levels of lactate dehydrogenase, immeasurable low levels of haptoglobin and very high levels of free haemoglobin in plasma. Coombs test was negative and no erythrocyte antibodies could be detected. Schistocytes could not be identified at blood microscopy. Biochemical screening revealed no antineutrophil cytoplasmic antibodies and the levels of immunoglobulins and complement factors were normal. The number of blood eosinophils was normal. The platelet number decreased from 422 x 10^9/l to 212 x 10^9/l at admission and then remained within the reference level. Antibody screening showed no signs of viral infection. Ultrasound examination of the kidneys revealed no abnormalities. A renal biopsy was not performed. Within 24 h after admission, she developed anuria and haemodialysis was initiated. Due to the slight reduction in platelet count, the patient was at first suspected of a microangiopathic haemolytic anaemia and plasma exchange was performed with substitution with fresh frozen plasma. For the next 18 days she received nine haemodialysis treatments, six plasma exchanges and 14 erythrocyte transfusions. During this period haemolysis ceased and the patient regained kidney function. Two months later she had normal kidney function with creatinine clearance of 116 ml/min and no signs of haemolysis.

The epidemiology of iron dextran hypersensitivity is well characterized [1–3], and it is well known that the drug may elicit a hypersensitivity reaction at the first exposure. Severe allergic reactions to iron dextran consist mainly of respiratory and cardiovascular symptoms. The presented patient twice demonstrated such typical immediate hypersensitivity symptoms, although these were relatively mild and easily reversed by hydrocortisone. The patient had not received any new medication and there were no signs of infections or other exogenous reasons known to induce acute severe haemolysis. Although the patient at the time of admission was suspected of microangiopathic haemolysis, there were insufficient biochemical findings supporting this diagnosis. Only a few days passed from the second exposure to iron dextran to when the patient developed haemolysis, and the condition can almost certainly be ascribed to iron dextran exposure. There are no previous published reports on haemolysis associated with infusion of iron preparations, but the World Health Organization's (WHO) Adverse Reaction Database (Uppsala Monitoring Centre) describes two patients with haemolysis probably due to iron dextran. However, the database also reports three patients with haemolysis due to iron gluconate treatment, suggesting that the reactions may not be related to a specific iron complex.

The mechanisms behind iron dextran-induced haemolysis in the presented patient are unknown. Several drugs can act as haptons and may, through their binding to specific erythrocyte receptors, induce production of complement-activating antibodies mediating severe extracellular haemolysis. Although intravenous administered iron complexes have not previously been implicated in immunological mediated haemolysis, the time frame for this mechanism is typically 1–2 weeks from the first drug exposure to active haemolysis, as was seen in our patient.

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**Acute renal failure in a patient with West Nile viral encephalitis**

SIR,

West Nile (WN) virus is increasingly recognized as an important human pathogen in the North America. Meningitis or encephalitis develops in approximately 1 in 150 infected persons. Rare extraneurological manifestations include myocarditis, pancreatitis and hepatitis. Acute renal failure (ARF) is a very rarely reported manifestation of WN virus infection. We describe a case of reversible ARF secondary to biopsy-proven acute tubular necrosis (ATN) in a patient with WN virus encephalitis. To our knowledge, this is the second report of ATN in a patient with WN virus infection.

**Case**

A 76-year-old man with known history of diabetic nephropathy and hypertension was admitted to hospital in New York City in October 2005 with fever and confusion. He reported fatigue and abdominal discomfort 3 days after being bitten by mosquitoes. Two weeks later he presented with high fevers, chills and confusion.

