MEDIBEATS
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In Metabolism

Not all hypokalemia require only potassium supplement for correction

60-year-old female, with no comorbid condition presented with bilateral symmetric poly arthritis involving knee, elbow and shoulder and lower backache since past 6 months. She developed weakness of both lower limbs so much that she had difficulty in walking and could not do daily activity without support. She also lost significant weight in past 2 months. There was no history of fever, focal neurological deficit, malena, neck pain and cough. On examination her blood pressure was normal. Other vitals and systemic examination were also normal. There was no swelling/ redness over small joints. On investigations, she had metabolic acidosis, hypokalemia (serum potassium – 2.1 meq/l), and normal anion gap. RA factor was positive. We start supplementing sodabicarb, and replace her potassium but it worsened the situation and she developed extreme vomiting. UGI endoscopy done which showed pangastritis. Urinary pH was 7 and potasium was high, suggesting renal loss possibly renal tubular acidosis (RTA). After supplementing potassium her potassium raised to 3.4 meq/l. She was discharged on potassium and sodabicarb supplementation along with DMARD.

After 4 days she readmitted through emergency with severe vomiting and found to have potassium of 2.38 meq/l. She was treated with intravenous potassium and sodabicarb and responded well. Indomethacin and spironolactone were added for RTA. Oral potassium chloride was on hold as patient was not tolerating it this time. Gradually she improved on oral sodabicarb and spironolactone.

She is now in our OPD follow up since past 6 months, maintaining serum potassium in range with minimal amount of syp potassium chloride, sodabicarb tablets and spironolactone. Her joint pain improved markedly on DMARD.

Conclusion: While working a patient of hypokalemia, it is important to look for associated metabolic derangement and urinary pH, and potassium that guide further management.

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