history was significant for CKD and Type 2 DM. Patient was started on subcutaneous heparin and the potassium rose to 6 mEq/L at the end of the day. Patient was started on Fludrocorticosterone and the potassium started drowntrending. Due to ischemic findings, patient was given heparin again which made the potassium rise again thus making it the clear cause of hyperkalemia in this patient. Although all patients who receive heparin may have reduced aldosterone levels, most are able to compensate through increased renin production and therefore remain asymptomatic. However, patients on prolonged heparin therapy or those unable to adequately increase renin production (e.g., patients with diabetes or renal insufficiency) may develop hyperkalemia. Fludrocotisone can be used as a potential treatment for the hospitalized patients who need to be on the heparin treatment.

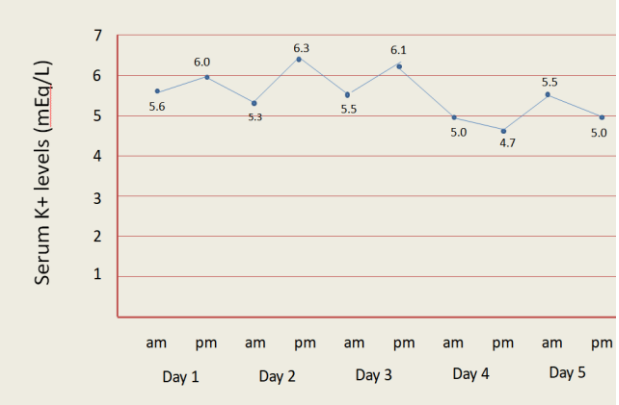
KEYWORDS: Fludrocorticosterone, drowntrending, Creatinine.

INTRODUCTION
Acute renal failure (ARF) can occur in about 5% of hospital admissions and up to 30% of admissions into the intensive care units.[1] Hyperkalemia is a potentially life-threatening condition in which serum potassium exceeds 5.5 mmol/l. It can be due to reduced renal excretion, excessive intake or leakage of potassium from the intracellular space. In addition to acute and chronic renal failure, hypoaldosteronism, and massive tissue breakdown as in rhabdomyolysis, are typical conditions leading to hyperkalemia. Symptoms are non-specific and predominantly related to muscular or cardiac dysfunction.[2] Heparin-induced hyperkalemia is less well-recognized than other side effects of heparin therapy.[3] Heparin has the potential to induce hyperkalemia by several mechanisms, including decreased aldosterone synthesis, reduction in number and affinity of aldosterone II receptors, and atrophy of the renal zona glomerulosa.[4]

CASE REPORT
A 73 year old male was admitted to the hospital from ED with altered mental status and found to have Acute on Chronic kidney injury complicated by uremia. Past medical history was significant for CKD stage 3 with baseline creatinine 1.3 mg/dL, CHF with reduced ejection fraction, Type 2 DM, CAD, Prostate cancer s/p prostatectomy and Gout. Past medication history is significant for Ramipril, Metoprolol, Indomethacin and Colchicine which were discontinued after he was admitted. Labs on admission were Na+ 132 mEq/L, K+ 5.6 mEq/L, Creatinine 3.19 mg/dL. Patient was started on IV Normal Saline, IV furosemide, PO Fludrocortisone, PO Sodium bicarbonate and Subcutaneous heparin with his evening K+ rising to 6 mEq/L. K+ came down trending to 5.3 mEq/L on day 2 in the morning but rose back to 6.3 mEq/L after he received another dose of subcutaneous Heparin.

On day 3, K+ levels were 5.5 mEq/L in the morning but later patient developed EKG changes which were

considered likely to be NSTEMI for which he was given IV heparin and K+ levels went back to 6.1 mEq/L in the evening. Later his EKG changes resolved and heparin was withheld completely due to hyperkalemia and his K+ levels trended down over the course of stay in the hospital with continuation of initial treatment of hyperkalemia.



DISCUSSION

Heparin-induced hypoaldosteronism leading to hyperkalemia is an uncommon adverse effect. It appears as though heparin blocks an enzymatic step in the synthesis of aldosterone, and reduced aldosterone levels may be evident as early as four days after initiation of therapy. Although all patients who receive heparin may have reduced aldosterone levels, most are able to compensate through increased renin production and therefore remain asymptomatic. However, patients on prolonged heparin therapy or those unable to adequately increase renin production (e.g., patients with diabetes or renal insufficiency) may exhibit signs of hypoaldosteronism, such as hyperkalemia.[5]

Aldosterone suppression results in natriuresis and less predictably in decreased excretion of potassium. Greater than normal serum potassium levels occur in about 7% of patients, but marked hyperkalemia generally requires the presence of additional factors perturbing potassium balance (in particular, renal insufficiency, diabetes mellitus, or the use of certain medications). Heparin-induced increases in serum potassium need to be better anticipated by clinicians. Serum potassium levels should be monitored periodically in patients being given heparin for 3 or more days, and in patients at relatively high risk for hyperkalemia, the monitoring interval should probably be no greater than 4 days.[6]

Fludrocortisone promotes potassium excretion by its direct actions on the renal distal tubules. It results in a significant and rapid decrease in serum potassium even with continued heparin administration and acute renal failure.[4] Therefore it can be used as a potential treatment for the hospitalized patients who need to be on the heparin treatment.

CONCLUSION

Heparin is a very unknwon cause of hyperkalemia in hospitalized patients. It is imperative for the physicians to recognize this association and focus on the judicious use of heparin to prevent thromboembolism. Heaprin decreases the production of aldosterone so using fludrocortisone in patients who develop hyperkalemia on heaprin therapy is important to treat hyprekalemia while keeping patients on heparin therapy.

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