On examination, he was alert, but confused. His blood pressure was 170/80 mm Hg and temperature 39°C. The rest of the examination was unremarkable. Serum sodium was 127 mmol/l, potassium 4.8 mmol/l, urea 15.7 mmol/l and creatinine 168 mmol/l. His baseline creatinine level was 150 μmol/l. Complete blood counts were normal, and urinalysis showed haematuria and proteinuria. Computed tomography (CT) of the brain and chest were unremarkable. Intravenous antibiotics were started and stopped when cultures of cerebrospinal fluid (CSF), blood and urine remained negative after 72 h. On day 2 he became lethargic. Lumbar puncture showed clear CSF, with 0.7 g/l protein, 2.6 mmol/l glucose, 68 red cells per cubic millimeter, 154 white cells per cubic millimeter with 85% lymphocytes and negative Gram stain. New York State Department of Health subsequently reported WN virus IgM antibody from CSF by capture enzyme immunoassay. The patient remained...
confused and agitated with stable vital signs. MRI of the brain demonstrated cerebral atrophy. Four days later, serum creatinine rose to 583.4 μmol/l and urea to 26 mmol/l. Urine volume never decreased; urinalysis showed turbid colour, with proteinuria, 2-4 white cells, 22-24 red cells, 2-5 coarse granular casts per high-power field, no eosinophils and tested negative for myoglobin. Abdominal ultrasonography was normal. Renal biopsy on the ninth hospital day showed acute tubular necrosis (ATN) and early diabetic nephropathy (Figure 1). Over the next five days renal function and mental status returned to baseline and the patient did not require dialysis therapy. By day 12 the patient was ambulatory and was discharged home. The patient has remained well throughout the 7-month period of close clinical observation; he has been afebrile and his serum creatinine level has remained stable at 150 μmol/l.

Comment

Most WN virus infections in humans are subclinical, with overt disease occurring in 1 out of every 100 infections. Rare manifestations include myocarditis, pancreatitis and fulminant hepatitis—the involved organs are sites of high viral replication [1]. Some support exists for a renal tropism of WN virus. WN virus has been detected in high titres by viral replication [1]. Some support exists for a renal tropism of WN virus. Some support exists for a renal tropism of WN virus. Some support exists for a renal tropism of WN virus. Some support exists for a renal tropism of WN virus. Some support exists for a renal tropism of WN virus.

In contrast to a prior reported fatal case of WN virus encephalitis and renal failure [3], our patient had no traditional risk factors for ATN such as hypotension, clinically evident hypovolaemia, endogenous or exogenous nephrotoxins and he did not require dialysis.

The chronological sequence of renal failure and recovery in the context of WN encephalitis suggest that the renal injury is due to direct viral and/or immune-mediated damage. WN virus is an emerging infectious disease, and as more cases are reported, it is possible that renal involvement will be recognized among the presentations of WN virus infection. Our case report may help to elucidate the mechanisms of renal impairment in WN virus infection.

Conflict of interest statement. None declared.


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Inequality of renal replacement therapy in the low-income countries

Sir,

As Grassmann and associates [1] have shown, chronic renal failure (CRF) patients treated by renal replacement therapy (RRT) worldwide are increasing rapidly. The cost of RRT, especially the cost of dialysis, has been growing rapidly with an increasing numbers of RRT patients [2], becoming a financial problem even in Euro–American countries and Japan. More seriously, developing countries have been struck by a surge in the RRT population and a sharp rise in reimbursement costs for RRT. Although policy makers and care providers are trying to precisely analyse this upward trend of RRT patients, sufficient data cannot be collected, due to absent or insufficient registries, past secrecy of middle and eastern European countries and the civil wars in some African countries.

Grassmann and associates [1] have enthusiastically collected demographic data from 122 countries and show us the relationship between GDP (gross domestic product) and the RRT population. However, they noted that data could not be collected from several sub-Saharan countries such as Botswana, Namibia, Congo, Zimbabwe, Burkina Faso and Ethiopia, and from some of the Asian countries, including Cambodia and Bhutan. As for the six sub-Saharan countries mentioned above, Naicker [3] recently made a report on their present status of RRT population/pmp (per million population) as 4.1, 13.5, 0.74, 7.8, 0.9 and 0.07, respectively. In addition, Sitprija [4] reported that one of the Asian countries, Cambodia, had RRT population of 4.5/pmp.

When I visited Bhutan, a small Himalayan country located between India and China, in May 2005, I had an opportunity to visit the Jigme Dorje Wangchuk National Referral Hospital in the metropolis Thimpu. The hospital had a clean, well-organized 3-bed haemodialysis (HD) unit, equipped with Fresenius 4008 machines. Dr Giri was taking care of 16 CRF patients on HD (9.6/pmp). These patients did not have to pay for treatment, as health care and education expenses in Bhutan are covered by the government. However, geographic conditions do not allow...