the diagnosis given the ultrasound finding, whereas renal gammagraphy is used to find out whether the parenchyma is functioning correctly. Treatment is performed using laparoscopic and endoscopic pyeloplasty in any of its variants. At present, robotic surgery has proven its utility in obtaining good pyeloplasty results for primary and secondary stenosis, both for children and adults and for different causes.[5]

In summary, when a young patient presents with AHT (related or not to HK), one should consider that it may owe to a PJS. Abdominal CT scans are a good diagnostic method for assessing this condition.

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Successful treatment with sodium thiosulfate for calcific uraemic arteriolopathy

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To the Editor,

A dermatological manifestation of chronic kidney disease (CKD) is calcific uraemic arteriolopathy (CUA) or calciphylaxis. It is an anatomopathological entity characterised by necrosis of the skin and adipose tissue due to incorrect calcium salt deposits.[1] Morbidity and mortality of calciphylaxis is high due to the complications associated with it: sepsis and ischaemia. Different clinical entities can manifest calciphylaxis: rheumatoid arthritis, inflammatory bowel disease, neoplasias, CKD, systemic lupus erythematosus or HIV infection.[1] Its treatment has to be aggressive. Sodium thiosulfate has shown improvements in skin lesions caused by calciphylaxis.

Our patient was a 78-year-old man with history of high blood pressure, diabetes mellitus type 2, dyslipidaemia, CKD of unknown origin treated with haemodialysis three times a week for 3hr, mineral and bone disorder associated with CKD (MBD-CKD), auricular fibrillation and ischaemic heart disease. His usual treatment was sevelamer, enalapril, aspirin, acenocumarol and insulin.

He was admitted to hospital for painful skin erythematous lesions with necrotic edges on both lower limbs, measuring 5x6cm. Physical examination: good general condition, body mass index: 23; blood pressure 150/63mm Hg; heart rate 64bpm; no fever. Cardiopulmonary and abdominal auscultation: painless. Lower limbs: normal pulse, with no sign of deep vein thrombosis and showing previously described lesions. Analyses: parathyroid hormone (PTH): 826.3pg/ml, calcium: 8.9mg/dl; phosphorus; 7.40; creatinine: 9.8mg/dl; albumin: 3g/dl; urea: 156mg/dl; C-reactive protein; 4.3. A cervical ultrasound and parathyroid gammagraphy were performed showing parathyroid hyperplasia free of adenomas. The radiological study (bone series and supra-aortic trunks Doppler) showed vascular calcifications on the ascending and descending aorta. Given the suspected calciphylaxis, we performed a biopsy of one of the lesions, with results compatible with calciphylaxis: lesion with abundant calcium deposits compared with the walls of small vascular structures. Swollen septa due to fibrosis (Figure 1). No signs of necrosis are observed. Lastly, we performed a technetium-99 gammagraphy which did not show that the calciphylaxis spread to the bones.

We considered MBD-CKD to be the cause of calciphylaxis and intensified the treatment for it: daily dialysis of 4hr was started with low calcium (2.5mEq/l) in the haemodialysis solution and high flux dialyser. The treatment was intensified with calcium-free phosphate-binders: lanthanum carbonate: 750mg/8hr and sevelamer: 1600mg/8hr, and PTH control with calcimimetics: 60mg/24hr. Acenocoumarol was withdrawn and treatment was started with 80ml of sodium thiosulfate at 25% (20g) following haemodialysis (three times per week). Lesions improved 2 months later (Figure 2). Analytical parameters upon discharge: P: 3.6mg/dl; total Ca: 8.9mg/dl; PTH: 406.90pg/ml.

