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.*

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 y 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 positive 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 emboli[6] 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.

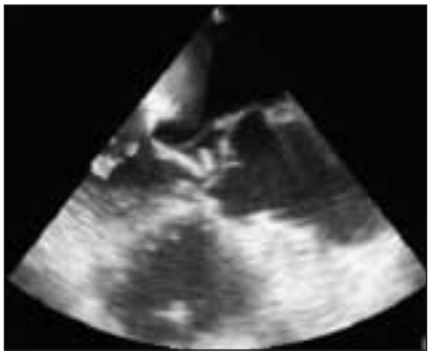

Figure 1.

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 thise infrequent diagnosis.

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Late-onset hemothorax after left jugular vein catheterization for hemodialysis

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

preferred vascular access because it is associated to lower risks and complications (15% of mechanical, infectious or thrombotic complications). The most frequent mechanical complications are arterial puncture and secondary hematoma. By contrast, hemothorax and pneumothorax incidence is lower than 0,0.2% and it happens immediately after the puncture.[2]

We present a 72 year-old woman with a history of high blood pressure and CRF secondary to nephroangiosclerosis in on hemodialysis since 2000. The patient had a permanent catheter in within the right jugular vein, which was placed 5 years ago. The catheter was non-functioning, permeation was not possible with the use of urokinase and the catheter had to be removed. Due to the lack of an internal vascular accesses, a temporary catheter, which was shorter than usual (16 cm instead of 19 cm), was placed in the left jugular vein. An X-ray film confirmed that the catheter was appropriately placed. Nineteen days later it was removed because it did not work properly and was changed by means of a metal wire (16 cm). No radiological control was made. During the following hemodialysis sessions the catheter flow was always lower than 200 mL/min, and the catheter was repeatedly manipulated.

The patient came 29 days later to hemodialysis referring dyspnea and pain in on her right scapula. At the beginning of the session clear fluid was obtained through the arterial branch and hematic fluid through the venous branch. A chest X-ray film was made (fig. 1), which showed right pleural effusion. Dialysis was performed without heparin through a right femoral access. Fifteen minutes after the end of hemodialysis the patient referred sudden right chest pain and dyspnea and she suffered cardiopulmonary arrest. On physical exam right pulmonary hypoventilation was evident. A decrease of hemoglobin value was detected on blood analysis, and the right pulmonary field was opaque on chest X-ray. The diagnosis of massive hemothorax was suspected, a drain tube was placed and resuscitation maneuvers were initiated. The patient was referred to the ICU, where she spent 72 hours and after that she was admitted to our Department with no consequences.

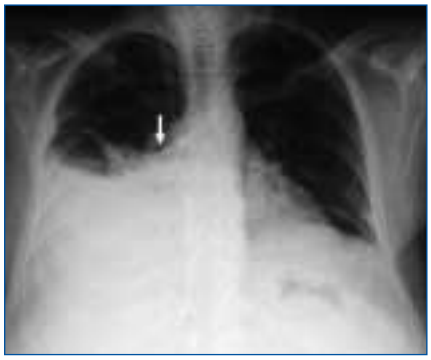

Figure 1. Chest X-ray showing right pleural effusion due to impaction of the left jugular catheter within the pleural space.

Internal jugular vein catheterization is not free from complications. Massive hemothorax is not frequent, occurs immediately after the puncture and commonly after catheterization of the subclavian vein. In the reported case, hemothorax happened 29 days after catheterization of the vascular access. That was due to the removal of the left jugular catheter, that was occluding a fistula created by the impact of the catheter.

Several facts were determinants for perforation into the interpleural space:[3] canalization of the left jugular vein instead the right one (that was occupied by a permanent catheter, which had to be removed), the replacement by a short catheter (16 cm) and the various manipulations of the catheter because of the low flow. Massive hemothorax was the consequence of the catheter removal, and perhaps this procedure should have been done with some precautions (removal in the ICU).

The widespread use of catheters for hemodialysis in patients with difficult vascular accesses increases the morbimortality. It is mandatory the referral of the patient to the nephrology department, and the cooperation of vascular surgeons and radiologists to achieve appropriate vascular accesses.

A radiological control should be made after catheterization and replacement of the catheter to assess the correct position and the lack of complications, although some authors do not agree with this measure.[4]

In summary, when placing temporary catheters the appropriate length, the correct position after placement and replacement should be considered, and manipulations should be avoided.

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Renal failure secondary to lymphoplasmocytic lymphoma-Waldenström's macroglobulinemia

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To the editor: Waldenström's macroglobulinemia (WM) is a lymphoproliferative disease characterized by the secretion of an IgM type monoclonal immunoglobulin. It occurs in patients with ymphoplasmocytic lymphoma and also in patients with other small B cell lymphomas. Typically, there is a lymphoplasmocytic infiltration of the bone marrow, lymph nodes, spleen, and occasionally of other tissues and organs like the kidney. Whether lymphoplasmocytic lymphoma and WM are the same condition is subject to controversy.[1,2]

Renal disease is basically due to IgM depositions in glomerular basal mem-