Sir,

Fibrillary glomerulonephritis (FGN) and immunotactoid glomerulopathy (ITG) are well known glomerular diseases with Congo-red-negative deposits [1–4]. While FGN is characterized by randomly arranged microfibrils around 20 nm in diameter with no underlying systemic disorder, ITG is defined by orderly arranged microtubular deposits, usually >30 nm in diameter with a hollow core; patients with ITG tend to have underlying lymphoproliferative diseases.

Microtubular monoclonal immunoglobulin deposits with a diameter smaller than that commonly observed in ITG have a tendency to have underlying lymphoproliferative disorders, and Touchard et al. coined the term ‘GOMMID’ [5].

GOMMID is not fully understood. Touchard et al. [5] proposed to include the following criteria: (i) the presence of organized microtubular deposits (15 nm in width) in glomeruli without thrombi in the glomerular capillaries and/or renal arterioles; and (ii) monotypic immunoglobulin deposits, irrespective of the presence of detectable monoclonal immunoglobulin in patient serum and/or urine, type I cryoglobulinaemia or immunoproliferative disorder [5]. Our case satisfies these criteria and features both pathological and clinical aspects of GOMMID.

The relationship between malignant lymphoma and GOMMID is not fully understood. Touchard et al. [5] showed that leukaemia/lymphoma cells were the major source of glomerular immunoglobulin deposits from immunofluorescence and electron microscopic studies. In the present case, although we failed to observe lymphoma cells with electron microscopy, their role was strongly suggested by the correspondence of the isotype of monotypic immunoglobulin deposited in the glomeruli with that of the serum paraprotein. On the other hand, the different patterns of Congo red staining, positive in the lymph node and negative in the glomeruli, were remarkable in this case. It would be suggested that each immunoglobulin deposition was related to the production of amyloid- or non-amyloid-forming κ light chains derived from different plasma cell clones. Diversity of the tissue microenvironment might also explain such different patterns of immunoglobulin deposition [7].

Case. A 46-year-old Japanese man presented with lymph node swelling and IgG-κ monoclonal gammopathy. Interestingly, although the patient developed AL amyloid deposition (κ type) in a cervical lymph node, no amyloid was recognized in the kidney.

Discussion. GOMMID was proposed to include the following criteria: (i) the presence of organized microtubular deposits (15 nm in width) in glomeruli without thrombi in the glomerular capillaries and/or renal arterioles; and (ii) monotypic immunoglobulin deposits, irrespective of the presence of detectable monoclonal immunoglobulin in patient serum and/or urine, type I cryoglobulinaemia or immunoproliferative disorder [5]. Our case satisfies these criteria and features both pathological and clinical aspects of GOMMID.

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Conflict of interest statement. None declared.
Rituximab is an alternative in a case of contra-indication of cyclophosphamide in Wegener’s granulomatosis

Sir,

We read with interest the report by Ferraro et al. [1] on the effectiveness of rituximab in refractory Wegener’s granulomatosis (WG). We report a case of relapsing WG with remission under rituximab, whereas cyclophosphamide was contra-indicated.

WG was diagnosed in February 2002 in a 57-year-old hypertensive patient with fatigue, renal failure (creatinine 334μmol/l), haematuria and serum PR3-ANCA at a titre of 1090 U/ml (positive >20). Renal biopsy disclosed nephroangiosclerosis and necrotizing glomerulonephritis. High dose corticosteroids and 12 cyclophosphamide pulses resulted in remission, but renal insufficiency led to dialysis. Maintenance therapy with azathioprine was given from August 2002 to July 2003. In February 2004, relapse occurred with fatigue and haemoptysis related to alveolar haemorrhage, with positive PR3-ANCA at a titre of 200 U/ml. Corticosteroids and a first cyclophosphamide pulse were administered but stopped because platelet count fell at 74,000/mm³. The alkaline phosphatase level was 334 UI/l (normal <100). Portal hypertension was demonstrated with grade II oesophageal varices due to veno-occlusive disease associated with nodular regenerative hyperplasia of the liver, probably induced by azathioprine. The patient received four weekly administrations of 375 mg/m² of rituximab, inducing complete remission. Mycophenolate mofetil (1.0 g/d) was subsequently administered as maintenance therapy. No relapse has occurred to date.

This case highlights the interest of rituximab in WG, in the case of contra-indication of cyclophosphamide. A few case reports show that rituximab is effective in WG [1–3], but prospective studies are necessary in order to evaluate its indications, since rituximab seems favourable as compared to other drugs.

Conflict of interest statement. None declared.

Sir,

Although uncommon, percutaneous renal biopsy can precipitate acute renal failure from ureteral obstruction, hypotension or parenchymal compression by perinephric haematoma [1–4]. We report a case of acute renal failure following percutaneous renal biopsy resulting from torsion and kinking of the main renal artery secondary to anatomic displacement of the affected kidney by a large retroperitoneal haematoma.

A 50-year-old man with a history of diabetes mellitus, hepatitis C and orthotopic liver transplantation presented with renal insufficiency of several months duration. The patient’s blood pressure was 128/49 mmHg, serum creatinine concentration (Scr) was 1.8 mg/dl and the urine albumin to creatinine ratio was 3.5 mg/mg. Additional serological work-up, renal sonogram and urine microscopy were unremarkable. Three months later, the Scr had increased to 2.6 mg/dl and a percutaneous kidney biopsy was performed.

Following the biopsy, the patient developed decreased blood pressure and increasing left flank pain. An emergent