specificity for the proteinase 3 antigen. One year after the diagnosis, the patient was asymptomatic.

JC is an ischaemic symptom of fatigue or pain with mastication caused by the narrowing or obstruction of the facial branches of the external carotid (which irrigate muscles used in mastication), and which is present in 45% of patients with TA. Other, less common causes of JC are primary amyloidosis, polyarteritis nodosa (PAN), Churg-Strauss syndrome (CSS), Takayasu's arteritis, GW, hairy cell leukaemia, McArdle's disease, crioglobulinaemia associated with vasculitis, and carotid atherosclerosis.

Compromise of the temporal artery associated with JC has been shown with a very low frequency with PAN, CSS, Takayasu's arteritis, crioglobulinaemia associates with vasculitis, primary amyloidosis and WG.[2] To increase the complexity of the issue, TA may affect the kidneys and lungs just as WG can.[2] In turn, TA can be associated with other types of vasculitis (such as CSS, PAN and WG),[4] with rheumatoid arthritis, primary biliary cirrhosis and neoplasias. The association of WG with other types of vasculitis, such as CSS and AT, has also been described.

It is interesting to recall that among the causes of giant cells in a temporal artery biopsy, we find systemic lupus eritematous, isolated angeitis of the central nervous system, Takayasu's arteritis, and TA. A compromised temporal artery without giant cells has also been described in some cases of systemic vasculitis such as hypersensitivity angeitis, crioglobulinaemia, CSS, WG and PAN.

There are ten patients described in the literature who had WG and an initial clinical profile compatible with TA.[1-4] All of these patients were older than 60 and had JC with or without sudden loss of sight, severe headache with or without double vision, or polymyalgia rheumatica upon diagnosis. The GSV was high at the onset of symptoms in all patients. Biopsy of the temporal artery showed TA in two patients, arteritis without giant cells in four patients, and for the rest it was negative to normal, as with our patient. Within six months, the ten patients developed renal and/or pulmonary lesions characteristic of WG, with typical histologies in the biopsy or positive ANCA.

In summary, we can state that there are five different categories to describe a vasculitis-induced compromised temporal artery, which are: 1) temporal arteritis without giant cells due to multiple entities; 2) temporal arteritis with giant cells, whether caused by TA or not; 3) TA concurrent with WG or other forms of vasculitis; 4) WG with a clinical profile resembling temporal vasculitis but with a negative biopsy (our patient); and 5) TA with clinical characteristics of WG (very uncommon).

Documenting the different histological types of vasculitis that produce similar clinical manifestations emphasises the importance of obtaining a biopsy, whether diagnostic or prognostic, given that treatments may be very different.

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Massive hepatic haematoma in patient on haemodialysis

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Dear Editor:

Patients on periodic HD have an elevated risk of spontaneous haemorrhages or haemorrhages caused by minimal trauma to the retroperitoneal, renal, pericardial, mediastinic and subdural areas. Multiple factors affect predisposition to bleeding, including platelet dysfunction, anti-platelet agents and anticoagulants.[1,2] The development of a subcapsular hepatic haematoma is an exceptional complication that can occur spontaneously or with minimal closed trauma.[3,4] We present a patient aged 77 years who in 2003 was diagnosed with chronic kidney disease second to myeloma kidney (IgG Kappa). Treatment with melfalan/prednisone decreased the circulating paraprotein and improved the medullar affectation. In February 2004, the patient began HD. During the last two years, the patient received two cycles of melfalan/prednisone, and later, bortezomib (Velcade®). He remained stable, with the disease under control and without anaemia with EPO. From April to July 2006, he received conjugated oestrogen (Equin®) (20mg/day) for uraemic thrombopathy. Lately, the patient has been taking aspirin as an anti-platelet agent. On 7 August 2006, eight hours after finishing the HD session, he experienced dizziness and fell, striking his right hypochondrium, and lost consciousness for several minutes. When he was admitted to the hospital he was conscious and aware of his surroundings; he complained of pain in his right scapula. Blood pressure was 55/35, cardiac frequency 72l/min and he showed sinus rhythm. Analytical tests showed: haematocrit 19%, haemoglobin 6.9g/dl, 300,000 platelets/mm^3, 10,000

