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C) BRIEF CASE REPORTS

Hypertensive urgency and stunting. What is the diagnosis?

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Dear Editor:
Tuberous sclerosis (TS) is a congenital anomaly of embryonic development, autosomal dominant inherited disorder, which has different forms of clinical expression. It is classified among the so-called phacomatoses: developmental anomalies susceptible to generating tumours or hamartomas of the nervous system. We present the case of a patient who was diagnosed by chance after a consultation in the Emergency Unit for another reason.

The case concerns a schoolgirl of 6 years and 6/12 months who attended the Emergency Unit due to an evanescent, occasionally petequial, rash which had been developing for several hours. Associated fever. Examination: weight 15.300kg (SD 6.3kg). Height 100cm (SD 16cm). Blood pressure: MID-160/120, MII-177/107, MSI- 169/118, MSD-169/110.

On physical examination the child showed no indication of severe illness. Afebrile. Dry skin, mucous membranes hydrated and somewhat pale. Dermal lesions associated with acute virosis. Rhythmic heart tones and strong II/VI systolic murmur heard in all positions. No findings in the abdomen: soft, depressible without pain, no organomegaly, sparse adipose tissue. Femoral pulse palpated. DTRs present. No meningeal signs. Hyperemic oropharynx.

Additional explorations: CBC: normochromic, normocytic anaemia. Leukocytosis, slight netrophilia, eosinophilia. Biochemistry: glucose 121mg/dL, urea: 135mg/dL, creatinine: 1.6mg/dL. Control: glucose: 81mg/dL, urea: 113mg/dL, creatinine: 1.1mg/dL, magnesium: 2.2, CRP: 215.8mg/l (N: 2-5). Control: 11.4, ABB: normal; serum iron: 64mg/dL (N: 45-156); lipid profile: normal; ESR: 4mm (N: 0-20); PTH: 75U/mcrl; infectious mononucleosis serology negative; pharyngeal culture: *Streptococcus pyogenes*; aldosterone: 50.10NG/dl, renin: 0.50ngml/h aldosterone/renin: 100.2 (<30). Coagulation test: normal. Urine: haematuria, mild proteinuria. Renal function test: glomerular filtration: 34ml/min/1.73m^2; calciuria: 3mg/kg/day; phosphaturia: 29mg/kg/day (N = 15-20); natriuria: 3.44mEq/kg/day (3.87 ± 1.3mEq/kg/day); kaliuria: 2.24 mEq/kg/day (1.73 ± 0.7mEq/kg/day). RTP: 64%; urine culture: negative; microalbunin/creatinine index: 3. 222mg/g; folic acid, vitamin B12 and eye depth: papillary pallor. No oedema of the papilla, clear edges. No other abnormalities were found; ECG: left ventricular hypertrophy. Echocardiogram: normal; left hand and wrist x-ray: osseous age of five years; immunoglobulins, C3 and C4 ANA and AMA normal; catecholamine: normal.

Renal abdominal ultrasound (Doppler): kidneys were within normal size range but with structural alterations. Focal lesions of bilateral renal parenchyma were present.

MRI of the abdomen, cranium and pelvis: angiomyolipomas with a sparse or almost undetectable quantity of intratumoral fatty tissue. Cerebral images compatible with small hamartomata in the white matter. Both suggestive of tuberous sclerosis. Genetic tests on TSC1 and TSC2 were both negative.

Evolution: improvement in blood pressure after starting treatment with ACEI and ARA II, being normal for the patient's age and size after use and with negative proteinuria.

Angiomyolipoma (AML) is the most common renal lesion in TS (34-80%), followed by renal cysts and polycystic disease. This is due to the fact that the TSC2 locus is adjacent to one of the polycystic kidney disease genes (PKD1) and adjacent deletions can produce both phenotypes.

