fluorodeoxyglucose activity in the thicken wall of AAA (Figure 1B). A renal biopsy specimen showed interstitial nephritis with patchy pattern infiltration of plasma cells (Figure 1C). Immunostaining of anti-IgG4 antibodies detected that a lot of IgG4-positive plasma cells infiltrated into the interstitium, compatible with IgG4-positive multi-organ lymphoproliferative syndrome (IgG4+MOLPS) (Figure 1D). He was treated with prednisolone (PSL) 40 mg daily. His clinical manifestations, laboratory data and imaging tests were all improved within 2 weeks.

IgG4+MOLPS is a new clinical entity, characterized by hyper-IgG4 gamma-globulinaemia, IgG4+ plasma cell infiltration in involved tissue with a favourable response to steroids, and includes Mikulicz’s disease, autoimmune pancreatitis (AIP), retroperitoneal and mediastinal fibrosis, interstitial nephritis and many other inflammatory conditions affecting multiple organs [1]. Though AAA is the most common type of aneurysm, IAAA is a rare variant of AAA, which is seen in 5–10% of all cases of AAA [2]. IAAA has some similarities to retroperitoneal fibrosis and had been previously considered to be one member of chronic periaortitis as well as idiopathic retroperitoneal fibrosis. Recently, IgG4+ IAAA has been proposed to be estimated as ‘IgG4-related periaortitis’ together with retroperitoneal fibrosis [3]. 18F-FDG uptake is caused by increased glucose utilization, observed not only in malignant cells but also in inflammatory tissue [4]. 18F-FDG PET/CT has been recently reported as being useful to diagnose and follow-up AIP and associated extrapancreatic lesions [4]. In the present case, steroid therapy could improve immediately the clinical manifestations of IgG4+MOLPS, such as interstitial nephritis, interstitial pneumonia and IAAA. 18F-FDG PET/CT is useful for monitoring both the disease activity of IgG4+MOLPS and the effect of steroid therapy.

**Conflict of interest statement.** None declared.

**Supplementary data**

Supplementary data are available online at http://ndtplus.oxfordjournals.org.

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**Reversible proteinuria after adalimumab discontinuation in a patient with Crohn’s disease**

Sir,

Renal complications are not infrequent in patients with inflammatory bowel diseases and seem to be more prevalent in patients with Crohn’s disease. The spectrum of renal complications in Crohn’s disease patients involves nephrolithiasis, amyloidosis, renal hypertension, glomerulonephritis (from minimal change nephropathy to rapidly progressive crescentic), tubulointerstitial abnormalities and iatrogenic complications related to medications such as aminosalicylates and cyclosporine.

Anti-tumour necrosis factor (anti-TNFα) agents, including adalimumab, constitute a new generation of biological agents used in the treatment of a variety of autoimmune diseases such as rheumatoid and psoriatic arthritis, ankylosing spondylitis and Crohn’s disease. We report herein a Crohn’s disease patient diagnosed with proteinuria that was totally reversed after discontinuation of adalimumab.

A 38-year-old male with terminal ileum Crohn’s disease diagnosed 6 months previously was admitted to our hospital due to a relapse. Before admission, the patient was administered methylprednisolone 32 mg/day with sub-optimal response. On admission, the patient underwent routine screening to exclude underlying infection prior to prescription of anti-TNFα agents. Subsequently, the patient was started on adalimumab induction scheme (160 mg subcutaneously). Two weeks afterwards, the patient complained of lower limb oedema, and laboratory screening revealed proteinuria of 1600 mg/day without any evidence of other extraintestinal comorbidity. Adalimumab was discontinued and patient was followed up. Proteinuria resolved completely 4 weeks after adalimumab discontinuation, but we decided no adalimumab rechallenge and no renal biopsy. The patient was started on therapy with azathioprine 150 mg/day and is currently in excellent clinical status.

To the best of our knowledge, this is the first report of a patient with Crohn’s disease and adalimumab-associated proteinuria. Treatment with adalimumab may lead to antibody formation, but renal complications are rare and have so far been reported only in 11 patients with rheumatoid arthritis [1–3] but not in patients with inflammatory bowel disease. Of interest, in rheumatoid arthritis patients, proteinuria has also been reported during therapy with other than adalimumab biological agents such as infliximab and etanercept [3].

