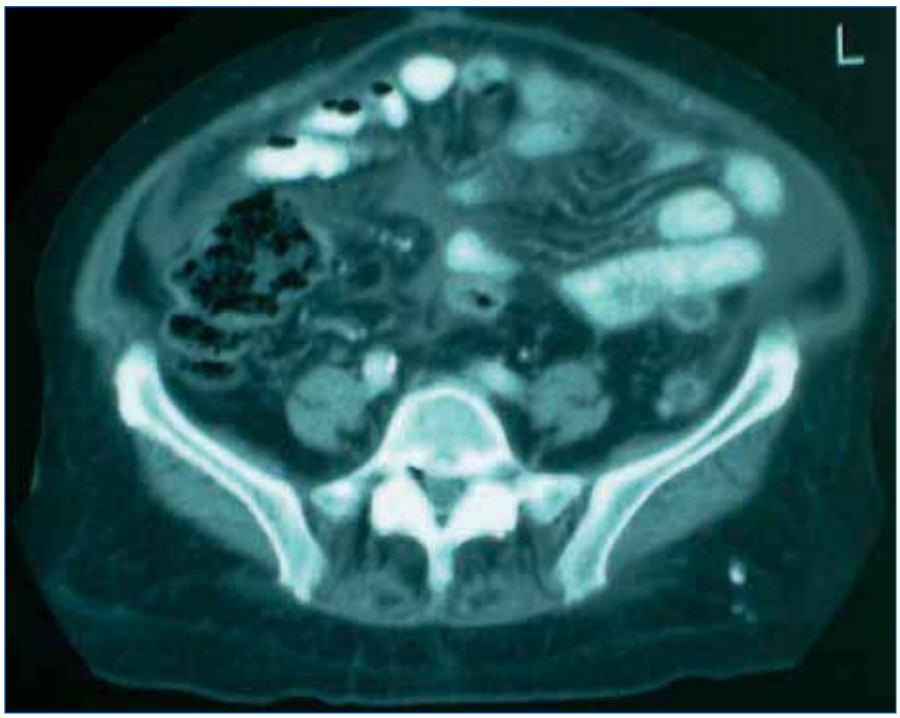

Figure 1. Abdominal CT scan with contrast. The arrow points to peritoneal thickening. The cross marks ascitic fluid. The star indicates increased vascularization in mesenterium.

attributed to the beneficial effects of tamoxifen.

The therapeutic approach proposed by the SP Japanese group would include transfer to hemodialysis, intestinal rest with parenteral nutrition, steroids, and surgery. Coadjuvant tamoxifen treatment has been used in some SP cases with encouraging results.

In this presentation form, diagnosis of fulminant SP is frequently difficult and based on suspicion. This is why immunosuppressive treatment is often delayed.[1,2] In cases where early immunosuppression had been administered, a more favorable course of the patient has been reported.[3]

By reporting this case we want to call attention to this acute inflammatory condition occurring after or during the resolution phase of a severe peritonitis. It should be suspected if an abscess is ruled out. It should be emphasized that early steroid administration clearly improved the course of the condition, and tamoxifen administration was effective as a coadjuvant.

