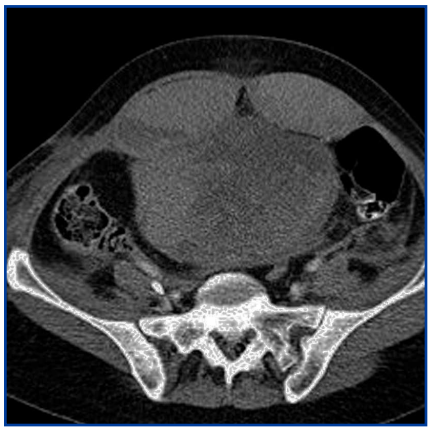

Figure 2. Abdominal CT: rectus haematoma and left hand side.

on haemodialysis, with evidence of negative anti-GBM Ab from the fourth plasmapheresis.

Disease caused by anti-GBM Ab is a medical emergency that needs to be ruled out in rapidly progressing renal failure, using an anti-GBM Ab test. Early treatment based on corticosteroids, cyclophosphamide and plasmapheresis appears to play a significant role in the recuperation of renal function.[1-3] Possible complications[2,3] are many. One complication is an alteration in coagulation, brought about by a loss of factors or the use of anticoagulants. However, a massive haematoma of the recti has not previously been described associated with plasmapheresis or disease caused by anti-GBM Ab.[2,3] Plasmapheresis is a delicate technique which should be carried out by experienced professionals, and is gradually being included in the activities of nephrologists.[4] Haematoma of the rectus sheaths is an uncommon process which can simulate intra-abdominal disease. The main predisposing factor is anticoagulation and a cough is the precipitating factor.[5,6] Diagnosis is based on clinical suspicion and confirmed with a CT.[5,6] Our patient, although showing normal coagulation, had received six plasmaphereses, and this raised for us the idea of citrate-induced coagulopathy, deficits in coagulation factors and uremic thrombopathy secondary to renal failure as possible causes.

Treatment for haematoma of the recti is initially conservative,[5,6] surgery should be reserved for progression, infection of the haematoma or haemodynamic instability. Arteriography with selective embolisation can be considered. In the case in question, the haematoma of the recti was completely resolved in forty days, despite requiring anticoagulation for the massive pulmonary embolism, and the patient survived without consequences, now having undergone a successful transplant.

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Multiple masses as a presentation of Wegener's disease

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Dear Editor:

Wegener's granulomatosis (WG) is a systemic vasculitis affecting vessels of small and medium diameter, the upper and lower respiratory tract, as well as the kidneys.[1] Although rare, systemic vasculitis can present itself as lesions resembling tumours, which can initially delay diagnosis and treatment.[2,3] We present the first such case of WG described in the literature, which presented with multiple masses in different locations.

37 year old woman who presented with weight loss, febricula, fatigue and night sweats. Symptoms had been present for six months. A physical examination revealed a palpable right supraclavicular mass and a mass on the abdominal wall at hypogastric level. The rest of the examination was normal. The laboratory showed a haematocrit of 23%, erythrocyte sedimentation rate (ESR) of 120mm in one hour (normal <20), with no microhaematuria or renal insufficiency. The chest x-ray showed a left apical mass which in the CT measured 6cm by 5cm with central necrosis, without cavitation, or hilar or mediastinal adenopathy. The antineutrophil cytoplasmic antibodies (ANCAs) were positive in high titres (810 AU, normal <10) and the enzyme immunoassay (ELISA) revealed specificity for the proteinase 3 antigen. A surgical biopsy of the right supraclavicular mass showed a necrotising granulomatous inflammation. A fibrobronchoscopy with bronchoalveolar lavage was performed, which showed up negative for neoplastic cells and infection; the transbronchial biopsy showed necrotising vasculitis. PPD and cultures were negative. Based on the above, the patient was diagnosed with WG. Treatment with cyclophosphamide 150mg/day and prednisone 60mg/day improved all manifestations. Disappearance of the pulmonary and hypogastric masses was

observed. The patient remained asymptomatic after one year.