leukocytes/mm^3 with the normal formula, PT 82%, INR 1.2 and fibrinogen 520mg/dl. Other data: Na 139mmol/l, K 6.7mmol/l, HCO_3 22mmol/l, urea 84mg/dl, Cr 7.5mg/dl, GGT 727UI/l, GOT 127UI/l, GPT 167UI/l, LDH 372UI/l and total bilirubin 0.4mg/dl. A thoracic-abdominal CT with contrast revealed a subcapsular hepatic haematoma measuring 10 x 7 x 20cm in the right lobe; the organ was displaced, but without underlying hepatic lesions (figure 1). The kidneys were atrophic and there was a moderate amount of free liquid (blood) in the pouch of Douglas. To reverse the hypovolaemia, we administered isotonic saline and blood transfusions (four packs of red blood cells) and gum for the hyperpotasaemia. Six hours after being admitted, his haemodynamics were stable and he underwent an HD session without heparin. In the following 48 hours he remained at rest and under observation with frequent haematological checks. Haemoglobin levels remained stable. Local discomfort required mild analgesics. Over the following days, HD was performed under close monitoring and without heparin. Eight days after admission, during a CT exam, the haematoma had not increased in size and remained within the subcapsular area. The patient was referred to his original unit and follow-up by a series of CTs showed the slow, progressive reabsorption of the haematoma over eight weeks, with no need for surgery.

The likely causes that would favour bleeding, in addition to the uraemia, could include the myeloma itself, the administration of oestrogens, use of heparin during HD, the anti-platelet agent, and lastly, the closed local trauma. Although the CT showed no sign of underlying hepatic lesions (adenoma, haemangioma or peliosis) the patient could suffer from lesions arising from hepatic peliosis that were undetected by the CT.[4,5] Hepatic peliosis , which is characterised by cystic blood-filled spaces in the sinusoids, is associated with the administration of androgenic-anabolic and other steroids, oral contraceptives and synthetic oestrogens. Its complications can include ruptured liver and spontaneous hepatic haematoma.[6-12] In this case, both the myeloma and the oestrogens could have favoured the development of hepatic peliosis,[8,10] making the liver more susceptible to spontaneous rupture or rupture from closed trauma.[3] Many hepatic haematomas that are haemodynamically stable are given conservative treatment without surgical intervention and have a success rate of more than 80%.[13,14] The only determining factor for it to be treated without surgical intervention is haemodynamic stability. In our patient, once his haemodynamics were stable, conservative treatment consisting of rest, transfusions, close monitoring and HD without heparin permitted favourable evolution and the resolution of the haematoma in two months.

To sum up, the development of a subcapsular hepatic haematoma in HD is a rare complication which, by producing a severe haemorrhage and shock, places the patient's life at risk and is a challenging situation. Conservative treatment with no surgical intervention is an option that permits recovery without problems. As far as we know, this is the first case of a patient undergoing periodic HD who has presented this complication.

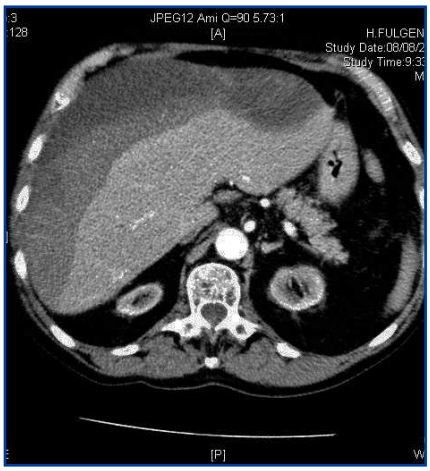

Figure 1. Contrast CT of the abdominal area. Subcapsular hepatic haematoma measuring 10 x 7 x 20cm at the level of the right lobe. Atrophic kidneys.

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Lithium poisoning and proteinuria in the nephrotic range

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Dear Editor:

The incidence of renal affectation in Systemic Sclerosis (SSc) is difficult to establish, because in the early stages and in its milder forms, the manifestations may be subclinical, and they may coincide with other pathologies, given the disease's long evolution. We distinguish between three types of nephropathy: acute, chronic and overlap syndrome (associating scleroderma and other rheumatic illnesses).[1-2]

Clinical case

Male patient, aged 36 years with a history of SSc since 2000, bipolar disorder, terminal ileitis, consumer of alcohol and marijuana.

