Case Report

Undetected hypoparathyroidism: An unusual cause of perioperative morbidity

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ABSTRACT

Routine investigation of serum calcium is not recommended in ASA one and two patients unless abnormalities of calcium metabolism are clinically suspected. The clinical features of hypocalcaemia can often be subtle and may manifest in the presence of associated factors. Hypoparathyroidism, an important cause of hypocalcaemia, often presents as soft tissue calcification (ostosis). Ligamentum flavum ostosis can present with compressive myelopathy requiring laminectomy. We report a case of ligamentum flavum ostosis and subclinical hypocalcaemia due to hypoparathyroidism, who went undetected pre-operatively resulting in significant post-operative morbidity.

Key words: Hypocalcaemia, hypoparathyroidism, ligamentum flavum ostosis

INTRODUCTION

Hypocalcaemia can be an important cause for delayed recovery in the post-operative period and can significantly influence post-operative morbidity.[1] Hypoparathyroidism is an important cause of hypocalcaemia.[2] We report a case of laminectomy and decompression of the spine with increased morbidity because of undetected pre-existing hypoparathyroidism. Consent of the patient was obtained before publication of this case report.

CASE REPORT

A 45-year-old male of 65 kg, presented with a history of low backache and weakness of both lower limbs for last 2 years, and urinary retention and constipation for the last 2 days. He had full power in the upper limb and Grade 3 power in the lower limbs. A magnetic resonance image (MRI) of the dorso-lumbar spine showed thickened ligamentum flavum indenting the thecal sac from D9 to D12 with cord oedema. Pre-anaesthetic examination was normal except for distended, but soft abdomen without clinical evidence of ascites or organomegaly. Bowel sounds were present. His urinary bladder was catheterized. Pre-operative investigations were normal. He was admitted for laminectomy and decompression of the spine.

Standard monitoring, including invasive blood pressure and arterial blood gas (ABG) was used intraoperatively. Anaesthesia was induced with propofol 140 mg, fentanyl citrate 150 μg and rocuronium bromide 50 mg and trachea wase intubated. Mechanical prophylaxis for deep vein thrombosis and warm air blanket were used intraoperatively. His post-induction ABG readings were normal. Anaesthesia was maintained with 2% sevoflurane in 50% O_2 in air, propofol infusion at 25 μg/kg/min and vecuronium bromide at 2 mg/h. The surgery lasted 5 h. Intermittent fentanyl (total 500 μg) was used for analgesia intraoperatively. Hydration was maintained with intravenous (IV) 3.5 L 0.9% saline, 0.5 L colloid (tetra starch).

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Noradrenaline at 2-4 μg/min was started in view of refractory hypotension. Blood loss was 250 ml. and urine output was 250 ml. ABG obtained 2 h post-induction showed metabolic acidosis. Severe canal stenosis and cord oedema was noticed from D9 to D12. Methylprednisolone was started as 30 mg/kg bolus followed by infusion at 5.4 mg/kg/h. At the end of surgery, the patient had inadequate motor power and respiratory effort. A repeat ABG showed metabolic acidosis. His blood sugar was 246 mg% and urine ketone was negative. Insulin infusion was started, and the patient was shifted to intensive care unit for elective ventilation. A central venous line inserted showed a central venous pressure of 1-2 cm H_2O. Overnight fluid therapy resolved acidosis and oliguria. Next day the patient was extubated. Kidney function test (KFT) revealed high normal values. Serum sodium, potassium, and thyroid status was found normal. At this time, his upper limb power was almost normal while power in both lower limbs was 1/5 requiring continuation of methylprednisolone. Six hours after extubation he developed tense abdominal distension and respiratory distress. His ABG revealed respiratory alkalosis. He was reintubated and ventilated. His chest X-ray and echocardiogram were normal. Due to inconclusive abdominal ultrasound report (USG) a computerized tomography of the abdomen was obtained which showed colonic gaseous distension without free fluid in the abdomen. Ryle's tube was inserted and injection neostigmine started at 2.5 mg/h for 2 h. On post-operative day (POD) 2 the patient re-developed deranged KFT along with high-grade fever. Blood, urine, and tracheal aspirate were sent for cultures and sensitivity and antibiotics upgraded as per culture reports. Sepsis and "pancreatitis profile" revealed serum procalcitonin 4.4 ng/ml, hypocalcaemia (serum calcium 4.1 mg%), hypoalbuminemia (2 g%), but normal serum amylase and lipase. Factitious hypocalcaemia was ruled out by serum ionized calcium 0.5 mmol/L. Further investigations revealed hyperphosphatemia, low parathormones and normal magesium levels. IV albumin was started to correct hypoalbuminemia. Due to refractory hypocalcaemia, high doses of IV calcium (450 mg elemental calcium 4th h), was required along with oral calcitriol 0.5 μg q8h. On POD 3 renal functions and acidosis started to resolve. Despite 2 days trial of neostigmine, prokinetics and laxatives, his abdominal symptoms persisted. Digital examination ruled out faecal impaction. Decompressive sigmoidoscopy was performed and total parenteral nutrition (TPN) was started.

