given procedure to the value that would be obtained by the conventional Nichols test.

The group that performed this study intends to prepare a longer publication on this subject in the near future.

We thank Dr. Martín Gil for his letter, that allowed us for a more detailed explanation about some issues that were possibly not sufficiently clear in the editorial.

1. De la Piedra C, Fernández E, González Casaus Mª L, González Parra E. Diferencias en la función de los péptidos paratiroideos ¿Qué estamos midiendo? *Nefrología* 2008; 28 (2): 123-128.

C. de la Piedra Gordo
Laboratorio de Bioquímica. Fundación Jiménez Díaz - UTE (Grupo Capio). Madrid.
Correspondence: *Concepción de la Piedra Gordo. cpiedra@fjd.es. Laboratorio de Bioquímica. Fundación Jiménez Díaz - UTE (Grupo Capio). Avda. de los Reyes Católicos, 2. 28040 Madrid. España.*

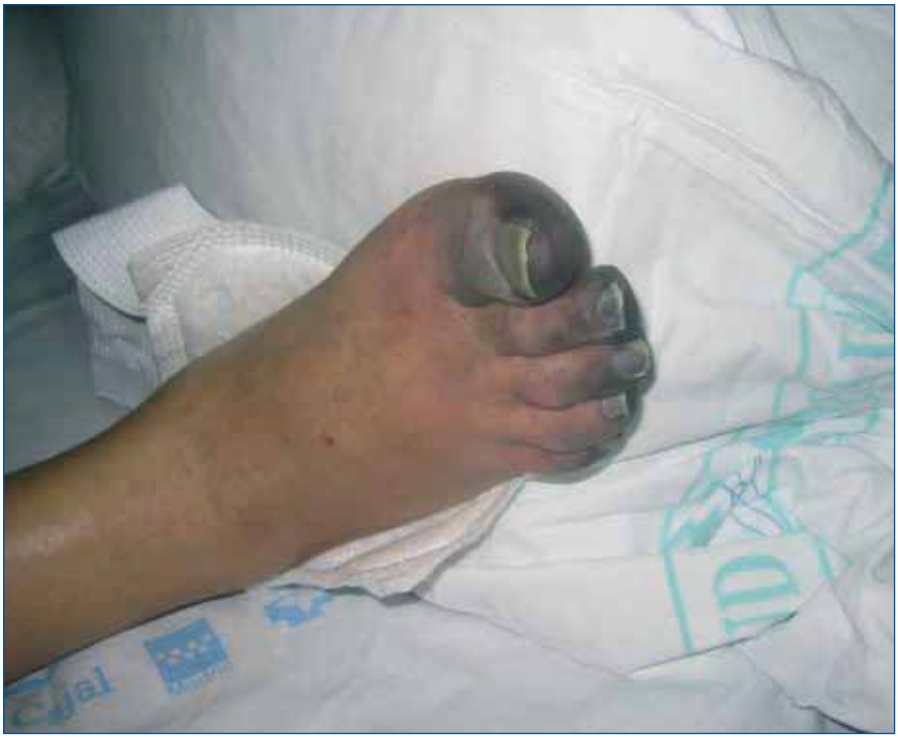

Figure 1. Distal cyanosis affecting toes resulting from microvascular thromboses.

Management of heparin-induced thrombocytopenia in a patient on hemodialysis complicated with thrombosis in the extracorporeal circuits

Nefrología 2008; 28 **(4)** 462-463

To the editor: Heparin-induced thrombocytopenia (HIT) is a hypercoagulability state resulting from production of antibodies against a heparin-platelet factor 4 complex that occurs in 0.2%-1% of patients receiving heparin.[1] The condition should be considered when a greater than 40% decrease from baseline is detected in platelet count between 4 and 10 days since the start of heparinization.[1,2] Mortality from thrombosis is high (30%), and urgent action consisting of heparin discontinuation and administration of an antithrombotic, lepidurin in our setting, is therefore required.[3]