In the literature, 89 cases of vasculitis presenting as a mass have been described.[2,3] In all of these cases the mass was present in only one location or organ, unlike our patient. The average age in the cases described was 50.5 + 15.8 years, and 51% were female. In 82% of cases, the 'tumour' was associated with constitutional symptoms and an elevated ESR. In approximately half of the patients, surgery was performed prior to diagnosis. The most frequent location for the masses was the breast (22%), followed by lesions in the central nervous system (16%). Another common location is the ovary (10%), caused by giant cell arteritis (GCA), and in the male, in the genitourinary system, caused by polyarteritis nodosa (PAN).

WG is the most common cause of vasculitic masses (32 cases), followed by GCA (18 cases) and PAN (17 cases), among others. WG is distinguished by the diversity of the locations in which the lesions (masses) are found, comprising twelve different positions (the most common being the breast and kidney). In patients with GCA, the masses are located only on the breast and ovary.

The purpose of this article is to draw attention to this unusual presentation of vasculitis. The improvement on old imaging methods, such as high-resolution tomography or nuclear magnetic resonance, among others, and the arrival of ANCAs, can assist with preoperative diagnosis. The inclusion of vasculitis in the differential diagnosis of lesions or masses leads to an earlier correct diagnosis and, consequently, the establishment of an appropriate treatment, avoiding unnecessary surgery.

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Long term hypopotassaemia associated with chlorthalidone

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Dear Editor:

Introduction

Chlorthalidone is a thiazide diuretic. Among its adverse effects is hypopotassaemia.[1]

Clinical case study

74 year old female suffering arterial hypertension for the last 20 years. Continuing treatment with chlorthalidone 50mg/24h for 20 years, with the addition a few months previously of omeprazole 20mg/24h and alprazolam 0.25mg/24h.

The patient presented with a potassium level of K 2.4mEq/l and pH 7.49 following a knee arthroplasty. Chlorthalidone was withdrawn and oral potassium was administered, normalising the potassium serum and pH. In an analysis of the past four years, the potassium levels were around 3.22mEq/l.

Physical examination was normal. Haemogram, routine biochemistry, cortisol, aldosterone, rennin, TSH and PTH, magnesium and chlorine were normal. In a 24 hour urine test, calcium, sodium and potassium of 53mmol/24h (normal: 20-125) were normal. Urological ultrasound normal.

After normalising the potassium and pH, the intake was withdrawn. Three days after having withdrawn the supply, the potassium was 3.45mEq/l and the pH was 7.49, which normalised after reintroducing the supply. One week later the potassium supply was withdrawn with levels of K 3.45 and pH 7.49; it was decided to continue treatment indefinitely and maintain subsequent follow-ups. The patient required potassium input for ten months, until it was definitively withdrawn.

Discussion

Among the severe adverse effects of chlorthalidone are hypopotassaemia and alkalosis.

Chlorthalidone and thiazide diuretics act primarily in the distal renal tubule, inhibiting the reabsorption of NaCl and increasing the reabsorption of Ca. The increase in Na and water in the cortical collecting tubule, or an increased flow rate, cause an increase in the secretion and excretion of K and H. Where hypopotassaemia is severe, an exchange of intracellular K and extracellular H takes place, bringing about alkalosis. Hypopotassaemia also causes the renal reabsorption of bicarbonate to increase. When plasma concentration exceeds the reabsorption capacity at proximal tubule level, it brings about bicarbonaturia; since Na is normally exchanged with K and H in the distal tubule, if this diminishes, the elimination of H increases, also producing alkalosis. When hypopotassaemia is severe, less than 2mEq/l, this impedes the tubular reabsorption of Cl, causing alkalosis to persist. In the case described, hypopotassaemia and alkalosis are probably secondary to a lasting renal tubular alteration associated with over twenty years of treatment with chlorthalidone, since treatment with potassium could be withdrawn after ten months.