were reduced. After these measures were taken, diarrhoea disappeared and kidney function recovered to basaline levels and 17 months later the patient was still asymptomatic.

Acute diarrhoea is a significant complication in solid organ transplant recipients (SOTR). The differential diagnosis of acute diarrhoea in these cases is difficult, since both microorganisms and immunosuppressive medication can be involved, especially mofetil mycofenolate.[1]

Our patient maintained a stable kidney function for 5 years after transplant with double immunosuppressive therapy. Drug levels remained stable, therefore it was ruled out that the diarrhoea could be secondary to immunosuppressive therapy. Suspected diagnosis was microorganisms; the usual ones were quickly ruled out, including *Clostridium difficile*[2] and CMV.

Cryptosporidium parvum is an intracellular protozoan that causes gastrointestinal disease worldwide, since it is an intestinal parasite of domestic and wild animals. This infection is more common in developing countries. The parasite is initially transmitted by the faecal-oral route, and in epidemics it has been associated with contaminated municipal water, person-to-person transmission and even animal-to-person transmission.[3]

Clinical symptoms of cryptosporidiosis depend on the host's immunological status. In immunocompetent subjects it causes self-limited diarrhoea, but in immunodepressed patients the infection can be prolonged and life-threatening, since there is no specific antiparasite drug.[4,5] In addition to intestinal involvement, in immunosuppressed patients, cases have been described with respiratory system, gallbladder and sclerosing cholangitis involvement.[6]

Microbiological diagnosis depends on the observation of the parasite in the faeces using a microscope and a modified Ziehl-Neelsen stain or modified Kinyoun stain that reveal the presence of 4-6 micron red oocysts. Several samples of faeces collected on subsequent days must be examined since oocyst shedding is intermittent.[7]

The cornerstone of treatment in immunodepressed patients is correction of electrolyte and acid base balance disorders.[4] This infection can be treated with nitazoxanide, paromomycin and azithromycin. However, clinical response is variable and the intestinal protozoan can be difficult to eradicate. It would seem that in a SOTR reduction of immunosuppression, together with antimicrobial treatment, can improve immunological status and achieve infection resolution.[8]

This case is interesting due to the importance of suspecting this infection in any immunodepressed patient who develops diarrhoea when no other cause is found.

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Received: 16 Apr. 2010 | Accepted for publication: 17 Apr. 2010

Antebrachial Skin Necrosis after Reconstruction of an Arteriovenous Fistula in a Patient on Haemodialysis

Nefrologia 2010;30(4):477-9

doi:10.3265/Nefrologia.pre2010.Apr.10299

Dear Editor,

The most frequent complications of arteriovenous fistula (AVF) type subcutaneous vascular accesses are infections, thrombosis and complications due to low flow, such as steal syndrome and stenosis, both intrinsic to anastomoses and venous return. An infrequent complication, but one that is difficult to treat, is calciphylaxis. It affects small peripheral arteries, causing very painful skin necrosis.[1-9] The case that we present below is unusual because,

although it presents some similarities with calciphylaxis, it differs in pathogenesis, treatment and resolution, since it is a surgical complication in a patient with a very calcified arterial bed.

The case is a 75 year old man, with a history of stage 5 chronic renal failure of unknown origin, who has been on haemodialysis for 7 years. Other history data of interest: chronic obstructive pulmonary disease, ischaemic stroke without sequelae, high blood pressure, former smoker, chronic atrial fibrillation anticoagulated with acenocoumarol, hypercholesterolemia, erosive duodenitis, appendectomy, several episodes of hyperpotassaemia, intermittent claudication, and femoropopliteal revascularization.

Regarding vascular access, haemodialysis was started through a right jugular tunnelised catheter, subsequently a humero-cephalic arteriovenous fistula (AVF) was performed in the left arm, but it did not function. Subsequently a radio-cephalic AVF was performed without problems in the right arm, and the catheter was removed. It functioned without any problems from the beginning, but progressively, during 2008, a very high recirculation index was detected (44.3%), with the consequent infra-dialysis. After consultation with the vascular surgery department, it was decided to carry out a reconstruction at a more proximal site, since the artery was very calcified. After surgery, there was a significant improvement of recirculation values, as well as analytical values corresponding to urea kinetics (KtV). On the 7th of May 2009, this vascular access suffered thrombosis; therefore a left jugular tunnelised catheter was implanted. Subsequently, on 18th August 2009, a new, more proximal, radio-cephalic AVF was performed in the right arm. A surgical complication developed, an expanding haematoma which affected the whole forearm. Progressively, the patient suffered pain and coldness of the right hand, with decreased pulses, compatible with steal syndrome. As to the antebrachial haematoma, its size did not decrease, and an ever greater area of tissue necrosis developed in the region of the surgical wound, reaching a size of 4 cm in length by 4 cm in width, with underlying granulation tissue surrounding the scab, as shown in Figure 1. In view of this situation, an arterial echo Doppler of the right upper limb was performed and a functioning AVF detected, with arterial steal, and a non-encapsulated haematoma. Pain increased, as also coldness and hand functional impotence, so we ligated the AVF on the 10th of September 2009. After intervention the symptoms of steal syndrome disappeared, the necrotic scab became detached and the antebrachial haematoma began reabsorption. For 2 months we proceeded to dress the underlying wound beneath the necrotic scab, until complete healing was achieved. Since then, the patient has been asymptomatic, and it was decided to maintain the left jugular tunnelised catheter as a permanent vascular access in view of the failed attempts to obtain other subcutaneous accesses.

