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# ROLE OF FLUDROCORTISONE IN TREATMENT OF HEPARIN-INDUCED HYPERKALEMIA: A CASE REPORT OF RARE ASSOCIATION

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## ABSTRACT
Heparin is an intravenous anticoagulant used in treating and prophylaxis of pulmonary thromboembolism, deep vein thrombosis, and post-myocardial infarction patients. A rare adverse effect of heparin reported is hyperkalemia, which manifests within a few days of therapy initiation. Here we report a case of a 71-year-old African American male patient presented to the ED via Dermatology Clinic for IV antibiotics for a stasis ulcer on his right lower leg, which has been exacerbating for the last seven months. Past medical history is significant for CKD stage IV with a baseline creatinine of 2.7 mg/dL secondary to uncontrolled hypertension, thoracic aortic dissection s/p repair three years ago, and essential thrombocytosis. On admission, his labs were Na+ 130 mEq/L, K+ 5.2 mEq/L, creatinine 2.98 mg/dL. The patient was started on subcutaneous heparin 5000 units twice daily, and K+ rose to 6.1 mEq/L. The patient was started on fludrocortisone, and his K+ started trending down slowly over days. Fludrocortisone (FCA) is a synthetic glucocorticoid with strong mineralocorticoid activity and moderate glucocorticoid activity.

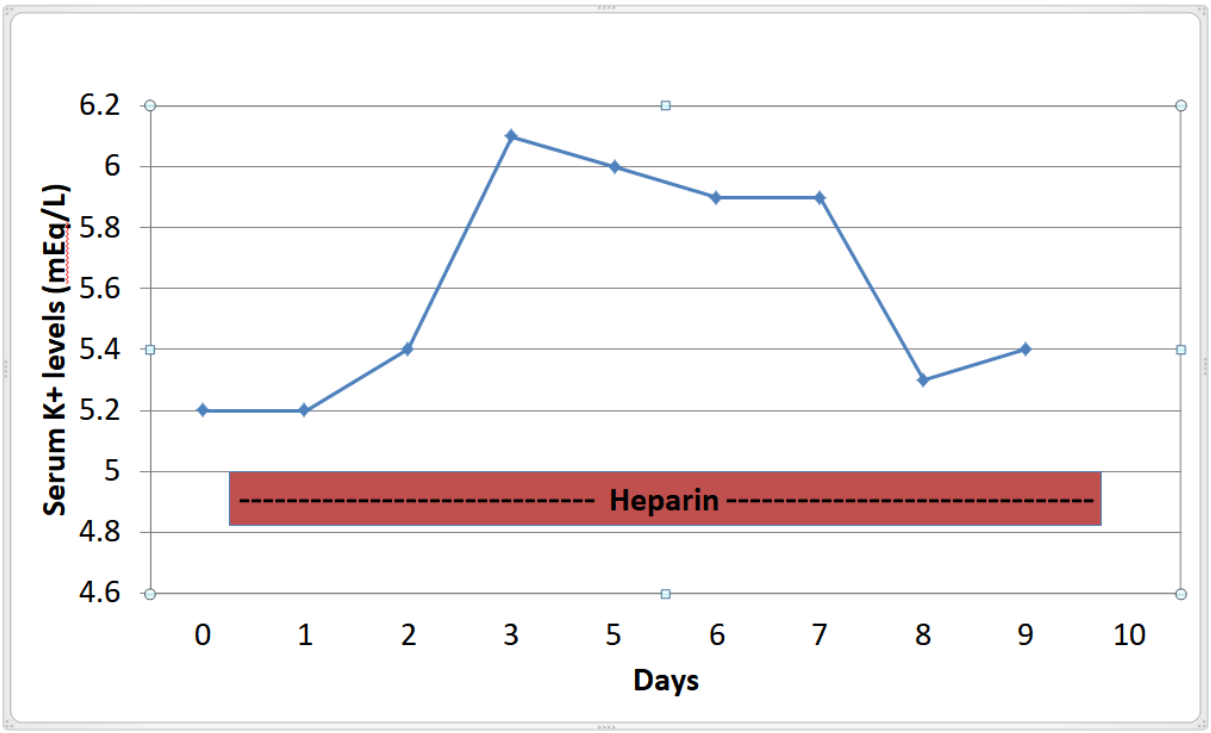
**KEYWORDS:** Pulmonary thromboembolism, deep vein thrombosis, and post-myocardial infarction patients.

