

subcortical atrophy. The MRI showed significant hyperintensity throughout the protuberance and in the pontine-mesencephalic union without diffusion restriction, which was therefore unrelated to ischaemia. Central pontine myelinolysis was then diagnosed. In the supratentorial area, she presented a significant hyperintensity in the periventricular white substance and an old lacunar infarct (Figure 1).

Clinical progress was satisfactory after considering a regime of repeated short, daily haemodialysis sessions, and the condition resolved in six days. Obviously, considering the patient's age and underlying condition, a sure diagnosis would be difficult to determine. However, we must underscore how unusual this acute neurological process was, following a year on dialysis, with no obvious changes in osmolarity, etc. However, it would seem that it could be related to aggressive dialysis sessions in a senile patient who may have been subjected to excessive ultrafiltration. The recovery time for the process, as well as the MR image, mean that we must consider a process of myelinolysis.

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Primary cytomegalovirus infection causing a kidney transplant patient to develop cryoagglutinins and cryoglobulins

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Dear Editor,

The development of cryoagglutinins and cryoglobulins following primary infection with cytomegalovirus (CMV) has been described in immunocompetent patients, and manifests in the infection's acute phase. Few examples of literature refer to the effect that this primary infection may have on a patient subjected to a high-risk kidney transplant (positive donor/negative recipient) following six months on

prophylactic valgancyclovir, which is universally accepted for this patient type.

We present the case of a 42-year old kidney transplant patient who developed mixed cryoagglutinins and cryoglobulins (IgM/IgG) in the context of a primary CMV infection two months after ending prophylactic treatment with valgancyclovir.

He received a kidney transplant from a cadaver donor in July 2007. The immunosuppressant regime consisted of basiliximab, steroids, tacrolimus and mycophenolate mofetil (MMF). During the first six months following the transplant, prophylaxis was performed with oral valgancyclovir in doses of 900mg/day. Two months after finishing treatment, he was seen for a mononucleosis-like syndrome and epigastric pain. Tests showed leucopenia (3100 leukocytes/mm³), hyperbilirrubinaemia (1.34mg/dl) and elevated LDH (277U/l) with normal transaminases and normal kidney function. CMV antigenaemia assay was performed, which was positive with a titre of more than 100 infected cells per 200,000 leukocytes. The patient was admitted for intravenous treatment with gancyclovir dosed at 5mg/kg/12 hours. During admission, the patient developed significant leucopenia that required use of granulocyte-macrophage colony-stimulating factor, cryoagglutinins at a 1/64 titre and IgM/IgG type cryoglobulins indicating type II cryoglobulinaemia, as well as monoclonal IgM kappa precipitate visible in the proteinogram. After evaluating the data with the help of the haematology department, we considered them secondary to the viral infection. MMF treatment was suspended, and we decided to replace the tacrolimus with mTOR inhibitors because of their beneficial action in eradicating CMV infection.¹ Treatment with gancyclovir was extended during 28 days, at which point a gradual decrease was observed in the antigenaemia titres until they became completely negative. Renal function continued to be stable at all times (Cr 0.9mg/dl). Cryoagglutinins



Figure 1. Craneal magnetic resonance image.

and cryoglobulins became negative two months after admission of the patient; the monoclonal spike was still present as late as seven months later.

The role that a primary infection with CMV or other viruses (HBV, VHC, HIV)² may play in the appearance of haematological and immunological abnormalities has been documented. Development of type II cryoglobulinaemia is one of the most frequently-documented forms in the literature.^{3,4} These abnormalities tend to be temporary and correlate with the progress of the infection. This type of anomaly may also develop in kidney transplant patients.⁵ It tends to revert following total elimination of the virus through antiviral treatment, and may complicate the treatment for these patients during the acute phase of the infection.

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A case of p-ANCA positive vasculitis with associated pericardial effusion

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Dear Editor,

Microscopic polyangiitis is a necrotising vasculitis primarily affecting the glomeruli (vasculitis limited to the kidney may be the only manifestation), and secondly, the lungs, but it is a systemic process that may affect small blood vessels in any part of the body. High blood pressure is the most common cardiovascular manifestation.

We present the case of a patient with sub-acute deterioration of renal function and affected pericardium who responded well to steroid treatment.

This 60-year old female patient with a history of diabetes mellitus that was diagnosed three years before, with no microangiopathy or macroangiopathy and high blood pressure during ten years which was treated with carvedilol, hydrochlorothiazide and valsartan. One year before, her creatinine level was 0.8mg/dl, GFR (MDRD) > 60 ml/min/1.73m, and systemic/urine sediment showed no abnormalities. In the six weeks prior to admission, she reported progressive weakness, followed by fever and dry cough. She did not present dyspnoea, chest pain, or diuresis alterations. The examination revealed blood pressure of 190/80mmHg, breathing well, with high jugular vein pressure, rhythmic cardiac sounds with pericardial rub, normal bilateral hemithorax ventilation and no oedema. The analysis showed haemoglobin levels of 7.7mg/dl, urea 217mg/dl, creatinine 4.5mg/dl,

potassium 3.6mEq/l, 98% baseline saturation; in the urine; proteinuria of +++ and 60 red blood cells/field. The ECG was normal. The chest radiography showed severe cardiomegaly (figure 1). An emergency echocardiography showed a moderate-to-severe pericardial effusion. Ultrasound showed no kidney alterations.

Treatment was started with doses of steroids at 1mg/kg/day, and the patient's state improved considerably in a few days. With regard to the laboratory results, the direct Coombs test was negative, haptoglobin was 4g/l, LDH 370U/l, C3 and C4 within the normal range, ANA was negative, ANCA was positive, with a p-ANCA pattern. Reactivity analysed by the ELISA test corresponded to antimyeloperoxidase of 146U/ml. Proteinuria was 2g/24 hours. The percutaneous renal biopsy of a total of 17 glomerules was compatible with focal proliferative glomerulonephritis, with crescents at 50% and significant tubulointerstitial involvement. A dose of IV cyclophosphamide was recommended, and the echocardiography was repeated (a day before cyclophosphamide bolus administration) with no signs of the pericardial effusion. After a stay of 18 days, she was discharged with a creatinine value of 1.8mg/dl.

In small-vessel vasculitis, it is possible to have primary or secondary cardiac involvement relating to high blood pressure, immunosuppressants, the



Figure 1. Cardiomegaly without other signs suggesting heart failure.