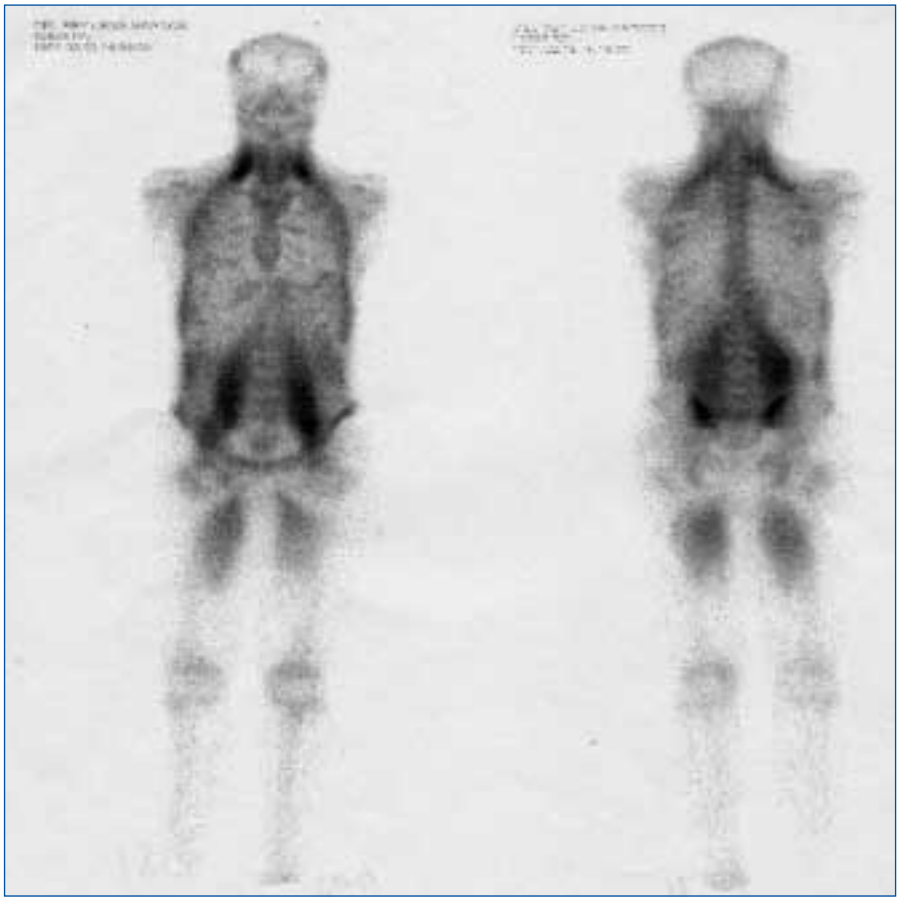

Figure 1. Scintigraphy with abnormal distribution of the radiotracer showing severe deposition at the abdominal muscle groups and the extremities compatible with rhabdomyolisis.

from the deltoid muscle was performed. The immunohistochemistry revealed type 2 carnitine-palmitoyl transferase (CPT) deficiency. The familial study disclosed that one sister was also affected.

Metabolic myopathies are a small percentage of rhabdomyolysis causes. However they are a preventable cause of acute renal failure, which very often goes unnoticed. CPT deficiency is the most frequent metabolic myopathy.[1,2] Lipids are an important energy source for resting muscles and during sub-maximal exercise. In case of CPT deficiency fatty acids do not enter in the mitochondria to be oxidized and no energy is obtained. The consequence is muscle destruction or rhabdomyolysis. This condition is the most frequent cause of recurrent myoglobinuria with no clear trigger and should be always suspected in these patients.[3]

The clinical picture consists of recurrent episodes of muscular weakness, myalgias, rhabdomyolysis or acute renal failure.[4,5] The episodes can be elicited by viral infections, exercise, prolonged fasting or fever. In two thirds of the patients the disease presents in the first or second decade.

The treatment is to avoid the factors that can trigger rhabdomyolysis, like prolonged fasting, to eating a low-fat high-carbohydrate diet, frequent meals and with excessive carbohydrates intake after exercise.

Renal acute failure is due to intratubular deposition of myoglobin. Early volume reposition,[6] and urine alkalinization[7] with calcium and potassium monitoring are fundamental to prevent it. Manitol use in acute renal failure is controversial, but in case of rhabdomyolysis it appears to reduce interstitial edema and to uptake free radicals. The beneficial effect has been demonstrated in patients with serum CK levels higher than 30,000 U/L.[8]

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Acute post-estreptoccocal glomerulonephritis in the elderly

Nefrología 2008; 28 **(1)** 113-114

To the editor: Post-streptococcal glomerulonephritis (PSGN) is the prototype of acute glomerulonephritis. It is heralded by a group A b-hemolytic streptococcus infection.[1,8] The evolution is benign in children, and poorer with older age[2] and in case of renal acute failure.[5] Approximately in 95% of the cases renal function is recovers in within 3-4 weeks. The evolution in the elderly is less predictable.[1,4,5] Irreversible renal failure occurs in less than 1% of children and in a higher percentage of adults.[6,7,9] The treatment consists of antihypertensive drugs, diuretics and antibiotics, and sometimes hemodialysis may be required.[1,3,8] Immunosuppressive therapy is indicated in case of glomerulonephritis with crescent formation.[5] Steroids appear to be efficacious in adults with PSGN and nephrotic syndrome.[3]