Renal histology, when performed in rheumatoid arthritis patients with adalimumab-related renal dysfunction, showed patterns of systemic lupus erythematosus-like...
syndrome [1], necrotizing and crescenting glomerulonephritis [3,5], minimal lesion glomerulopathy [2], extracapillary glomerulonephritis with IgA deposits or active follicular necrosis against a background of glomerular sclerosis [4].

It is noteworthy that, as with this Crohn’s disease case, favourable outcomes have been reported in all other rheumatoid arthritis cases. After adalimumab discontinuation and glucocorticoid [4] and/or immunosuppressive therapy [3,5], renal function recovered within a few weeks. Of interest, in one case proteinuria relapsed after adalimumab rechallenge [2].

In this case, the relative contributions of adalimumab and of the underlying Crohn’s disease to the development of proteinuria cannot be exactly determined. However, it is questionable whether in this particular patient adalimumab induction scheme was the only factor able to induce proteinuria. We believe that the complete reversibility of proteinuria after adalimumab discontinuation points towards adalimumab as a triggering factor to a strongly predisposed for a renal dysfunction individual.

As there have been some concerns regarding safety issues during administration of anti-TNF-α agents, it is of importance to understand the mechanism(s) of their interference in the normal kidney function. By this way, we may be able to properly screen and possibly identify high-risk groups before initiation of anti-TNF-α therapies. For the moment, careful patient selection for biological agents as well as regular follow-up can promptly diagnose and completely reverse rare or unexpected episodes.

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Lacking evidence for calcium-binding protein fetuin-A to be linked with chronic kidney disease-related pruritus (CKD-rP)

Sir,

Uraemic pruritus, now better known as chronic kidney disease-related pruritus (CKD-rP), is still a commonly experienced, tormenting and challenging symptom in patients with chronic kidney diseases [1].

There has been an ongoing discussion whether pruritus in chronic kidney disease is brought about by the common disturbance of calcium/phosphate homeostasis [2].

Recently it has been proposed that a vicious circle of metabolic derangements (malnourishment, inflammation, arteriosclerosis) may explain the exaggerated morbidity and mortality in a subset of haemodialysis patients [3]. Inflammation might be the most deleterious factor in this respect which, besides other factors, is related to the occurrence of CKD-rP and down-regulation of fetuin-A, an important calcium-binding circulating protein favouring tissue-calcifications in these patients [4].

Thus, we were interested in whether patients with CKD-rP display lower levels of serum fetuin-A.

Ten patients in a hospital-based haemodialysis centre complaining about CKD-rP were compared to another 12 patients who did not report to have suffered from CKD-rP at least 6 months prior to the interview. Patients with CKD-rP were asked to score the intensity of current pruritus using a visual analogue scale (VAS) ranging from 0 to 10. In both groups, 10 ml of blood was taken immediately after puncture of the arterio-venous fistula, and fetuin-A, 25-hydroxyvitamin D3 (25(OH)D3), total protein, albumin, calcium (corrected for serum albumin), phosphate, and high-sensitivity CRP (hsCRP) were measured.

Independent t-tests (continuous variables) and a χ²-test (gender) evaluated whether there were significant (P < 0.05) differences between patients with and without CKD-rP.

The mean pruritus intensity of patients with CKD-rP was 5.9 ± 1.9. After identifying one univariate outlier (hsCKP = 35.6) in the group of patients without CKD-rP, CRP was significantly higher in patients with CKD-rP. We failed, however, to find significant differences in serum-calcium, phosphate, fetuin A and 25-OH-Vitamin D3 between the two groups (Table 1). Additionally, there was no relationship between the intensity of CKD-rP and the metabolic factors measured (including CRP) in patients suffering from pruritus.

Although our study approach has methodological limitations (small number of patients, no matched pairs) the results suggest that neither calcium-binding protein fetuin-A levels nor 25(OH)D3 values were noticeably different in patients with CKD-rP compared to patients without CKD-rP. On the other hand, the marker for inflammation, CRP, was found to be significantly higher in patients with CKD-rP as shown before [5]. We hence believe that other inflammation-driven processes need to be studied in

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