CASE REPORT

Spontaneous Tumor Lysis Syndrome in an Infant: A Case Report

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Received: 13 August 2014 / Accepted: 19 December 2014 / Published online: 14 February 2015
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Abstract Tumor lysis syndrome has been observed in patients with malignancies with high cellular burden and high cell turnover, tumor sensitive to therapy, especially after initiating medical treatment. It very rarely occurs spontaneously. The case described here is of 6 months male infant who presented with fever since 1 month and loose stools associated with blood since 15 days. The laboratory investigations showed lactate dehydrogenase (LDH) of 6,192 IU/L and serum uric acid 18.2 mg/dl along with pancytopenia. The infant presented with electrolyte abnormalities and renal failure.

Keywords Spontaneous tumor lysis syndrome · Hyperuricemia · AML

Introduction

Malignancies with high cellular burden and high cell turnover can lead to tumor lysis syndrome (TLS) after various non surgical treatment modalities. It is a group of electrolyte instability presenting with a scenario of hyperuricemia, hyperphosphatemia, hyperkalemia, and hypocalcemia [1]. Spontaneous TLS—that is, TLS occurring in the absence of therapeutic intervention (including chemo radiation and immuno-therapy) is defined by the

presence of electrolyte abnormalities and renal failure. It is a rare occurrence [2]. In this paper we describe a case of pretreatment spontaneous TLS in a 6 months old infant, later diagnosed to be suffering from acute myeloid leukemia (AML).

Case Report

A 6 months old male infant was brought with complaints of fever since 1 month and loose stools associated with blood since 15 days. Fever was moderate grade, not associated with chills and rigors. Loose stools were watery to semisolid in consistency and black in color. Mother gave history of reduced cry and, decreased activity, reduced feeding and decreased urine output. Vomiting was absent and there was no yellowish discoloration of skin. No significant past or family history was noted for tuberculosis, seizures, bleeding disorders or malignancy in the family. The child was born at term by normal vaginal delivery, cried soon after birth, has been exclusively breast fed, attained milestones appropriate for age and has been vaccinated to date.

On Examination: poorly nourished infant had weak cry and decreased activity. The height was 60 cm, weight was 5.46 kg and head circumference was 40.5 cm. There was pallor but no cyanosis, icterus, lymphadenopathy or edema. Vitals were temperature of 101 °C, pulse rate was 160 bpm, blood pressure 88/60 mmHg and respiratory rate 62 per minute. Per abdomen: soft, non tender, hepatosplenomegaly was present, liver span was 7, 4 cm was below right costal margin. Cardiovascular system: S1 S2 heard. Ejection systolic murmur heard in aortic area. Respiratory system: trachea central. NVBS bilaterally heard. No added sounds.

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Investigations

Blood count on admission revealed Hb 2.7, WBC1800, platelets 3000. Peripheral smear showed anisopoikilocytosis and pancytopenia. Myeloblasts were seen while doing differential leucocyte count. Bone marrow aspiration cytology was advised but could not be performed due to insufficient sample.

LDH 6,192 IU/L, Uric acid 18.2 mg/dL, PT 17.7, INR 1.21, Creatinine 2.1 mg/dL, Urea 115 mg/dL, Calcium 5.9 mg/dL, Phosphorus 5.4 mg/dL, Sodium 131 mEq/L, Potassium 3.6 mEq/L, Bicarbonate 10.9 mEq/L, Chloride 99 mEq/L, Total bilirubin 0.3 mg/dL, Total protein 4.9 g/dL, Albumin 2.8 g/dL, AST 80 U/L, ALT 21 U/L, ALP 109 U/L.

Stool examination showed presence of occult blood in the specimen. Urine examination showed presence of protein [2+] and traces of blood. Urine microscopy showed presence of uric acid crystals. Ultrasonographic examination of abdomen showed presence of bulky bilateral kidneys with edematous appearance and altered echo texture. Fluid was seen in hepatorenal pouch. Sub acute intestinal obstruction was noted. Blood culture and sensitivity showed infection with *Staph aureus* and *Klebsiella*. The infant was diagnosed as having AML complicated with TLS and acute renal failure.

