thy without amputation (Doppler study of the lower extremities with distal compromise and parietal calcifications in bilateral arterial vessels.) No Deep Vein Thrombosis (DVT.) Echocardiography and valvular Doppler with slight deterioration of the RVSD and calcifications in the aortic and mitral valves.

From 2003 to 2005, the patient presented with slight-moderate secondary hyperparathyroidism with hyperphosphataemia that was difficult to treat due to lack of compliance with diet and phosphate binders. A diet low in P was indicated with proteins and calcium binders (first calcium carbonate, then calcium acetate or aluminium sporadically.) Normocalcaemia. KT/V sp greater than 1.4 (15 hrs HD/week) with calcium dialysate 3mEq/l.

Laboratory Data: average haematocrit 35%; haemoglobin 11g/dl; Alb. 3.4g/dl; Ca 8.7mg/dl; P 7.3mg/dl; PTH 538pg/ml; FAlc 350 IU.

The patient was treated, on a discontinuous basis, with oral calcitriol, which was then suspended due to hyperphosphatemia.

In 2006 the patient presented with symmetric ulcers in the lower distal extremities (legs and feet), pruritic and very painful. Some advanced with eschar and bacterial overinfection due to scatching. The patient was treated with local antibiotic and systemic antibiotics.

Collagen disease tests and coagulation studies were normal, cryoglobulin and anticardiolipin negative.

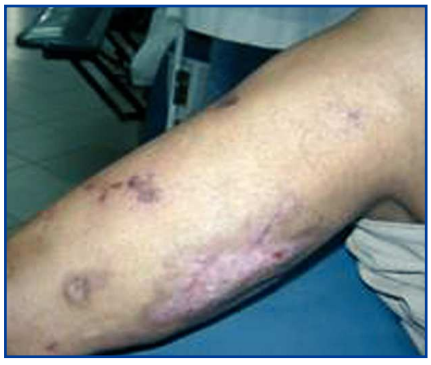

Figure 1. Resolution of calciphylaxis.

Skin biopsy showed necrosis of the superficial dermis and localised deposits of calcium in the middle arteriolar layer compatible with calciphylaxis.

Parathyroid hormone (PTH) levels were greater than 800pg/ml and the patient presented with hyperphosphatemia with normocalcaemia. Ultrasound of parathyroids only detected the lower glands. Scintography of the parathyroids with Tc99m and sestamibi detected hypercaptation in three glands.

Patient with caliphylaxis, PTHi greater than 800 and hight CaxP. Daily dialysis was indicated with dialysate calcium 2.5mEq/l. The patient did not improve, due to the persistence of injuries, and subtotal parathyroidectomy was indicated in 2006.

At the end of 2006, the lesions in the lower extremities persisted with PTHi 280pg/ml (range 150-435) and hyper-P. This was interpreted as persistence of HPT.

In February 2007, sevelamer was started, 6 capsules a day, daily dialysis and solution low in calcium (2.5mEq/l), low phosphate diet Oral ibandronate was added, 150mg/month.

Calcaemia controls remained within normal ranges. The lesions in the lower extremities improved and scarred after six months. However, the peripheral vasculopathy progressed and developed into dry gangrene.

Different results with the use of bisphosphonates are described in the literature. They have a potent inhibiting affect on osteoclastic activity and bone resorption, reducing vascular calcification. They also have an inhibiting affect on proinflammatory cytokines, allowing an improvement in the clinical picture.

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Hyperamylasaemia in a patient with multiple myeloma in haemodialysis

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Dear Editor,

An increase in amylase is a marker for acute pancreatitis. However in patients with Chronic Renal Failure (CRF) it is not uncommon to find increased levels, yet these rarely exceed two or three times the maximum normal limit.[1]

The link between hyperamylasaemia and neoplasia has been known for years and has been described in tumours of different histological strains[2] and in multiple myeloma also.[3,4] We present a patient with CRF secondary to myeloma kidney

in periodic haemodialysis (HD) who presented with hyperamylasaemia and rapid evolution of their disease.

