CASE REPORT

Radiotherapy-Related Tumour Lysis Syndrome in a Patient with Metastatic Adenocarcinoma of Unknown Origin

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ABSTRACT

Tumour lysis syndrome (TLS) is a rare but serious complication of cancer treatment. It is generally seen in patients with high tumour load or chemosensitive tumour after chemotherapy and is more common with haematological malignancies like leukaemia and lymphoma when compared to solid tumours. TLS occurring after radiotherapy (RT) in patients with solid tumours is very rare. We aimed to present TLS seen after RT for a vertebral tumoral mass in a patient with metastatic adenocarcinoma of unknown origin. A 78-year-old woman, who was diagnosed with adenocarcinoma of unknown origin, was hospitalized to undergo palliative RT for the vertebral mass. On the 1st day, 4 mg q6hour perioral dexamethasone was started. 300 cGy per session RT started on the 2nd day of hospitalization. After the fifth session of RT (after a total dose of 15 Gy), she developed TLS complicated with acute kidney injury requiring renal replacement therapy and she was successfully treated by haemodialysis. Close monitoring, even in patients with low risk for TLS and early administration of preventive modalities should be kept in mind.

Keywords: Radiotherapy, solid tumour, tumour lysis syndrome.

INTRODUCTION

Tumour lysis syndrome (TLS) is a rare but serious complication of cancer treatment, which occurs secondary to massive tumour cell lysis resulting in the release of large amounts of intracellular potassium, phosphate, and uric acid in to systemic circulation (1). TLS occurring after RT in patients with solid tumours is very rare (2). We aimed to present TLS seen after RT for a vertebral tumoral mass in a patient with metastatic adenocarcinoma of unknown origin.

CASE REPORT

A 78-year-old woman who had hypertension admitted with a complaint of fatigue. She had fatigue and severe back pain for one month. On physical examination, her general condition was good. She was afebrile; blood pressure was 120/80 mmHg, pulse was 78/min. Hepatomegaly was detected on abdominal examination. The remaining examination was unremarkable. Her laboratory findings were as follows: haemoglobin: 13.8 g/dL, leukocyte: 9200/μL, platelet count: 210×10³/μL, serum urea: 35 mg/dL, creatinine: 0,6 mg/dL, Na: 145 mmol/L, K: 4,2 mmol/L, uric acid: 5,1 mg/dL, calcium: 9,1 mg/dL, phosphorous: 3,9 mg/dL, LDH: 620 IU/L, albumin: 3.6 mg/dL, ALT: 51 IU/L, AST: 45 IU/L, ALP: 158 IU/L, GGT: 286 IU/L. Her hepatitis serology and autoimmune markers were negative. Thoracoabdominal computed tomography showed tumoral mass in liver, intraabdominal lymph nodes and also an expansile mass lesion involving the C6 vertebrae. The biopsy of hepatic mass revealed metastatic adenocarcinoma. As the primary source of the metastasis could not be defined, the patient diagnosed metastatic adenocarcinoma of unknown origin. The patient was hospitalized to undergo

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palliative RT for the vertebral mass. On the 1st day, 4 mg q6hour per-oral dexamethasone was started. Three hundred (300) cGy per session RT started on the 2nd day of hospitalization. After the fifth session of RT (after a total dose of 15 Gy), her laboratory findings were as follows: serum urea: 407 mg/dL, creatinine: 5.6 mg/dL, Na: 134 mmol/L, K: 7.6 mmol/L, uric acid: 13.2 mg/dL, calcium: 9.3 mg/dL, phosphorus: 10.8 mg/dL, albumin: 3.7 g/dL, LDH: 1520 IU/L, HCO_3: 16 mmol/L. RT was stopped and the patient underwent haemodialysis treatment after intravenous infusion of calcium gluconate and dextrose solution with insulin because her electrocardiography revealed peaked T waves. Intravenous saline infusion was administered before and during dialysis. After haemodialysis, her serum urea and creatinine levels were 197 mg/dL and 2.39 mg/dL, respectively and serum levels of potassium and bicarbonate were 5.1 mmol/L and 16 mmol/L, respectively. Intravenous saline infusion was continued and allopurinol was added. Her urine output was approximately 100–150 cc/h. Her laboratory findings were normalized gradually in three days without need for further dialysis. She completed 10 sessions of RT without any other complications.

DISCUSSION

TLS is a rare but serious complication of cancer treatment. The Cairo-Bishop criteria were defined for diagnosis and classification of TLS. Diagnosis of laboratory TLS requires that two or more of the following laboratory abnormalities occur within three days before or up to seven days after the initiation of chemotherapy (CT): hyperuricemia, hyperkalaemia, hyperphosphatemia and hypocalcaemia. Clinical TLS is diagnosed when laboratory findings were accompanied by an increased creatinine level, seizures, cardiac arrhythmia or death (3). In accordance with Cairo-Bishop criteria, TLS was the diagnosis in our patient represented with acute kidney injury.

High tumour burden, chemosensitivity of the tumour, advanced age, baseline renal dysfunction, oliguria, dehydration, exposure to nephrotoxic agents, severe leucocytosis, baseline elevation in serum uric acid, phosphorus and LDH levels are associated with a high risk of TLS (1, 3). The incidence with solid tumours consists only 1% of the TLS. The risk of TLS is considerably higher in some solid tumours such as neuroblastoma, germ cell tumours and small cell lung cancer and in most of them TLS is related to CT (4). The first case with TLS related to solid tumour after RT was reported by Tomlinson *et al*. The syndrome was occurred after treatment of metastatic medulloblastoma by radiotherapy with a dosage of 3 Gy (5). In our case, a total dose of 15 Gy lead to TLS. In a review including the cases between 1950 and 2014, a total of 121 patients with TLS secondary to solid tumours were reported. Eight of them occurred spontaneously, three detected after RT and one was after both RT and CT. The remaining cases were treated by CT. Spontaneous TLS was related to adenocarcinoma with unknown origin in two of the reported cases. The authors suggested that liver metastasis may be related to higher risk of TLS. This paper consists of a heterogeneous group of patients who had variable types of tumours and clinical and laboratory findings (6). The only risk factor was high serum LDH levels in our patient. Liver metastasis was mentioned as a risk factor for TLS based on observational data, but it remains speculative. In our case, the dosage of RT is relatively low when compared to previous studies.

TLS is a reversible complication especially when diagnosed early. Treatment consists of aggressive hydration, forced diuresis, allopurinol and rasburicase. In patients unresponsive to medical therapy, renal replacement therapy (RRT) is the next step in the treatment. The indications of RRT in TLS are similar to those in patients with other types of acute kidney injury, although somewhat lower thresholds are used for TLS, because there is a high potential risk for rapid potassium accumulation (1, 7). If RRT is initiated early, renal survival is excellent (8). When type of the tumour and type of the treatment were taken into account, our patient with relatively low risk for TLS developed the syndrome complicated with AKI requiring RRT and she was successfully treated by haemodialysis.

In conclusion, the relatively low incidence of TLS in solid tumours may be related to low number of risk factors as well as under-diagnosis of the syndrome. Close monitoring, even in patients with low risk for TLS and early administration of preventive modalities, including aggressive hydration first, should be kept in mind.

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