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Case Presentation
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Solid Tumor Spontaneus Tumor Lysis Syndrome: Two Cases

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Abstract

Tumour lysis syndrome (TLS) is a life-threatening oncological emergency. It commonly occurs in haematological malignancies, it rarely develops as a result of solid tumours. It usually develops after treatment but it may rarely develop spontaneously in solid tumours, spontaneous TLS is much less common. In this article we discussed two cases of TLS, spontaneously developing in colon adenocarcinoma and lung cancer, in the context of literature.

Keywords: Tumour lysis syndrome; Solid tumour; Acute kidney damage

Introduction

Tumor lysis syndrome (TLS) is a life-threatening oncological emergency. TLS is a metabolic disorder associated with treatment or spontaneous death from cell death. The result of cell disruption, a lot of uric acid, potassium and phosphate are released to the circulation. As a result of the precipitation of calcium-phosphate crystals hypocalcemia occurs. This clinically affects many organs as acute kidney damage, cardiac arrhythmias and seizures. The most commonly used diagnostic laboratory (two or more) and clinical (one or more) criteria for TLS are those proposed by Cairo-Bishop (Table 1)[1-2].

Spontaneous TLS is a rarely seen oncological emergency. Spontaneous TLS occurs in the absence of chemotherapy. Although spontaneous TLS is more common in haematological malignancies, it rarely develops in solid tumours. Small cell lung cancer, advance gastric cancer, breast cancer, pancreatic cancer, *hepatocellular carcinoma,* colon *carcinoma and ovarian cancer* from spontaneously TLS solid tumours have been described [3-8].

Case Presentation

Case 1

A 70-year-old woman is applied to our clinic with complaints of anorexia, nausea and vomiting. Follow-up without treatment with lung cancer for 4 years. On physical examination blood pressure was 110/60 mmHg, body temperature was 36.5°C, pulse was 78/min, bilateral crepitant rales in her lungs. Before treatment and after treatment laboratory examinations are given in Table 2. Blood gas, metabolic acidosis were present (pH 7,2, PCO_2 28 mmHg, HCO3 14 mEq/L), anion gap was found to be 19 mEq /L. The lesion (6 cm x 2 cm) with calcific foci advancing to the right lobe of the lower lobe was detected on thorax tomography; Positron emission tomography right lung and bilateral lymph node involvement were detected. Spontaneous TLS was diagnosed and treatment was started with hydration, sodium bicarbonate, allopurinol, anti-potassium treatment (potassium-restricted diet, insulin administered with glucose, salbutamol, sodium bicarbonate, kayexelate). The patient was treated successfully and discharged.

Case 2

A 56-year-old woman with conscious disability was admitted to the hospital with an acute renal failure. Approximately 3 years ago, she was operated with diagnosis of colon adenocarcinoma. Chemotherapy had been offered but the patient did not accept. On physical examination, she was conscious, non-cooperative, blood pressure 100/50 mmHg, body temperature 37.4°C, pulse 120/min. Before treatment and after treatment laboratory examinations are given in Table 2. Blood gas, metabolic acidosis were (pH 7,12 PCO_2. 22 mmHg, HCO_3 10 mEq/L), anion gap was found to be 45 mEq /L. The right liver lobe mass (9x8.6 cm), midline incision in situ mass (2.2 cm x

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Table 1: Tumor lysis syndrome diagnostic criteria.

Having 2 or more compared to laboratory criteria	Having one or more clinically
Uric acid ≥ 8.0 mg/dL or 25% increase from baseline	Creatinine ≥ 1.5 times the upper limit of normal
Potassium ≥ 6.0 mmol/L or 25% increase from baseline	Cardiac arrhythmia/sudden death
Phosphorus ≥ 4.5 mg/dL or 25% increase from baseline	New-onset seizure
Calcium ≤ 7.0 mg/dL or 25% decrease from baseline	

Table 2: Laboratory findings.

	Reference range	Case 1 (before treatment)	Case 1 (after treatment)	Case 2 (before treatment)	Case 2 (after treatment)
glucose (mg/dl)	82-126	127	110	145	124
blood urea nitrogen (mg/dl)	0-20	82	18	264	16
Serum creatinine (mg/dl)	0,5-0,95	3,3	1,0	22	0,7
calcium (mg/dl)	8,8-10,2	9,6	8,6	6,2	7,2
phosphor (mg/dl)	3,5-4,5	6,9	3,1	14	2,6
uric acid (mg/dl)	1-5,7	12	6,3	20	2,4
potassium (mmol/L)	3,5-5,5	7,2	5,3	11,6	3,7
Lactate dehydrogenase (IU/L)	135-214	570	220	1238	377

1.4 cm) were observed on tomography. During thorax tomography, malignant mass causing bone destruction (6 cm x 2 cm), solid nodule on left hilus were found. In the positron emission tomography test, the involvement of the activity was detected in the regions described in the tomography. Two sessions of hemodialysis were performed with acute renal failure and uremic encephalopathy. In addition, hydration, sodium bicarbonate, calcium gluconate, allopurinol and anti-potassium treatment were given. The patient was successfully treated.

