Haemospermia in malignant hypertension

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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.

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Myelinolysis or late-onset imbalance in dialysis

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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-