

Acute onset quadriparesis following oesophagectomy due to isolated hypophosphataemia

Sir,

A 65-year-old male weighing 50 kg with carcinoma oesophagus received neo-adjuvant chemotherapy (carboplatin and paclitaxel) and radiation and subsequently underwent transhiatal oesophagectomy under general anaesthesia after an elaborate preoperative workup which was unremarkable. His past medical and surgical history was unremarkable. All preoperative laboratory investigations including electrolytes were within normal limits. At the end of surgery, the patient was shifted to the surgical intensive care unit and the trachea was extubated uneventfully after 2 h. On postoperative day (POD) 2, high flow oxygen therapy was initiated via nasal cannula as he developed bibasal atelectasis, had poor incentive spirometry efforts and had a $\text{PaO}_2/\text{FiO}_2$ ratio of 175. On POD3, he developed sudden onset atrial fibrillation (AF) with fast ventricular rate which was managed with IV amiodarone. About 24 h after new onset AF, patient developed acute onset flaccid quadriparesis which raised a suspicion of thromboembolic phenomenon. This was ruled out on echocardiography (right heart function and chambers were normal with normal biventricular function) and a normal computed tomography scan of brain. Meanwhile, sample for serum electrolytes (sodium, potassium, chloride, calcium, magnesium and phosphate) was sent. Serum phosphate was 1.7 mg/dl (normal range

2.5–4.5 mg/dl), which was very low. Correction with intravenous injection Potphos™ (93 mg/ml of phosphorus and 170 mg/ml of potassium chloride) available as 15 ml vial (manufactured by Neon Laboratories Ltd.) was started. Values of other electrolytes were within normal limits. The acute onset quadriparesis recovered in 45 min of starting phosphorus correction. The patient was thereafter treated with IV Potphos™ 1 vial/day for 3 days and was later on advised to use Addphos™ sachet (available as 3.2 g sachet, manufactured by Steadfast Medishield Pvt. Ltd.), which contains 1.936 g of sodium acid phosphate, for 2 weeks. He was transferred to ward on POD 8. Serum phosphorus level measured prior to discharge was 3.5 mg/dl which was considered within normal range.

There could be numerous causes of hypophosphataemia in post-oesophagectomy patients. Prolonged use of antacids, poor nutritional status, re-feeding syndrome, acute respiratory alkalosis, and bronchodilator use can all contribute to hypophosphatemia in the early postoperative period.^[1] Severe hypophosphataemia has been demonstrated as a rare cause of early postoperative weakness.^[2] It is a documented complication after hepatic surgery with multifactorial cause including increased excretion in urine due to phosphatonins.^[3] To the best of our knowledge, similar acute onset symptoms of hypophosphataemia have not been described after oesophagectomy. We suggest monitoring phosphate levels in the early postoperative period for oesophagectomy patients. As suggested by Ianov *et al.*,^[4] phosphorus correction should be done slowly at the rate of 10–45 mmol in 6–8 h (10 mmol = 1395 mg phosphorus which is present in 15 ml of potassium phosphate infusion).

Consent was taken from the patient for publication in a medical journal for academic purpose without disclosing the name.

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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Nil.

Conflicts of interest

There are no conflicts of interest.

***Vibhavari M Naik, Mohammed Salman Saifuddin,
Abhijit S Nair, Basanth K Rayani***

Department of Anaesthesiology, Basavataarakam Indo-American Cancer Hospital and Research Institute, Hyderabad, Telangana, India

Address for correspondence:

Dr. Abhijit S Nair,
Department of Anaesthesiology, Basavataarakam Indo-American Cancer Hospital and Research Institute, Hyderabad - 500 034, Telangana, India.

E-mail: abhijitnair95@gmail.com

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