Discussion

TLS is caused by the circulatory release of intracellular electrolytes and metabolites, which is the result of cytotoxic chemotherapy or rarely spontaneous disruption of multiple tumour cells. TLS clinical manifestations may occur as a result of released potassium, phosphorus, uric acid to the circulation, increase in potassium, phosphorus, uric acid and decrease in calcium. Hyperpotassium may result in paraesthesia, paralysis, gastrointestinal symptoms, arrhythmia, convulsions and sudden death. Hyperuricemia, nausea, vomiting, loss of appetite, itching and laryngitis may occur. Hyperphosphatemia may result in nausea, vomiting, diarrhea and convulsions. Calcium phosphate build-up results in itching, joint pain and hypocalcaemia. Hypocalcaemia may result in paraesthesia, tetanus, carpal and pedal spasm, bronchospasm, seizures, anxiety, delirium, ventricular arrhythmia, heart block, hypotension and cardiac arrest. So, TLS is a serious condition that causes 20% to 50% of patients to die if they cannot be diagnosed or diagnosed late. TLS mortality in solid tumours is higher than hematological malignancies [1,9].

TLS is a very rare complication in solid tumours. TLS is rarely reported in lung cancer and colon cancer, but spontaneous TLS is much less common. The incidence of TLS in solid tumours was found to be less than 0.3%. Risk factors for TLS in solid tumours are high tumour volume, use of combination chemotherapy drugs, dehydration. Metastases, liver metastases and rapidly proliferating tumour, exposure to nephrotoxic drugs or uric acid excretion inhibiting drugs, extrinsic compression of the urinary tract by the tumour, infection or urinary obstruction, high white cure cell (>50,000 cells/μL) , classically high lactate dehydrogenase and uric acid levels have previously been defined as renal dysfunction [10-11]. In our two cases, high tumour volume and high lactate dehydrogenase were present. We had an additional metastasis in one of our case.

TLS and spontaneous TLS are rarely seen in small cell lung cancer. There are publications in the form of case presentations in the literature. Five cases have been published about small cell lung cancer. The sex of the patients, 3 male, 2 female, as a laboratory, hyperpotassium and hyperphosphatemia in 5 patients, hyperuricemia in two patients, hypocalcaemia in one patient, serum creatinine increase in three cases have been identified [12-15].

The ideal treatment approach for minimizing TLS morbidity and mortality is to anticipate cases and take protective measures. One of the main pillars of TLS protection in patients with cancer is to give adequate fluid to the patient. As a result of hydration, uric acid, phosphate and potassium levels drop dilutionally, renal blood flow, glomerular filtration rate and urine volume increase. Intravenous fluid hydration, xanthine oxidase inhibitor (allopurinol and febuxostat) or rasbikuraz are both involved in the prevention and treatment of hyperuricemia complications for adults. The alkalization of urine to increase the solubility of uric acid is no longer recommended as a routine treatment since it causes simultaneous precipitation of calcium phosphate. Calcium gluconate is recommended in the treatment of symptomatic hypocalcaemia. In patients with symptomatic hypocalcemia who do not respond to treatment, dialysis is indicated. Application of anti-potassium treatment in case of hyperpotassium if there is cardiac involvement in the electrocardiogram, calcium gluconate is given, intravenous infusion of insulin and glucose, salbutamol nebula, kayexelate and acidosis, if present sodium bicarbonate infusion are given. If it is resistant to therapy, it is haemodialysis indications. Treatment for hyperphosphatemia is difficult. Phosphate binders are given in the treatment but they are less effective. The best treatment of hyperphosphatemia is renal replacement therapy. Renal replacement therapy, severe acidosis, severe hyperuricemia, excessive volume loading unresponsive to diuretic therapy and severe acute renal damage are also indicated [16-18].

As a result, spontaneous TLS rarely develops in solid tumours, especially in small cell lung cancer and colon cancer. Risk factors for TLS need to be assessed in patients with solid tumours. Closely monitoring the clinical and laboratory values and taking the necessary precautions during risky outcomes will reduce TLS more frequently in cancer cases.

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