Differential diagnosis with calciphylaxis is necessary, since the patient's risk factors predisposed to this disorder, and the skin lesion had certain similarities. However, since the cause-effect relation with surgery was evident, and hand ischemia caused by the steal syndrome caused the patient intense pain and functional impotence, we opted for AVF ligature. It led to the complete recovery of hand ischemia and the skin necrosis, after opportunely applying dressings, with no sequelae. If the case had not resolved, it would have been necessary to perform a biopsy to rule out calciphylaxis. There are no similar cases described in the scientific literature, the most similar disorders are pressure ulcers, caused by external pressure in bedridden patients or patients with reduced mobility.[10-15] Pressure in our case was caused from the subcutaneous compartment due to a space impingement on the surgical wound area caused by the expanding haematoma, which, together with defective vascularisation due to severe arterial calcification, caused skin necrosis.

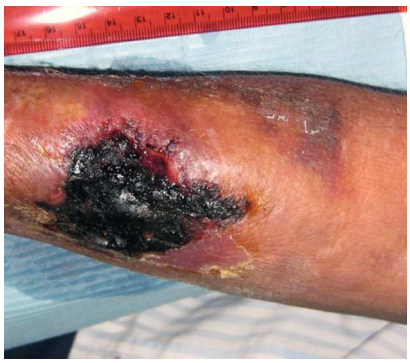

Figure 1. Skin necrosis.

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Received: 15 Apr. 2010 | Accepted for publication: 17 Apr. 2010

Interstitial Pneumonitis caused by Everolimus

Nefrologia 2010;30(4):479-80
doi:10.3265/Nefrologia.pre2010.Apr.10337

Dear Editor,

mTOR inhibitors are immunosuppressors that block proliferation signal and have a lower nephrotoxic potential. However, a series of adverse reactions have been described, especially with sirolimus, among which the most outstanding is pneumonitis.

We present the case of a 70 year old man, suffering from hypertension and dyslipidaemia, who received his first kidney transplant from a deceased donor in March 2009, due to chronic kidney disease secondary to chronic pyelonephritis. Immunosuppressive treatment consisted of basiliximab, mycofenolate, everolimus and steroids. The patient presented good evolution, with creatinine on discharge at 1.7 mg/dl.

The patient is admitted 4 months after transplant surgery with a fever over 10 days, cough and dyspnoea. On physical examination poor general condition is noted with chills, tachycardia, bilateral crepitation and oxygen saturation of 93% (arterial blood gas: pH 7.50, pCO_2 26.8, pO_2 60, HCO_3 20.9 mmol/l). The patient also presented non-pruriginous erithematous exanthema on the back and chest, with lip herpes.

Laboratory findings were 3,980 WBC/μl (eosinophils 29.3%), haemoglobin 12.1 g/dl, platelets 276,000, creatinine 1.79 mg/dl, plasma sodium 138 mEq/l, plasma potassium 3.9 mE/l, and plasma levels of everolimus of 14 ng/ml. On the chest X-ray a bilateral interstitial pattern was seen and the chest CT showed pulmonary nodules and infiltrates with thickening of the bilateral interstitium (Figures 1 and 2). Antigen studies and cultures of cytomegalovirus (CMV) were negative, as also pneumococcal and *Legionella* antigen studies. A bronchoscopy was performed and signs of chronic bronchopathy were seen with negative BAl and BAS. Empiric treatment with wide spectrum antibiotics and antivirals was begun, without any clinical improvement and with radiological worsening. Therefore, suspecting pneumonitis due to everolimus, we decided to discontinue this drug and introduce tacrolimus. On the day after discontinuing everolimus the patient presented clinical and radiological improvement with resolution of the condition.

To summarise, knowing that pneumonitis may appear with all mTOR inhibitors can lead to rapid discontinuation of the drug, preventing unnecessary diagnostic and therapeutic action in kidney transplant patients.

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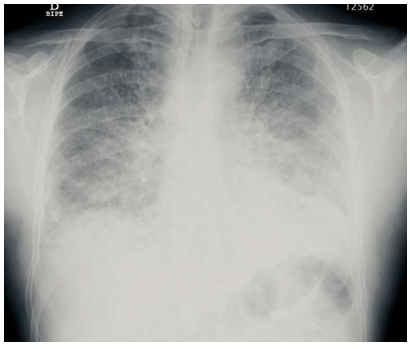

Figure 1. Chest X-ray .

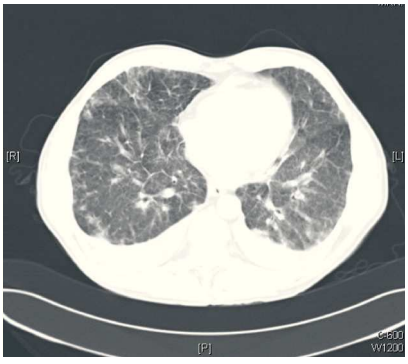

Figure 2. Chest CT