We present a 78 years old male who was admitted because of cardiac failure of 48 hours and oligo-anuria, together with renal function worsening in the last week. The blood analysis revealed

Cr 4.2 mg/dL (former Cr was 1.4 mg/dL), microhematuria and proteinuria of 500 mg/dL. Two weeks ago he had acute pharingotonsillitis and gout, and treatment with NSAIDS and colchicine was prescribed. He had a history of high blood pressure, coronary heart disease and prostatic hyperplasia. On physical exam he was eupneic, had no fever and appeared in good health status. Blood pressure was high, basal crackles were heard in lungs and pitting edemas were evident in both legs. Laboratory findings disclosed glomerular filtration worsening, hyperuricemia, hypoalbuminemia, and mild anemia and leukocytosis. ESR, rheumatoid factor and ASLO values were elevated. The level of C3 was decreased and other immunological parameters were normal. Lipid levels and proteinogram were normal. The Bence Jones selective proteinuria was negative as well as the urine culture. Abdominal ultrasound showed normal looking kidneys, and the echocardiography was also unremarkable. The X-ray film showed pulmonary vascular redistribution. The analysis of pharyngeal exudate disclosed beta-hemolytic Streptococcus.

We initiated treatment with furosemide and antihypertensive drugs and negative balances were achieved. ACE inhibitors and NSAIDS were discontinued. The clinical evolution was favorable, blood pressure was controlled, but renal function did not change. When we received the results of the immunological study we suspect a diagnosis of PSGN and initiated treatment with oral amoxycillina clavulanate for 2 weeks. Renal function further worsened and a renal biopsy was performed on the 20th day of in-hospital stay.

The pathological study revealed diffuse proliferative mesangial and capillary glomerulonephritis (11% glomerular sclerosis, 22% epithelial crescents, diffuse increase of mesangial and endothelial cellularity, and chronic inflammatory infiltrate in the interstitium. Immunofluorescence showed basal membrane and mesangial granular depositions of C3, IgG, IgA, C4, C1q. (fig. 1)

Prednisone (1 mg/kg/day) was initiated, penicillin G sodium was intravenously administered during fourteen days and hemodialysis was required. When the patient was discharged he had lost 6 kg of weight, pharyngeal swab exam was negative, C3 fraction had increased and renal function had improved (Cr 3.1 mg/dL). Steroid treatment was maintained for 17 months and the blood pressure was controlled with 4 drugs. The complement value was normal after two months and the ASLO levels were normal after 6 months. The patient has currently normal blood pressure, good health status and his renal function is stable (Cr. 1.8 mg/dL).

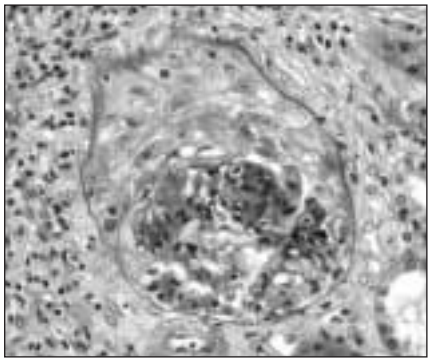

Figure 1. Widespread extra-capillary hypercellularity (epithelial crescent) and glomerular mesangial proliferation. PAS x 40 approx.

There are few reported cases of PSGN in the elderly, perhaps because the incidence of the disease is very low in this age. For this reason the evolution is not uniformly described, although it is generally accepted that renal biopsy should be performed early, as the clinical picture can be mistaken with other conditions and also because the outcome can be poor. It is indicated to initiate immunosuppressive therapy either with prednisone or with other agents. The decision of the agent and the duration of the therapy should be individualized.

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Hemoperitoneum and endocarditis

Nefrología 2008; 28 **(1)** 114-115

Summary

The incidence of hemoperitoneum varies from 6% to 57% in premenopausal women. Bloody peritoneal dialysate may be the result of the peritoneal dialysis procedure or may be due to factors unrelated to renal disease. The Libman-Sacks endocarditis was described for the first time in 1924, is characterized for verrucous lesions in the surfaces valves and has been intimately associated with the presence of antiphospholipid antibodies. We send a case of a patient in program of Dialysis peritoneal that presented an Libman-Sacks endocarditis and hemoperitoneum.

Key words: *Antiphospholipid syndrome. Endocarditis. Peritoneal dialysis. Hemoperitoneum.*

Resumen

La incidencia de hemoperitoneo en diálisis varia del 6% hasta el 57% en mujeres premenopáusicas. El sangrado peritoneal puede ser el resultado de un proceso relacionado con la diálisis o no estar relacionado con la enfermedad renal. La endocarditis de Libman-