A 72-year-old male patient who experienced an acute myocardial infarction during coronary bypass surgery is reported here. The patient required insertion of an aortic counterpulsation balloon and administration of unfractionated heparin (UFH). Three days later, fever, hypotension, and renal failure requiring hemodialysis occurred. Four days later, circuit thrombosis, not prevented by prostacyclin addition, was detected. Platelet count decreased to 42,000(mL from a baseline value of 210,000/mL. On the following day, the patient showed cyanosis in the fingers of both feet (Figure 1), impossibility of hemodialysis persisted, and a right femoral thrombosis was detected by echo Doppler. HIT was suspected and confirmed by platelet aggregation tests and ELISA. Heparin was discontinuation

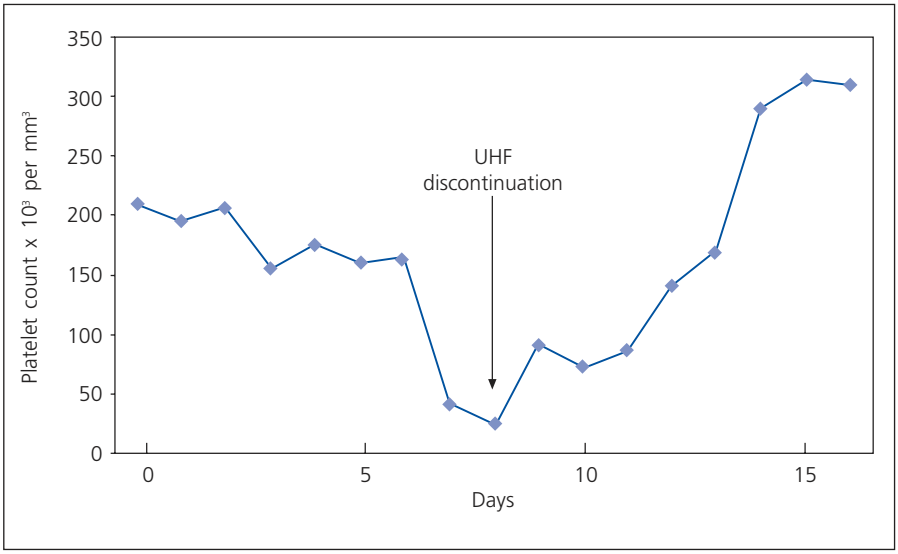

Figure 2. Evolution of the platelet count.

nued, and a lepidurin bolus of 0.1 mg/kg was administered. Activated partial thromboplastin time (APTT) increased from 36" to 72", and hemodialysis was normally performed. APTT was monitored every 2 hours to maintain it between 2 and 3 times the control value (33"). A new lepidurin administration (0.05 mg/kg) was not required until 16 hours later. When renal function improved, a continuous lepidurin infusion (0.005 mg/kg/h) was started. Platelet function recovery was noted at 48 h (fig. 2). Seven days later the patient was stabilized and treatment was started with acenocoumarol. Six days later, international normalized ratio (INR) was 2.8, and lepidurin was therefore discontinued.

Lepidurin is the therapeutic option for patients with HIT in our setting. Lepidurin is an antithrombotic drug that inactivates thrombin directly, so that it also has a bleeding risk[3] and requires monitoring. Recent studies suggest that the doses recommended by the supplier are too high.[3,4] This recommendation is relevant in renal failure patients because lepidurin excretion depends exclusively on the glomerular filtration rate.

The reported case alerts about two significant issues in clinical practice. The first issue is that HIT should be considered as a potential cause of thrombosis of the hemodialysis circuit in patients treated with heparin. Moreover, in patients with renal impairment lepidurin should be started at low doses, and drug levels should be frequently monitored.

1. Warkentin TE, Greinacher A. Heparin-induced thrombocytopenia: recognition, treatment and prevention. The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126: Supl.: 311S-337S.
2. Arepally GM, Ortel TL. Heparin-induced thrombocytopenia. *N Engl J Med* 2006; 355: 809-17.
3. Lubenov N, Eichler P, Lietz T, Greinacher A. Lepidurin in patients with heparin-induced thrombocytopenia: results of the third prospective study (HAT-3) and a combined analysis of of HAT-1, HAT2 and HAT3. *J Thromb haemost* 2005; 3: 2428-36.
4. Tardy B. Predictive factors for thrombosis and major bleeding in an observational study in 181 patients with heparin-induced thrombocytopenia treated with lepidurin. *Blood* 2006; 108: 1492-6.

