Perioperative management of patient with Conn's syndrome and severe hypokalaemia: How low is too low?

Sir,

The presence of hypokalaemia in patients with systemic hypertension is highly suggestive of aldosteronism.[1] We present the successful perioperative management of a patient with persistent hypokalaemia secondary to an adrenal incidentaloma scheduled for left-sided laparoscopic adrenalectomy and cholecystectomy.

A 56-year-old male patient presented with symptoms suggestive of gall stone disease since 2 months. On evaluation, he was found to have asymptomatic hypokalaemia (serum potassium 2.0 mEq/L). He was a known case of hypertension which was controlled with oral amlodipine 5 mg twice daily and oral metoprolol once daily. Computed tomography of the abdomen revealed a left adrenal neoplasm measuring 2.7 × 2.4 cm. There was no uptake on metaiodobenzylguanidine scanning; urinary catecholamines and dexamethasone suppression test were negative. Echocardiography (ECG) revealed concentric left ventricular hypertrophy and mildly dilated left atrium with grade I diastolic dysfunction. On Holter monitoring, left anterior fascicular block, poor R wave progression and intermittent atrial fibrillation were noted. Oral spironolactone 50 mg once daily and oral potassium chloride 40 mEq twice daily were started to correct hypokalaemia.

Preoperatively, the other biochemical investigations were within normal range (serum Na: 145 mEq/L and serum Cl: 100 mEq/L). ECG showed sinus bradycardia (54/min) with prolonged QT interval (0.48 s), broad QRS complexes and right bundle branch block with left anterior hemiblock.

Daily serum potassium levels were monitored prior to surgery. All values were below 2.5 mEq/L. On the morning of the surgery, the patient was premedicated with oral alprazolam, spironolactone, metoprolol and amlodipine. The morning electrolytes were serum Na 146 mEq/L and serum K^+ 2.4 mEq/L. After routine monitoring was established, subarachnoid block was performed at L3–L4 space with intrathecal 5 mg (heavy) bupivacaine and 300 mcg morphine. Right internal jugular cannulation using ultrasound guidance and right radial catheter were secured for venous pressure and invasive blood pressure monitoring, respectively. Anaesthesia was induced with fentanyl, propofol and atracurium followed by tracheal intubation and maintained on oxygen, nitrous oxide with isoflurane. Train of four (TOF) was used for monitoring the neuromuscular blockade.

Left adrenalectomy was performed after localisation of the adrenal mass laparoscopically in the left lateral position. Occasional hypertensive responses were managed with intravenous nitroglycerin infusion (1–5 mcg/kg/min). The procedure lasted for 120 min. Care was taken to avoid hyperventilation. Serum potassium levels were monitored hourly intraoperatively along with arterial blood gas analysis. No potassium replacement was given. At the end of the procedure, trachea was extubated after observing adequate tidal volume and muscle power (TOF ratio ≥0.9). The patient was transferred to the intensive care unit for further monitoring and O_2 therapy. Potassium levels remained between 2.3 and 2.8 mEq/L. Oral potassium chloride was supplied for 1 week postoperatively before it normalised.

Primary hyperaldosteronism can be caused by unilateral adrenal adenoma or bilateral hyperplasia.[2] Anaesthetic concerns for this patient included prolonged action of neuromuscular blocking agents because of hypokalaemia and metabolic alkalosis. When compared with patients with essential hypertension, the other problems are hypernatraemia, intravascular volume depletion and abnormal renal functions.[3] In addition, acute preoperative potassium correction was associated with risk of cardiac arrhythmias. Intraoperative acute replacement of potassium would cause alteration of intracellular: extracellular ratio of potassium *in vivo* (30:1). Serum K^+ levels were maintained between 2.3 and 2.8 mEq/L. Ventilation strategies to avoid hypocarbia and metabolic alkalosis were used to decrease the severity of hypokalaemia intraoperatively. Allard[4] suggested that in patients with chronic hypokalaemia, dangers of hypokalaemia during intraoperative period are less than that of iatrogenic hyperkalaemia from overadministration or too rapid administration of potassium.

During surgery, adrenal gland handling may lead to catecholamine release from the adrenal medulla with resultant haemodynamic variations. Hypertensive peaks can occur due to carbon dioxide pneumo-insufflation

leading to adrenergic activation and also due to tumour handling and resection. This type of hypertensive peaks is quite common in cases of Conn's syndrome even after thorough preoperative blood pressure control as reported by Gockel I *et al*. in their series.[5] Postoperatively, serum potassium was replaced with oral potassium chloride postoperatively for 1 week only.

We conclude that severe hypokalaemia in a patient with primary hyperaldosteronism can be managed with aldosterone antagonist and oral potassium chloride alone preoperatively. During intraoperative period, acute correction of K^+ is not necessary and may lead to arrhythmias.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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