ter trajectory and accumulating within the pleural cavity (fig. 1b). A right infraclavicular incision was made for direct compression of the subclavian vein and the application of hemostatic material. The vein leakage was seen to disappear, and a right pleural drain was placed - followed by a favorable clinical and radiological course.

CASE 2

A 19-year-old male presented with chronic renal failure secondary to acute renal failure in the context of meningococcemia. A left radiocephalic fistula was prepared on March 27, 2007, with poor venous development. In view of the need to start dialysis, tunneled catheter placement was programmed for May 18, 2007. Following vein puncture and the drawing of blood of venous appearance, the catheter was positioned but was found to function poorly. The chest X-rays showed the catheter tip to be located in a right paravertebral position, with no firm evidence of pneumothorax (fig. 1c). Emergency surgical removal was carried out, with no evidence of leakage at phlebographic control.

Since 1996, we access the right internal jugular vein as described by Apsner et al.[2] for the placement of tunneled catheters, since localization is easy and the procedure has few complications. Puncture is carried out at the confluence between the right internal jugular vein and the subclavian vein, at the so-called right innominate vein. We use fluoroscopic control only in those cases presenting insertion difficulties. In the 10 years during which we have applied this technique, there have been few complications – the above two cases being the only examples of extravascular positioning registered in our experience.

In our review of the subject, we have found few cases of extravascular placement of central venous catheters for hemodialysis.[3] Complications of this kind could be avoided by using ultrasound or fluoroscopic control on a systematic basis.

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Improvement of intradialytic arrhytmias after combined conductivity and ultrafiltration profiling without secondary overhydration

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To the editor: Hemodynamic instability (arrhythmias, hypotension) is one of the most frequent complications found during dialysis. For years, the usefulness of ultrafiltration and/or conductivity profiling to prevent the appearance of such problems has been the subject of debate.

We report the case of a 75-year-old male initially subjected to peritoneal dialysis due to chronic renal failure secondary to diabetic kidney disease who required transfer to hemodialysis because of peritonitis, with a poor course one year after treatment. After starting hemodialysis, and as a result of nutritional problems, dry weight began to decrease (over 4 kg), with good hemodynamic tolerance. However, after two months the heart rate – which at the start of the session was 60-70 bpm – suddenly increased in the last hour to 110-120 bpm. In some cases this situation was accompanied by severe hypotension. Continuous electrocardiographic monitorization of several hemodialysis sessions was thus decided. In the first three hours the patient showed sinus rhythm with a heart rate of 60-70 bpm, though after the third hour rapid atrial fibrillation was recorded that only reverted after conclusion of the dialysis session. The previous and posterior blood pressure values remained at 120-130/70-80 mmHg. In view of this situation, the dialysis machine conductivity and ultrafiltration parameters were changed during the session, applying a descending logarithmic profile for conductivity (start 15.7 mS/cm, end 13.8 mS/cm) and ultrafiltration (dialysis previously being carried out with a constant conductivity of 14.2 mS/cm). This measure improved tolerance during the sessions, with no severe hypotension and presenting a stable heart rate. Over the following four months the patient gained 2 kg in dry weight, but the blood pressure did not increase (110-120/70 mmHg); no antihypertensive medication was needed, and no edema or other signs of volume expansion were noted. Likewise, there were no increases in pre-dialysis sodium level (134-135 mEq/l in the determinations with constant conductivity at 14.2 mS/cm versus 135 mEq/l in those made with the exponential profile).