The child was infused with intravenous fluids and antibiotics. Several pints of packed cells and platelets were transfused. Infant was treated with allopurinol to reduce the uric acid levels for treatment of ARF and TLS and was subsequently started on appropriate chemotherapy. In spite of rigorous treatment schedule the infant developed septicemia and succumbed to death.

Discussion

TLS was first noted by Bedrna and Polcak, described in 1929. It is an emergency in oncology which results in metabolic imbalance due to the fast devastation of malignant cells leading to pouring of intracellular substances into the blood stream. This state is frequently linked with malignant tumors with an increased growth ratio [3]. TLS is characteristically related with poorly differentiated, high-grade lymphomas and acute lymphoid leukemia but is less often reported in AML and other malignancies of blood origin [2].

Since an array of biochemical derangements is associated with TLS, it is apt to have laboratory criteria to diagnose the emergency condition. The presence of two or more abnormal laboratory tests such as hyperkalemia, hyperuricemia, hyperphosphatemia, hypocalcaemia confirms presence of TLS [4]. Hande and Garrow proposed 25 %

increase or decrease from the baseline concentrations defines the hyper- or hypo states for laboratory diagnosis of TLS [5]. Clinically TLS was defined as laboratory-defined TLS accompanied by any of the following such as Creatinine level > 2.5 mg/dL, Potassium level > 6 mEq/L, Calcium < 6 mg/dL or development of a life-threatening arrhythmia [1].

Cairo and Bishop proposed laboratory and clinical criteria for TLS [6]. To fulfill the diagnosis, at least two laboratory criteria must be present for 3 days before treatment or up to 7 days after treatment. A clinical diagnosis consists of one clinical symptom in addition to two laboratory criteria. Our patient met the laboratory diagnosis of TLS based on his hyperuricemia and hypocalcemia.

The infant presented to us with an extremely elevated LDH and uric acid. LDH levels > 1,000 IU/L often hints to the occurrence of TLS. An elevated LDH points to a high tumor burden, thereby making it as one of the probable predictors of TLS [5].

The occurrence of renal failure is attributed to the crystallization of uric acid at low pH. The molecules like purine nucleobases that are liberated during the lysis of the tumor cells are degraded into uric acid through the action of xanthine oxidase in the liver. When the uric acid filtration capacity of the kidneys is surpassed by its production, it leads to precipitation of uric acid crystals in the renal tubules [3]. The mainstay of prevention of TLS consists of hydration as it promotes the excretion of uric acid and other metabolites.

Conclusion

Tumour lysis syndrome is potentially fatal, thus it is crucial to recognize the presence of laboratory and clinical findings that suggest the diagnosis. Provision of suitable prophylactic treatment for TLS to the high risk patients will surely aid in preventing mortality associated with this emergency condition. In infants immature renal system can lead to life threatening situation due to TLS, but timely intervention is highly efficient to prevent the adverse outcomes.

References

1. 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. *Tumori*. 2010;96(6):1040–3.
2. Riccio B, Mato A, Olson EM, Berns JS, Luger S. Spontaneous tumor lysis syndrome in acute myeloid leukemia: two cases and a review of the literature. *Cancer Biol Ther*. 2006;5(12):1614–7.

3. Froilán Torres C, Castro Carbajo P, Pajares Villarroya R, Plaza Santos R, Gómez Senent S, Martín Arranz MD, et al. Acute spontaneous tumor lysis syndrome in a patient with Crohn's disease taking immunosuppressants. *Rev Esp Enferm Dig.* 2009;101(4):288–94.
4. McBride A, Westervelt P. Recognizing and managing the expanded risk of tumor lysis syndrome in hematologic and solid malignancies. *J Hematol Oncol.* 2012;13(5):75.
5. Chubb EA, Maloney D, Farley-Hills E. Tumour lysis syndrome: an unusual presentation. *Anaesthesia.* 2010;65(10):1031–3.
6. Kekre N, Djordjevic B, Touchie C. Spontaneous tumour lysis syndrome. *CMAJ.* 2012;184(8):913–6.