Haemospermia in malignant hypertension

Nefrologia 2010;30(2):266

Dear Editor,

Specialised literature informs us that hypertension may accompany haemospermia. This association may be considered merely statistical, but the fact that it appears in more severe hypertension, such as malignant hypertension, cannot.[1]

We present the case of a 36-year old man who visited the hospital due to emitting blood in the semen. Apart from the presence of untreated hypertension which had been present for ten years, there were no other relevant data. Examination, laboratory testing, cultures and imaging techniques did not provide any data regarding its origin. Furthermore, the urological history was uneventful, with no history of trauma or infection, and there were no accompanying clinical profiles, prior medications or sexual habits that could be considered abnormal. The patient had a blood pressure of 220/140mmHg, and was asymptomatic with the following relevant data: ocular fundus with oedema of the optic nerve, exudates and haemorrhages, ECG in which we observed ventricular hypertrophy with a systolic overload, renal failure (Cr > 3mg/dl) with proteinuria ++++. In a previous analysis, renal function had been normal. The usual hormonal and radiology studies to rule out secondary hypertension were negative. He was identified as a malignant hypertension patient, and it was believed that a kidney biopsy would not be indicated or appropriate.

Haemospermia is generally a self-limited, benign process which is idiopathic in many cases. In others, it is secondary to aggressive urological examination, exacerbated sexual activity or excessive sexual continence, or rarely, a tumour. Underlying hypertension is described in at least 6% of all cases.[2] Coinciding haemospermia and malignant hypertension has occasionally been described by several authors.[3,4] An association between asymmetrical ectasia of the seminal vesicle (seminal ectasia can cause haemospermia) and severe hypertension can also be described.[5] Although the physiopathology of the process behind this association is not clear, presence of haemospermia in these acute forms of high blood pressure, which has already been described, may not be a mere coincidence.

1. Close CF, Yeo WW, Ramsay LE. The association between hemospermia and severe hypertension. Postgrad Med J 1991;67:157-8.
2. Ahmad I, Krishna NS. Hemospermia. J Urol 2007;177:1613-8.
3. Fleming JD, McSorley A, Bates KM. Blood, semen and innocent man. Lancet 2008;371:958.
4. Bhaduri S, Riley V. Hematospermia associate with malignant hypertension. Sex Transm Inf 1999;75:200.
5. Weidner W, Jantos CH, Schumacher F, et al. Recurrent hemospermia-underlying urogenital anomalies and efficacy of imaging procedures. Br J Urol 1991; 67:317-23.

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Myelinolysis or late-onset imbalance in dialysis

Nefrologia 2010;30(2):266-7

Dear Editor,

The first description of myelinolysis dates from 1959. Since then, little progress has been made in determining its definitive cause, although we know the risk factors or the processes from which it arises. The most commonly reported form occurs in relation with rapid correction of hyponatraemia.[1] It has also been described in alcoholism, liver transplant, and more rarely, following haemodialysis sessions not always related with changes in natraemia.[2-4] In the latter, imbalance syndrome is much more common. However, there are circumstances that may cover both processes, especially during infancy.[3] The clinical profile is characterised by an unexplained drop in level of consciousness, inability to look up, motor deficit in all four limbs, pseudobulbar syndrome, etc. In the proper clinical context, an MRI showing several indicative findings, especially at the protuberance,[5,6] is an aid to diagnosis. The definitive diagnosis is by anatomical pathology. The course of the syndrome may be fatal even when it spares sensitivity and level of consciousness (locked-in syndrome), and treatment is purely conservative.

Our 82-year old female patient, on haemodialysis throughout the past six months due to chronic renal failure (CRF) secondary to amyloidosis, was admitted for heart failure. She underwent an emergency dialysis session with 2500 ultrafiltration, and 24 hours later, a second ultrafiltration of 2000 given the scarce clinical and radiological signs of improvement. The patient, who initially presented a stable neurological state, began to show a strange clinical profile when finishing the second session of acute dialysis. It consisted of a low, fluctuating level of consciousness, absence of language ability, fixed gaze looking forward and loss of strength in the right arm and both legs. Specialised examination pointed to a dialysis imbalance process. Pre- and post-haemodialysis Na levels did not show significant abnormalities (136 to 132mEq/l), and neither were the changes in other parameters (urea, etc) outside of the normal range. We performed a cranial TC which detected an old thalamic lacunar infarct and cortico-

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 myelinosis.

1. Lohr JW. Osmotic demyelination syndrome following correction of hyponatremia: association with hypokalemia. Am J Med 1994;96(5):408-13.
2. Huang WY, Weng WC, Peng TI, Ro LS, Yang CW, Chen KH. Central pontine and extrapontine myelinolysis after rapid correction of hyponatremia by hemodialysis in a uremic patient. Ren Fail 2007;29(5):635-8.
3. Aydin OF, Uner C, Senbil N, Bek K, Erdoðan O, Gürer YK. Central pontine and extrapontine myelinolysis owing to disequilibrium syndrome. J Child Neurol 2003;18(4):292-6.
4. Kim J, Song T, Park S, Chois IS. Cerebellar peduncular myelinolysis in patient receiving hemodialysis. AJR Am J Roentgenol 2004;182(3):809-16.
5. Kumar S, Fowler M, González-Toledo E, Jaffe SL. Central pontine myelinolysis, an update. Neurol Res 2006;28(3):360-6.
6. Aðildere AM, Benli S, Erten Y, Co?kun M, Boyvat F, Ozdemir N. Osmotic demyelination syndrome with a dysequilibrium syndrome: reversible MRI findings. Neuroradiology 1998;40(4):228-32.

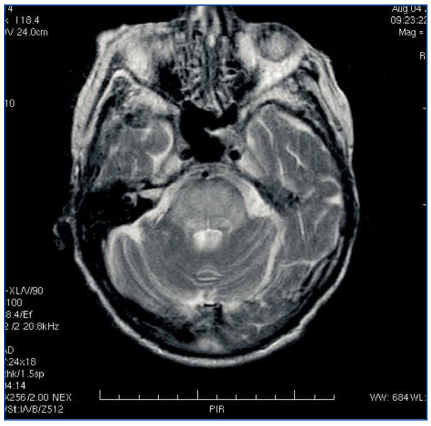

Figure 1. Craneal magnetic resonance image.

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

Nefrologia 2010;30(2):267-8

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 leucopoenia (3100 leukocytes/mm^3), 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 leucopoenia 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