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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P. Young, B.C. Finn, M.E. Ceballos, M. Iturraspe, M. Forrester
Buenos Aires British Hospital. Buenos Aires. Argentina.
Correspondence:
Pablo Young
Hospital Británico de Buenos Aires.
Buenos Aires (Argentina).
pabloyoung2003@yahoo.com.ar

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.

Hypopotassaemia can be present in hiperaldosteronism and rennin-producing tumours, Bartter's syndrome[2] and Gitelman's syndrome.[3]

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F.J. Nicolás Sánchez, R.M. Sarrat Nuevo, T. Soler Rosell, A.M. Gort Oromí
Internal Medicine Department. Santa María Hospital. Lleida, Spain.
Correspondence:
Francisco José Nicolás Sánchez
Servicio de Medicina Interna. Hospital de Santa María. Lleida.
fnicolas@comll.cat

Septic shock caused by *Streptococcus bovis* in a haemodialysis patient

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Dear Editor:

In patients with chronic renal failure in substitutive treatment, cardiovascular pathology is assumed to be the primary cause of morbi-mortality, followed by infection.[1] Hypoalbuminaemia, catheter holders and other factors predispose these patients to infections which are much more serious than in the general population.[2]

Streptococcus bovis is a Lancefield group D streptococcus which has properties in common with enterococcus and some streptococci of the *Viridans* group, although it differs from these in habitat and clinical significance. It is usually found in bovine livestock intestine and can contaminate through faeces, water and food of animal origin.[3] As a human pathogen, it gives rise to bacteremia and endocarditis, particularly in elderly patients with chronic disease. It is associated with adenoma, colorectal carcinoma and liver disease; it therefore appears to be related to the habitual point of entry for bacteria: the gastrointestinal system.[4]

Streptococcal peritonitis has been described in patients in substitutive treatment with peritoneal dialysis; however, the *Viridans* group is more common.[5]

We describe the case of a 77 year old patient suffering from terminal chronic renal failure as a result of probable nephrosclerosis, who had started periodical haemodialysis four years earlier, with significant associated comorbidity: myocardial ischaemia, aneurysm of the abdominal aorta subject to intervention ten years previously and HCV-positive antibodies. Splenectomised for thrombocytopenic purpura since the age of 60. Three months prior to admission the patient presented with gastroenteritis with fever after ingesting shellfish (oysters) which required hydration and was treated with ciprofloxacin.

Having previously been well and having eaten out twice that day, the patient attended at dawn, presenting with elevated fever and general ill health. After taking blood cultures and a basic laboratory test, empirical treatment with cefotaxime and gentamicin was started. Pancytopenia was detected, which had not been present four days previously at the monthly checkup. A session of haemodialysis was required for hyperpotassaemia. Hypotension persisted despite vasoactive drugs, and the patient died within hours.

Streptococcus bovis was later isolated in the blood culture.

We were struck by the speed of the condition's progress in a patient who had been in a good clinical situation beforehand.

The HCV-positive serology and the previous gastroenteritis were possible predisposing factors in the appearance of streptococcus-induced bacteremia. Given that no autopsy was performed, we cannot know if valvular lesions (endocarditis) or lesions on the prosthetic aorta were present, or if organic intestinal pathology existed.

Faced with bacteremia in patients with renal failure which also presents liver or intestinal disease, we should consider the possibility of *Streptococcus bovis* and begin early treatment with penicillin or vancomycin. A speedy introduction of specific antibiotic therapy is fundamental in avoiding the onset of septic shock, which in our case had a fatal outcome in a few hours.

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P. Galindo, A. Pérez, C. Ferreira, E. Zarco, E. Vaquero
Department of Nephrology.
Virgen de las Nieves University Hospital.
Granada. Spain.
Correspondence:
Pilar Galindo
Servicio Nefrología.
Hospital Universitario Virgen de las Nieves. Granada.
pglaura@telefonica.net