2. Laouad I, Buchler M, Weestel PF *et al*. Replacement kidney lipomatosis after renal transplantation. *Transplantation* 2005; 79: 496–498
3. Ambos MA, Bosniak MA, Gordon R *et al*. Replacement lipomatosis of the kidney. *AJR Am J Roentgenol* 1978; 130: 1087–1091
4. Yagci C, Kosucu P, Yorubulut M *et al*. Renal replacement lipomatosis: ultrasonography and computed tomography findings. *Eur Radiol* 1999; 9: 1599–1601
5. Chang SD, Coakley FV, Goldstein RB. Case report: renal replacement lipomatosis associated with renal transplantation. *Br J Radiol* 2005; 78: 60–61

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Doxycycline for haematopoietic stem cell transplantation-related thrombotic microangiopathy

Hassane Izzedine[1]
Alain Baumelou[1]
Chems Gharbi[1]
Edward Bourry[1]
Jean Paul Vernant[2]

[1]Department of Nephrology, Pitié-Salpétrière Hospital, Paris, France
[2]Department of Hematology, Pitié-Salpétrière Hospital, Paris, France
E-mail: hassan.izzedine@psl.aphp.fr

Transplantation-associated thrombotic microangiopathy (TA-TMA) is a devastating consequence of allogeneic haematopoietic stem cell transplantation (HSCT) with a mortality rate of 60–90%. None of the interventions used, as used up till now in idiopathic thrombotic thrombocytopaenic purpura (TTP) (fresh frozen plasma transfusion, plasma exchange and steroids), were effective to treat TA-TMA [1,2]. We report a dramatic improvement of TA-TMA in two HSCT patients [conditioning, cyclophosphamide, total body irradiation, graft-*versus*-host disease (GVHD) prophylaxis] using doxycycline.

A 36-year-old woman with Hodgkin's lymphoma received an allogeneic HSCT in December 2002. Twelve months later, she developed a biopsy-proven TMA (proteinuria, 3 g/day, microscopic haematuria, oliguric acute renal failure with creatinine level at 680 μmol/L; haemoglobin Hb, 6.3 g/dL; schistocytes; platelet count, 35 × 10^9/L; LDH, 1754 IU/L). The serum complement proteins were at normal levels, no mutations of the membrane cofactor protein were found and a plasma ADAMTS13 activity was found at 40%. Steroids, plasma exchange, fresh frozen plasma transfusion, vincristine and haemodialysis were tried with a partial response (haemoglobin, 7.3 g/dL, platelet 70 000/mm^3 both after treatment). Doxycycline 200 mg daily was added for a suspected gastrointestinal *Bartonella* infection. Within two months, haemoglobin and platelet count rose without transfusion to 10.8 g/dL and 234 000/mm^3, respectively. Despite improvement of haematological parameters, the patient remained dialysis-dependent. The second patient had a similar haematologic disease and course under doxycycline prescribed for a bartholinitis.

Five patients with TTP and *Bartonella*-like erythrocyte inclusions, successfully treated with doxycycline, experienced recurrence of their TTP following cessation of treatment [3]. TA-TMA has a multi-factorial aetiology of endothelial damage. Doxycycline targeting the adherens junction on endothelial cells prevents vascular hyperpermeability [4]. Doxycycline as a potential treatment of TA-TMA warrants further studies.

Conflict of interest statement. None declared.

1. Choi CM, Schmaier AH, Snell MR *et al*. Thrombotic microangiopathy in haematopoietic stem cell transplantation: diagnosis and treatment. *Drugs* 2009; 69: 183–198
2. Michael M, Elliott EJ, Ridley GF *et al*. Interventions for haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura. *Cochrane Database Syst Rev* 2009; 1: CD003595
3. Tarantolo SR, Landmark JD, Iwen PC *et al*. *Bartonella*-like erythrocyte inclusions in thrombotic thrombocytopenic purpura. *Lancet* 1997; 350: 1602
4. Fainaru O, Adini I, Benny O *et al*. Doxycycline induces membrane expression of VE-cadherin on endothelial cells and prevents vascular hyperpermeability. *FASEB J.* 2008; 22: 3728–3735

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The spot urine protein/creatinine ratio is a simple, rapid and inexpensive method for monitoring patients with light-chain multiple myeloma

Protein electrophoresis of a 24-h urine collection (UPEP) is considered the standard method for following up patients with light-chain multiple myeloma [1]. The serum-free light-chain assay (SFLCA) has increasingly been used in this population [2], and in individual patients tracks well with proteinuria [3]. In addition, the SFLCA is also generally more sensitive than urine studies including immunofixation electrophoresis for detecting minimum residual light-chain disease [4,5]. However, the SFLCA is expensive, and due to inter-patient variation in the renal metabolism of light chains, the amount of proteinuria cannot be predicted by the SFLC concentration [1,5–7]. As proteinuria correlates better with renal dysfunction than SFLC and may be caused by factors other than light chains, serial measurement of urinary proteinuria is still considered essential [7].

The spot urine protein/creatinine ratio (SUPCR) has increasingly replaced the 24-h urine in patients with proteinuria from a variety of causes [8], but has not been examined in patients with multiple myeloma. As free light chains have a half-life of 2–6 h [9], the SUPCR is theoretically ideally suited to measure response to treatment within days of beginning therapy, and moreover, can be inexpensively and serially measured with rapidly available results. In this report, five patients with predominantly light-chain multiple myeloma were followed up by SUPCR and SFLCA. In Patient 1 and 2 (Figure 1A and B), progressive disease and subsequent response to therapy were accurately detected by SUPCR and in agreement with changes in the SFLCA. In Patient 3 (Figure 1C), bortezo-