and a higher ESA dosage those with an accepted 'accelerated atherosclerosis' and clinical or subclinical problems determining worse results in terms of mortality, previously hyporesponsive to the ESA (ferric state actually representing a deficit or decreased availability from the deposits, acute inflammation or chronic microinflammation, secondary hyperparathyroidism, among other factors)?

Recently, we are reaching a crucial moment and are currently analysing a prospective, phase IV, multicentre, open, non-controlled study, to assess the effectiveness of Cuban rhEPO. We are assessing haemoglobin levels and rhEPO doses employed over a period of 12 months, the type of response over time (variability), and adverse events. We included 617 patients from 15 nephrology departments throughout Cuba.[4]

This study highlights problems in controlling haemoglobin levels and rhEPO doses similar to those detected in other international studies.[5]

I have summarised my opinion based on the current evidence, as a strategy for guaranteeing efficient ESA use with minimum risks and in line with good clinical practice:

1. Avoid blood transfusions.
2. Start rhEPO treatment in renal anaemia patients with haemoglobin of 10g/dl.
3. Keep haemoglobin levels between 11.5g/dl and 13g/dl.
4. Never try and reach the latter by increasing rhEPO doses.
5. Question rhEPO doses over 8000U/week.
6. Use the best intravenous iron products available, depending on the elements of iron metabolism for each patient.
7. Increase the clinical method, scientific and rigorous search of the factors concerning a lack of response that are associated with ESA, undertake energetic and effective actions on this, and on those well identified mortality factors for patients with stage 5 CKD.

In summary, we must be careful in our prescription and assess the risk-benefit for each haemoglobin level, in accordance with each patient's characteristics and needs. We must consider that an inadequate EPO response or using it at a high dosage is a risk marker for mortality.

We must not forget that stage 5 CKD patients are becoming increasingly more heterogeneous with regards epidemiological and clinical aspects and related comorbidities.

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B) BRIEF CASE REPORTS

Listeria monocytogenes: an infrequent cause of peritonitis in peritoneal dialysis

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To the Editor,

Peritoneal infections are a serious complication in peritoneal dialysis and can affect the clinical state of the patient and technique viability.[1] Gram positive bacteria are most frequently involved (coagulase negative *Staphylococcus* [40%-60%], *Staphylococcus aureus* [10%-20%] and *Streptococcus* [10%-20%]). Of all peritonitis, 5%-20% are due to gram negative organisms. Other germs, which represent less than 5% of cases, are other bacteria, fungi and protozoa.[1]

There are not many cases of *Listeria monocytogenes* peritonitis published in the literature, and they generally affect immunocompromised patients.[2-12]

We present the case of a patient undergoing peritoneal dialysis due to heart failure resistant to diuretics. This is the first case of *Listeria monocytogenes* infection in the peritoneum in our hospital.

We present a 64-year-old man who underwent an operation for tetralogy of Fallot when he was younger. He later developed a severe right heart failure and eventually became resistant to diuretics. This caused

him to be admitted to hospital on several occasions for anasarca and acute renal failure. He was rejected for a heart transplant because he had severe pulmonary hypertension. Given this situation, he was entered onto a peritoneal ultrafiltration programme (May 2006), having a single night exchange of 2l of icodextrin.

The patient arrived at the emergency department with abdominal pain, moderate diarrhoea and cloudy peritoneal drainage fluid. He did not complain of fever, vomiting or focal neurological or infectious signs. He had no family history of food borne diseases and he was not aware of having made any mistakes with the dialysis technique, which would have caused the equipment to become unsterile. This patient had already suffered two other peritonitis, with negative peritoneal fluid culture. He was treated successfully with wide spectrum antibiotics (vancomycin and ceftazidime), recovering without any major problems.

