

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 pharyngotonsillitis 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 amoxicillina 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

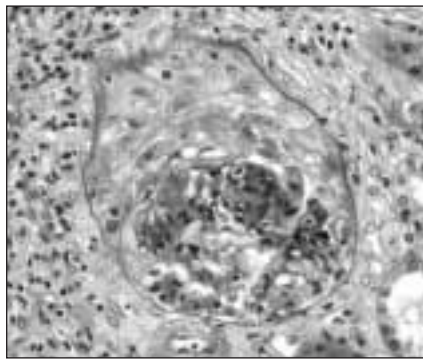


Figure 1. Widespread extra-capillary hypercellularity (epithelial crescent) and glomerular mesangial proliferation. PAS x 40 approx.

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).

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.

- Rodríguez-Iturbe, B. Glomerulonefritis endocapilar aguda. En: Hernando L, Aljama P, Arias M (Ed): *Nefrología Clínica* 2.ª Edición. Editorial Médica Panamericana. pp. 308-314, Madrid-España, 2004.
- Lavjay Butani. Prolonged hypocomplementaemia after post-streptococcal glomerulonephritis. *Nephrol Dial Transplant* 16: 869, 2001.
- Raff A, Hebert T, Pullman J, Coco M. Crescentic post. streptococcal glomerulonephritis with nephritic syndrome in the adult: is aggressive therapy warranted? *Clin Nephrol* May; 63 (5): 375-80, 2005.
- Hsu YH, Yang AH, Chen TW, Huang TP. Focal segmental glomerulosclerosis after

poststreptococcal glomerulonephritis in the elderly: a case report. *Zhonghua Yi Xue Za Zhi* (Taipei) May; 61 (5): 301-5, 1998.

- Vijayakumar M. Acute and crescentic glomerulonephritis. *Indian J Pediatr* Dec; 69 (12): 1071-5, 2002.
- Baldwin DS. Poststreptococcal glomerulonephritis. *Am J Med* 1077; 62: 1
- Pinto SW, Sesso R, Vasoncelos E y cols. Follow-up of patients with epidemic poststreptococcal glomerulonephritis. *Am J Kidney Dis* 38: 249, 2001.
- Couser WG. Glomerulonephritis. *The Lancet* May (9163): 509-515, 1999.
- Melby PC, Musick WD, Luger AM, Khanna R. Poststreptococcal glomerulonephritis in the elderly. Report of a case and review of the literature. *Am J Nephrol* 7 (3): 235-40, 1987.

R. Pérez Morales, J. Pérez Martínez, F. Llamas Fuentes and E. Andrés Mompean

Department of Nephrology. University Hospital of Albacete.

Correspondence: Rosa Pérez Morales rosa5ve@hotmail.com. Hospital de Albacete. Calle Hermanos Falcó. 02002 Albacete.

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 varía 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-

Sacks se describió por primera vez en 1924, se caracteriza por lesiones de tipo verrugoso implantadas en las superficies valvulares y está íntimamente ligada a la presencia de anticuerpos antifosfolípido. Enviamos el caso de una paciente en programa de Diálisis peritoneal que presentó una endocarditis de Libman-Sacks, y cursó con hemoperitoneo.

Palabras clave: Síndrome antifosfolípido. Endocarditis. Diálisis peritoneal. Hemoperitoneo.

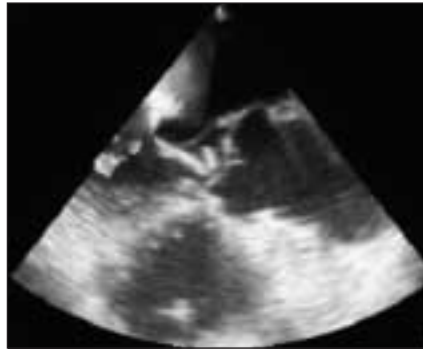


Figure 1.

To the editor: The incidence of hemoperitoneum in patients in dialysis varies from 6% to 57% in postmenopausal women.¹ The appearance of blood in the peritoneum can be related either with renal disease and dialysis or not.

Libman-Sacks endocarditis was first described in 1924. It is characterized by vegetating lesions implanted on valvular surfaces and is closely related to the presence of anti-phospholipid antibodies.^{2,3} We report a patient in peritoneal dialysis, who developed Libman-Sacks endocarditis and hemoperitoneum.

The patient is a 51 year-old woman with CRF secondary to IgA mesangial nephropathy, who initiated renal replacement therapy by peritoneal dialysis in May of 2006. She had a history of natural interruption of a pregnancy on the sixth month in 1981 and thrombosis in arteriovenous fistula in 1988.

