

A) COMMENTS ON PUBLISHED ARTICLES

Page kidney Doppler ultrasound

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To the Editor,

We read with great interest the paper published in the journal NEFROPLUS by Dr Montoya et al.¹ in relation to Page kidney. This is an excellent review describing this rare condition in great detail, both diagnostically and therapeutically.

In this letter, we would like to provide additional information to that already offered by the authors regarding an ultrasound finding of interest that we have recently described.²

In our study we described a transplant patient under antiplatelet aspirin therapy that underwent percutaneous nephrostomy to resolve an ureterovesical stenosis while definitive surgery was being planned. Oliguria and impaired renal function were noted 48h after placing the nephrostomy tube. Blood pressure was unchanged. Two-dimensional ultrasound revealed a large

subcapsular haematoma, while the Doppler colour ultrasound showed preserved renal perfusion with normal arterial and venous flow. The pulsed Doppler ultrasound showed the existence of an increase in intrarenal resistance with reversal of diastolic flow throughout the kidney. The patient underwent surgery within 24 hours. The subcapsular haematoma was evacuated, and therefore, diuresis was recovered, renal function was normalised and the Doppler pattern returned to normal.²

Diastolic flow reversal in pulsed Doppler ultrasound is traditionally characteristic of renal vein thrombosis.³ However, it also can be presented, reversibly, by acute rejection and severe acute tubular necrosis,³ anticalcineurinic toxicity⁴ and, in our experience, Page kidney.³

Lastly, it is of great interest to perform a Doppler ultrasound after each renal intervention in order to diagnose associated complications. It is also important to know that the reversal of diastolic flow in the Doppler does not necessarily indicate that there is a renal vein thrombosis.

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M. Rivera Gorrin

Nephrology Department. Ramón y Cajal Hospital. IRYCIS. Madrid, Spain

Correspondence: Maite Rivera Gorrin

Servicio de Nefrología.
Hospital Ramón y Cajal. IRYCIS.
Carretera de Colmenar km 9,100.
28034 Madrid.
mriverag.hrc@salud.madrid.org
mriverago@gmail.com

B) BRIEF PAPERS ON BASIC RESEARCH AND CLINICAL INVESTIGATION

Lactic acidosis and linezolid-induced pancytopenia

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To the Editor,

Linezolid¹⁻³ is an antibiotic in the oxazolidinone group with a tricyclic structure responsible for its efficacy against methicillin-resistant staphylococci. It inhibits protein synthesis by using a

different site from other antibiotics. It is active³ against numerous microbes, such as staphylococci, including methicillin-resistant ones, streptococci, enterococci and other gram-positive types, like *Corynebacterium* and *Listeria*, as well as some anaerobes such as *Clostridium difficile*.

It is being increasingly used in current clinical practice for the treatment of nosocomial pneumonia caused by MRSA or *Streptococcus pneumoniae*, community pneumonia due to gram-

positive organisms and complicated and uncomplicated skin infections, including diabetic foot infections without concomitant osteomyelitis.

60% is metabolised³ by the liver and 30% by the kidney, therefore the dosage does not require adjustment in moderate renal or hepatic failure, although there is no experience in serious failures.

Like any drug, it has side effects.¹⁻³ The most frequently reported (between 1%

and 10%) are gastrointestinal problems, headache and abnormalities in laboratory tests such as pancytopenia and lactic acidosis. Central nervous system disorders and bacterial or fungal superinfection appear less frequently.

We present the case of a 79-year-old man with a history of with drug-controlled hypertension, benign prostatic hyperplasia and chronic kidney disease stage 4 in outpatient nephrology follow-up for 8 months. He underwent surgery for a prosthetic left knee in a local hospital on 18th March 2010, and needed subsequent surgical cleaning for prosthetic infection on April 26th, when antibiotic treatment with linezolid and levofloxacin was started. He was discharged on 28th May. The patient maintained treatment with linezolid for 7 weeks. The germ causing the prosthetic infection was unknown.

