Renal thrombotic microangiopathy induced by β-interferon

Sir,

We read with interest the recent case report ‘Minimal change disease with interferon-β therapy for relapsing remitting multiple sclerosis’ [1]. In this paper, the authors include renal thrombotic microangiopathy (TMA) among rare side effects of interferon (IFN) therapy, more frequently described with IFN-α [2]. We report here our experience about this topic.

A 36-year-old white female with a 3-year history of multiple sclerosis and normal blood pressure and renal function was admitted for acute renal failure and pulmonary oedema. Three months previously, she started subcutaneous IFN-β1a treatment of 22 μg thrice weekly. On admission, physical examination showed high blood pressure and severe pleuropericarditis without neurological or dermatological findings. Laboratory tests revealed microangiopathic haemolytic anaemia. Other immunological and microbiological laboratory tests were unremarkable. A renal biopsy disclosed signs of TMA; among 43 glomeruli, light microscopy revealed focal ischaemic signs and mild mesangial cell proliferation; vessel narrowing with thrombosis and thickening of arteriolar walls and intimal onion skin-like swelling; light interstitial lymphomonocytic infiltration and thickening of arteriolar walls and intimal onion skin-like swelling. Immunofluorescence showed mesangial IgM, C1q and fibrinogen staining. A diagnosis of haemolytic–uraemic syndrome was made. She was treated with transfusions, haemodialysis, plasma exchange and methylprednisolone i.v. followed by oral prednisone. Her cardiac function improved, and haematological signs progressively disappeared, but renal function did not recover. IFN-β treatment was discontinued. She is now receiving peritoneal dialysis treatment. IFN-α is known to cause a variety of renal lesions, including TMA [3,4], but to our knowledge, our observation is the first report of TMA induced by INF-β.

Editorial note: This letter had been sent to Aravindan A. et al., but we did not receive a response.

Conflict of interest statement. None declared.

1Nephrology and Dialysis, A.R.N.A.S. Civico and Di Cristina, Palermo, Italy
2Nephrology and Dialysis, A.R.N.A.S. Civico and Benfratelli, Palermo, Italy
3Nephrology and Dialysis, A.R.N.A.S. Civico and Di Cristina, Palermo, Italy
4. Badid C, McGregor B, Faivre JM et al. Renal thrombotic microangiopathy induced by interferon-α. Nephrol Dial Transplant 2001; 16: 2469–2471
5. Monsein LH, Davis M. Radionuclide imaging of a rectus sheath hematoma caused by insulin injections. Clin Nucl Med 1990; 15: 539–541
6. Yoshida T, Harayama T, Kusunoki C et al. A case of type 2 diabetes mellitus with a giant subcutaneous hematoma caused by rough insulin injection. J Japan Diab Soc 2008; 51: 929–932
7. Kahara T, Kawara S, Shimizu A et al. Subcutaneous hematoma due to frequent insulin injections in a single site. Intern Med 2004; 43: 148–149
8. Arendt-Nielsen L, Egekvist H, Bjerring P. Pain following controlled cutaneous insertion of needles with different diameters. Somatosens Mot Res 2006; 23: 37–43
9. Y oshida T, Harayama T, Kusunoki C et al. Renal thrombotic microangiopathy induced by interferon-β therapy for relapsing remitting multiple sclerosis. NDT Plus 2010; 3: 132–134
10. Magee CC. Renal thrombotic microangiopathy induced by interferon-α. Nephrol Dial Transplant 2001; 16: 2111–2112
11. Vacher-Coponat H, Opris A, Daniel L et al. Thrombotic microangiopathy in a patient with chronic myelocytic leukaemia treated with α-interferon. Nephrol Dial Transplant 1999; 14: 2469–2471
12. Badid C, McGregor B, Faivre JM et al. Renal thrombotic microangiopathy induced by interferon-α. Nephrol Dial Transplant 2001; 16: 846–848

Membranous glomerulonephritis with superimposed ANCA-associated vasculitis: another case report

Dear Sir,

We report here another case of primitive membranous nephritis with superimposed anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, in addition to 10 cases recently reported by Nasr et al. [1]. This association has since been reported in relation with systemic lupus erythematosus, hepatitis B or C virus infection and treatment with penicillamine, hydralazine and propylthiouracil [2–5].

A 67-year-old Caucasian male was presented at the emergency department with anorexia, nausea and vomiting. Routine laboratory tests revealed severe renal failure and a consultation with a nephrologist was requested. Blood pressure was 170/100 mm Hg, and urine output over 24 h was 2.2 L. Medical history was remarkable for hypertension (in treatment with β-blockers) and possible upper respiratory infection about 4 weeks before admission (treated with amoxicillin 2 g/day orally). Urinalysis revealed haematuria (+++) and non-selective proteinuria (4.8 g/24 h), in front of seric albumin levels of 2.6 g/dL. Skin examination revealed no significant lesions.

LAC, ANA, anti-DNA, ENA, HBsAg, anti-HCV, cryoglobulins, complement levels, ANCA and serum protein electrophoresis were normal. Perinuclear ANCA was positive at 1:40.

A renal biopsy was performed, and sampling for LM included 11 glomeruli, three of which were globally sclerotic. Light microscopy revealed the presence of extracapillary proliferation which compressed the glomerular tuft and vasculitis with fibrinoid necrosis of the arterial wall. Cellu-