Case Report

Tumor lysis syndrome developing intraoperatively

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Abstract

Tumor lysis syndrome is a potentially life threatening condition which is most commonly encountered in patients being treated with chemotherapy. We report a case of spontaneous tumor lysis syndrome that developed intraoperatively in a patient with undiagnosed Burkitt's lymphoma. Characteristic electrolyte disturbances and white emulsion like urine following laparotomy and tumor handling intraoperatively suggested the diagnosis. This is a rare perioperative complication and the report emphasizes the importance of being vigilant in recognizing the same.

Key words: Continuous venous-venous hemodialysis, Burkitt lymphoma, intraoperatively, tumor lysis syndrome, ventricular arrhythmia

Introduction

Tumor lysis syndrome (TLS) is an infrequent presentation in operating room requiring prompt management. We describe a case of TLS which presented with sudden ventricular arrhythmia during a laparotomy in a patient of lymphoma. Intensive resuscitation in the operating room followed by emergent hemodialysis in the intensive care unit was needed to manage the complications.

Case Report

A 40-year-old man was admitted to the hospital with one-week history of abdominal distension and pain. Patient had clinical signs of intestinal obstruction with presenting complaints of constipation with vomiting of one-week duration and abdominal tenderness on palpation. Blood investigations were normal except for leukocytosis with a white cell count of 15.4 × 10^9/l. Serum biochemistry tests were abnormal, showing a potassium level of 5.8 meq/l, serum creatinine level of 1.8 mg/dl, and a phosphate of 2.54 mmol/l. Liver enzyme tests were also deranged with an alanine transaminase (ALT) of 65 IU/l, aspartate transaminase (AST) of 156 IU/l and lactate dehydrogenase (LDH) of 4321 IU/l.

Computed tomography revealed extensive soft tissue lesions involving the abdominal and pelvic cavities and retroperitoneum [Figure 1]. Bilateral pleural effusion was detected and fluid analysis and cytology report was suggestive of lymphomatous pathology. Emergency laparotomy was then planned to relieve obstruction and simultaneously confirm the diagnosis and stage the disease. Preoperative hyperkalemia was treated with calcium gluconate and insulin and dextrose.

Cefixime 1.5 g and metronidazole 500 mg intravenous (IV) were administered. Pre-emptive analgesia was given with fentanyl 100 mcg IV and diclofenac 75 mg IV. Rapid sequence induction/intubation was done with propofol (120 mg) and

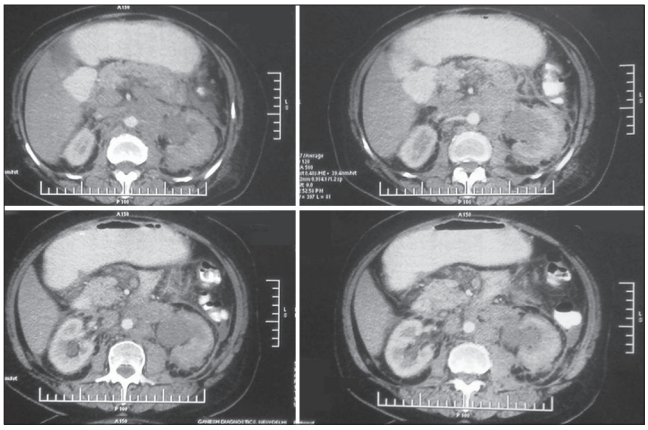

Figure 1: CT scan

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rocuronium (70 mg) IV. Succinylcholine was avoided in view of hyperkalemia. Anesthesia was maintained using a mixture of isoflurane (0.4 – 1%), air and oxygen. Atracurium (5 mg IV) was administered intermittently, as per the requirement, to maintain neuromuscular blockade. Total of 4 l of fluid (3 l crystalloid, 1 l colloid) was infused intraoperatively.

Intraoperative finding disseminated peritoneal deposits throughout the abdomen. Multiple biopsies from different areas were taken to confirm the diagnosis. Debulking of the mass obstructing the bowel was done to relieve the intestinal obstruction. The operation progressed uneventfully except for occasional ventricular ectopics. An episode of life-threatening ventricular arrhythmia, lasting for 30 min, occurred at the closure of abdomen. During the episode, recurrent and alternating ventricular fibrillation and ventricular tachycardia occurred, which were treated promptly by repeated defibrillation and anti-arrhythmic agents (amiodarone 150 mg over 10 min) in the operating room.