J. M. César[a], A. Candelar[b], M. T. Tenorio[c], A. Chinear[a], R. Saizr[b] y F. Liañor[c]
Servicios de [a]Hematología, [b]Anestesia y [c]Nefrología. Hospital Ramón y Cajal. Madrid.
Correspondencia: *Jesús César Pérez. jcesar.hrc@salud.madrid.org. Hospital Ramón y Cajal. Ctra. Colmenar, km. 9,1. 28034 Madrid. España.*

A 48-year-old male with renal infarction and thrombophilia

Nefrología 2008; 28 **(4)** 463-464

To the editor: Acute renal infarction results from renal artery occlusion by embolic or thrombotic events.[1] Renal embolism is most commonly caused by thrombi released from the heart in patients with heart disease. Thrombosis is usually due to trauma, or to generalized atherosclerosis in elderly patients. A less common cause are hypercoagulability states, associated to an increased risk of venous, or more rarely arterial, thrombosis. Such association is stronger in patients aged < 55 years and women.[2]

The case of a 48-year-old male patient whose father had died from a stroke is reported. The patient had a personal history of HBP, dyslipidemia and former smoking. In 1994 he experienced an inferolateral non-Q wave AMI, and in 2001 he was admitted to hospital for an ischemic stroke. He was then diagnosed a mutation in the prothrombin (FII 20210) and methylenetetrahydrofolate reductase (MTHFR) genes, with normal homocysteine levels. Patient was again admitted to hospital in 2002 for non-Q wave AMI. He was being treated wit ramipril, acetyl salicylic acid, omeprazole, pravastatin, and metoprolol.

He attended the emergency room for a sudden, continuous pain in the periumbilical region and right flank for the past 72 hours. Pain was irradiated to the right renal fossa and associated to nausea, vomiting, and dark urine.

Physical examination revealed abdominal tenderness in the periumbilical region and right flank.

Laboratory tests showed elevated GOT, GPT, and LDH levels with normal renal function, electrolytes, and coagulation. Urine analysis found proteinuria (30 mg/dL). Chest and abdominal X-rays were unremarkable.

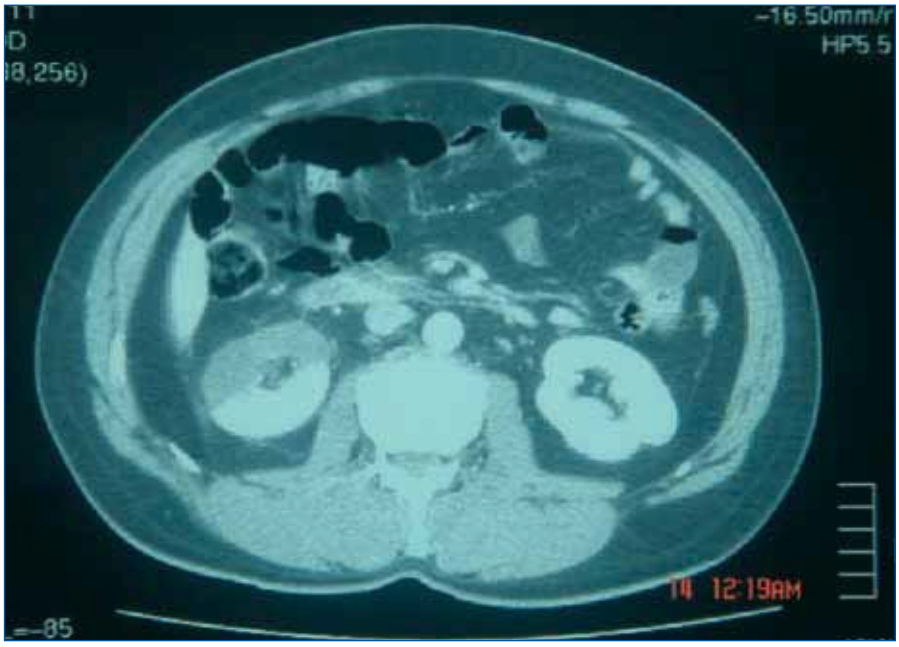

Figure 1. Abdominal CT scan with contrast showing patchy hypodense areas in the right kidney.