defect, and can be either congenital or acquired.[1-3,5-8] Its prevalence is hard to estimate and varies from 1.6% and 2.9% to 10%, according to studies.[3,5,6] It appears more commonly in females and in the right hemithorax,[5] with a tendency to manifest at the start of the peritoneal dialysis technique, although it is not unusual for it to appear months or even years later. There are two factors that are combined in its aetiology and pathogenesis: on the one hand, the increase in intra-abdominal pressure and, on the other, the existence of continuity solutions in the pleuro-peritoneum, either congenital or acquired.[7,8] Peritonitis episodes can also worsen congenital diaphragmatic defects, although we have only found two reported cases and both are quite old, confirming the scarceness of this possibility.[2,3] Clinical results are diverse, depending on the amount of pleural effusion. In some studies, up to 25% of the cases go unnoticed.[5] In others, there may be paroxysmal dyspnoea, thoracic pain and coughing, together with a decrease in ultrafiltration. The examination showed abolition of breath sounds, dullness on percussion and no transmission of vocal vibrations. Diagnosis was via chest X-ray and pleural fluid analysis (with higher glucose concentration than plasmatic). Stainings can be used (methylene blue) and peritoneography with contrast medium to reveal the existence of pleuro-peritoneal communications, although these methods can cause peritoneal irritation. Currently, the most common technique applied is peritoneal scintigraphy with technetium-99.[4,7] Several therapeutic options have been reported: transitory haemodialysis with a return to peritoneal dialysis with low volumes or with a cycler, pleural tap, pleurodesis with chemical substances or autologous blood transfusion and direct surgical repair or thoracoscopy.[3,5-8] Nonetheless, treatment results are not very encouraging and in a high proportion of cases, the patient is definitively transferred to dialysis.[3,5-7]

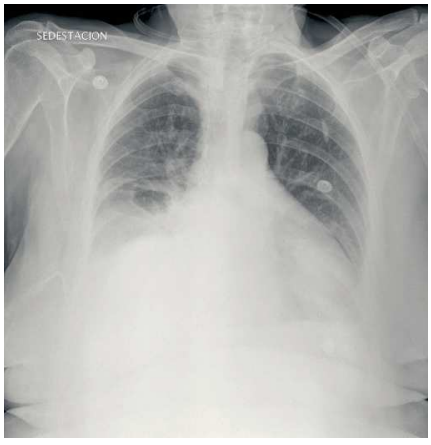

Figure 1. Hydrothorax.

In our patient, advanced in age, the increase in intra-abdominal pressure due to the peritoneal dialysis gave rise to, firstly, a hernia. This increase in pressure with the structural weakness of the diaphragmatic parietal peritoneum caused by peritonitis generated pleuro-peritoneal communication, giving rise to hydrothorax. Given these complications haemodialysis was chosen as the most appropriate extrarenal purification technique, although it was against the patient's will.

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V. Oviedo Gómez, I. Sánchez García, P. Martín Escuer, E. Hernández García, J. Martín Gago, F. Sousa Pérez, J.M. Monfá Bosch

Nephrology Department. Palencia Health Care Complex. Palencia, Spain

Correspondence: Victoria Oviedo Gómez
Sección de Nefrología.
Complejo Asistencial de Palencia.
Avda. Donantes de sangre, s/n. 34005 Palencia.
voviedo71@hotmail.com
majuelo55@yahoo.es

Membranous glomerulonephritis secondary to Hashimoto's thyroiditis

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Dear Editor,

Thyroid pathology has a high prevalence in the general population, although autoimmune aetiology is rare. It is associated with a variety of glomerulonephritis types in very few cases, the most frequent being membranous glomerulonephritis.[1,2] We present a case of membranous glomerulonephritis secondary to Hashimoto's thyroiditis.

