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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 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 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.

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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-

lar crescents were present exclusively in three glomeruli. The crescents were accompanied by foci of fibrinoid necrosis with endocapillary and extracapillary fibrin. Tubular atrophy and interstitial fibrosis were absent. Interstitial inflammation was present, focal and accompanied by tubular degenerative changes. There was evidence of necrotizing vasculitis. IF revealed granular, segmental to global glomerular capillary wall positivity for IgG, kappa and lambda. Weaker staining for C3 was detected. Staining for fibrinogen highlighted areas of glomerular fibrinoid necrosis. Immunofluorescence shows intense staining of the arterial wall for IgG.

The final diagnosis was 'membranous glomerulonephritis with superimposed ANCA-associated vasculitis and extracapillary proliferation'. The patient started a 6-month course of methylprednisolone (1 g i.v.) for three consecutive days at months 1, 3 and 5, followed by methylprednisolone *per os* alternated with cyclophosphamide *per os*. The patient is in partial remission. Creatinine fell to 1.6 mg/dL while proteinuria reduced to 1.2 g/24 h. Treatment ended on September 2010.

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Coagulase-negative staphylococcus—oftentimes a virulent masquerader

To the Editor,

Septic arthritis caused by *Staphylococcus lugdunensis*, a coagulase-negative species, is a rare entity. As of 2008, there were only 58 confirmed cases [1] of bone and joint infections caused by this particular staphylococcal species, potentially due to the fact that many microbiology labs identify but do not speciate coagulase-negative staphylococcus [2]. Despite the presence of confirmed cases of bone and joint infections due to *S. lugdunensis* in the literature, we believe that this is the first case of septic arthritis caused by this organism in ESRD patients with long-term indwelling venous catheters. We present our observations below.

A 60-year-old African American male with ESRD and an indwelling venous catheter for access presented to his primary care physician with a 2-week history of pain, swelling and warmth in his right knee. He reported a low-grade fever of 100°F. He was initially treated with prednisone by his primary care physician, and due to unresolving symptoms, he underwent an arthrocentesis which showed 46 333 cells per cubic millimeter, no visible crystals, and cultures that grew a coagulase-negative staphylococcal species, later identified as *S. lugdunensis*. As such, the patient was admitted. Blood cultures drawn from the indwelling venous catheter grew identical organisms, and antibiotics were started empirically in the form of vancomycin. Orthopedic consultation was obtained, and the patient underwent arthroscopic irrigation and debridement of the right knee, during which frank pus was noted within the right knee. Knowing that *S. lugdunensis* is a virulent pathogen, we performed an echocardiogram showing vegetation in the mitral valve leaflet which indicated a subacute versus an old, healed vegetation [3].

In our patient with septic arthritis, the source for *S. lugdunensis* was most likely the indwelling venous catheter as it grew the identical organism. What is striking about this organism is that in contrast to other coagulase-negative species, *S. lugdunensis* has been associated with aggressive infections likely due to the fact that it shares many of its virulence factors with *Staphylococcus aureus* such as proteases, lipases and hemolysins [1,4]. A learning point from this case is to ask microbiology labs to speciate out coagulase-negative staphylococci in the appropriate clinical scenario. The likelihood of *S. lugdunensis* isolated from a clinical culture specimen being a pathogen versus a contaminant varies anywhere from 91% to 30% according to published data [5]. Missing *S. lugdunensis* could be catastrophic for the patient, as the bacteria can cause acute endocarditis, septic arthritis and vertebral discitis, especially since the bacteria is usually pan-sensitive to antibiotics. As microbiology labs speciate more coagulase-negative staphylococci, we are likely to identify *S. lugdunensis* more frequently in clinical infection.

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