When he was admitted, signs of distension and pain were noted upon abdominal palpation. Analytical tests showed: leukocyte count: 8900/μl, 84% neutrophils, haemoglobin: 12.1g/dl; platelets: 163 000/μl; urea: 60mg/dl, creatinine: 1.7mg/dl; normal hepatic enzymes; peritoneal leukocytes count: 8800/μl, 96% neutrophils. The Gram staining of the peritoneal fluid revealed single and short-chain bacilli. Empirical intraperitoneal antibiotic treatment was started with vancomycin and ceftazidime. Small, translucent, grey colonies with a discreet beta haemolysis area were found in the peritoneal fluid on blood agar plate cultures following aerobic incubation at 37ºC (pH 7.2-7.4), indicating *Listeria monocytogenes*. Faecal samples were cultivated. They were negative for *Listeria*, although it was provided after antibiotic treatment had been started which could have halted its growth. Initial antibiotics were substituted for intravenous ampicillin and intraperitoneal gentamicin. The infection started responding to the antibiotics after 72 hours. Specific antibiotic therapy was maintained for three weeks.

Listeria monocytogenes is the only *Listeria* among the seven known species that can infect human beings. It is an aerobic gram positive germ (in certain circumstances can also behave like an anaerobic one) which cannot form spores. Despite being present in the environment, it does not usually cause humans to become ill. An incidence of 0.7 cases/100 000 has been calculated.[13] The elderly, newborns, cancer patients, cirrhotic and immunocompromised patients are more susceptible to contracting a *Listeria* infection. The most common clinical signs include meningitis, endocarditis, gastroenteritis, miscarriage and bacteraemia. Peritonitis caused by *Listeria* is a rare, dangerous form of the complication. Spontaneous forms of peritonitis caused by *Listeria* are well known, especially in cirrhosis patients.[13] Around 50 cases have been published and most of them describe Spanish patients.[14] The geographical predilection in Spain is not entirely understood, but may be due to eating habits including the consumption of incorrectly pasteurised dairy products or raw fruit and vegetables.[14]

For patients undergoing dialysis, peritonitis caused by *Listeria* is very rare. Table 1 shows the cases published to date in the medical literature. All cases have occurred in immunocompromised patients, due to illness or medication.

It has been reported that natural killer cells (non-antigen first line of defence) in heart failure patients are in a situation of anergy and respond less to the molecules that usually stimulate them, such as interleukin-2 and interferon-gamma. These patients are therefore considered to be at greater risk of developing infections given their immunocompromised situtation.[15]

Listeria is one of the most virulent pathogens which cause food borne diseases. It has a mortality rate of 20%-30%, higher than almost all other food borne illnesses. It was difficult to find the source of infection in our patient, but given that he lives in a rural area it is possible that he ingested incorrectly pasteurised dairy products, which would have caused this germ to colonise the intestine. The bacteria would have then invaded the mucosa, reaching the peritoneum. Unfortunately, this has not been confirmed, given that the faecal culture sample was provided after antibiotic treatment had been started. However, the hypothesis mentioned above seems the most plausible.[12] Furthermore, chronic heart failure could have been involved in this process. In this situation, the patient would have intestinal oedema, increased permeability and would be more susceptible to bacterial invasion.[16]

The antibiotic treatment of choice for *Listeria* infection is penicillin or ampicillin, which can be administered in combination with aminoglycosides or not.[12] However, there is no clear indications as to which treatment is better for peritonitis caused by *Listeria*, or how long treatment should last. Vancomycin may not be effective since *Listeria* is an intracellular microorganism.[5,6,9] Both trimethoprim-sulfamethoxazole and erythromycin have been used successfully for patients allergic to penicillin.[2,12] For most cases, peritonitis responds rapidly and effectively to antibiotics, without needing to withdraw the peritoneal catheter.

In summary, we must remember that *Listeria monocytogenes* is a germ that can cause peritonitis in patients undergoing peritoneal dialysis, where rod-shaped gram positive bacteria can be found, even in patients considered immunocompetent. Above all, prevention is the best weapon to combat this zoonosis.