CUA consists of a hydroxyapatite deposit in the skin and soft tissues with

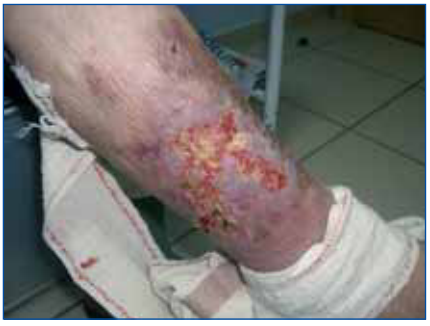

Figure 1. Lesion on right leg prior to treatment with thiosulfate.

risk of necrosis. In CKD, CUA physiopathology is due to an alteration in phosphocalcic metabolism, uraemic state, increase in PTH (although there are CUA cases following parathyroidectomy)[2], calcium-based phosphate binders and a high calcium concentration in the dialysis solution.[3] Phosphate and calcium are bound producing vascular, skin and organic calcifications.[4]

Other predisposing factors are: female sex, obesity,[5] hypoalbuminaemia (<2g/dl), diabetes mellitus, C and S protein deficiency,[6] oral anticoagulants (they inhibit synthesis of 4.8-gamma-carboxyglumate)[7], intravenous iron and vitamin D due to its intestinal action on calcium reabsorption.[8]

Lesions caused by CUA are often on the abdomen and the calf area, given their abundance of subcutaneous tissue. The lesion is similar to *livedo reticularis* progressing to ulceration. Calcium deposits in skin are deposited in the dermis and subcutaneous tissue. Physio-pathogenitically, high levels of urea and phosphorus cause smooth muscle cells to convert into osteoblast cells that also increase osteopontin levels, which together with proinflammatory and free radical molecules, make it easier for phosphorus to adhere to calcium.[1,7,9,10] This magma is mostly concentrated around calcium deposits that are also found in the arterioles[1] and media of the vessels.

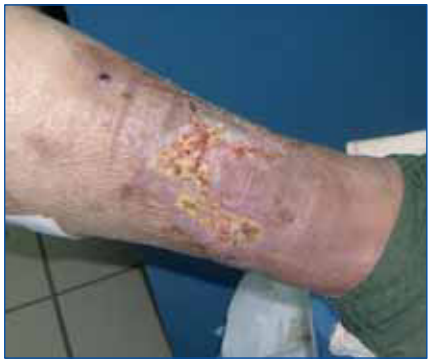

Figure 2. Lesion on right leg after two months of treatment with sodium thiosulfate.

The diagnosis is essentially clinical. Lesion biopsy is not recommended given the risk of infection and ulceration.[10] Gammagraphy with technetium-99 is used to diagnose whether it has spread to the bones.[10] Calciphylaxis has a morbidity and mortality of 80% given the risk of infection and necrosis.

The CUA therapeutic approach must be aggressive, controlling its associated alterations (BMD-CKD control), avoiding agents that could strengthen it, and curing the lesion and infectious complications.[2] BMD-CKD control will be performed using calcium-free phosphate binders, low calcium haemodialysis and peritoneal dialysis solutions (CUA can develop above 4mEq/l[11]) and implementing daily haemodialysis. PTH control will be performed using calcimimetics and vitamin D, preferably vitamin D analogues given that they have a lower calcifying and hyperphosphatemic effect.[2] Parathyroidectomy will be reserved for cases resistant to drug treatment.

Dermal CUA lesions that have no ulcerations improve with corticoids.[10] However, for those with ulceration, the hyperbaric oxygen chamber is effective against anaerobic organims.[11] The sodium thiosulfate (antidote against cynade, used in skin treatments for acne and pityriasis versicolor, and protection against carboplatin and cisplatin toxicity) has proven a successful therapeutic measure in CUA lessions.[5,14] Sodium thiosulfate inhibits calcium salt precipitation and dissolves calcium deposits.[12,13] It does not have any effects on levels of calcium, phosphorus or PTH. The recommended dose is 20g I.V., three times a week during a minimum of 6 months.[2,14] Its administration could lead to metabolic acidosis, osteoclast activation, volume overload and hypotension.

In summary, CUA is a serious entity among our patients. Its treatment has to be aggressive. Sodium thiosulfate is a valid therapeutic agent for treating CUA lesions. It is safe to use and the benefits obtained mean that it can be considered a first-line drug for CUA lesions.

