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LETTERS TO THE EDITOR

Sequential growth of a cerebral haematoma after dialysis

Crecimiento secuencial de hematoma cerebral tras hemodiálisis

Dear Editor:

Patients with chronic renal failure have an increased incidence of ischemic and hemorrhagic strokes[1] due to the high frequency and synergy of multiple vascular risk factors (arterial hypertension, diabetes mellitus, malnutrition, platelet dysfunction, treatment with erythropoietin). The incidence is highest in the advanced stages on haemodialysis (HD) support (up to 13 ictus/1,000 patients-year)[2,3], with a relative increase in the proportion of cerebral haemorrhages.

We report the case of a hypertensive 52-year-old woman with chronic renal failure due to cortical necrosis (disseminated intravascular coagulation from foetal death), who had been in a HD programme for 2 years and was on the waiting list for kidney transplantation. She was diagnosed with chronic hepatopathy due to the hepatitis C virus, with recent viral response to interferon and ribavirin and in treatment with erythropoietin, Caosina (calcium carbonate), Becozyme and Seguril.

She came to our centre with symptoms of sudden onset of right limb claudication and sleepiness, 3 days after a HD session (with heparin) that had taken place without incident. On examination we observed: blood pressure of 180/111 mmHg, mixed dysphasia, homonymous hemianopia and right hemiparesis (NIHSS-15). Computed tomography (CT) revealed a deep haematoma in the left basal ganglia (volume 42 ml; fig. 1A). Creatinine was 10.29 mg/dl, urea was 193 mg/dl, and ureic nitrogen was 90.1 mg/dl; haemostasis was normal and platelet count was 110,000. She was admitted into the stroke unit with supportive care and was prescribed a HD session without heparin. After 3 h, we observed deterioration in her level of consciousness, slurred speech and motor deficits (NIHSS-21) with growth of the haematoma (87 ml; fig. 1B) in the control CT scan. The evolution was unfavourable, with symptomatic crises requiring

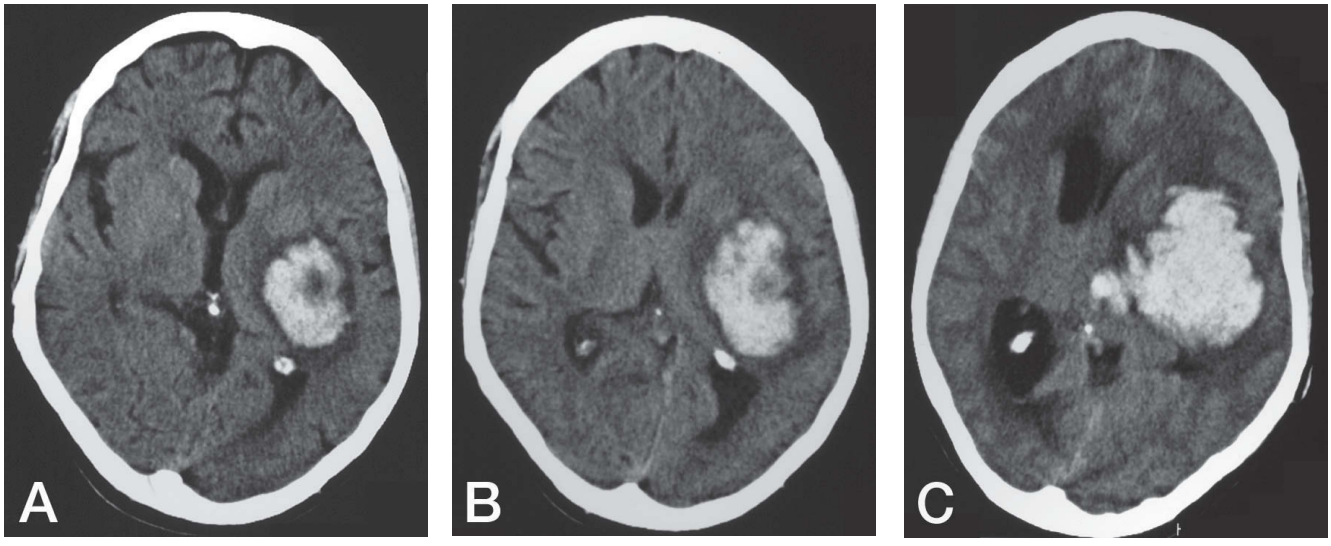

Figure 1 CT scan: sequential growth of spontaneous deep haemorrhage in left basal ganglia (day +1 [A], +2 [B] and +5 [C]).