77 year old male patient diagnosed with Lambda light chain-associated multiple myeloma in November 2002. At this time he presented with creatinine 2.8mg/dl and Bence Jones proteinuria. The patient received different lines of treatment (vincristine-adriamycin-dexamethasone; melphalan-prednisone; bortezomib) with clinical responses that were increasingly poorer. Administration of chemotherapy drugs was discontinued two years later. In July 2004, the patient started HD treatment, 12 hours a week with an average Kt/v of > 1.2. Residual diuresis was maintained at 700-1000cc/day.

The patient was admitted in November 2007 due to the deterioration of his general health with intense asthenia, epigastrium pain and significant bone pain with difficulties in deambulation. Physical examination found BP 140/80mmHg, no fever, pale skin and mucosa and without any other significant findings. Blood tests: Hb 7.9g/dl; hematocrit 23.3%; platelets 56,000/mm;[3] leukocytes 6000/mm^3 with normal formula. ESR 84 mm during the first hour; no schistocytes or blastos were observed in the peripheral blood. Iron 93μg/dl; ferritin 6603ng/ml; transferrin saturation rate 45%. Creatinine 7.91; urea 130; Ca 9.30; P 2; uric acid 7.5 (mg/dl); total proteins 6.50; albumin 3.32g/dl; IgG 370; IgA 43; IgM 15; IgD 1 (mg/dl.) Serum immunofixation: Lambda monoclonal band. C-reactive protein 13.8mg/dl. GOT 56; GPT 33; GGT 480; alkaline phosphatase 139, LDH 780, amylase 3779; lipase 449 (IU/L.) Two months before, amylase levels were normal. Amylasuria 276 IU/L, with urinary excretion of amylase 6%.

Ultrasound and abdominal Computerised Axial Tomography (CAT) did not show findings of pancreatic disease. Due to the persistence of hyperamylasaemia, the pancreatic enzymes were studied: pancreatic isozyme 132; salivary isozyme 3607 IU/l. In light of the patient's general condition, treatment was started with high doses of steroids and morphine chloride with the aim of decompressing the bone marrow and alleviating pain in the bones, which were increasing. The clinical situation worsened and the patient died 10 days after admission.

Acute pancreatitis is the first diagnosis suspect with respect to an acute increase in amylase levels. Its incidence in CRF is six times greater than in the general population.[5] Hypercalcaemia, hyperparathyroidism, dyslipidaemia and treatment with diuretics are predisposing factors. In HD, cases of pancreatitis associated with haemolysis have also been described.[6] In the present case, the clinical picture and the abdominal CAT ruled out acute pancreatitis.

Macroamylasaemia is a biochemical abnormality of unknown aetiology. It is characterised by the presence of amylase complexes attached to proteins, which form a molecule with greater molecular weight, which accumulate in the plasma, since filtration decreases.[7] Amylase levels are normally lower than two times the normal limit. Diagnosis is established by determining the molecular weight of the serum amylase, with immunological techniques, and indirectly, by determining the fractional excretion of amylase in the urine. A value < 1% is indicative of macroamylasaemia. The absence of monoclonal peak in the blood and urinary excretion of amylase of 6% exclude this diagnosis.

Paraneoplastic hyperamylasaemia is a rare manifestation of multiple myeloma. A total of 21 cases have been described with an average age of 66. It is more commonly associated with the IgA type (40%), followed by IgG (30%) and it has only been described in two patients with light chain myeloma.[3,4] The lambda light chain isotype predominates in proportions of 3:1 in comparison with the kappa type and amylase detected in all saliva originating cases. It is the tumour cells that cause the ectopic segregation of salivary amylase and the secretion mechanism is similar to that of immunoglobulins.[8] It has been seen in patients with hyperamylasaemia the myeloma is more aggressive, it has greater extramedullary extension, a poorer response to chemotherapy treatment and therefore lower survival rate (< 24 months.) Amylase in the myeloma has been used as a marker for progression and response to treatment.

Although the incidence of acute pancreatitis in patients with CRF is high, the appearance of hyperamylasaemia of salivary origin forces to considering paraneoplastic origin.

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