showed the analytical changes depicted in Table 1 and the patient has not presented new episodes of fluid retention.

DN is a common complication of diabetes and is currently an important public health problem, as diabetic renal disease is the main cause of terminal chronic kidney disease in Western countries.[3] Diabetic patients with a history of DN who develop slow-onset proteinuria, are not usually subjected to a biopsy, on the assumption of the presence of DN. However, non-diabetic glomerular disease may also develop in diabetic patients, which is why a renal biopsy may be indicated.[4] In our case, the patient had longstanding diabetes mellitus, which was poorly controlled metabolically. We are unaware whether he had proteinuria prior to his first admission, although the onset of anasarca and fluid retention was sudden, so we decided to perform a renal biopsy and the diagnosis was DN.

In the medical literature cases of NS due to minimal change disease have been reported in diabetic patients.[5,6] In the case described by Donaire et al, the suspicion of a cause other than diabetes was founded on the short history of diabetes, the absence of retinopathy and the fact that a previous check-up proved negative for proteinuria.[5] Although in our case the established diagnosis was DN, the sudden onset of symptoms with severe proteinuria which led to fluid retention on more than one occasion and subsequently spontaneous remission, and then a proteinuria of less than 0.5g/24 h during follow-up, suggested the possibility that the patient might have a comorbid minimal change nephropathy. This might have gone unnoticed during the histological analysis when an underlying DN substrate was found and electron microscopy test was not performed. The patient might have had an unrelated infectious process prior to his first admission. In fact, he had developed cutaneous lesions on his upper limbs. This process could have been triggered by an immune mechanism, leading to an increase in glomerular permeability and, subsequently, severe NS with spontaneous remission some months later.

To conclude, we described a case of NS with clinical symptoms indicating a minimal change aetiology, which could have gone unnoticed in the renal biopsy because we found a DN histological substrate associated to the base pathology (long-term diabetes mellitus).

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Table 1. Follow-up of laboratory results

	Baseline	First admission	Second admission	Visit (first month)	Consultation (second month)	Visit (third month)	Consultation (fifth month)
PCR (mg/dl)	1.5	2.2	2	1.8	1.7	1.6	1.6
Albumin (g/dl)	3.8	3	2.7	2.2	2.5	3.1	4
Cholesterol (mg/dl)	172	270	310	273	171		104
Proteinuria (g/24h)		26	17	5.95	5.06	0.47	0.34
CrCl (ml/min)		41	40	34	42	34.7	47

PCR: plasma creatinine; CrCl: creatinine clearance.

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Immunotactoid glomerulopathy and tuberculosis: a novel association

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To the Editor,

Tuberculosis is associated with a variety of glomerular manifestations. However, association with immunotactoid glomerulopathy has never been reported. We encountered a case of 37-year-old gentleman with such a novel presentation.

A 37-year-old gentleman presented with swelling all over the body for 6 weeks. His past history revealed recent history of pulmonary tuberculosis 9 weeks back. He was presently on 2 drug anti-tubercular treatment (ATT) (isoniazid and rifampicin) after first 8 weeks of 4 drugs, which additionally included pyrazinamide and ethambutol. At the time of diagnosis of tuberculosis, he was also found to have

stage 1 hypertension and was started on hydrochlorthiazide 12.5 mg daily. There was no history of hematuria, past renal disease or any other systemic disorder. Physical examination revealed pitting edema and no other notable findings.

Laboratory data showed hemoglobin 10.1 g/dl, white cell counts of 6800, blood urea 68 mg/dl, serum creatinine 1.4 mg/dl, eGFR by MDRD formula 59 ml/min/1.73 m^2, protein 4.5 g/dl, albumin 1.7 g/dl, cholesterol 356 mg/dl, Hepatitis-B and C and HIV-1 and 2 negative, ANA and cryoglobulins negative, normocomplementemia, urine –, protein 3+, RBC 4-6 and WBC 1-2/hpf, casts-nil and 24-hr urine protein 4.8 g (non-selective). Liver functions tests were within normal limits. An ultrasound guided renal biopsy was performed.

