Avens Publishing Group
J Androl Gynaecol
October 2015 Vol.:3, Issue:2

Open Access Case Report

Journal of Androlgy & Gynaecology

A Case of Tumor Lysis Syndrome after Docetaxel Administration for Recurrent Ovarian Cancer

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Submission: 24 August, 2015
Accepted: 21 October, 2015
Published: 25 October, 2015

Keywords: Tumor lysis syndrome; Ovarian cancer; Docetaxel; Rasburicase; Allopurinol

Abstract

Tumor Lysis Syndrome is a rare complication of the treatment of solid malignancies. A 51 year old female developed this condition one week after undergoing docetaxel chemotherapy for progressive, recurrent ovarian cancer. She was diagnosed based on classic laboratory disturbances including increased creatinine, hyperphosphatemia, hypercalcemia, hyperkalemia, and hyperuricemia. The patient was successfully treated with aggressive intravenous hydration, allopurinol, and rasburicase. Although uncommon, clinicians should be aware of this condition so that proper identification occurs and treatment can be implemented promptly.

Abbreviations

TLS: Tumor Lysis Syndrome; CA125: Cancer Antigen 125; CT: Computed Tomography; EKG: Electrocardiogram; ICU: Intensive Care Unit; BID: Twice daily

Introduction

Tumor Lysis Syndrome (TLS) is an emergent condition resulting from massive tumor cell lysis. The release of intracellular contents into the blood stream causes hyperkalemia, hyperuricemia, hyperphosphatemia, and acute kidney injury due to the precipitation of uric acid and phosphate crystals in the renal tubules. TLS is most commonly encountered in the treatment of aggressive hematopoietic malignancies. However, there are rare reports of this syndrome described after treatment of solid tumors. We report a case of TLS in a patient with recurrent ovarian cancer shortly after receiving docetaxel chemotherapy.

Case Presentation

A 51 year old woman was admitted to the hospital with general malaise. She had a history of stage IV a papillary serous ovarian cancer and had received docetaxel 7 days prior for recurrent, platinum resistant disease.

The patient was an otherwise healthy female who was originally diagnosed with stage IVa disease one year prior to current presentation with initial presenting complaint of dyspnea. Malignant pleural effusions were confirmed with cytology and extensive abdominopelvic disease was detected on subsequent PET/CT imaging. Tumor markers obtained were significant only for a CA 125 of 1066. Following initial cytoreductive surgery, the patient was treated with dose-dense paclitaxel (80 mg/m^2, cycles days 1, 8, and 15) and carboplatin (AUC 6, cycle day 1). This regimen was given at 28 day intervals with one week free of chemotherapy for recovery [1]. Carboplatin dosing was calculated using adjusted body weight (0.4(actual body weight – ideal body weight) + ideal body weight) as this patient had a BMI over 25 (BMI 27.4, BSA 1.78). Creatinine clearance was calculated using the Cockroft Gault equation with a minimum creatinine value of 0.7 mg/dL. The Calvert formula was used for Carboplatin dosing.

She received 6 cycles of dose-dense paclitaxel and carboplatin, with an initial improvement in her CA125. However, CT imaging at completion of 6 cycles demonstrated residual disease involving the chest, abdomen and pelvis and her CA125 plateaued. She was diagnosed with platinum resistant disease and was switched to a 28 cycle day regimen of gemcitabine (800 mg/m^2, cycle days 1 and 8) and bevacizumab (15 mg/kg, cycle day1) of which she received 2 cycles. Her CA 125 count continued to rise despite this treatment, thus she was changed to a 28 cycle day regimen of liposomal doxorubicin (40 mg/m^2, cycle day 1) and bevacizumab (15 mg/kg, cycle day 1). After 3 cycles of therapy, her disease failed to respond as evidenced by a rising CA 125. As a result, she was changed to docetaxel at a dose of 75 mg/m^2 and received one cycle uneventfully.

Seven days after her first docetaxel administration, the patient presented with fatigue and decreased oral intake. She was admitted for supportive therapy for suspected chemotherapy-associated malaise. On the evening of hospital day #1 the patient reported increased shortness of breath. She was noted to be pale, hypotensive, and hypoxic. An EKG revealed new onset right bundle branch block. She was transferred to the ICU for critical care monitoring given her worsening clinical picture.

TLS was diagnosed after laboratory evaluation revealed acute renal insufficiency (2.4 mg/dL) and severe electrolyte abnormalities (phosphorus 5.7 mg/dL, calcium 9.7 mg/dL, potassium 7.0 mmol/L and uric acid 10.1 mg/dL). Treatment with allopurinol, rasburicase, and aggressive hydration was promptly initiated. She received a single dose of intravenous rasburicase and started on allopurinol 300 mg BID. Six hours following administration of rasburicase, her uric acid had decreased to 2.9 mg/dL. She was continued on allopurinol for 3 days as her uric acid level continued to decrease. As her electrolytes

ISSN: 2332-3442

normalized, her clinical status stabilized. She was transferred out of the ICU on hospital day #10. She continued to recover clinically and was discharged to home in stable condition on hospital day #14.

Discussion

TLS is an infrequent complication of the treatment of solid malignancies. It is most commonly encountered in the treatment of aggressive hematopoietic cancers. At risk malignancies include those with high proliferative rates, large tumor volumes, and high chemosensitivity [2]. Initiation of cytotoxic treatment leads to the rapid lysis of tumor cells, with high intracellular levels of potassium, phosphate, and nucleic acids. Nucleic acids are broken down into uric acid, which, along with phosphate crystals, precipitate in the renal tubules, leading to acute kidney injury [3].

Intravenous hydration is a cornerstone of treatment to provide renal perfusion and increase urine output, which helps eliminate uric acid and phosphate [3]. Hypouricemic agents are also indicated. Allopurinol decreases uric acid formation, while rasburicase degrades uric acid to the more water soluble allantoin [4]. Rasburicase is more effective in normalizing the serum uric acid levels as compared to allopurinol [5,6].

TLS is commonly seen in the treatment of hematopoietic malignancies and prophylaxis with rasburicase for high risk patients is recommended [7]. Isolated reports of TLS in solid tumors have been described in the literature with three involving ovarian neoplasms. This includes a case report of TLS during induction chemotherapy with carboplatin and cyclophosphamide in a patient with serous ovarian adenocarcinoma [8]. A second case report describes TLS in a patient receiving salvage topotecan for recurrent serous ovarian cancer [9]. In both cases, the patients were treated using intravenous hydration and allopurinol. The first case referenced additionally utilized parenteral bicarbonate and furosemide in the patient's management [8,9]. A third case report documents fatal TLS after receiving carboplatin and paclitaxel chemotherapy for recurrent ovarian cancer with documented intravascular spread [10]. To our knowledge, TLS has not been reported in the literature as a complication of the treatment of ovarian cancer with docetaxel. Several isolated cases of TLS after the use of docetaxel for other solid tumors (including esophageal, lung and prostate) are reported [11-13].

While TLS remains a rare complication of the treatment of ovarian cancer, prompt recognition and treatment are essential to recovery and the avoidance of permanent renal injury. In the case presented, appropriate treatment with hydration and hypouricemic agents quickly restored normal laboratory parameters and renal function. No lasting sequelae from TLS occurred in this patient. Given her recurrent disease and excellent clinical response in CA 125, a second dose of docetaxel was considered. After discussion with the patient and her family, another cycle was administered along with prophylactic allopurinol 300 mg orally once. She tolerated the course without complication. Although rare in the field of gynecologic oncology, practitioners should be aware of the clinical and laboratory manifestations of TLS so that patients may benefit from the timely initiation of treatment.

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