

level of 8.8 mEq/L to levels in the range 6.0 to 7.0 mEq/L. Nevertheless, within 48 h of administration of fludrocortisone 100 µg bid by mouth, plasma potassium was restored to the normal range (3). Further investigations revealed a serum creatinine level of 191 µmol/L (2.16 mg/dL), and a random serum cortisol of 423 nmol/L (15.3 µg/dL) (3). Tacrolimus is another inducer of Type 4 renal tubular acidosis (RTA), and the resulting hyperkalemia may be refractory to intravenous furosemide, oral sodium polystyrene, intravenous sodium carbonate, and glucose-insulin infusion (4). In the report by Sahu et al. (2017) (4), however, serum potassium fell from 6.2 mmol/L (6.2 mEq/L) to the normal range 24 h after the administration of fludrocortisone 0.1 mg/day. With continued use of fludrocortisone, the serum potassium subsequently fell to 4.5 mmol/L (4.5 mEq/L) (4).

Heparin-induced hyperkalemia also has a Type 4 RTA-dependent etiology, and it also resolves after administration of oral fludrocortisone (5). The association of hyponatremia (116 mmol/L; 116 mEq/L) and hyperkalemia (7.2 mmol/L; 7.2 mEq/L) (the latter resistant to intravenous insulin/glucose infusion and to oral cation exchange resins) was attributable to the heparin analogue, pentosan, in a 65-year-old woman with a mistaken provisional diagnosis of adrenal crisis. Both her hyperkalemia and hyponatremia responded to the combination of intravenous hydrocortisone, 100 mg every 6 h, intravenous sodium bicarbonate infusion, and nebulized salbutamol, with the consequence that, 7 days later, her serum sodium had risen to 144 mmol/L (144 mEq/L), and serum potassium had fallen to 3.4 mmol/L (3.4 mEq/L). Her random pretreatment serum cortisol was > 1650 nmol/L (59.81 µg/dL), thereby negating the provisional diagnosis of adrenal crisis (6). Accordingly, there might be a role for the use of high-dose hydrocortisone to exploit the mineralocorticoid action of that agent for the purpose of bringing down the serum potassium level if the patient is too ill to take oral fludrocortisone (7).

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## RESPONSE TO LETTER TO THE EDITOR



### To the Editor:

Thank you for the interesting comments by Dr. Jolobe in this issue of the Journal (1). Although his review of the use of mineralocorticoid treatment of hyperkalemia has a plausible mechanism of action, the limitation to refractory hyperkalemia excludes this strategy for the overwhelming majority of emergency department (ED) cases. Our study investigated the initial ED treatment of hyperkalemia, and had a primary endpoint defined as the serum potassium level after 4 h of treatment (2). Because the definition of refractory hyperkalemia requires a failure of standard therapy, it is outside the purview of our study to provide insight regarding treatment with mineralocorticoids. Further, we are not aware of a single prospective study using mineralocorticoid therapy for the ED treatment of hyperkalemia. We cannot support the use of mineralocorticoid therapy as an appropriate intervention until there are prospective safety and efficacy data in the ED environment.

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## MEDICO-LEGAL ANALYSIS OF TWO RECENT CASES OF BODY PACKING



### To the Editor:

We read with interest the two recent reports published in your journal, discussing the unusual case of a body packer concealing both a synthetic cannabinoid and cannabis,