On light microscopy, glomeruli exhibited varying degrees of mesangial expansion, negative silver staining and congo red staining and some thickening of peripheral capillary walls. Immunoflourescence was positive only for IgG in mesangium and peripheral capillary walls. Electron microscopy showed microtubules >30 nm arranged focally in parallel in mesangium suggesting immunotactoid glomerulopathy (ITG) (figure 1). Further work-up showed a negative serum and 24-hr urine immuno-fixation electrophoresis. Imaging studies done for lymphoproliferative disease as an etiology were negative too.

He was treated with ATT for a total of 6 months. His blood pressures were kept under control with ramipril 10 mg daily. His proteinuria decreased to 1.1g/day at 6 months. At 2 years of follow-up, his serum creatinine is 3 mg/dl with eGFR of 23. We offered a repeat renal biopsy during this period which the patent did not consent.

ITG is distinct rare morphologic entity characterized by microtubular glomerular deposits often ranging from 34 to 49 nm in diameter organized in parallel arrays. It usually occurs in older individuals presenting with nephrotic syndrome,

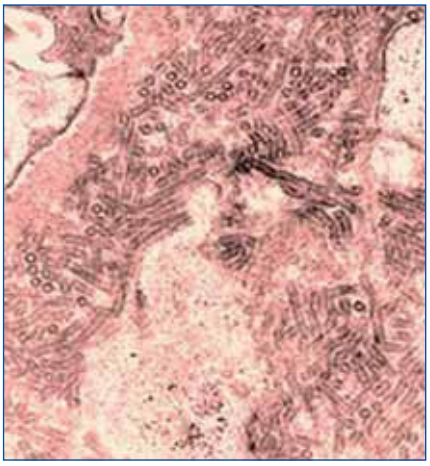

Figure 1. Microtubular deposits of >30 nm seen in mesangium (on electron microscopy magnification x15000).

hematuria and renal insufficiency. The term was introduced by Schwartz et al in 1980's, where they described this disease as a glomerular disease characterized by highly organized crystalline structure of immune deposits in absence of systemic diseases such as amyloidosis, cryoglobulinemia, paraproteinemia, and systemic lupus erythematosus[1]. In most instances, an underlying lymphoproliferative disorder is found. Association with HIV, sickle cell disease, hypereosinophilic syndrome and recurrence in transplanted kidneys has been reported[2-5].

Our case showed a temporal association with tuberculosis. Though tuberculosis or its treatment is shown to be linked to a variety of glomerular diseases such as amyloidosis, minimal change disease, IgA nephropathy, and collapsing glomerulopathy[6-9] but as causality with ITG has never been reported. It is difficult to prove whether tuberculosis per se caused ITG, however treatment of tuberculosis resulted in partial remission.

The exact pathogenesis of ITG remains to be elucidated. Like lymphoproliferative diseases, tuberculosis is also an inflammatory disorder. It might be possible that immune dysregulation in tuberculosis or systemic inflammatory mediators cause defects in critical podocyte cellular functions involved in clearance of filtered and retained immunoglobulins. This would end up in formation of immunotactoids.

The treatment strategies for ITG have been variable, though there has been a case of ITG exhibiting nephrotic syndrome successfully treated with corticosteroids and antihypertensive therapy[10]. We did not subject our patient to steroids as there was a potential risk of flaring tuberculosis with high doses of corticosteroids. However, we did not try rituximab as data for this agent is limited at present[5].

Our patient was relatively young as compared to most other cases and progressed to chronic kidney disease stage 4 over a span of 2 years. The natural course of the disease is progression to end-stage renal disease (ESRD) within 7 months to 10 years. However, in a recent report a patient with a diagnosis of ITG developed acute kidney injury (AKI) and ESRD within 1 week of initial presentation[11].

To conclude, to the best of our knowledge, our case is the first report of an association of ITG with tuberculosis. There could be a possible causal relationship between mycobacterial infections and ITG. In addition to search for lymphoproliferative disorder and HIV, tuberculosis as an etiology should be kept in mind in a case of ITG.