The use of conductivity and ultrafiltration profiling during hemodialysis has been studied by a number of authors. The objective of such profiling is to improve hemodynamic tolerance by preventing vascular depletion secondary to sodium loss during dialysis.[1] However, the different series found in the literature report quite variable results – reflecting the use in many cases of very different profiles. Some studies have reported no significant differences in hemodynamic tolerance on applying combined conductivity and ultrafiltration profiles.[2] Other studies involving linear profiles starting with high conductivities (15-15.5 mS/cm) and ending with values close to 14 mS/cm have observed a reduction in hypotensive episodes – though at the

expense of an increased volume expansion (increase in blood pressure before and after dialysis).[3-5]

In other cases the applied profile exhibited end conductivity values lower than those of the serum sodium concentration before dialysis. In this way elimination is secured of the excess sodium that may have diffused in the first phase of the dialysis session, achieving a neutral balance without inducing volume expansion.[6,7]

Likewise, series have been published in which conductivity and ultrafiltration begin at very high values close to 15.8-15.9 mS/cm, followed by a rapid exponential reduction until ending at values close to 14 mS/cm. In this way most of the ultrafiltration takes place in the first moments of the session, when conductivity is very high, though on quickly reducing the sodium concentration, accumulation and subsequent overhydration are avoided.[8]

In sum, the use of combined conductivity and ultrafiltration profiling may prove useful for controlling hemodynamic instability (arrhythmias, hypotension) during hemodialysis, though great care is required on selecting the starting and ending conductivities, in order to avoid volume expansion.

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Large kidney due to levofloxacin

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To the editor: Tubulointerstitial nephritis was first described by Councilman[1] in 1898. It is an infrequent clinical-pathological entity with an estimated incidence of 8-14% among patients subjected to biopsy for acute renal failure of indeterminate origin.[2] The triggering factors of this disease comprise neoplasms, drugs and infections. We present the case of a patient who manifested with acute tubulointerstitial nephritis and a large kidney (nephromegaly) secondary to levofloxacin.

CLINICAL CASE

The clinical case corresponds to a 67-year-old male. His personal history included allergy to nicotinamide, ischemic heart disease (requiring three coronary stents, with follow-up comprising 6 catheterizations – the last performed one year before admission) and dilated myocardiopathy secondary to the latter. His usual treatment consisted of pantoprazole 40 mg/24 h, carvedilol 25 mg/12 h, aspirin 100 mg/24 h, amlodipine 5 mg/24 h, isosorbide dinitrate 20 mg/8 h, and simvastatin 20 mg/24 h.

The patient reported to the Emergency Service with right-flank abdominal pain irradiating to the hypogastrium, and accompanied by vomiting and fever. Physical examination revealed positive right-side fist-percussion as sole significant finding. The emergency complementary tests showed creatinine 4 mg/dl, leukocytosis (16,000/µl) and urine sediment with leukocyturia. The chest and abdominal X-rays showed no anomalies. The study was completed with abdominal ultrasound, which revealed an enlarged right (15 cm) (fig. 1) and left kidney (14.5 cm) – the latter organ also showing two cortical cysts. Based on the above data, admission to the Nephrology ward was decided, and a more complete evaluation with laboratory tests was made – the immune and tumor marker parameters proving negative. The blood and urine cultures were also negative. Fluid therapy and broad-spectrum antibiotic treatment was prescribed, resulting in clinical and analytical improvement (creatinine 1.6 mg/dl); hospital discharge was thus decided. The patient was posteriorly readmitted with this same clinical presentation on two further occasions, with the clinical diagnosis of acute pyelonephritis refractory to medical treatment. During the admissions, the study was completed with blood cultures, urine cultures, and gallium gammagraphy. In view of the persistence of the bilateral nephromegaly, the possibility of amyloidosis was discarded by a rectal and abdominal adipose tissue biopsy, which proved negative. Tuberculosis was likewise ruled out by negative Mantoux tests and specific cultures. A bone marrow aspirate to evaluate possible myeloma was also negative. Thus, due to the suspicion of disease circumscribed to the kidneys, a renal biopsy was performed, revealing a diffuse interstitial inflammatory infiltrate composed of T lymphocytes, numerous plasma cells and few β lymphocytes. These findings were compatible with acute tubulointerstitial nephritis, as a result of which treatment with prednisone was started.