Underwent treatment with D-penicilamine for the SSc for several years, which was discontinued a year ago due to gastric distress. Usual treatment: nifedipine, deflazacort, lithium and olanzapine.

In January 2008, the patient was referred for renal failure (RF) study, due to recent presentation of plasma creatinine at about 2mg/dl, proteinuria 300mg/dl and hypoalbuminaemia 2.36g/dl.

Physical examination showed limited flexion of the fingers; the rest was normal. BP: 123/76mmHg.

Analytical tests showed: haemoglobin, 11.5g/dl; urea, 138; creatinine, 4.2; cholesterol 157mg/dl. Total proteins, 5.4; albumin 2.6g/dl. PTH, 119pg/mml. Widespectrum immunology screen negative except for ANCA-anti-MPO+. Proteinuria, 4.2g/día; sediment, 1,042 red blood cells per high-power field. Creatinine clearance 25ml/min. In studies prior to 2000, anti-scl 70 and ANA+ were prominent. The ultrasound showed kidneys measuring 11.5cm with an increase in cortical echogenicity.

Given the presence of RF and proteinuria in the nephrotic range, we started treatment with ramipril and recommended discontinuing the lithium.

15 days later, the patient came to the Emergency Room with nausea, vomiting, and abdominal pain that had been evolving over several days. He presented a lithaemia of 4.4 mEq/l,; crp 5.8; urea 178mg/dl. Hydration treatment was started, despite the fact that it increased lithaemia; for this reason, the patient underwent emergency haemodialysis. In subsequent checks, the lithaemia was below 1.5meq/l, so it was not necessary for him to undergo additional haemodialysis sessions.

As glomerular disease was suspected, we performed a kidney biopsy. The anatomical pathology showed 10 glomerules, of which two were sclerotic and six had focal and circumferential epithelial crescents and diffuse mesangial proliferation and capillary lumens obliteration with a mixed interstitial inflammatory infiltrate. Negative immunofluorescence test (figures 1 and 2).

We started immunosuppression with three methylprednisolone tablets, followed by 1mg prednisone/kg/day and 900mg of cyclophosphamide in a monthly bolus, and followed up on the patient.

We arrived at the clinical opinion that it was probably overlap syndrome scleroderma/vasculitis with ANCA-anti-MPO+ with stage IV CKD secondary to proliferative mesangial glomerulonephritis with extracapillary proliferation.

Discussion

Hybrid forms of scleroderma/ANCA-vasculitis, sometimes associated with glomerulonephritis, are described in up to 10% of all cases. This overlap syndrome may occur after a period of treatment with D-penicilamine which varies from five months to five years. For some authors, this association represents a new entity that is related with this drug. Although our patient had gone at least a year without taking it, we can establish the hypothesis that the D-penicilamine could have acted latently as an antigenic factor, and then triggered the extracapillary form of vasculitis.[3-7]

1. Tomioka M, Hinoshita F, Miyauchi N, Et al. ANCA-related crescentic glomerulonephritis in a patient with scleroderma without marked dermatological change

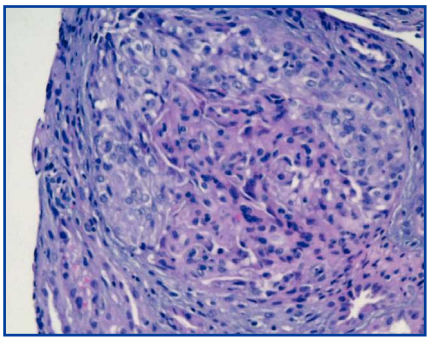

Figure 1. Aematoxylin-eosin stain, 20 x 10. Glomerule in which we see a circumferential crescent with a ruptured Bowman's capsule.

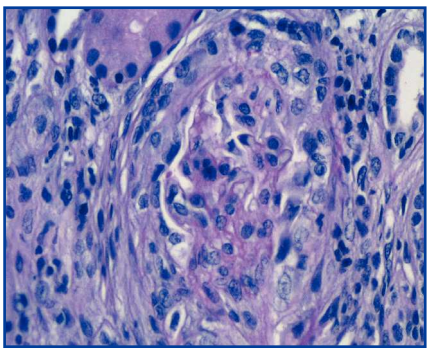

Figure 2. PAS stain, 20 x 10. Glomerule in which we observe a crescent with mixed inflammatory infiltrate obliterating the capillary lumens.