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Sarcoidosis: diagnosis from the renal function and hypercalcaemia study

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To the Editor,

Sarcoidosis is a multi-systemic granulomatous disease of unknown aetiology, which is characterised by the presence of non-caseating epithelioid granulomas. Renal involvement is uncommon in sarcoidosis and, in cases where it does occur, it is associated with hypercalcaemia, hypercalciuria, increased levels of calcitriol and parathyroid hormone (iPTH) suppression.[1]

We present the case of a 64-year-old male patient with a family history (patient's father) of emphysema. Incidents of note in his medical history include various episodes of macrohaematuria when the patient was 15, pleuritis at the age of 30, rhinitis at the age of 60 and glaucoma. He was admitted to the nephrology department with suspected renal failure. The patient presented toxic syndrome and had been vomiting and suffering from diarrhoea for two months. The only notable findings during the physical examination were a painful, enlarged spleen and high blood pressure (162/90mm Hg). The following analytical findings were of note: haemoglobin: 11.7mg/dl, calcium: 12.0mg/dl, phosphorus: 3.0mg/dl, iPTH: 0.3pg/ml (normal values 10-65pg/ml), alanine aminotransferase (ALT): 22U/l, aspartate aminotransferase (AST): 69U/l, gamma glutamyl transpeptidase (GGT): 69U/l, ferritin: 495ng/ml, uric acid: 7.0mg/dl, urea: 56mg/dl, creatinine: 2.13mg/dl, estimated glomerular filtration rate (eGFR): 33ml/min, proteinuria: 0.334g/24 hours and in the sediment there were only 10-20 erythrocytes per field. Calciuria was 896mg/24h. Angiotensin converting enzyme (ACE) levels: 167U/l (normal range 8-55), 25-(OH)-vitamin D3: 69pg/ml (normal range 9-52), 1,25-(OH)2-vitamin D3: 89pg/ml (normal range 15-60pg/ml). The other biochemical parameters, and the immunological and tumour marker results were normal. The chest X-ray revealed an interstitial pattern at the base of the right lung. In the thoraco-abdominal computed tomography (CT) scan, the lung parenchyma analysis showed diffuse, non-specific interstitial reinforcement in both lungs. The abdominal exploration revealed small inflammatory/reactive retroperitoneal adenopathies, homogenous spleen enlargement and bilateral renal microlithiasis. A renal ultrasound scan confirmed the morphology, position and size of the kidneys to be normal. Gammagraphy with gallium revealed moderately severe inflammation of the parotid glands and the base of the right lung. The renal histology tests detected 13 diagnostically useful glomeruli. Three of them were completely sclerotic, and the rest had preserved their structure and morphology. Focal ischaemic ondulations and minimal mesangial segmental increases were identified. Glomerular cell proliferation was not observed. No granulomas were observed, and patches of interstitial fibrosis and tubular atrophy, which together accounted for 10% of the cylinder, were identified. Two interlobular arteries without morphological changes were identified. Immunofluorescence assays using anti-IgG, IgA, IgM and C1q, C3, kappa and lambda sera were negative. Pulmonary histology samples obtained by fibrobronchoscopy and transbronchial biopsy showed the presence of a non-caseating granuloma.

Sarcoidosis was diagnosed and prednisone was administered, starting with a dose of 1mg/kg body weight and progressively reducing the dose from the first month onwards. After three months, the constitutional syndrome disappeared, progressive weight gain was achieved and renal function improved significantly (creatinine 1.3mg/dl and eGFR 58.8ml/m). The patient's calcaemia (calcium 8.9mg/dl) and anaemia (Hb 13.0mg/dl) were corrected and his iPTH (32pg/ml) and ACE (13U/l) levels were normal.

Sarcoidosis is a multi-systemic disease of unknown aetiology and the pulmonary and lymphatic systems are the most commonly affected (30%-60% of cases). Hypercalcaemia (2%-10%) and hypercalciuria (6%-30%) can cause nephrocalcinosis, lithiasis and renal insufficiency. The prevalence of tubulointerstitial nephritis ranges from 7% to 27%, although chronic renal failure develops in less than 1% of cases, according to a number of retrospective studies.[2] Sarcoidosis patients often have high levels of vitamin D and ACE, which are synthesised by the epithelioid cells of the granuloma.[3,4] In the case that we present the clinico-radiological involvement was minimal and the diagno-