the majority coincide that at concentrations greater than 100mg/dl, this treatment is warranted, although others reduce this value to 80mg/dl. In any case, clinical and laboratory alterations will indicate the need for haemodialysis in the majority of cases. In this manner, patients with haemodynamic alterations, acute renal failure, severe neurological alterations, and/or severe metabolic acidosis that do not respond to conservative treatment should be started on extra-corporeal depuration treatment.

There is currently no consensus regarding the type of dialysis that should be administered. Warthall, et al[3] described reduced salicylate concentrations by 77% to 84% using continuous veno-venous haemodiafiltration for a mean 11 hours, whereas Lund, et al[4] described similar results using conventional haemodialysis followed by continuous dialysis for 12 hours. In our case, we achieved a 51% reduction using conventional haemodialysis for four hours, which demonstrates the usefulness of this technique in the acute phase. We believe that more studies would be appropriate on this subject, although the results currently available seem to indicate starting treatment with conventional haemodialysis in severe cases or patients with important clinical/laboratory repercussions, since we can achieve a significant reduction in toxin levels within a short period of time, and afterwards the patient can be evaluated for continued depuration treatment with continuous techniques, according to the serum concentrations of salicylates and the previously mentioned alterations.

### Conflicts of interest

The authors have no conflicts of interest to declare.


1. O´Malley GF. Emergency department management of the salicylate-poisoned patient. Emerg Med Clin North Am 2007;25(2):333-46.
2. Minns AB, Cantrell FL, Clark RF. Death due to acute salicylate intoxication despite dialysis. J Emerg Med 2011;40(5):515-7.
3. Wrathall G, Sinclair R, Moore A. Three cases report of the use of haemodiafiltration in the treatment of salicylate overdose. Hum Exp Toxicol 2001;20:491-5.
4. Lund B, Steifert SA, Mayersohn M. Efficacy of sustained low-efficiency dialysis in the treatment of the salicylate toxicity. Nephrol Dial Transplant 2005;20(7):1483-4.



**C. Ruiz-Zorrilla López[1], B. Gómez Giralda[1], J. Sánchez Ballesteros[2], M. García García[2], A. Molina Miguel[1]**

[1] Nephrology Unit. Río Hortega Hospital. Valladolid, Spain

[2] Intensive care unit Río Hortega Hospital. Valladolid, Spain.

**Correspondence:** C. Ruiz-Zorrilla López
Unidad de Nefrología.
Hospital Río Hortega. Dulzaina, 2. Spain
47012 Valladolid.
carlosruizzorrilla@hotmail.com


## B) BRIEF CASE REPORTS

# Chronic hypercalcaemia in haemodialysis patients


Nefrologia 2011;31(6):752-4
doi:10.3265/Nefrologia.pre2011.Jun.10974


**To the Editor,**

The increase of adynamic bone disease in haemodialysis (HD) patients can hinder the diagnosis of other diseases that progress with latent hypercalcaemia. Here we present a n elusive relevant case.

A male patient of 73 years with terminal renal failure (TRF) secondary to diabetic nephropathy that started HD in June 2008. The patient smoked 10 cigarettes/day, and did not consume alcohol. The patient had long-term arterial hypertension (AHT), type 2 diabetes mellitus for 45 years on treatment with insulin, diabetic retinopathy, dyslipidaemia, chronic ischaemic heart disease tending towards acute myocardial infarction (AMI), stage III b-IV chronic ischaemia of the lower limbs requiring femoropopliteal bypass in December 2009, 80% stenosis of the left carotid artery, widespread vascular calcifications, chronic bronchopathy, obstructive sleep-apnoea syndrome, small (lacunar infarcts of the thalamus and hemiprotuberance) and large (previous stroke in the left hemisphere) vessel ischaemic brain disease, and polyarthrosis with severe degeneration of the lumbar spinal column.

