

1. Monsein LH, Davis M. Radionuclide imaging of a rectus sheath hematoma caused by insulin injections. *Clin Nucl Med* 1990; 15: 539–541
2. 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
3. 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
4. Arendt-Nielsen L, Egekvist H, Bjerring P. Pain following controlled cutaneous insertion of needles with different diameters. *Somatosens Mot Res* 2006; 23: 37–43

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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- β -1a treatment of 22 μ g 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 thrombi and thickening of arteriolar walls and intimal onion skin-like swelling; light interstitial lymphomonocytic infiltration and focal tubular atrophy. 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.

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1. Aravindan A, Yong J, Killingsworth M *et al.* Minimal change disease with interferon-beta therapy for relapsing remitting multiple sclerosis. *NDT Plus* 2010; 3: 132–134
2. Magee CC. Renal thrombotic microangiopathy induced by interferon-alpha. *Nephrol Dial Transplant* 2001; 16: 2111–2112
3. 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
4. Badid C, McGregor B, Faivre JM *et al.* Renal thrombotic microangiopathy induced by interferon- α . *Nephrol Dial Transplant* 2001; 16: 846–848

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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, ANCAs 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-