Case Report

Tumor lysis syndrome developing intraoperatively

Ankur Verma, Ruchi Mathur, Munish Chauhan, Prashant Ranjan

Department of Anaesthesiology, Dharmshila Cancer Hospital and Research Centre, Vasundhara Enclave, 1Swami Dayanand Hospital, Shahdara, 2Indraprastha Apollo Hospital, Delhi, 3Fortis Hospital, Noida, Uttar Pradesh, India

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...
was found to be raised to 16.7 mg/dl (normal 3.1–8.3 mg/dl).

The deposits of uric acid crystals. The plasma uric acid level decreased and on changing the catheter patient passed around 500 ml of white emulsion like urine. Urine microscopy revealed decreased and on changing the catheter patient passed around 500 ml of white emulsion like urine. Urine microscopy revealed

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 1 ml/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.

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 ectopies. 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 occurred, 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 to 5 mmol/l.

Conservative management with aggressive hydration, alkalinization and diuresis with correction of electrolyte abnormalities via targeted therapy or through hemodialysis is required for the management. 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.
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.

References

1. Dagher R, Kreissman S, Robertson KA, Provisor A, Bergstein J, Burke K, et al. High dose chemotherapy with autologous peripheral blood progenitor cell transplantation in an anephric child with multiply recurrent Wilms' tumor. J Pediatr Hematol Oncol 1998;20:357-60.
2. Tosi P, Barosi G, Lazzaro C, Liso V, Marchetti M, Morra E, et al. Consensus conference on the management of tumor lysis syndrome. Haematologica 2008;93:1877-85.
3. Chanimov M, Koren-Michowitz M, Cohen M, Pilipodi S, Bahar M. Tumour lysis syndrome induced by dexamethasone. Anesthesiology 2006;105:633-4.
4. Wester JP, Breumelhof R, Geers AB, Meuwissen OJ. Acute tumour lysis syndrome due to mono-therapy with a corticosteroid in a patient with non-Hodgkin's lymphoma. Ned Tijdschr Geneeskd 1997;141:1621-3.
5. Yang SS, Chan T, Dai MS, Lin SH. Steroid induced tumour lysis syndrome in a patient with pre-leukaemia. Clin Nephrol 2003;59:201-5.
6. Malik IA, Vellozo R, Khurshid M, Khan A. Radiation induced tumour lysis syndrome in patients with leukaemia. J Pak Med Assoc 1992;42:191-3.
7. Farley-Hills E, Byrne AJ, Brennan L, Sartori P. Tumour lysis syndrome during anaesthesia. Pediatr Anesth 2001;11:233-6.
8. Levin M, Cho S. Acute tumour lysis syndrome in highgrade lymphoblastic lymphoma after a prolonged episode of fever. Med Pediatr Oncol 1996;26:417-8.
9. Marenco JP, Nervi A, White AC. ARDS associated with tumour lysis syndrome in a patient with non-Hodgkin's lymphoma. Chest 1998;113:550-2.
10. Farley-Hills E, Byrne AJ, Brennan L, Sartori P. Tumour lysis syndrome during anaesthesia. Pediatr Anesth 2001;11:233-6.
11. Lee MH, Cheng KI, Jang RC, Hsu JH, Dai K, Wu JR. Tumour lysis syndrome developing during an operation. Anaesthesia 2007;62:85-7.
12. McDonnell C, Barlow R, Campisi P, Grant R, Malik D. Fatal peri-operative tumour lysis syndrome precipitated by dexamethasone. Anaesthesia 2008;63:652-5.
13. Rheingold SR, Lange BJ. Oncologic emergencies. In Pizzo PA, Poplack DG, editors. Principles and Practice of Pediatric oncology. 4th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2002. p. 1193-4.
14. Stucky LA. Acute tumor lysis syndrome: Assessment and nursing implications. Oncol Nurs Forum 1993;20:49-57.
15. Truini-Pittman L, Rossetto C. Pediatric considerations in tumor lysis syndrome. Semin Oncol Nurs 2002;18(3 Suppl 3):17-22.
16. Jeha S. Tumor lysis syndrome. Semin Hematol 2001;38 (4 Suppl 10):4-8.
17. Hochberg J, Cairo MS. Tumor lysis syndrome: Current perspective. Haematologica 2008;93:9-13.
18. Rimmer JM, Horn JF, Gennari FJ. Hyperkalaemia as a complication of drug therapy. Arch Int Med 1987;147:867-9.

How to cite this article: Verma A, Mathur R, Chauhan M, Ranjan P. Tumor lysis syndrome developing intraoperatively. J Anaesth Clin Pharmacol 2011;27:561-3.

Source of Support: Nil, Conflict of Interest: None declared.