Three years before starting HD, and with treatment including thiazide and oral calcitriol, the patient was hospitalised for severe hypercalcaemia (13.5mg/dl), with neurological and digestive symptoms. In the subsequent analysis, we observed hilar lymph nodes of a non-pathological size and isolated reticular pulmonary parenchymal opacities, homogeneous hepatosplenomegaly, angiotensin-converting enzyme (ACE) on the upper limit of its normal range (three measurements taken between 30U/l and 60U/l, under normal conditions, normal range: 8-55). All other tests were negative, and the patient was diagnosed with exogenous intoxication by vitamin D and thiazide, and was treated with parenteral pamidronate, with an excellent clinical evolution and no levels of 1,25 $(OH)_2$ vitamin D. Since then, the patient has been asymptomatic, with suppressed intact parathyroid hormone (iPTH) levels and a spontaneous tendency towards hypercalcaemia, for which he was diagnosed with adynamic bone disease (ABD). During the month of July 2009, the patient suffered a progressive condition of asthenia, anorexia, night sweating, disorientation, irritability, amnesic altera-

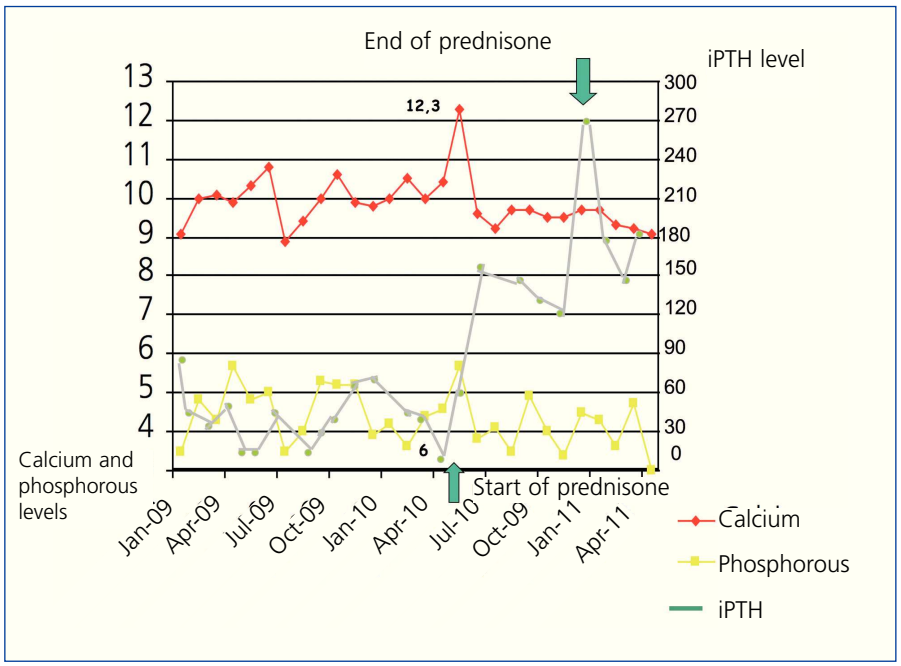


**Figure 1.** Evolution of calcium, phosphorous and intact parathyroid hormone (iPTH) levels.

tions, apathy, apraxia, and motor difficulty for walking. We also observed cortico-subcortical atrophy, leukoaraiosis, and dilation of the ventricular system, and after performing a lumbar puncture with negative biochemical and microbiological results, we ruled out normotensive hydrocephalus, infectious disease, and degeneration of the central nervous system (CNS). The patient's symptoms were explained by small-vessel encephalopathy and a conservative treatment plan, evolving slowly towards chronicity. We did not administer oral calcium, vitamin D, or its derivatives, with dialysis provided with a calcium bath at 2.5mEq/l. In May 2010 the patient had no clinical variations, with hypercalcaemia at maximum values of 12.2mg/dl and iPTH at 6pg/ml (Figure 1). We performed a complementary study with the following results: normal thyroid profile, negative Mantoux (negative Booster), chest x-ray indicative of chronic obstructive pulmonary disease (COPD), normal levels of parathyroid hormone-related peptide (PTHrP), normal alkaline phosphatase levels, levels of 1,25 $(OH)_2$ vitamin D reduced to 10pg/ml, normal plasma protein immunoelectrophoresis and proteinogram, negative immunology, bone scan and imaging demonstrated osteopaenia and vascular calcifications with no indications of osteolysis, normal tumour markers, and ACE at 157U/l. The full-body computed tomography (CT) showed several bilateral mediastinal hilar lymph nodes (Figure 2). Pulmonary fields had ground glass densities with nodular morphology in the middle lobe, periportal, perigastric lymph nodes and in the celiac trunk and left para-aortic and interaortocaval spaces, and spirometry with mixed ventilation abnormalities that were mostly restrictive in nature.