In August of 2006 she was admitted because of a clinical picture compatible with stroke (facial palsy and dysarthria). During the in-hospital stay an endocarditis on native aortic valve was detected by heart ultrasound. Doppler echocardiography is considered elective for the diagnosis of endocarditis. The frequency of valvular involvement is 18-50%.⁴ The frequency of valvular disease detection is even higher on transesophageal echocardiography.⁵ In the reported case thoracic echocardiography showed an enlarged vegetation emerging from the left coronary veil that caused mild aortic regurgitation (fig. 1).

The patient had no fever. On physical exam no cardiac murmurs were heard. Blood cultures, viral serologies, and ANA and anti-DNA antibodies were negative. Lupus anticoagulant was posi-

tive in two occasions. Cranial magnetic resonance imaging disclosed images compatible with cerebral infarctions in the frontal and left occipital lobes, and in the right cerebellar hemisphere, probably due to embolization from endocardial vegetation. The electrocardiogram showed also subendocardial ischemia probably related to microemboli. Libman-Sacks endocarditis is usually asymptomatic, although the vegetations fragments can produce distant emboli⁶ as in this patient. She additionally presented an isolated episode of mild hemoperitoneum in the same context as the embolisms with unremarkable ultrasound findings. The commonest causes of hemoperitoneum are: of gynecological origin, after transplantation, with catheter replacement or related to an increase in physical activity. Mild bleeding can also appear in pancreatitis, peritoneal sclerosis and after performing a colonoscopy.

Broad-spectrum antibiotic therapy was administered with no improvement in echocardiographic images. Anticoagulant treatment with warfarine was initiated. The hemoperitoneum was not a contraindication as it was very small.

The approach to the patient with valvular disease includes prophylaxis of endocarditis, anti-platelet or anticoagulant therapy, and surgical valvular replacement in selected cases with severe valvular impairment. The role of steroids in the evolution of the valvular disease is not yet completely known.⁷

We witnessed the vegetation disappearance with anticoagulant therapy on echocardiographic control.

In this case, the natural pregnancy interruption, the history of thrombosis, the positive lupus anticoagulant and the

excellent evolution with anticoagulant therapy would confirm these infrequent diagnosis.

1. Greenberg A, Bernardini J, Piraino BM et al. Hemoperitoneum complicating chronic peritoneal dialysis: single-center experience and literature review. *Am J Kidney Dis* 19: 252, 1992.
2. Turiel M, Sarzi-Puttini P, Peretti R, Bonizzato S, Muzzupappa S, Atzeni F, Rossi E, Doria A. Five-year follow-up by transesophageal echocardiographic studies in primary antiphospholipid syndrome. *Am J Cardiol* 96: 574, 2005.
3. Farzaneh-Far A, Roman MJ, Lockshin MD, Devereux RB, Paget SA, Crow MK, Davis A, Sammaritano L, Levine DM, Salmon JE. Relationship of antiphospholipid antibodies to cardiovascular manifestations of systemic lupus erythematosus. *Arthritis Rheum* 54: 3918, 2006.
4. Cervera R, Font J, Paré C, Azqueta M, Pérez-Villa F, López-Soto A, Ingelmo M. Cardiac disease in systemic lupus erythematosus: prospective study of 70 patients. *Ann Rheum Dis* 51: 156-9, 1992.
5. Roldan CA, Shivaly BK, Lau CC, Gurule FT, Smith EA, Crawford MH. Systemic lupus erythematosus valve disease by transesophageal echocardiography and the role of antiphospholipid antibodies. *J Am Coll Cardiol* 20: 1127-34, 1992.
6. Roldan CA. Valvular disease associated with systemic illness. *Cardiol Clin* 16: 531, 1998.
7. Fluture A, Chaudhari S, Frishman WH. Valvular heart disease and systemic lupus erythematosus: therapeutic implications. *Heart Dis* 5: 349-53, 2003.

A. Sastre López, V. Mascarós Ferrer, V. Íñigo Vanrell and J. M. Gascó Company
Nephrology Department. Son Llàtzer Hospital. Palma de Mallorca.

Correspondence: Aránzazu Sastre López
aranchasastre@hotmail.com. Hospital Huca. Avda. Fernández Ladreda, 30. 24005 León.

Late-onset hemothorax after left jugular vein catheterization for hemodialysis

Nefrología 2008; 28 (1) 115-116

To the editor: Temporary or permanent central venous catheterization is commonly performed in hemodialysis patients when an internal vascular access is not available. Internal jugular vein catheterization is nowadays the