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Table 1. Characteristics of 11 cases of peritonitis caused by Listeria monocytogenes in patients undergoing peritoneal dialysis

Ref.	Age / Sex	Underlying disorders	Drugs used	Previous peritonitis episodes	Signs	Antibiotic treatment	Duration of treatment	Catheter withdrawal	Results
[2]	71/F	ITP	Prednisone	None	Abdominal pain, cloudy fluid	I.V. and I.P. erythromycin and I.V. co-trimoxazole (allergic to penicillin)	Not known	No	Cure
[3]	50/F	SLE	Prednisone, azathioprine	*Staphylococcus aureus* 11 months before	Abdominal pain, cloudy fluid	Gentamicin + ampicillina ampicillin	4 weeks	No	Cure
[4]	31/F	SLE	Prednisone	Not known	Abdominal pain, cloudy fluid	Vancomycin at start; ampicillin once vancomycin failed	Not known	No	Cure
[5]	53/M	Wegener's Granulomatosis	COX 25mg	No	Abdominal pain, cloudy fluid	I.P. Ampicillin and P.O. pivampicillin following vancomycin failure	3 weeks	No	Cure
[6]	60/M	LLC	Prednisone	Not known	Fever, abdominal pain, cloudy fluid	Oral amoxicillin 1g and I.V. following vancomycin failure	10 days	No	Curación
[7]	67/M	Cirrhosis		None	Abdominal pain, cloudy fluid	Ampicillin following vancomycin and gentamicin failure	Not known	No	Not known
[8]	38/F	CGN, kidney transplant lost		Negative culture for 2 years, gram positive 1 year ago	Abdominal pain, cloudy fluid, nausea, mild diarrhoea	Tobramycin + amikacin	2 weeks	No	Cure
[9]	64/M	Polymyositis	Prednisone	None	Abdominal pain, cloudy fluid	I.P. vancomycin + gentamicin, after failure, I.P. ampicillin and gentamicin	10 days + 4 weeks	No	Cure
[10]	38/F	SLE	Prednisone, azathioprine	None	Fever, cloudy fluid, septic shock	I.V. ampicillin + amikacin	4 weeks	No	Cure
[11]	68/M	Prosthetic, valves, EHD, ischaemic renal failure kidney failure		None	Abdominal pain, lcloudy fluid, fever, nausea, vomiting, shock	I.P. vancomycin and netilmicin	3 weeks + 6 weeks	No	Peritonitis resuelta pero fallecimiento por insuficiencia cardíaca descompensada
[12]	28/F	SLE		Negative culture 11 months ago, CNS and SV 4 months ago	Abdominal pain, cloudy fluid, nausea, conjunctivitis	I.P. Cephalosporins and ampicillin	3 weeks	No	Cure
OC	64/M	TF, EHD, ischemic renal failure		Two episodes with negative cultures	Abdominal pain, cloudy fluid and IV ampicillin	I.P. vancomycin + ceftazidime, then I.P. gentamicin.	3 weeks	No	Cure

OC: our case; M: male; F: female; ITP: idiopathic thrombocytopenic purpura; SLE: systemic lupus erythematosus; CLL: chronic lymphocytic leukaemia; CGN: chronic glomerulonephritis; TF: tetralogy of Fallot; EHD: end stage heart disease; COX: cyclophosphamide; CNS: coagulase-negative staphylococcus; SV: streptococcus viridans; IP: intraperitoneal; P.O.: per os; I.V.: intravenous.

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Arterial hypertension induced by pyeloureteral stenosis in horseshoe kidney

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To the Editor,

Horseshoe kidney (HK) was first described by Berengario da Carpo in 1552. Thirty-three percent of cases are asymptomatic and the remaining may present with complications such as multicystic renal dysplasia, obstructive uropathies, hydronephrosis, lithiasis, infections and neoplasias such as renal carcinoma and Wilms' tumour or urothelial tumour.[1]

We present the case of a 27-year-old male, who came to the emergency department for abdominal pain located in the left flank. From his medical record, we discovered that he had a recent history of arterial hypertension (AHT). His blood pressure was 160/90mm Hg in the physical examination. We performed an abdominal ultrasound (not shown) in which cystic images were observed on the superior pole of the left kidney, which presented with a mild cortical atrophy. Given that a kidney disease was suspected, a computerised tomography (CT) scan was performed, showing that the cystic images corresponded to dilation of the pyelocaliceal system (Figure 1) in a HK (Figure 2). We performed a left pyeloplasty which resolved the obstructive problems: the patient being asymptomatic at present.