We detected bronchitis and chondritis in the bronchoscopy. We then performed a bronchoalveolar lavage and mediastinal fine-needle aspiration with negative cytology for malignancy and no granulomas or lymphoid cellularity. The culture for mycobacteria also resulted negative. The lymphocytic subpopulations in the outflow from the lavage were within normal ranges, with a CD4/CD8 index of 2.22.

Diagnosed with pulmonary sarcoidosis, the patient was treated with oral prednisone at 10mg/day for three months with a progressively descending dose until reaching 5mg/48 hours as a maintenance dose, which was sustained for 18 months. The clinical evolution and progress of laboratory parameters (ACE of 36U/l in September 2010) were satisfactory; the patient had improved cognitive state and increased iPTH levels, with normalised hypercalcaemia. The patient is currently on an HD regimen.

Multiple causes can explain hypercalcaemia in a patient on HD (the use of calcium chelating agents, dialysed with a high-calcium bath, use of vitamin D and derivatives, poorly controlled secondary hyperthyroidism, ABD, and osteomalacia). One uncommon cause is sarcoidosis, a granulomatous disease of unknown origin, in which an extra-renal synthesis of calcitriol is produced by activated macrophages.[1] The disease normally involves constitutional symptoms (weight loss, fever), arthralgia, asthenia, and pulmonary symptoms (reticular opacities and bilateral pulmonary hilar lymph node). It tends to respond to corticosteroid therapy that should be maintained for at least one year. Extra-thoracic symptoms occur in only 30% of cases. Cases mentioned in HD patients are rare in the medical literature[2-5] and tend to be diagnosed based on hypercalcaemia with systemic and pulmonary symptoms. Our patient had increased ACE levels (75%), neurological symptoms (5%), hepatosplenomegaly (20%-25%), and hypercalcaemia (10%-20%). We had

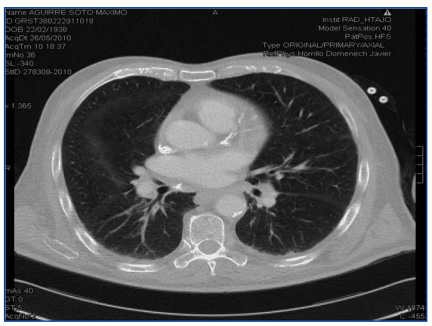

**Figure 2.** Chest computed tomography. Hilar lymph nodes.

previously observed reticular pulmonary opacities, with no increase in ACE levels or symptoms in other areas that would suggest this diagnosis. Additionally, the patient, being diabetic, had widespread calcifications and multiple risk factors that could explain the vascular encephalopathy; the chronic pneumopathy had already been considered as ABD. The patient's clinical evolution was latent and progressive, with no elevations in ACE levels or true hypercalcaemia until three years after the first crisis. The non-specific constitutional symptoms and the sum of causes that all could have explained the patient's condition hindered making the proper diagnosis. After treatment, iPTH levels were stable between 180pg/m and 270pg/m, which was surprising. We also want to bring attention to the fact that cases of hypercalcaemia right on the limit are not always due to ABD, which we believe to be adequate reason to consider other possible diagnoses when the circumstances call for it.

### Conflicts of interest

The authors have no conflicts of interest to declare.


