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CASE REPORT

ACUTE RENAL FAILURE FROM SPONTANEOUS ACUTE TUMOR LYSIS SYNDROME: A CASE REPORT AND REVIEW

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ABSTRACT

Acute tumor lysis syndrome (ATLS), a condition which results from a rapid destruction of tumor cells with massive release of cellular breakdown products, has been well described. However, only a few cases of spontaneous ATLS have been reported in the literature. Acute renal failure (ARF) from spontaneous ATLS has been reported only in three patients who were diagnosed to have Burkitt's lymphoma, adenocarcinoma, and acute myeloid leukemia. We report a similar case of a patient with non-Hodgkin's lymphoma, who developed ARF from spontaneous ATLS. ARF can complicate the clinical course of spontaneous ATLS. Since only one patient survived, patients who develop ARF from spontaneous ATLS have a poor outcome. This paper illustrates the need to anticipate the development of ARF, despite aggressive therapy, in a patient with spontaneous ATLS. Prospective studies on renal function prior to and during therapy are required in order to develop a clinical profile reliably detecting patients at risk for developing renal failure and subsequent complication.

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Key Words: Acute tumor lysis syndrome; Lymphoma; Renal failure

INTRODUCTION

Acute tumor lysis syndrome (ATLS) is characterized by hyperuricemia, hypocalcemia, hyperkalemia, hyperphosphatemia, lactic acidosis, and azotemia. Spontaneous development of ATLS, prior to initiation of treatment, was reported in only five patients (1–5). Four of those patients, in Table 1, developed acute renal failure (ARF) (1–4). A MEDLINE search (1966–January 2001) leads one to believe this is the second reported case of ARF from spontaneous ATLS due to non-Hodgkin's lymphoma (NHL).

Case: A 71-year-old woman presented with a two-week history of anorexia, weight loss and painless jaundice. Physical examination revealed blood pressure 100/70 mmHg, pulse 85/min, respiration rate 18/min, temperature 37°C, icteric skin, bilateral basal rales, ascites, splenomegaly, and pedal edema. Chest radiograph (CXR) showed enlarged mediastinum. Abdominal computed tomography revealed a large abdominal mass involving the pancreas and the inferior vena cava, ascites, and splenomegaly. Fine needle aspiration biopsy of the mass showed large cell NHL. Bone marrow biopsy showed infiltration with the malignant cells.

Treatment included hydration with intravenous fluids, allopurinol, cyclophosphamide, and corticosteroids. Laboratory values are summarized in Table 2. On day five, the patient developed oliguric ARF, the creatinine level had increased from 1.3 to 4.3 mg/dl (normal range 0.5–1.5), phosphorus value had increased from 4.6 to 13.3 (normal range 2.5–4.5), and potassium level had increased from 3.9 to 7.1 mEq/l (normal range 3.5–5). Calcium level had decreased from 8.5 to 6.5 mg/dl (normal range 8.5–10.5) and uric acid level, from 8 to 33.7 mg/dl (normal range 2.5–8). The lactic dehydrogenase (LDH) level was 20270 U/l (normal range 60–250). The lactic acid level was 18 mmol/l (normal range 0.6–2.2). Hemodialysis was initiated for treatment of ARF. On day six, the patient developed respiratory failure, which required ventilatory support. CXR showed pulmonary edema. Arterial blood gas analysis, while the patient was receiving fraction of inspired oxygen (FiO_2)

Table 1. Cases of ARF from Spontaneous ATLS

References	Age (*y*)	Gender	Tumor	Outcome
Cittenden et al. (1)	50	Male	Adenocarcinoma	died
Jasek et al. (2)	83	Female	Burkitt's lymphoma/leukemia	died
Lotfi et al. (3)	63	Female	Acute myeloid leukemia	survived
Feld et al. (4)	72	Male	Adenocarcinoma	died
Our patient	71	Female	Non-Hodgkin's lymphoma	died

