letters to the editor

- Krishnamurthy S, Samanta D, Yadav S. Renal amyloidosis secondary to childhood tuberculosis: a report of two cases. J Postgrad Med 2009;55:121-3.
- Mori S, Matsushita Y, Arizono K. Minimalchange nephrotic syndrome associated with isoniazid in anti-tuberculosis chemoprophylaxis for a patient with rheumatoid arthritis. Intern Med 2011;50:253-7.
- Ortmann J, Schiffl H, Lang SM. Partial clinical remission of chronic IgA nephropathy with therapy of tuberculosis. Dtsch Med Wochenschr 2010;135:1228-31.
- Rodrigues CE, Sette LH, Torritani J, Malheiros DM, Titan SM, Barros RT, et al. Tuberculosis-associated collapsing glomerulopathy: remission after treatment. Ren Fail 2010;32:143-6.
- 10. Kinomura M, Maeshima Y, Kodera R, Morinaga H, Saito D, Nakao K, et al. A case of immunotactoid glomerulopathy exhibiting nephrotic syndrome successfully treated with corticosteroids and antihypertensive therapy. Clin Exp Nephrol 2009:13:378-84.
- Jain S, Chhabra D. A case of immunotactoid glomerulopathy with rapid progression to end-stage renal disease. Scientific World J 2009;9:1348-54.

A. Gupta, A. Khaira

Division of Nephrology. University of Ottawa. Ottawa, Ontario (Canada). **Correspondence:** A. Gupta Division of Nephrology. University of Ottawa, Riverside Drive, K1G0E8, Ottawa, Ontario. Canada. parthankur@yahoo.com parthpreeti@rocketmail.com

Sarcoidosis: diagnosis from the renal function and hypercalcaemia study

Nefrologia 2011;31(3):371-2

doi:10.3265/Nefrologia.pre2011.Mar.10832

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 noncaseating 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-

letters to the editor -

sis was confirmed by transbronchial biopsy. The analytical profile was indicative of sarcoidosis (hypercalcaemia, hypercalciuria, high levels of vitamin D and ACE and substantial iPTH suppression).

Renal function impairment in sarcoidosis is generally due to hypercalcaemia, hypercalciuria and nephrocalcinosis, although nephrolithiasis, glomerulopathies and interstitial nephritis (with or without sarcoid granuloma) form part of the spectrum of renal pathologies in sarcoidosis.¹

Corticosteroids⁵ are the treatment of choice and in the case presented here a good response was obtained. Renal involvement without the lungs being affected is very rare² and in this case it was not possible to establish that this was the case until the lung biopsy was performed. When we are faced with a case of renal failure associated with hypercal-caemia, sarcoidosis should be suspected, even though there is no clinical manifestation of lung pathology.

- Gobel U, Kettritz R, Schneider W, Luft F. The protean face of renal sarcoidosis. J Am Soc Nephrol 2001;12(3):616-23.
- Baughman RP, Teirstein AS, Judson MA, Rossman MD, Yeager H Jr, Bresnitz EA, et al. Clinical characteristics of patients in a case control study of sarcoidosis. Am J Respir Crit Care Med 2001;164(10 Pt 1):1885-9.
- Sharma OP. Vitamin D, calcium, and sarcoidosis. Chest 1996;109(2)535-9.
- Romer FK. Angiotensin-converting enzyme in sarcoidosis. Acta Med Scand 1979;206(1-2):27-30.
- 5. Nunes HBD, Valeyre D. Sarcoidosis treatment. Rev Prat 2008;58(10):1099-104.

O. Ibrik¹, R. Samon¹, A. Roda¹, R. Roca¹, J.C. González¹, J. Viladoms¹, J. Vilaseca², M. Serrano²

¹ Servicio de Nefrología. Hospital de Mollet. Mollet del Vallès. Barcelona.

² Servicio de Neumología. Hospital de Mollet. Mollet del Vallès. Barcelona.

Correspondence: O. Ibrik

Servicio de Nefrología. Hospital de Mollet. Pau Casals, 20, 3.º-1.ª. 08150 Mollet del Vallès. Barcelona.

22721aii@comb.cat oibrik@yahoo.es

Membranous glomerulonephritis in a patient with syphilis

Nefrologia 2011;31(3):372-3

doi:10.3265/Nefrologia.pre2011.Mar.10819

To the Editor,

La glomerulonefritis membranosa Membranous glomerulonephritis (MGN)¹ is the second most prevalent renal pathology to be identified in biopsies. One of the most common causes of nephrotic syndrome in the adult population, it is characterised by the formation of immune complexes, predominantly IgG and complement, on the subepithelial side of the glomerular capillaries, and this is associated with increased proteinuria.²

In general, its aetiology is idiopathic or primary and, less frequently, secondary (immunological, infectious, drug and medication-related, or neoplastic).

Unfortunately, it is difficult to distinguish primary from secondary forms by histological means,² so explicit clinical information, including the age of the patient, history of exposure to medicines or toxic substances, serological tests and suspected neoplasias which are linked to the pathology, is required.

The importance of serological tests lies in their ability to confirm the diagnosis. In the case of syphilis screening, non-treponemal tests are performed: the VDRL (Venereal Disease Research Laboratory) and RPR (rapid plasma reagin) tests. If the results are positive, the more specific treponemal tests are performed to confirm the diagnosis: FTA-ABS (absorption of fluorescent antibodies by Treponema) and MHA-TP (Treponema pallidum microhaemagglutination). They must be repeated three and six months later to ensure the response to treatment.

The case which concerns us is relevant, owing to the small number of publications on the association between syphilis and MGN.

The patient was a 27-year-old, white, Caucasian male with a history of cryp-

torchidism, adenoidectomy and amygdalectomy in childhood. He was an active smoker, a social drinker and a homosexual. Two months before being assessed by our department and, coinciding with a slight pharyngodynia, an induration had appeared in the patient's right groin, as well as ulcerated serpiginous lesions on the penis and a whitish urethral discharge, which was initially treated with azithromycin. While waiting for the serological results, maculopapular lesions were observed in the surrounding area on the thighs and trunk. They spread to the patient's feet and hands, progressing through different phases with no signs of fever, and accompanied by oedema of the lower limbs and genitals, with a slight increase in the abdominal perimeter and a decrease in diuresis, which is why the case was reported to us. The patient's urine was normal in colour, with no evidence of dysuria or blood in the urine. Blood pressure (BP) was within normal limits.

The analytical findings of note were as follows: urea: 61mg/dl; creatinine: 1.73mg/dl; normal ions; total protein: 4.4g/dl; albumin: 1.8g/dl; total cholesterol: 295mg/dl, HDL: 61mg/dl, LDL: 206mg/dl, triglycerides: 140mg/dl and normal hepatic enzyme levels. Significant findings in the urine analysis included proteinuria: 13.4g at 24h, 250 red blood cells per microlitre and a negative leukocyte count. The haemogram and coagulation were normal, except for an FTP of 762g/l. Autoimmunity assays: antinuclear antibodies (ANA) and antineutrophil cytoplasmic antibodies (ANCA) negative; complement and protein tests were normal. Serology tests for hepatitis B (HBV), hepatitis C (HCV) and human immunodeficiency (HIV) viruses were negative. Positive 1/32 titre RPR (rapid plasma reagin) and FTA (anti-Treponema antibody) results.

Renal ultrasound showed the kidneys to be normal in size. The echocardiogram was within normal limits and no lung parenchyma changes were detected in the chest X-ray.