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Spontaneous remission of nephrotic syndrome in a patient with diabetic nephropathy and Parkinson's disease

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To the Editor,

Parkinson's disease (PD) is a common neurodegenerative disease that can be caused by mitochondrial dysfunction, oxidative stress, apoptosis or inflammation.[1] Between 50% and 80% of PD patients show intolerance to glucose, which can be exacerbated by levodopa treatment.[2] We describe the case of a patient with PD and poorly controlled diabetes mellitus, who was initially treated with anti-diabetic drugs and later required insulin therapy and who came for consultation with a nephrotic syndrome (NS).

The patient was a 74-year-old man with a 9-year history of diabetes mellitus (initially treated with anti-diabetic drugs and for the last 3 years with insulin); diagnosed with infarctional ischaemic heart disease and post-infarction angina, he had undergone double coronary bypass surgery. Previous episodes of deep vein thrombosis and pulmonary thromboembolism, and hypercoagulability had been confirmed (heterozygotic mutation of homocysteine gene). For 10 years he had had PD, which was being treated with carbidopa/entacapone/levodopa, ropinirole and rasagiline. Other medical conditions included prostate adenoma, hiatus hernia and chronic renal failure with previous plasma creatinine levels of 1.4-1.5mg/dl.

The patient was referred to the emergency department by his GP, owing to symptoms of anasarca. In the days prior to his visit to the emergency department he had noticed a decrease in the frequency of diuresis accompanied by weight gain. He did no report blood-stained or dark-coloured urine. A week before he had developed very itchy petechiae on his arm and the back of his hands.

The physical examination revealed that the patient's general condition was good, and he was conscious and orientated. There was slight jugular vein ingurgitation and his blood pressure was 150/78mm Hg. Body temperature was normal. As far as the rest of the examination is concerned, notable symptoms included pitting oedema of the lower limbs and signs of venous insufficiency.

In the complementary tests, the blood analysis showed: haematocrit: 44%, leukocyte: 7060, platelets: 152 000; pH: 7.32, bicarbonate: 28mEq/l, glucose: 241mg/dl, creatinine 2.2mg/dl and calcium 7.7mg/dl. The rest of the on-the-spot analysis was normal.

In the routine blood analysis the findings were as follows: uric acid: 11.8mg/dl, cholesterol: 297mg/dl, triglycerides: 141mg/dl, albumin: 1.9g/dl, total protein: 5.2g/dl, LDH: 629U/l, glycosylated haemoglobin: 8.5%. Immunological analysis: C-reactive protein 1.9mg/dl; rheumatoid factor, ASLO, ANCA, antinuclear antibodies, anti-Ro, anti-La, anti-Sm and anti-RNP antibodies were within normal limits. Tumour markers, including ACE, CA19-9, AFP and PSA were acceptable. Blood electrophoresis: hypoproteinaemia, reduced albumin levels, raised alpha-2 and beta globulins with a polyclonal increase in gammaglobulins. Thyroid hormones were normal. Serological tests for the hepatitis C and HIV virus were negative. HBsAg positive, anti-HBc and anti-HBs negative; hepatitis B virus DNA less than 2000 copies/ml. Herpes virus 1-2 IgG positive.

The urine analysis on admission showed proteins +++, blood ++ and the presence of casts (cylindruria). Protein quantification in 24-hour urine was 13g/24 h.

Chest X-ray: enlarged heart with no signs of acute heart failure. Electrocardiogram: sinus bradyarrhythmia at 50bpm. A Doppler ultrasound scan showed no pathological findings.

Given the patient's history of poorly controlled diabetes mellitus and his admission owing to recent fluid retention, it was decided that a renal biopsy should be performed. Our findings were as follows: six glomeruli, two of which were completely sclerotic. In two of the other four glomeruli, focal, nodular lesions of the glomerular tuft (Kimmelstiel-Wilson nodules) were identified. The result of the immunofluorescence assay was negative. Moderate interstitial fibrosis associated with tubular atrophy and chronic inflammatory infiltrate was observed. The vascular component presented no lesions. The definitive diagnosis was nodular glomerulosclerosis with a morphological substrate of diabetic nephropathy (DN).

With this diagnosis, the initially established treatment, which consisted of diuretics, irbesartan, atenolol, statins and oral anticoagulants, was maintained and the patient was discharged.

Twelve days later the patient was re-admitted for fluid retention, and he responded favourably to diuretic treatment. Subsequent outpatient follow-up