We present the case of a woman aged 66, admitted to our department with proteinuria, generalised oedemas and new-onset hypertension, resistant to prescribed treatment. Analytical tests showed: urea 78mg/dl, creatinine 1.1mg/dl; MDRD-4 formula creatinine

clearance 48ml/min; total proteins 4.6g/dl; albumin 1.2g/dl. Protein profile compatible with nephrotic syndrome. Cholesterol: 484mg/dl. Triglycerides: 180mg/dl. LDL: 386mg/dl. Autoimmunity and complement within normal ranges. Negative circulating immune complexes. TSH: 10.17mU/l, T4: 0.78mg/dl. Anti-microsomal antibodies: 84U/ml. Antithyroglobulin antibodies: 4U/ml. Anti-TSH receptor antibodies: 1.6U/l. Proteinuria to 10g/24 hours. An ultrasound-guided renal biopsy was performed with an anatomopathological result of membranous glomerulonephritis. The gynaecological study was normal, as was the abdominal ultrasound and the thoracic-abdominal-pelvic CT scan. Membranous glomerulonephritis probably secondary to Hashimoto's thyroiditis was diagnosed and treatment with levothyroxine, statin and dual blockade of the renin-angiotensin system was commenced.

During follow-up, treatment had to be suspended with ACE inhibitors due to intolerance, despite which there was a progressive decline in proteinuria up to values of 1.5g/24 hours after 6 months of hormonal treatment, and TSH, T3 and T4 figures normalised.

Hashimoto's thyroiditis treatment is not clear; hormone substitution therapy is recommended in cases of thyroiditis with clinical repercussion, although there is no clear consensus in the subclinical cases.[3] Our case shows how normalisation of the thyroid hormone values by administering levothyroxine has a linear repercussion in relation to the proteinuria detected. Even so, not all the cases described present this relationship.[4]

Lastly, we deem it necessary, as other authors have stated, to perform an analysis of the thyroid hormones in every patient with nephrotic syndrome as part of an aetiological screening process.[5]

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C. Ruiz-Zorrilla López, B. Gómez Giralda, A. Rodrigo Parra, A. Molina Miguel
Nephrology Department.
Río Hortega University Hospital. Valladolid, Spain.
Correspondence: Carlos Ruiz-Zorrilla López
Sección de Nefrología.
Hospital Universitario Río Hortega. Valladolid.
carlosruizzorrilla@hotmail.com

"Horseshoe kidney", renal adenocarcinoma and nephrotic syndrome

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Dear Editor,

"Horseshoe" kidney (HK) is one of the most common congenital malformations of the genitourinary system.[1] It is often associated with other renal or extrarenal anomalies, among which are tumours[2] and several types of glomerular diseases.[3] We describe a patient with HK, renal adenocarcinoma (RAC) and nephrotic syndrome with morphological substrate of focal segmental glomerulosclerosis (FSG) as well as the possible relationship between the two.

We present the case of a 38-year-old male patient referred for nephrotic syndrome. He is a smoker, has Hodgkin's disease and ischaemic heart disease. He is found to have a HK in a routine ultrasound.

A week before admittance (October 2007), he starts showing symptoms of oedemas. The biochemistry showed: proteinuria 18g/24h, serum albumin 2.1g/dl and hyperlipaemia, associated with microhematuria and serum creatinine 1.6mg/dl. Examination results: blood pressure 103/60mmHg, body mass index (BMI) 21kg/m^2 and generalised oedemas. Normal haemogram and coagulation study. ANA, anti-DNA, ANCAS, anti-MBG antibodies, cryoglobulins, lupus anticoagulant, anticardiolipin antibodies and hepatitis B and C antivirus antibodies as well as human immunodeficiency virus (HIV), all negative. The ultrasound and CT scan confirmed HK, with a tumour on the upper pole of the right kidney (Figure 1a). The cystography was normal. A nephrectomy with exeresis of the inferior isthmus is performed. Following surgery there was localised bleeding and partially recovered acute renal failure (creatinine 3mg/dl).

The piece removed in the nephrectomy measured 14x6cm with a tumour measuring 4x4.5cm. The microscopic study confirms renal adenocarcinoma with a pattern of chromophobe and eosinophilic cells (Figure 1b). The renal vein was not infiltrated.

The optical microscope study of an adjacent wedge displayed larger glomeruli, some of which were hyalinised. In the rest, there was an increase in the mesangial matrix and capillary collapse with several foam cells, interstitial fibrosis and infiltration of inflammatory cells. Mesangial and parietal deposits of IgM and C3 were