Case Report

Tumour lysis syndrome

A 57-year-old man with relapsed non-Hodgkin's lymphoma (NHL) was admitted for salvage chemotherapy with dexamethasone, ara-C and cisplatin. Shortly after cisplatin was initiated he developed a cough, wheeze, nausea, vomiting, tachypnoea and tachycardia. The biochemical findings are shown in Table I. He was treated by pushing intravenous fluids and potassium-binding resin, followed by allopurinol and calcium supplementation. His laboratory results improved over the next 48 hours (Table I). The patient subsequently tolerated two cycles of the same chemotherapy with aggressive hydration and allopurinol. He will have CT scan re-staging and stem cell transplantation in the future.

The main differential diagnosis was cisplatin hypersensitivity and tumour lysis syndrome (TLS). The biochemical features (hyperuricaemia, hyperkalaemia, hyperphosphataemia and hypocalcaemia) were typical of TLS. TLS is a life-threatening oncological emergency caused by rapid lysis of malignant cells, either spontaneously or induced by chemotherapy. TLS typically occurs with high-grade lymphomas and other haematological malignancies.[1] Manifestations of TLS are variable, including renal failure, muscle cramps, weakness and seizures.[2]

The cornerstone of TLS management is to push fluids to increase excretion of urate and phosphate.[3] Hyperkalaemia must be managed urgently. Drugs that lower serum urate have a key role in the management of TLS (Fig. 1). Allopurinol reduces the formation of urate, but does not affect

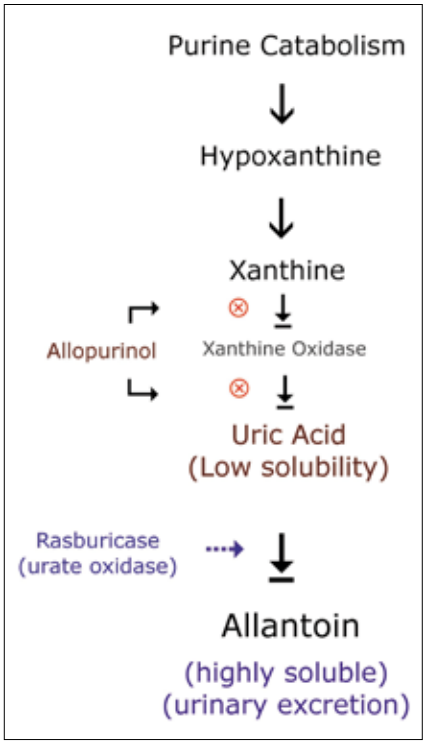

Fig. 1. Purines are catabolised to hypoxanthine and xanthine by the action of enzyme xanthine oxidase, which is inhibited by allopurinol. These intermediaries are further converted to uric acid, which is the final step in purine catabolism in humans. However, in most other mammals (but not in humans) urate oxidase enzyme converts uric acid to the more soluble allantoin. Rasburicase is a recombinant form of urate oxidase.

circulating urate or crytals deposited in the tissues[4] (Fig. 2). Rasburicase, a recombinant urate-oxidase enzyme, converts urate to soluble allantoin. Although rasburicase lowers uric acid more than allopurinol, there is currently no evidence of clinical benefit and it is extremely expensive.[5]

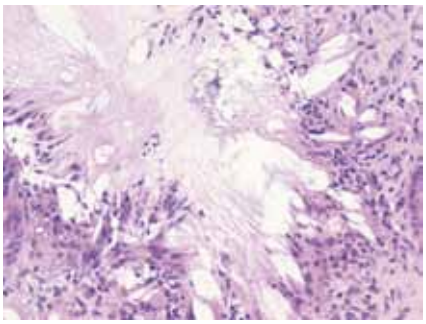

Fig. 2. Haematoxylin and eosin stained section of urate nephropathy with interstitial elongated urate crystals with an inflammatory response on the periphery (X10 magnification). (Slide courtesy of Helen Wainwright, Anatomical Pathology, University of Cape Town.)

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References available at www.cmej.org.za

Table I. Relevant biochemical findings during admission

	Admission	3 hrs after chemotherapy	12 hrs after chemotherapy	48 hrs after allopurinol	Normal ranges
Potassium	4.6	6.0	6.6	4.5	3.3 - 5.3 mmol/l
Phosphate	0.83	3.22	3.50	0.88	0.80 - 1.40 mmol/l
Uric acid	0.50	0.85	0.89	0.55	0.23 - 0.46 µmol/l
Calcium	1.81	1.70	1.40	1.67	2.05 - 2.56 mmol/l
Urea	4.7	14.4	23.8	11.9	3.6 - 7.0 mmol/l