1. Motoyama K, Inaba M, Emoto M, Morii H, Nishizawa Y. Sarcoidosis initially manifesting as symptomatic hypercalcemia with the absence of organic involvement. Intern Med 2002;41(6):449-52.
2. Huart A, Kamar N, Lanau JM, Dahmani A, Durand D, Rostaing L. Sarcoidosis-related hypercalcemia in 3 chronic hemodialysis patients. Clin Nephrol 2006;65(6):449-52.
3. Yamada S, Taniguchi M, Tsuruya K, Lida M. Recurrent sarcoidosis with psoas muscle granuloma and hypercalcaemia in a patient on chronic hemodialysis. Nephrology (Carlton) 2009;14:452-3.
4. Kuwae N, Oshiro Y, Nakazato S. Sarcoidosis and hypercalcemia in a patient undergoing hemodialysis. Nippon Jinzo Gakkai Shi 2003;45(2):98-103.
5. Tanaka K, Tanimoto H, Sakai K, Arai K, Mizuiri S, Hasegawa A, et al. Recurrence of sarcoidosis in a hemodialysis patient confirmed by anormal calcium metabolism. Clin Calcium 2005;(Suppl 1):83-5.



**J.A. Martín Navarro, M.J. Gutiérrez Sánchez, V. Petkov Stoyanov**

Nephrology Department. Del Tajo Hospital. Aranjuez. Madrid, Spain

**Correspondence:** J.A. Martín Navarro
Servicio de Nefrología. Hospital del Tajo.
Avda. Amazonas Central, s/n.
28300 Aranjuez. Madrid. Spain
juanmartinnav@hotmail.com
jantonio.martinnav@salud.madrid.org


## Fulminant oligosecretory multiple myeloma


Nefrologia 2011;31(6):754-6

doi:10.3265/Nefrologia.pre2011.Jun.10976


**To the Editor,**

Multiple myeloma (MM) is a clonal proliferation of plasma cells with the production of monoclonal immunoglobulins. This disease can be diagnosed as a result of a variety of clinical manifestations, including bone pains (lytic lesions), a major increase in plasma proteins, and/or the presence of monoclonal protein in blood/urine samples, and signs and symptoms indicative of malignancy, including anaemia, hyperviscosity syndrome, hypercalcaemia, and renal failure. Previous studies have mentioned mortality rates between 10% and 20% in the first two months of its appearance.[1]

Here we describe the case of one patient, previously healthy, with recently diagnosed oligosecretory MM, who developed fulminant symptoms one week after detection, having just received the first dose of chemotherapy.

The patient was a 69-year old female. The only relevant history was a peptic ulcer several years before, and the patient did not take any medications chronically or have a family background of kidney disease. The patient was also waiting for an operation to treat a crural hernia. Two months before seeking emergency treatment, the patient's plasma creatinine level was 0.9mg/dl, with no anaemia, and with normal urine parameters and chest x-ray results.

The patient was admitted to the emergency room with a compromised general state of health, anorexia, and nausea.

Anamnesis did not indicate a decreased water intake, but the rhythm of diuresis did, as well as the appearance of nocturia, which previously had not occurred. The patient also had a cough with bloody sputum, dyspnoea upon light exertion, and orthopnoea.

Upon questioning, the patient commented that she had received an anti-flu shot one month prior, and had later started treatment with oral calcium and paracetamol (this treatment had been abandoned in the last 15 days).

The physical examination indicated general poor state of health. The patient had a baseline $O_2$ saturation of 78% and jugular ingurgitation. Pulmonary auscultation revealed crepitation up to mid-level, and the abdomen was globular with pitting oedema in the limbs. The rest of the physical examination was normal.

The blood analyses carried out in the emergency room revealed: creatinine: 5.5mg/dl, Na: 136mEq/l, K: 5.4mEq/l, calcium: 10.9mg/dl, pH: 7.30, $HCO_3$: 19mEq/l, haemoglobin: 8.8g/dl, haematocrit: 27%, leukocytes: 13 300, and platelets: 133 000. The coagulation analysis was normal.

Urine analysis (systematic) resulted in: proteins ++, blood +++, sediments >40 red blood cells/field, ionogram: Na 41mmol/l, K 52mmol/l.

A chest x-ray taken when the patient was hospitalised indicated bilateral increased densities with butterfly patterns (Figure 1), and the abdominal ultrasound detected homogeneous hepatosplenomegaly and kidneys with normal size and morphology, with no dilation of the urinary tract.

With the available data indicating rapidly progressing renal failure, anaemia, and bloody sputum, as well as the increased densities in the chest x-ray, we