Further Section

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Answers

Pseudohyperkalemia and excessive K intake were not present in this patient. Furthermore, neither the creatinine clearance nor the urine output were low enough to account for decreased K excretion. Thus, K redistribution and/or defects in renal K secretion were the primary possibilities. Severe digoxin toxicity can cause K shift from the intracellular fluid, but was not present. While tissue damage or necrosis leading to hyperkalemia was possible, particularly in view of the history of deep venous thrombosis, pulmonary embolism, and metastatic carcinoma, there was no clear evidence for this phenomenon. Various perturbations in the renin-aldosterone axis of this individual seemed much more likely. In view of the abnormal renal function studies, renal aldosterone resistance secondary to some form of tubulo-interstitial renal disease might have been present. Against that possibility were the very low fractional sodium excretion (0.3%), the elevated blood urea/creatinine ratio, and the elevated urine osmolality which all pointed to prerenal azotemia. More plausible was the possibility that the patient had low distal renal tubular sodium delivery which impaired potassium secretion despite adequate aldosterone and increased plasma potassium. A remote consideration in this patient was hypoaldosteronism due to heparin inhibition of adrenal aldosterone release.

The urine potassium concentration is often difficult to interpret during evaluation of patients with either hypo- or hyperkalemia. A new approach [1-3] improves on the traditional assessment of urine potassium excretion by allowing clinicians to adjust for an elevation in the urine potassium secondary to water reabsorption in the medulla. This test is rapid and assesses the transtubular potassium concentration gradient (TTKG), a semiquantitative reflection of the potassium secretory process in the cortical collecting duct. By correcting for medullary water abstraction (dividing by the ratio of urine to plasma osmolality), the urine potassium concentration should approximate that in the lumen of the cortical collecting duct in situations where the urine is hypertonic (in the presence of antidiuretic hormone). Thus:

\[ \text{TTKG} = \frac{[K]}{[K]} \times \frac{\text{osmp}}{\text{osmp}} \]

In our clinical example, the first TTKG calculation was 2.4. From our experience in rats and humans, a value of 4 or less indicates low potassium secretion, whereas values of 6 or greater suggest stimulated potassium secretion in the cortical collecting duct and usually reflect high renal aldosterone action. The finding of a low TTKG value confirms the presence of an abnormality in the renin-aldosterone-renal axis, but does not differentiate between the more likely possibility of decreased distal renal tubular response to aldosterone (caused by low sodium delivery) versus a low circulating aldosterone level due to heparin inhibition of adrenal aldosterone release or a low angiotensin II level caused by severe illness.

(3) Since reduced distal nephron sodium delivery was a likely and readily reversible cause of hyperkalemia in this patient, and since the plasma potassium of 6.0 mmol/l was potentially life-threatening due to cardiac arrhythmias, treatment was aimed at correcting the pathophysiologic abnormality. We recommended increasing distal nephron sodium delivery by administering
furosemide and intravenous isotonic saline. In addition, pericardiocentesis was performed with removal of 480 ml of sanguinous fluid. Use of a loop diuretic was an essential component of therapy because intravenous sa-

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line and pericardiocentesis alone probably would not have immediately increased distal nephron sodium delivery. These measures designed to increase the cardiac output in conjunction with furosemide resulted in an increased urine flow and urine sodium. High renal aldosterone activity was restored as shown by a rise in the calculated TTKG to 6.3. (This would not have occurred in heparin-induced hyperkalemia.) Increased potassium excretion resulted in a fall of the plasma potassium to 4.6 mmol/l. While increased urine flow could have increased potassium excretion by itself, it should not have raised the TTKG. Interestingly, the urine potassium concentration did not change significantly, a finding which exemplifies the problem of using this value in isolation to assess patients with hyperkalemia.

References