Over next 2 days gastrointestinal (GI) symptoms, procalcitonin and total leucocyte count started to decrease. Serum calcium increased to 7.7 mg%.

On POD 9 the patient was extubated. Eighteen hours later, he was reintubated and ventilated due to severe tachypnoea and decreased consciousness. Pulmonary embolism was ruled out by doing a USG Doppler lower limbs and echocardiography. MRI spine did not reveal any fresh changes. Cerebrospinal fluid analysis ruled out meningitis. On POD 13 he was tracheostomised.

Patient showed steady improvement from next day. He was weaned off the ventilator fully conscious with good power in all limbs, normal calcium and albumin levels, and no GI symptoms. He was later decannulated and discharged with on-going treatment and advice for follow-up.

DISCUSSION

Ossification of the ligamentum flavum is a rare cause of thoracic myelopathy. Neurologic improvement and good recovery usually occurs following decompression laminectomy.[3] However in our case, there was considerable post-operative morbidity.

Our patient must have been hypovolemic pre-operatively which would have masked hypoalbuminemia. This got manifested once the patient became volume repleted. The patient had been harbouring urinary tract infection (UTI) which was missed in the absence of urine microscopic examination. This got aggravated perioperatively due to stress of surgery and hyperglycaemia.

Hyperglycaemia in our non-diabetic patient was because of steroids and sepsis due to UTI. The decreased urine output, and metabolic acidosis was the combined result of sepsis and intraoperative usage of noradrenaline.[4,5] Antidiuretic hormone release due to stress of surgery[6] may have added to oliguria.

The manifestations of hypocalcaemia can be varied depending upon its rapidity of development.[2] Spasticity is a known clinical manifestation of hypocalcaemia, which in our case manifested as a tense abdomen. As calcium is

an important mediator of excitation-contraction coupling, colonic distension and paralytic ileus is possible due to hypocalcaemia.[7,8]

The best-studied treatment of acute colonic pseudo-obstruction is IV neostigmine, which leads to prompt colon decompression in the majority of patients after a single infusion.[9] However in our case, actual improvement started to occur only after normalisation of calcium levels.

Alkalosis and hypokalaemia aggravate the clinical manifestations of hypocalcaemia.[10] In this patient, pain and discomfort of tense abdominal distension possibly caused hyperventilation and respiratory alkalosis exaggerating the respiratory muscle fatigue due to hypocalcaemia. Hypokalaemia due to steroid and insulin compounded the respiratory muscle weakness.

Methylprednisolone, with predominantly glucocorticoid activity, decreases calcium absorption from the gut and increases its excretion in the urine.[11] It also causes hypokalaemia and alkalosis by excretion of potassium and hydrogen ions through renal tubules.[12]

Increased serum free fatty acids due to TPN lower serum ionized calcium.[10] Sepsis causes hypocalcaemia mediated by inflammatory cytokines.[13]

Our patient was diagnosed as primary hypoparathyroidism, with severe hypocalcaemia, sepsis and acute renal failure. The aetiological diagnosis was established after doing a 'pancreatitis profile' in view of fever, abdominal symptoms, and acute kidney injury (AKI), which revealed hypocalcaemia. Hypoparathyroidism is an important cause of hypocalcaemia. A review into the literature of hypoparathyroidism revealed hyperostosis as a feature due to chronic hypocalcaemia.[2] This explained the ligamentum flavum thickening.To the best of our knowledge, this is the first report correlating hypoparathyroidism, paralytic ileus and AKI.

CONCLUSION

Hypocalcaemia though not an uncommon entity is frequently overlooked. Its presence along with its etiological and aggravating factors should always be sought where recovery gets delayed.

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