Blood sample sent for laboratory examination revealed severe hyperkalemia 6.6 mmol/l, hypocalcemia (0.86 mmol/l) and deranged serum creatinine (1.9 mg/dl) but normal arterial blood gases. During the resuscitation, intravenous calcium gluconate, sodium bicarbonate, rapid-acting insulin and dextrose solutions were also administered to treat the hyperkalemia. After restoration of normal rhythm and perfusion, the patient was transferred to the intensive care unit (ICU) for further management. In the ICU, controlled ventilation was continued. Sedation and analgesia were provided using propofol and morphine infusions respectively. Ventricular tachycardia recurred, which was cardioverted.

Postoperative blood results showed that the potassium had risen to 7.6 mmol/l, creatinine to 2.4 mg/dl and phosphate to 2.64 mmol/l, while the total calcium concentration had fallen to 1.67 mmol/l (normal range 2.2–2.7 mmol/l). A presumptive diagnosis of TLS was made and in view of uncontrolled hyperkalemia, continuous venous-venous hemodialysis (CVVHD) was started. After the initiation of CVVHD, the arrhythmias settled, and 6 h later, serum potassium levels decreased to5 mmol/l.

Within 8 h of arrival on the ICU, the urine output of the patient decreased and on changing the catheter patient passed around 500 ml of white emulsion like urine. Urine microscopy revealed the deposits of uric acid crystals. The plasma uric acid level was found to be raised to 16.7 mg/dl (normal 3.1–8.3 mg/dl). The diagnosis of TLS was confirmed and the treatment was commenced with rasburicase, a recombinant urate oxidase enzyme (0.2 mg/kg/day), in conjunction with allopurinol and vigorous hydration to treat hyperuricemia. Blood uric acid dropped to 9.1 mg/dl 2 days after the operation. Fluid resuscitation and control of electrolyte disturbances continued producing a urine output in excess of 1ml/kg/h.

The patient was weaned off from the ventilator on the third postoperative day. Over the next three days, the uric acid level reduced to 3.2 mg/dl, the serum phosphate level decreased and the appearance of the urine returned to normal. No further treatment for hyperkalemia was necessary and patient was weaned off from CVVHD one day later. The pathologic assessment confirmed the diagnosis of Burkitt's lymphoma. Chemotherapy was initiated and patient was discharged home with advice of regular follow-ups.

Discussion

TLS is an oncologic emergency requiring prompt attention to the management of potentially life-threatening complications due to metabolic derangements resulting from massive lysis of rapidly proliferating malignant cells, and is characterized by hyperuricemia, hyperkalemia and hyperphosphatemia. Hyperphosphatemia may lead to hypocalcemia with resultant tetany or other potentially life-threatening complications. TLS usually develops shortly after the start of effective cytotoxic therapy and may lead to complications in the cardiovascular, renal or neurological systems acute renal failure and death. Renal tubular precipitation of uric acid is a major, but not the only factor, leading to renal failure in TLS.[1]

Chemotherapy agents, employed for the treatment of the hematological malignancies such as Burkitt's lymphoma and acute lymphoblastic leukemia, are known to cause TLS.[2] Other reported triggering factors include corticosteroid therapy, radiotherapy, anesthesia and pyrexia.[3-12] Hills *et al.* described the case of a two-year-old child with high grade B-cell lymphoma who developed TLS in a radiology suite while undergoing transcutaneous ultrasound guided biopsy under anesthesia.[10] A similar case report of TLS in a six-year-old child with high-grade lymphoma undergoing laparotomy has been described by Lee *et al.*[11]

Conservative management with aggressive hydration, alkalinization and diuresis with correction of electrolyte abnormalities via targeted therapy or through hemodialysis is required for the management.[13-16] Intravenous fluid replacement is required in an attempt to maintain adequate urine output. Hyperuricemia is controlled through the use of the hypouricemic agents like allopurinol or rasburicase, with the former inhibiting xanthine oxidase and thus blocking uric acid formation, and the later catalyzing the breakdown of uric acid to allantoin.[17]

Patients at risk for TLS should have close electrocardiogram monitoring and the biochemical profile should be checked regularly by measurement of electrolytes and other parameters. Surgery can trigger TLS in patients with pre-existing risk factors like high tumor burden and altered biochemical profile, so tumor handling should be minimal. Anesthetic agents predisposing to hyperkalemia,[18] such as depolarizing neuromuscular blocking agents should be used with caution in patients with high tumor burden.

To conclude, critical cardiovascular complications requiring repeated resuscitation and emergency CVVHD were highlights of this case. Prompt diagnosis and meticulous intensive care of patients with life-threatening TLS are important to minimize mortality associated with it.

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