Table 2. Laboratory Values of Our Patient

Laboratory Data	Day One	Day Five	Normal Range
Creatinine	1.3	4.3	0.3–1.5 mg/dl
Phosphorous	4.6	13.3	2.5–4.5 mg/dl
Potassium	3.9	7.1	3.5–5 mEq/l
Calcium	8.5	6.5	8.0–10.5 mg/dl
Uric acid	8.5	33.7	7.5–8 mg/dl
Lactic dehydrogenase	120	20270	60–250 U/l
Lactic acid	1.5	18	0.6–2.2 mmol/l

of 100%, revealed pH value 6.828 (normal range 7.35–7.45); PCO_2, 36 torr (normal range 35–45); and PO_2, 126 torr (normal range 80–100). The patient developed hypotension. Therapy with vasopressors was initiated. On day seven, the hemoglobin level had decreased from 10 to 5.3 g/dl (normal range 12–16), and the platelet count had decreased from 75 to 30 k/ul (normal range 180–400). Prothrombin time had increased from 19 to 56 s (normal range 9–11.5), and partial thromboplastin time had increased from 37 to greater than 100 s (normal range 23–33). D-Dimer level was more than 5 ug/ml (normal range 0.0–0.5), and the fibrinogen level was 201 mg/dl (normal range 200–400).

Fresh frozen plasma and platelets were transfused. Permission for the insertion of a pulmonary artery catheter was denied. On day eight, the patient had a cardiac arrest and expired. A request for autopsy was not granted.

DISCUSSION

Spontaneous ATLS was first reported in 1977 by Crittenden et al. in a 50-year-old man with widespread adenocarcinoma of gastrointestinal tract origin, who developed hyperuricemic ARF (1). The patient died 12 days after presentation (1). A following case was reported by Jasek et al. of an 83-year-old woman, diagnosed with Burkitt's leukemia/lymphoma, who developed ARF from spontaneous ATLS (2). Treatment was only supportive and she expired shortly after diagnosis (2). Lotfi et al. reported a 63-year-old woman who was diagnosed to have acute myeloid leukemia, developing ARF from spontaneous ATLS. The patient survived after institution of allopurinol and chemotherapy (3). Finally, Feld et al. reported a 72-year-old man with adenocarcinoma who developed spontaneous ATLS and ARF (4). The patient refused treatment and died eight days after presentation (4).

ATLS occurs spontaneously (1–5) or as a complication of rapid lysis of tumors following surgery, chemo-, radio-, immuno- (7,8), hormonal (9) or

intrathecal therapy (10), transcatheter chemoembolization of hepatocellular carcinoma (11), and after a sustained episode of fever in a patient with high grade lymphoblastic lymphoma (12). ATLS may reoccur in the same patient (5,13). Feld et al. hypothesized that the most likely mechanism for the development of spontaneous ATLS in a solid tumor is rapid tumor necrosis or cell turnover as opposed to a treatment effect (4). Kalemkerian et al. reviewed the risk factors associated with the development of ATLS and the outcome of 25 patients with small cell carcinoma and solid tumors (6). Risk factors include pretreatment renal insufficiency, elevated serum LDH, and hyperuricemia (6). Nine of 25 (36%) patients died during the treatment of ATLS (6). ATLS can result in oliguric ARF. Hyperuricemia and hyperphosphatemia contribute to the development of oliguric ARF in patients with ATLS (14).

ATLS may be avoided and treated with allopurinol and intensive hydration. Alkalinization therapy is not recommended because it is thought that changes in pH may cause hypocalcemia by favoring the complexation of calcium and phosphate (15). Hyperkalemia may be treated with oral sodium–potassium exchange resin, combined insulin–glucose therapy or hemodialysis. Hemodialysis is often necessary to control life-threatening hyperkalemia or volume overload, while tumor burden is controlled with cytoreductive therapy (16). Furthermore Pichette et al. reported the use of high dialysate flow rate continuous arteriovenous hemodialysis (CAVHD) of 4 l/h and found CAVHD to be a more potent form of treatment than conventional intermittent hemodialysis (17).

In a following paper, Agha-Razii et al. concluded that the use of continuous veno-venous hemodiafiltration (CVVHDF) flow rate of 2.5 l/h can achieve excellent solute clearances and metabolic control (18), and that CVVHDF is an ideal treatment for ARF in tumor lysis syndrome (18). Our patient and three of the four reported patients with ARF from spontaneous ATLS (Table 1) expired (1,2,4), which reflects a poor impact of ARF on prognosis in patients with spontaneous ATLS.

CONCLUSION

Spontaneous ATLS can result in ARF. The natural history and mortality of ATLS are still not clearly defined. Although ATLS occurs infrequently, early recognition and management by a team approach in the intensive care unit may improve short-term prognosis in susceptible patients. Our patient and other reported cases illustrate that ARF can complicate the clinical course of spontaneous ATLS. Patients who develop ARF from spontaneous ATLS have a poor outcome.

Prospective studies on renal function prior to and during therapy are required in order to develop reliable clinical profiles to detect patients at risk for developing renal failure and subsequent complication.

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