Acute renal failure due to tumor lysis syndrome in a HIV seropositive patient with Castleman’s disease

Sir,

Castleman’s disease (CD), a rare lymphoproliferative disorder, is characterized by histological features of lymph node hyperplasia and capillary proliferation. Conditions such as minimal-change, membranous, mesangiprolifemative, membranoproliferative glomerulonephritis, thrombotic microangiopathy and renal amyloid have been reported with CD [1]. Acute renal failure (ARF) due to tumour-lysis syndrome (TLS) has not been reported in these patients.

Case. A 34-year-old Caucasian male was admitted to the Houston VA medical center with nausea, right upper quadrant and epigastric pain. His past medical history was significant for the diagnosis of HIV infection 11 years previously. He had refused therapy until 6 months prior to this admission when he developed constitutional symptoms and fever. Initial blood tests were as follows: serum creatinine 5.4 mg/dl, uric acid 12.9 mg/dl, phosphorus 3.1 mg/dl and serum LDH 361 U/l. Acute pancreatitis was also noted. Baseline serum creatinine and uric acid were not elevated. Renal ultrasound showed normal renal sizes with no evidence of hydronephrosis. Cervical lymphadenopathy and renal amyloid have been reported with CD [1].

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Introduction of cyclophosphamide and prednisone resulted in rapid destruction of tumour cells and worsening of TLS, manifested by a dramatic rise in the serum levels of creatinine, phosphorus (12 mg/dl), uric acid (20 mg/dl) and LDH enzyme (550 U/l). Cervical lymphadenopathy and pancreatitis improved. Haemodialysis was continued for 10 days along with allopurinol. With the resolution of TLS, renal function slowly improved over 6 weeks.

Comment. CD has hyaline-vascular and plasma cell variants and clinically could be localized or multicentric. While localized CD is a benign lymphoproliferative disorder, the multicentric type is associated with infections, multi-organ failure and malignancies [2]. It is postulated that Kaposi’s sarcoma associated virus (HHV-8) could play a crucial role in producing IL-6 and releasing angiogenic factors resulting in lymphoplasmacytotic proliferation [2]. Renal involvement in CD has clinical presentations that vary from nephrotic syndrome to antimonyeloperoxidase-antibody-positive rapidly progressive glomerulonephritis [3] and end-stage renal disease from renal amyloidosis [4].

Even though TLS is common in haematologic malignancies after chemotherapy, in rapidly growing tumours hyperuricaemia could be seen even without the introduction of chemotherapy or radiotherapy, an entity recognized as ‘spontaneous’ TLS. We postulate that our patient initially may have had spontaneous TLS and that subsequent renal recovery was hampered by chemotherapy-induced TLS.

Conflict of interest statement. None declared.

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Molecular forms of adiponectin in uraemic plasma

Sir,

Adiponectin is a recently found anti-atherogenic plasma protein secreted by adipocytes. Plasma adiponectin level is reduced in patients with coronary artery disease [1], type 2 diabetes mellitus [1] and obesity [2]. In contrast to these high-risk groups, plasma adiponectin has been reported to be elevated in haemodialysis patients [3]. Since uraemic plasma is known to contain not only intact forms but also fragments of some peptide hormones such as parathyroid hormone, it is an important question whether adiponectin in uraemic plasma is intact or not. To answer the question, we analysed its molecular forms.

Plasma samples were taken from two patients (patients A and B) on maintenance haemodialysis and a healthy volunteer; both 42-year-old men without diabetes, obesity or coronary artery disease. Fresh plasma was fractioned by gel filtration using a 10 × 300 mm column of Superose 6 HR (Amasham Biosciences, Tokyo) and 31 mM Tris–HCl buffer (pH 7.2). An aliquot of each 0.5 ml fraction was assayed for adiponectin by ELISA [2]. Subsequently, another aliquot of the fractioned plasma was subjected to SDS-polyacrylamide gel electrophoresis (PAGE) in reducing condition. Western blotting was done using anti-adiponectin monoclonal