## INTRODUCTION
Heparin is an intravenous anticoagulant used in treating and prophylaxis of pulmonary thromboembolism, deep vein thrombosis, and post-myocardial infarction patients. It is now being replaced by low-molecular-weight heparin (LMWH) because of certain advantages like equal efficacy, increased bioavailability, and less frequent dosing interval. Also, monitoring of activated partial thromboplastin time is not required. The significant adverse effects of these drugs are bleeding, thrombocytopenia, alopecia, and osteoporosis. A rare adverse effect of heparin reported is hyperkalemia, which manifests within a few days of therapy initiation.[1]

**Case Report 2:** A 71-year-old African American male patient presented to the ED via Dermatology Clinic for IV antibiotics for a stasis ulcer on his right lower leg, which has been exacerbating for the last seven months. Patient-reported having associated low-grade fevers and pain. However, he denied chills, nausea, vomiting. Past medical history is significant of CKD stage IV with a baseline creatinine of 2.7 mg/dL secondary to uncontrolled hypertension, thoracic aortic dissection s/p repair three years ago, and essential thrombocytosis. Past medication history was significant for Labetalol 800 mg and Nifedipine 90 mg daily for hypertension.

On admission, his labs were Na+ 130 mEq/L, K+ 5.2 mEq/L, creatinine 2.98 mg/dL. The patient was admitted and given IV cefepime 1000 mg, IV ertapenem 1000 mg, vancomycin 1300 mg, PO Ibuprofen 800 mg one tablet for pain, and subcutaneous heparin 5000 units twice daily. On day 2, Antibiotics were discontinued due to suspecting stasis ulcer over an infection. The patient developed Acute Kidney injury, and creatinine rose to 3.34 mg/dL, and serum K+ levels 5.2 mEq/L subsequently rising to 3.41 mg/dL, and K+ levels 6.1 mEq/L on day 4 when he was started to be treated for Acute Kidney injury and hyperkalemia.

The patient was given insulin/dextrose and started on IV furosemide and Sodium Chloride 0.45%. His K+ dropped to 5 mEq/L but went back up to 6 mEq/L on day 5 when he was shifted to Normal Saline and Sodium bicarbonate 1300 mg BID. The patient was started on fludrocortisone, and his K+ started trending down slowly over days. IV furosemide was discontinued on day six and was changed to PO torsemide 20 mg day 8-10. His Serum K+ levels are 5.3 on day 10.




**Serum Lytes**:

| Days | Na+ (mEq/L) | K+ (mEq/L) | Creatinine (mg/dL) | CO2 (mEq/L) |
|---|---|---|---|---|
| 1 | 130 | 5.2 | 2.98 | 23 |
| 2 | 131 | 5.2 | 3.34 | 21 |
| 3 | 130 | 5.4 | 3.27 | 21 |
| 4 | 133 | **6.1** | **3.43** | 22 |
| 5 | 131 | 6.0 | 2.99 | 23 |
| 6 | 130 | 5.9 | 2.75 | 26 |
| 7 | 130 | 5.9 | 2.97 | 26 |
| 8 | 131 | 5.5 | 3.05 | 28 |
| 9 | 130 | 5.4 | 3.13 | 29 |
| 10 | 130 | 5.3 | 3.43 | 28 |

## DISCUSSION

A reversible effect on aldosterone mediates Heparin-induced hyperkalemia through blockage of an enzymatic step in the synthesis and angiotensin II receptors in the adrenal gland.[2] The most important mechanism of heparin-induced hypoaldosteronism involves reducing both the number and affinity of angiotensin II receptors in the zona glomerulosa.[3] Prolonged heparin administration has caused a marked reduction in the width of adrenal zona glomerulosa, thereby leading to hyperkalemia and natriuresis. These side effects are significant in the elderly, renal insufficiency, and diabetic patients.[4]

Fludrocortisone (FCA) is a synthetic glucocorticoid with strong mineralocorticoid activity and a moderate glucocorticoid activity. It stimulates the Na-K-ATPase activity and increases the potassium secretion from the gastrointestinal tract. Potassium is also shifted intracellularly, which also play an essential role in the potassium-lowering effect of FCA. When used in the dose range of 0.1–0.3 mg/day, FCA reduced serum potassium.[5]

## CONCLUSION

Heparin induces hyperkalemia by reducing the levels of aldosterone. Patients with comorbidities are at significant risk of developing hyperkalemia while on heparin therapy. The physicians need to recognize this association, and prompt therapy with fludrocortisone is necessary to control potassium levels in patients who have to be kept on heparin to prevent thromboembolism.

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