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CASE REPORT

Tumour lysis syndrome: A rare acute presentation of locally advanced testicular cancer — Case report and review of literature

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Abstract Tumour lysis syndrome (TLS) is a potentially fatal complication of malignancy or its treatment. This uncommon syndrome comprises laboratory findings of hyperuricaemia, hypocalcaemia, hyperkalaemia and hyperphosphataemia. A literature search revealed a total of eight patients, with testicular cancer, who had TLS. All these patients had metastatic disease. We present a unique case of a 47-year-old gentleman we saw in clinic, who presented with a rapidly growing right groin mass and acute breathlessness, and discuss the diagnosis and management of TLS. TLS is extremely rare in testicular cancer but necessitates the awareness of urologists. TLS can occur spontaneously in testicular malignancy. Cell lysis in a rapidly proliferating germ cell tumour is a possible mechanism. The prompt identification and institution of management for TLS is crucial to improve clinical outcomes.

1. Introduction

Tumour lysis syndrome (TLS) is an oncological emergency, which is characterized by the derangement of cellular metabolism that can lead to acute renal impairment, metabolic acidosis, cardiac rhythm disturbances, seizures and death [1]. Massive cell lysis results in the release of large amounts of uric acid, potassium and phosphate into the systemic circulation. Hyperkalaemia, if untreated, may cause muscle weakness or paralysis and more significantly, cardiac arrhythmias and ultimately death [1]. Hyperphosphataemia is often associated with hypocalcaemia due to the precipitation of phosphate with the calcium in tissues. The resultant secondary hypocalcaemia may result in

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neuromuscular symptoms such as tetany, laryngospasm and cardiac arrhythmias. Both hyperuricaemia and hyperphosphataemia potentiate the risk of acute kidney injury by way of uric acid precipitation and calcium phosphate deposition in the renal tubules, further aggravating the electrolyte imbalance [2]. Additionally, the products of cell lysis trigger the release of cytokines, resulting in a systemic inflammatory response syndrome and frequently multiorgan failure [1].

TLS most commonly occurs after the initiation of cytotoxic therapy in patients with aggressive lymphoma or leukaemia. However, it may also occur spontaneously with other types of tumours that have a high proliferative rate, tumour burden or sensitivity to cytotoxic agents. In the setting of testicular cancer, a literature search only revealed eight incidences of TLS in testicular tumours, of which seven were metastatic and one had nodal involvement [3,4]. TLS occurred spontaneously in three patients, all of who had metastatic testicular seminomas [3]. The presence of metastatic disease would be compatible with an increased tumour burden which consequentially increases the risk of TLS occurring, either spontaneously or after the initiation of chemotherapy.

We present the case of a 47-year-old gentleman who was referred for a right groin swelling.

2. Case report

The patient was referred for progressive right groin swelling of 1-year duration, which had recently rapidly increased in size over the past 2 weeks. He reported a history of an undescended right testis, for which he underwent no previous surgical intervention. He reported good appetite and denied any weight loss. Physical examination revealed a 15 cm $\times$ 10 cm firm right groin lump, not extending into the scrotum. The right testis was not palpable in his scrotum. A CT abdomen/pelvis scan done prior to the clinic consultation revealed a 15 cm $\times$ 11 cm $\times$ 16 cm right groin solid mass, which was partly cystic with septations and calcification, suggestive of gonadal malignancy. There was no lymphadenopathy, metastasis or ascites in the abdomen. The right testis was not seen within the scrotum (Fig. 1). During the consultation, he was noted to be dyspnoeic and tachypnoeic but able to speak in full sentences. Upon further probing, he reported decreased effort tolerance, breathlessness and bilateral lower limb swelling for the past 2 days. He was admitted for an urgent CT pulmonary angiogram (CTPA). The CTPA performed excluded pulmonary embolism and lung metastases.

After admission, he was noted to be more tachypnoeic and struggling to speak in full sentences on the ward. An arterial blood gas (ABG) performed on 4 L/min of oxygen revealed severe metabolic acidosis with a pH of 7.08 and a lactate $>$10.9 mmol/L. He was promptly transferred to the intensive care unit (ICU) and intubated to provide mechanical ventilatory support. TLS was provisionally diagnosed and subsequently confirmed on laboratory findings. His blood results revealed hyperuricaemia, hyperphosphataemia, hyperkalaemia and an elevated creatinine and urea. Testicular tumour markers revealed a raised lactate dehydrogenase (LDH) $>$3800 U/L, and β subunit of human chorionic gonadotropin (β-hCG) 37 IU/L, with a normal α-fetoprotein (AFP) 3 μg/L. Post-intubation, he had a decreased level of consciousness, with a Glasgow coma scale score of 3 out of 15 despite receiving no intravenous sedation.

His electrolyte abnormalities were addressed systematically. For the hyperkalaemia, he received intravenous insulin, calcium gluconate and calcium resonium. For the hyperuricaemia, oral allopurinol via a nasogastric tube and intravenous rasburicase were administered. In view of the refractory hyperkalaemia and worsening metabolic acidosis despite intravenous sodium bicarbonate and multiple fluid challenges, a vascular catheter was inserted in his right internal jugular vein; he was started on continuous renal replacement therapy (CRRT). His systolic blood pressure dropped to 40–60 mmHg during CRRT, from approximately 110 mmHg. It was refractory to intravenous 5% albumin boluses as well as intravenous noradrenaline. CRRT was discontinued after 60 min as the persistently low blood pressure resulted in the formation blood clots in the vascular catheter. Additional inotropic support in the form of intravenous adrenaline was started. Despite the best treatment, the patient had a cardiac arrest and died.

3. Discussion

3.1. Incidence

This case illustrates a rare presentation of a testicular malignancy. To our knowledge, this is the first case of a non-metatstatic testicular malignancy, with no nodal involvement to present with spontaneous TLS. TLS most

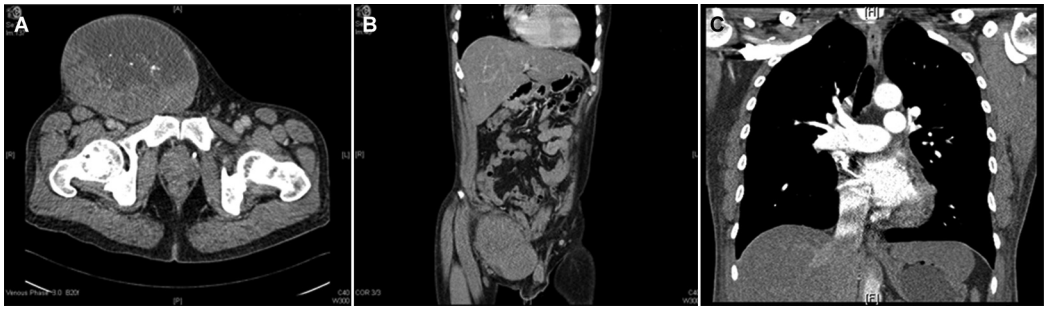

Figure 1 (A) (B) CT abdomen/pelvis revealed a 15 cm $\times$ 11 cm $\times$ 16 cm right groin mass. There was no lymphadenopathy or metastasis present. (C) CT pulmonary angiogram revealed no pulmonary embolism or lung metastasis.