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Virology Question and Answer Scheme (VIROQAS)

A case of bilateral ureteric obstruction in an allogeneic stem cell transplant recipient

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Case presentation

Mr. MN, a 19 year old male, was treated for Acute Lymphocytic Leukaemia initially according to the UKALL12 regimen, and then with Bone Marrow Transplantation after total body irradiation, CamPath and cyclophosphamide conditioning. Following transplantation he initially made a good recovery, but at week 2 post-transplant began to suffer from mucositis and haemorrhagic cystitis. The cystitis failed to improve with conservative management including intravenous hydration, bladder irrigation and modification of his immunosuppressive regimen. He received 1 dose of intravesical cidofovir. During this period he received standard acyclovir prophylaxis and showed no signs of graft-vs.-host disease.

On day 17 his renal function deteriorated acutely and severely, with creatinine rise from 89 to 287 over a 12 hour period, with concurrent anuria. The acute presentation and severity of the renal failure combined with anuria raised the suspicion of post-renal obstructive failure. CT imaging of the renal tract revealed mild dilatation of the proximal ureters bilaterally but no urolithiasis or mass lesions. However blood was noted within the severely thick walled bladder, consistent with severe haemorrhagic cystitis (Fig. 1). Subsequent bilateral nephrostomies showed multiple filling defects within the collecting systems and ureters secondary to clots (Fig. 2). Due to the patient's acutely deteriorating clinical condition he was admitted to the intensive care unit where haemofiltration was initiated. MN was also treated with a short course of intravenous human immunoglobulin. The ureteric obstruction was treated with the insertion of bilateral ureteric drains under radiographic guidance which alleviated the acute renal failure with a return of creatinine and urea levels to baseline levels over the subsequent 72 h without further haemofiltration.

Concurrent with these events, the patient became acutely short of breath with widespread wheeze and hypoxia. This required intubation and mechanical ventilation. A plain chest radiograph showed no signs of consolidation or collapse to account for this deterioration in respiratory function (Fig. 3), and computed tomography examination revealed new ground glass opacity with some centrilobular ground glass nodules consistent with infective or inflammatory bronchiolitis. PCP and viral infections would be considered the most likely causes though ultimately the imaging is organism non-specific (Fig. 3a and b). Bronchoscopy was undertaken, and bronchoalveolar lavage samples obtained for diagnostic analysis.

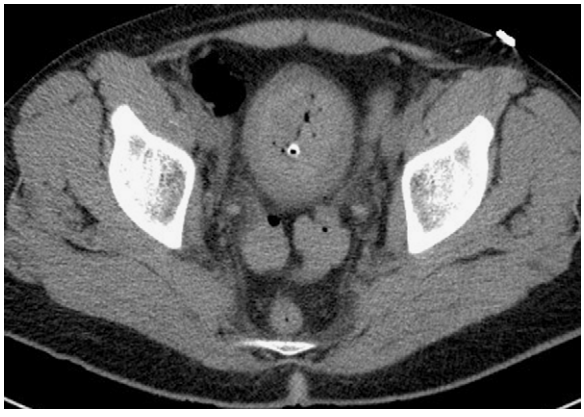

Fig. 1. Unenhanced CT examination shows high attenuation blood within the bladder which is significantly thick walled. There are multiple small intravesical air locules which are more likely to relate to the inflammatory process than catheterisation.

