Post-operative severe hypokalemia mimicking myocardial ischemia

Sir,

Perioperative ST segment depression with tachycardia is often attributed to be of ischemic origin.[1] It becomes all the more important in patients with diabetes and hypertension where patients stand higher risks of coronary diseases and silent ischemic events. Although, a high index of suspicion should be maintained for ischemia, but the possibility of alternative diagnosis should not be negated without appropriate investigation.

We present a case of 28-year-old female patient with Cushing's syndrome manifesting unexplained painless ST depression with tachycardia post-operatively after an uneventful intra-operative bilateral laparoscopic adrenalectomy. She had no significant medical history until 6 months, when she developed cushingoid features with unexplained weight gain. A diagnosis of Cushing syndrome due to bilateral adrenal hyperplasia was made and subsequently was confirmed radiologically as well as biochemically. She was receiving amlodipine, insulin for hypertension, diabetes, respectively and both conditions were fairly controlled. No history of coronary artery disease (angina, palpitations and diaphoresis) or effort limitation was found. Pre-operatively hemogram, biochemistry (potassium = 3.5 Meq/l) and electrocardiogram (ECG) were unremarkable. Due to prior hypokalemia she was receiving potassium chloride syrup and

was continued pre-operatively. She received polyethylene glycol solution for bowel preparation prior to surgery and morning potassium was 3.3 Meq/l. Laparoscopic bilateral adrenalectomy was performed uneventfully. During the surgery, she received hydrocortisone (100 mg, intravenous, after adrenal removal) and normal saline as maintenance fluid. In recovery half-an-hour after surgery, she developed unexplained tachycardia and ST depression [Figure 1]; however, the patient reported no complaints, seemed comfortable with no chest or surgical site pain.

Being diabetic possibility of silent ischemic episode was considered due to associated severe tachycardia (130 beats/min). Quantitative troponin-I was normal suggesting the absence of myocardial ischemia.[2] Twelve lead ECG showed global ST depression, which is unlikely to be caused by ischemia.[3] Arterial blood gas showed severe hypokalemia (1.9 Meq/l), lactate of 0.2 mmol/l with all other values well within normal range. In the absence of possibility of ischemia (normal troponin, lactate) global ST depression was attributed to severe hypokalemia. This was further confirmed by normalized ST depression with potassium correction through the central line in next 6-8 h. The probable cause of precipitation of severe hypokalemia in our patient with already potassium on the lower side was multifactorial. Use of bowel preparation is known to cause potassium depletion.[4] Corticosteroids used during the bilateral adrenalectomy also precipitate hypokalemia by potassium internalization and urinary excretion.[5] Perioperatively we used glucose insulin neutralizing drip; although, potassium was added to the solution (as per standard regimen), but it is still known to cause hypokalemia due to inter-individual response variability.[6] Normal saline (avoiding gluconeogenic-ringer lactate) as maintenance fluid can also contribute to hypokalemia.

Hypokalemia is a rare, but well-known cause of ST depression with tachycardia.[7] Although limb muscle weakness can be associated, it may not however become apparent in a non-ambulatory immediate post-operative patient. Such a global ST depression associated with severe post-operative hypokalemia has not been reported previously. In asymptomatic patients with new onset, unexplained global ST depression in the post-operative period, hypokalemia should be ruled out as many of the above precipitating factors are often present in many surgical patients.

Figure 1: Post-operative ST depression with tachycardia

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