

# Acute spontaneous tumour lysis syndrome in a patient with non-Hodgkin's lymphoma: a rare case report

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Metabolic disorders such as hyperuricaemia and hyperuricaemic renal failure, hyperkalaemia and hyperphosphataemia in hematological malignancies with high tumour burden and high turnover and rarely in solid malignancies are defined as acute tumour lysis syndrome. It frequently occurs after treatment (chemotherapy / radiotherapy) and rarely occurs before treatment. In this case, it is called spontaneous acute tumour lysis syndrome. We aimed to remind this rare clinical entity with high mortality risk by presenting a patient with non-Hodgkin's lymphoma and acute spontaneous tumour lysis syndrome.

**Key Words:** Acute spontaneous tumour lysis syndrome, non-Hodgkin lymphoma, renal failure

## INTRODUCTION

Tumour lysis syndrome (TLS) is a serious complication of cancer treatment. Acute TLS, which is a clinical picture with life-threatening metabolic disorders and caused by rapid destruction of tumour cells, occurs in haematological malignancies with large tumour mass (acute lymphocytic leukaemia, non-Hodgkin's lymphoma) and rarely in solid malignancies. Metabolic disorders are characterized by hyperuricaemia, acute renal failure, hyperkalaemia, hyperphosphataemia and hypocalcaemia. Although TLS is most commonly seen in chemosensitive tumours in the first days of chemotherapy or radiotherapy, it may rarely develop spontaneously in the tumours with high proliferation.

Acute spontaneous tumour lysis before chemotherapy is rarely encountered in acute leukaemia and high-grade non-Hodgkin's lymphoma which shows high-grade mitotic activity<sup>[1-3]</sup>. There is a positive correlation of the development of acute renal failure (ARF) with tumor size, and elevated serum LDH and uric acid levels<sup>[2]</sup>.

## CASE REPORT

An 80-year-old male patient was admitted to the general internal medicine clinic due to a swelling in his neck. It was found out that the swelling in his neck had been there for about 6 months, he had lost 7-8 kg within the last 6 months, and his night sweats had increased for the last one month. Bilaterally palpable cervical and supraclavicular lymph nodes were detected on physical examination. His past medical history revealed that the patient

underwent subtotal gastrectomy in 2006 due to gastric adenocarcinoma (T<sub>1</sub>N<sub>0</sub>M<sub>0</sub>). He did not receive adjuvant chemotherapy and radiotherapy since it was early stage disease. The supraclavicular lymph node of the patient who was hospitalized in our clinic with the pre-diagnosis of lymphoma was excised. Histopathological diagnosis was CD20 positive diffuse large cell non-Hodgkin lymphoma – a form of high-grade lymphoma. Staging positron emission tomography (FDG-18 PET / CT) showed multiple lymph nodes in the supradiaphragmatic and infradiaphragmatic lymphatic stations with conglomerated-mass appearance and intense / hyperintense metabolic changes, and also in the spleen, and foci in the left proximal humerus, left eighth costa and left part of sacrum in the skeleton.

Spontaneous tumour lysis syndrome was suspected in the patient who had hyperuricaemia, azotaemia, and hyperphosphataemia in blood and who developed oliguric acute renal failure rapidly on the third day of admission. (Table 1) Intravenous fluid replacement, urine alkalization, forced diuresis and allopurinol therapy were initiated immediately. Despite the treatment, severe metabolic acidosis developed and the patient received hemodialysis treatment. However, the clinical picture of acute renal failure worsened and mental confusion developed rapidly, and the patient died in the intensive care unit on the fifth day of treatment.

## DISCUSSION

In haematologic and solid tumours, metabolic disorders such as hyperuricaemia and hyperuricaemic renal failure, hyperkalaemia, hyperphosphataemia with hypocalcaemia

are defined as acute tumour lysis syndrome. Spontaneous occurrence of this syndrome before treatment is rarely observed while it frequently occurs after treatment. Known risk factors for tumor lysis syndrome include elevated LDH ( lactic dehydrogenase) level which is twice more than normal, solid tumors with high proliferation ability, total tumour volume larger than 10 cm, high tumour burden sensitive to chemotherapy or radiotherapy and high white count acute leukaemia (WBC>100,000 cell/µl).

Spontaneous acute tumour lysis syndrome developing without any precipitating factor was reported in the literature for Burkitt's lymphoma [3], diffuse large B cell lymphoma and Richter's syndrome [4], anaplastic large T-cell lymphoma [5], acute myeloid leukaemia [6] acute lymphoblastic leukaemia [7], metastatic germ cell tumour [8], lung adenocarcinoma [9], squamous cell carcinoma [10], breast cancer [11], gastric cancer [12], cholangiocarcinoma [13], myelofibrosis [14], multiple myeloma [15] and myelodysplastic syndrome [16].

What is laid emphasis on the pathogenesis of tumour lysis syndrome is the release of intracellular metabolites (nucleic acid, potassium, phosphorus, uric acid) and cytokines after rapid tumour necrosis. These metabolites cause hyperuricaemia, hyperphosphataemia and hyperkalaemia in blood. The precipitation of calcium-phosphate crystals and uric acid crystals, which develop secondarily to hyperphosphataemia, in the renal tubules causes obstructive nephropathy and renal failure. This metabolic state may cause cardiac dysrhythmias, mental confusion, seizure and sudden death other than acute renal failure. Meanwhile, released cytokines may cause systemic inflammation and multiple organ failure.