Angiomyolipoma is a benign tumour of the renal cortex, characterised by the presence of mature or immature fatty tissue, vascular wall and smooth muscle though with the capacity to provoke severe haemorrhage, replacement of renal parenchyma and mass effect, which can induce pain and may compromise renal function.[1] Renal failure is less frequent, and is generally associated with glomerulosclerosis secondary to hyperfiltration as a result of surgery or tumoral invasion, particularly by cysts.[2] Some patient groups have indicated that there are variants of angiomyolipoma which have the capacity for metastatic growth.[3]

Despite being benign there is a possibility of malignant transformation.[4] Furthermore, the tumour can occasionally relapse in

patients who have already been subject to intervention.

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Peritonitis caused by *Saccharomyces cerevisiae* in an ambulatory peritoneal dialysis patient

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Dear Editor:

Saccharomyces cerevisiae (S. cerevisiae) is a yeast used habitually in breadmaking and alcoholic fermentation.[1] Its isolation as a pathogen in humans is infrequent. This bears relation to its capacity to colonise the digestive tract and to its use as a probiotic in the treatment and prevention of diarrhoea associated with *Clostridium difficile*, and in other illnesses.[2]

We describe a case of peritonitis caused by *S. cerevisiae* in an ambulatory peritoneal dialysis patient. The case concerns a 59 year old male diagnosed 25 years ago with type 2 diabetes mellitus with photocoagulated retinopathy, arterial hypertension and asymptomatic nephrolithiasis in the lower pole of the left kidney which had evolved into advanced renal failure, for which the patient was included in a programme of peritoneal dialysis.

The patient attended the Emergency Unit presenting with retrosternal pain, nausea, vomiting, dysphagia with solid foods and diffuse abdominal pain. Fifteen days previously a diagnosis of peritonitis with negative cultures had been made by the nephrology department. This was treated empirically with vancomycin and ceftazidime. Cloudy liquid persisted over the following days. The patient was admitted and peritoneal liquid was sent to the biochemistry and microbiology departments, where it was cultured using the usual means. The cellular count was 350 leukocytes, 46% of which were polymorphonuclear.

After 24 hours the microbiology lab sent a preliminary report showing a result of *Candida sp*. with species pending; the nephrology department was also informed by telephone. The patient was initially treated with fluconazole and 5-fluorocytosine.

The following day the yeast was identified, using the VITEK 2 system, as *S. cerevisiae*. This identification was confirmed using the API ID 32C system (both from BioMerieux). In addition, an antimycogram was carried out using the SENSITITRE system, it being sensitive to all the tested antifungal drugs (amphotericin B, fluconazole, itraconazole, ketoconazole, 5-fluorocytosine, voriconazole and caspofungin), and this provided a definitive report. When we reported the isolation of this fungus to nephrology, they informed us that the patient was a baker.

Given this result, antifungal treatment was modified, suspending fluconazole and treating the infection with 5-fluorocytosine (500mg every 12 hours, following a loading dose of 2g on the first day) and lipsomal amphotericin B (70mg ev. on the first day, 150mg ev. on the second and 200mg ev. from the third day),. The patient showed a good level of tolerance to the treatment. After five days the liquid cell count was lower, and after fourteen days it was normal, with liquid showing as clear. Following twenty days of treatment the patient was healthy, and was discharged.

Although *S. cerevisiae* is not a common pathogen, it has been principally involved in various clinical processes such as fungaemia associated with catheters, arthritis, peritonitis, disseminated infection in advanced AIDS and in neutropaenia.[3]

We have found three published cases of peritonitis caused by this yeast in ambulatory peritoneal dialysis patients.[4-6]

Our patient could have been infected by this yeast, given that he and his wife were in daily contact with the fungus through being bakers. In the published cases, no reference is made as to what could have been the source of the infection.

Amphotericin B is the drug of choice in empirical treatment.[7] Our strain was susceptible, in vitro, to all the tested antifungal drugs. According to the bibliography consulted, *S. cerevisiae* is usually susceptible in vitro to amphotericin B and 5-fluorocytosine, whereas there are some strains which are resistant or potentially resistant to the action of derived azoles.[8] Therefore, when this yeast is isolated, it is advisable to modify