On June 10 the patient visited the emergency department of our hospital for malaise, asthenia, anorexia, vomiting and weight loss. Upon arrival, the patient was in good general condition, without signs of poor peripheral perfusion. Blood pressure was 118/52mm Hg, temperature of 36.5°C and the heart rate was 72 beats/min. There was a slight increase in local temperature of the left knee, with minimal effusion. Laboratory tests included the following: urea 184mg/dl, creatinine 2.6mg/dl, potassium 5.7mmol/l, leukocytes 4000/ μ l, with 61.3% neutrophils, haemoglobin 9g/dl, platelets 45000/ μ l, lactic acid 3.11mmol/l, pH 7.25 and bicarbonate 18.5mmol/l. The patient was admitted to orthopedic surgery.

Given these laboratory findings and the absence of signs of systemic infection justifying lactic acidosis or pancytopenia, they were considered as side effects of linezolid. This was therefore withdrawn without any antibiotic alternative.

Progressive improvement of the pancytopenia and renal function after hydration of the patient were reported in subsequent days, so the patient was transferred to the hospital where underwent surgery. Upon discharge, the following results were found: creatinine 1.51mg/dl, total leukocyte count 5300/ μ l, haemoglobin 9g/dl and platelets 59 000/ μ l.

Although linezolid is a very effective antibiotic in cases refractory to conventional antibiotics, its possible side effects must always be taken into account. In this case it caused pancytopenia and lactic acidosis, which improved after withdrawal of the drug.

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T. Carbajo, M. Fenolosa, R. Pons, C. Calvo
Nephrology Department. Castellón General Hospital, Spain

Correspondence: Teresa Carbajo
Servicio de Nefrología.
Hospital General de Castellón.
tenguerengue@hotmail.com

Another patient with a natural history of diabetic nephropathy: current situation and means of prevention

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To the Editor,

Diabetic nephropathy (DN) is the leading cause of chronic kidney disease (CKD) in developed countries,¹ with an increasing incidence and prevalence. We describe the effects of diabetes mellitus (DM) on the kidney after

years of natural development (without prior renoprotective treatment and poor metabolic control).

CASE REPORT

A 60-year old man with a previous history of type 2 DM for more than 20 years, treated with antidiabetic agents initially and insulin for the last 5 years, and with poor metabolic control. Non-proliferative diabetic retinopathy. Ex-smoker of 30 cigarettes per day until 5 years ago.

The patient visited the emergency department complaining of generalised oedema of one month duration, dyspnoea, and 2-3 times nocturia. On physical examination, the patient was in good general condition, was conscious and oriented. Blood pressure was 160/110mm Hg, heart rate 80 beats/min. Jugular venous distension. Cardiac auscultation was rhythmic. Pulmonary auscultation showed minimum crackles in the bases. Pitting oedema to knee. The rest of the examination was normal.

Blood analysis revealed glucose 200mg/dl, urea 52mg/dl, creatinine 1.2mg/dl, uric acid 5.3mg/dl, cholesterol 313mg/dl, triglycerides 144mg/dl, albumin 1.9g/dl, total protein 4.4g/dl, calcium 7.6mg/dl, phosphorus 3.2mg/dl, iron 41 μ g/dl, and ferritin 155ng/ml. The haemoglobin was 11.6g/dl, haematocrit 34.3%, and the rest of the blood count and coagulation tests were normal. The thyroid hormones, antibodies, viruses (HIV, hepatitis B and C) and PSA were normal. Haemoglobin A_{1c} was 8.3%. The immunological test, including immunoglobulin, complement, rheumatoid factor, ASO, ANA, anti-DNA, ANCA and C-reactive protein were also normal.

The urinalysis revealed protein +++, blood + glucose ++ and negative nitrite. Proteinuria in 24-hour urine