Appropriate intravenous fluid replacement, urine alkalization, correction of metabolic acidosis and electrolyte imbalance, and decreasing uric acid synthesis form the basis of treatment in the patients with acute tumor lysis. Also, rasburicase, a recombinant urate oxidase inhibitor, was shown to reduce mortality and morbidity in acute tumour lysis [17]. Haemodialysis can be performed in the patients with life-threatening hyperkalaemia or persistent oliguria despite aggressive intravenous hydration.

**CONCLUSION**

Acute spontaneous tumor lysis syndrome is a rare metabolic emergency with high mortality risk. For early recognition and prevention of this syndrome, it should be kept in mind that it may occur in the patients with high risk before chemotherapy treatment and may have a mortal course.

References

1. Lee GR, Bithell TC, Foerster J, Athens JW, Lukens JN .Wyntrobe's Clinical Hematology. 9th ed. Lea&Febiger, Philadelphia, London.1993.

**Table 1. Biochemical and hematological parameters of our patient**

Parameters	1st day	3th day	5th day
Urea (mg/dl)	83	112	182
Creatinine(mg/dl)	1	1.6	3.1
Uric acid (mg/dl)	6.5	8.3	9.3
Potassium (mEq/L)	4.9	4.8	5.8
Calcium (mg/dl)	9.8	10.3	10.2
Phosphorus(mg/dl)	3.5	5.4	6.1
LDH(IU/L)	737	1100	1831
ALP (IU/L)	250	281	584
Haemoglobin (gr/dl)	8.3	10.7	10.5
Haematocrit (%)	26.4	32.8	32.2
WBC (cell/µl)	12100	16100	18600
Platelets (cell/µl)	121000	111000	53000

LDH : Lactic dehydrogenase

ALP : Alkaline phosphatase

WBC : White blood cells

2. Cohen LF et al. Acute tumor lysis syndrome. A review of 37 patients with Burkitt's lymphoma. Am J Med.1980;68:486-91.

3. Jasek A M et al. Acute spontaneous tumor lysis syndrome. Am J Hematol. 19- 94;49:488-92.

4. Tufan A, Unal N, Koca E, Onal I, Aksu S, Haznedaroglu I. Spontaneous tumor lysis syndrome in a patient with diffuse large B cell lymphoma and Richter syndrome. Ann Hematol.2006;/85:/183-4.

5. Hsu HH, Huang CC. Acute spontaneous tumor lysis in anaplastic largeT-cell lymphoma presenting with hyperuricemic acute renal failure. Int J Hematol. 2004;/79:/48-51.

6. Lotfi M, Brandwein JM. Spontaneous acute tumor lysis syndrome in acute myeloid leukemia ? A single case report with discussion of the literature. Leuk-Lymphoma. 1998;/29:/625-8.

7. Akoz A.G et.al.Unusual case of spontaneous acute tumor lysis syndrome associated with acute lymphoblastic leukemia: A case report and review of the literature. Acta Oncologica. 2007; 46: 1190-1192.

8. D'Alessandro V, Greco A, Clemente C, Sperandeo M, De Cata A, Di Micco C, et al. Severe spontaneous acute tumor lysis syndrome and

- hypoglycemia in patient with germ cell tumor. *Australas Med J*. 2013 Mar 31;6(3):168-71.
9. Feld J, Mehta H, Burkes RL. Acute spontaneous tumor lysis syndrome in adenocarcinoma of the lung. *Am J Clin Oncol*. 2000;23:491-3.
  10. Shenoy C. Acute spontaneous tumor lysis syndrome in a patient with squamous cell carcinoma of the lung. *QJM*. 2009 Jan;102(1):71-3.
  11. Sklarin NT, Markham M. Spontaneous recurrent tumor lysis syndrome in breast cancer. *Am J Clin Oncol*. 1995;18:71-3.
  12. Goyal H, Sawhney H, Bekara S, Singla U. Spontaneous acute tumour lysis syndrome in gastric adenocarcinoma : a case report and literature review. *J Gastrointest Cancer*. 2014 Dec;45 Suppl1:208-11.
  13. Ali AM, Barbaryan A, Zdunek T, Khan M, Voore P, Mirrakhimov AE. Spontaneous tumor lysis syndrome in a patient with cholangiocarcinoma. *J Gastrointest Oncol*. 2014 Apr;5(2):E46-9.
  14. Sile S, Wall BM. Acute renal failure secondary to spontaneous acute tumor lysis syndrome in myelofibrosis. *Am J Kidney Dis*. 2001;38:1-4.
  15. Saravu K, Kumar S, Shastry AB, Kurien A, Prabhu R, Kumar R. Spontaneous tumour lysis syndrome in a case of multiple myeloma - A rare occurrence. *Australas Med J*. 2013 Mar 31;6(3):168-71.
  16. Feng Y, Jiang T, Wang L. Hyperuricemia and acute kidney injury secondary to spontaneous tumor lysis syndrome in low risk myelodysplastic syndrome. *BMC Nephrol*. 2014 Oct 11;15:164.
  17. Hagino T. Tumor lysis syndrome. *Gan To Kagaku Ryoho*. 2010 Jun;37(6):984-8.