HK is the most common type of renal fusion anomaly. It appears in 1 out of every 400 births, with a higher incidence in men (2:1). The isthmus is usually located anterior to the large abdominal vessels. Hydronephrosis due to obstruction in the pyeloureteral junction is observed in a third of all HK, being factors which contribute to the upper ureter entering the renal pelvis and isthmus or blood supply anomalies.[2] It has been documented that Wilms' tumours, clear-cell, neuroendocrine,[3] and urothelial carcinomas and nephroblastomas can be found in HK. It can be associated with congenital, genitourinary, bone, gastrointestinal, myelomeningocele and cardiovascular anomalies. Pyeloureteral junction stenosis (PJS) is the most common congenital alteration of the upper urinary tract, and is most associated with HK. In most cases, PJS is due to a destruction of the muscular fibres and an increase in the amount of collagen in the pyeloureteral junction. The most common clinical sign is lumbar back pain, but for chronic obstruction, activation of the renin-aldosterone system would lead to vasoconstriction of the afferent arteriole, with a consequent reduction in renal blood flow and AHT developing. In spite of this, given hydronephrosis and HK, the most common cause is lithiasis, followed by PS.[4] HK is diagnosed using imaging tests. CT with modern multidetectors can perform a multiplanar reconstruction and confirm

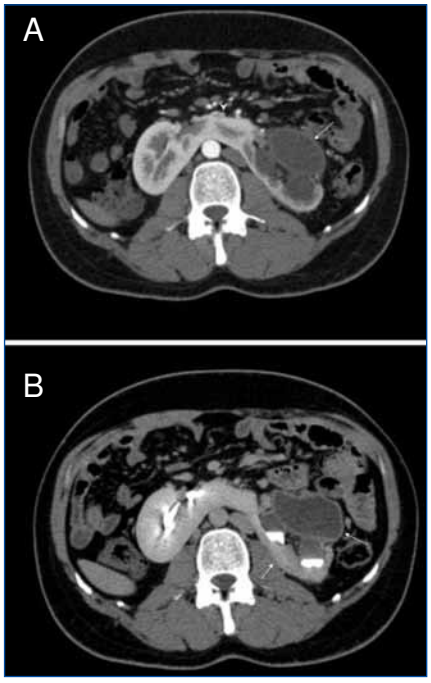

Figure 1A y B. 1A and B. Abdominal CT scan after administering I.V. contrast agent. A) nephrogenic phase. Cystic formation in the left kidney at the height of the hilum (arrow). B) Excretory phase. The passage of the contrast agent is observed, confirming that it corresponds to dilation of the pyelocaliceal system.

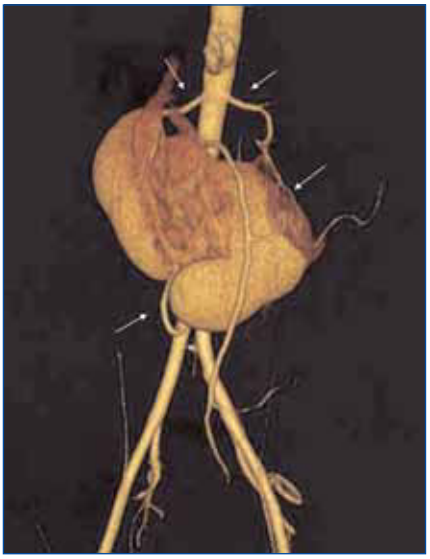

Figure 2. 3D reconstruction of the previous test; horseshoe kidney with multiple accessory arteries (arrows).