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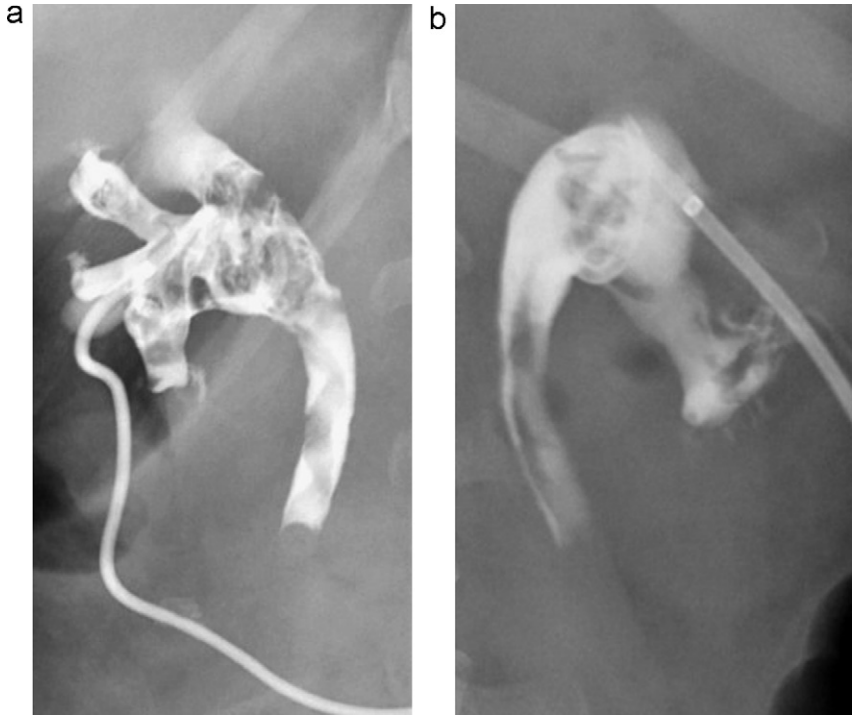

Fig. 2. Bilateral nephrostogram shows multiple filling defects in the collecting systems and upper ureters secondary to clots.

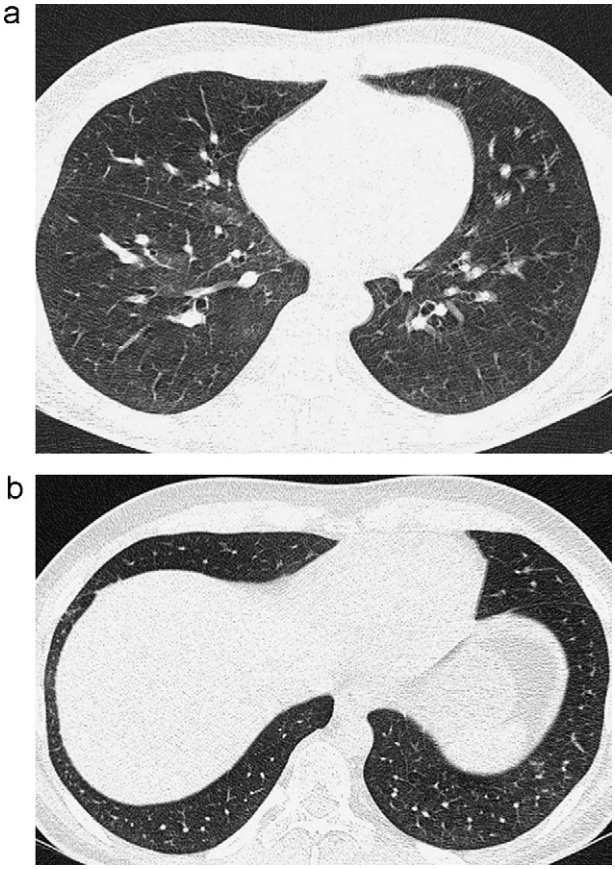

Fig. 3. (a) Computed tomography shows ground glass changes in the right lung and (b) centrilobular ground glass nodules in the left base.

MN recovered well from the pneumonitis and was successfully extubated after 6 days. 4 Weeks after insertion of the ureteric stents, MN's ureters were sufficiently clear as to allow normal urine passage, and they were removed successfully. Renal function returned to normal prior to discharge from hospital.

What is the likeliest viral cause for the haemorrhagic cystitis seen in MN?

How might this relate to the acute renal failure?

Could the same virus be responsible for the pneumonitis?