letters to the editor

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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 by the nephrology been made 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, 5fluorocytosine, 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 5fluorocytosine (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.⁴⁶

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

letters to the editor

treatment if it has been started with any derived azole, as was done in the case of our patient. The data referring to treatment of this fungus is scarce, since, as we have said, its isolation is uncommon.

Although fungal peritonitis in peritoneal dialysis patients at times requires the withdrawal of the peritoneal catheter, in our case this was not necessary, since following the treatment the patient developed well.

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Nephrotic-range proteinuria in a patient with primary antiphospholipid syndrome

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Dear Editor:

Nephrotic-range proteinuria is a rare form of presentation of primary antiphospholipid syndrome (PAPS). Renal manifestations of PAPS can be acute or chronic, accompanied by hypertension and progressive renal failure.¹ Chronic kidney disease (CKD) with a reduction in glomerular filtration is evident in the majority of studies, with an incidence range of 56-100%. Principal treatment is based on anticoagulation with an INR >3.² On rare occasions treatment with immunosuppressant drugs has been tried, with a variable response.³

Clinical case

31 year old patient with paranoid schizophrenia and depressive syndrome, mixed dyslipidaemia, smoker and drinker. Thrombosis of the femoral veins, up to the inferior vena cava, and left renal vein; plasma creatinine (PCr) of 2.7mg/dL, in treatment with acenocoumarol (INR 2-3).

The patient attended consultation with negative hypercoagulability, PCr 2.1mg/dL and proteinuria of 2g/24h. The ultrasound showed the right kidney at 13.6cm and the left at 10.5cm, with some cortical scarring. The patient reported a sedentary lifestyle with dyspnoea of moderate strength. Arterial tension normal, with moderate obesity. Isotope renogram with Tc^{99m} showed renal asymmetry with minor cortical scarring.

Laboratory tests: platelets 100-130 10^3/ul; urea: 50; creatinine: 1.7; uric acid: 10.4; triglycerides: 676; cholesterol: 303mg/dL; albumin 3.5g/dL; remainder normal. Immunological test was negative except for positive lupus anticoagulant in several findings. Proteinuria: 9g/24h, normal sediment. CrCl: 100ml/min.

Given the patient's clinical characteristics, a right renal biopsy using microlumbotomy was proposed, but refused. We started treatment with enalapril, allopurinol, atorvastatin and hygiene and dietary measures (figure 1).

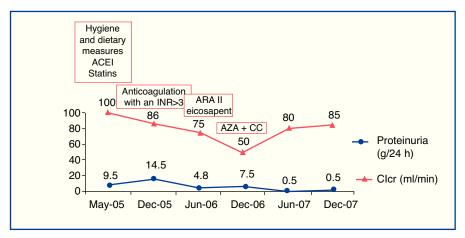


Figure 1. Evolutionary chart showing established treatments: representation of creatinine and proteinuria clearance alongside patient follow-up, with the response obtained.