sedation and endotracheal intubation, signs of transtentorial herniation (absence of verbal response and eye opening, decerebrate rigidity of the right limbs) and systemic complications (fever, supraventricular tachycardia and haemodynamic instability). Due to electrolyte alterations, she required a new HD session on the third day, which conditioned a further neurological deterioration (unresponsive right mydriasis, absence of motor response), with progression of the haemorrhage (volume, 135 ml; fig. 1C) in the control CT scan. The next HD session took place on the fifth day, and on the sixth day she died due to cerebral death.

Haemodialysis, applied to patients of increasingly advanced age and with elevated comorbidity, increases the already high risk of brain haemorrhage in patients with nephropathies, as described in prospective series in countries with low transplantation rates (Japan, United States). This observation is attributed to the use of anticoagulants, fluctuations in volemia and increases of perilesional oedema through osmotic changes during the procedure[2-4]. These are brain haematomas with a high probability of rebleeding (5-10 times higher) and early mortality (80% in the first month, twice that of the general population)[5]. The low level of consciousness on admission, the large size and irregularity of the haematoma, the hypertension (especially diastolic), the diabetes mellitus, the increase in fibrinogen and prothrombin time have all been considered as poor prognostic factors. Surgical treatment is not recommended due to the high risk of rebleeding. It is necessary to maintain the renal replacement therapy, although haemodialysis may condition the growth of brain haematomas even when performed without heparin, as illustrated by our case. Techniques such as continuous haemofiltration or the use of nafamostat mesilate instead of heparin could help to reduce this risk[5,6].

This work was presented as a communication (poster) at the LX Annual Meeting of the Spanish Society of Neurology (November 2008) and at the Sixth Meeting of the Madrid Association of Neurology (October 2008).

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Epileptic seizure as a first sign of a cardiac angiosarcoma

Crisis epiléptica como primera manifestación de angiosarcoma cardíaco

Dear Editor:

Primary cardiac tumours are a rare entity. Their incidence varies between 0.0017 and 0.033% depending on the series consulted[1,2]. Only 0.2% of cases are found in systematic autopsies and the majority are benign (90%)[1]. Among primitive malignant cardiac tumours, sarcomas stand out for their frequency (76-78%), and angiosarcoma is the most common in adults[1-3], prevailing between the second and fifth decade of life[3] and in the male gender[2-4]. Two thirds of angiosarcomas are located in the right atrium, although those located in the left atrium have a better prognosis. Case report: Caucasian male, 45 years old, with no known allergies or relevant medical history. Ex-smoker (10 cigarettes a day for two years) having no toxic habits. Family history included the death of his father at age 57 due to gastric cancer, of his mother at age 65 due to melanoma with disseminated bone disease, of his brother at age 45 due to lung neoplasm, and of his sister at age 42 due to brain metastasis with primary lung tumour and of another sister at age 57 due to liver sarcoma. Although he was previously well, he presented a focal seizure (clonus, left hemisphere) with subsequent generalisation. The neurological examination observed dysarthria and left distal crural paresis (4/5) with Babinski sign. Cardiac auscultation was absolutely normal. The complementary tests showed no significant alterations in blood count, biochemistry, immunity, coagulation or blood gases. The serological tests performed to rule out brain damage caused by infections (HIV, cytomegalovirus, toxoplasma) were negative. An increase in serum enolase (33.6 ng/ml) was observed, along with normality of other tumour markers. The neuroimaging tests performed (computed tomography [CT] and cerebral magnetic resonance imaging [MRI]) (fig. 1) revealed an image compatible with right parietal solitary metastasis with slight concomitant bleeding. A chest CT (fig. 2) revealed metastatic lung nodules and a tumour in the right atrium, shown in the echocardiography as a rounded neoformation of 59?42?30 mm, with a broad base of implantation, variable density, polycyclic edges and with small, mobile areas on its surface. The remaining study results (bronchoscopy, bone