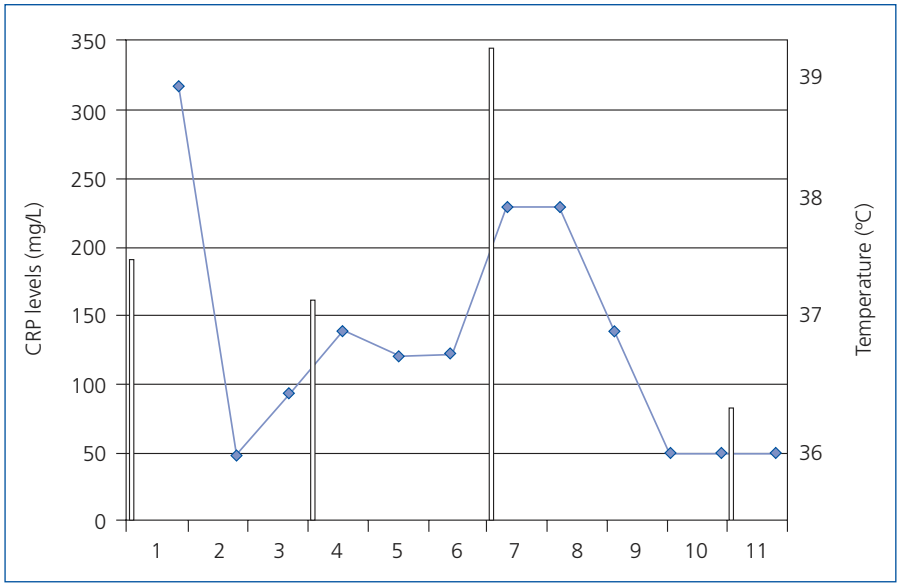

Figure 2. CRP and temperature values during the episode. Successive days are represented in the abscissa axis. Peritoneal catheter was removed on day 1. On day 6, treatment was started with corticosteroid and tamoxi fen.

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Sepsis induced by hafnia alvei in a kidney transplant patient

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To the editor: Immunosuppression associated to transplant involves a risk of infection.[1]

A 69-year-old female patient with a history of CRF secondary to adult polycystic liver and kidney disease who had been on HD since 1982 is reported. She was implanted a cadaveric kidney graft in 1986. Baseline immunosuppression consisted of cyclosporine, azathioprine, and steroids. Other rele-

vant history data included HBP, chronic virus C hepatitis, and allergy to quinolones.

In 2007, the patient attended the emergency room complaining of fever, dry cough, and some abdominal discomfort.

On physical examination, the patient was found to be in a good general condition, with a baseline SatO of 94%, BP values of 130/60 mmHg, and a temperature higher than 38 ºC. The only remarkable findings in cardiopulmonary auscultation were crackling sounds, particularly in the left base. Cysts of her underlying disease were palpated in the abdomen.

Results of blood laboratory test son admission included: Creatinine 1.5 mg/dL, WBC 5090 (82% neutrophils), platelets 95,000, prothrombin activity 55%. Results of all other blood tests and urine analysis were normal.

Chest X-rays showed blurring of left base, and patient was admitted to hospital with a diagnosis of left basal pneumonia.

After collecting samples for culture (blood, urine, sputum), empirical treatment was started with cefotaxime.

At 24 hours of admission, the patient experienced impairment of her general condition, severe arterial hypotension, oliguria, and coagulopathy, requiring hemodynamic support with dopamine and colloids, furosemide infusion, and vitamin K treatment. A CT scan of the abdomen and pelvis found no added disease to cysts.

Because of severity of the condition, cefotaxime was replaced by imipenem and clarythromycin. Blood culture results reported isolation of penicillin-resistant and imipenem-susceptible *Hafnia alvei.*

Measures taken achieved clinical stability and improvement of the patient.

Immunosuppression, necessary to prevent rejection of transplanted organs, implies a predisposition to experience severe infection.[1] Rubin et al reported a calendar of infections depending on the transplant period: hospital-acquired infections are common in the first month, opportunistic infections predominate between the first and sixth months, and any community-acquired infection may occur after 6 months.[2]

A community-acquired *H. alvei* infection occurring in a kidney transplant patient 20 years after transplant is reported here.

Members of the *Hafnia* genus are Gram-negative bacilli belonging to the family Enterobacteriaceae that occasionally cause infection in humans. Though this species has been known since 1954, for decades it was considered to belong to the genus *Enterobacter*, and was therefore called «*Enterobacter hafniae*» or «*Enterobacter alvei*». Recent DNA and biochemical studies defined these organisms as members of a separate genus with a single species, *Hafnia alvei*.[3]

The gastrointestinal tract of humans and animals, particularly mammalians, but also birds, may be a reservoir for *Hafnia.*[3] Epidemics of *H. alvei* infection related to the poultry industry are well documented in the literature.[4] In our case, the patient was questioned again and said that she was in continuous contact with poultry, but had no awareness that her animals were ill.

Most patients colonized or infected by *H. alvei* have underlying diseases such as cancer (particularly hematological tumors), surgery, trauma, pulmonary disease, cirrhosis, hepatitis, or pancreatitis.[5] In the case reported, CRF for polycystic liver and kidney disease, chronic virus C hepatitis, and immunosuppression also existed. In this patient, comorbid conditions and repeated contact with poultry may have promoted infection development.

Very little is known about this microorganism as a pathogen in animals and humans. It may cause a great variety of systemic infections, including septicemia and pneumonia.

A literature review revealed that half the patients with bacteremia were immunocompromised because of tumors, hepatic disease, or HIV infection.[3] In transplant patients, association was only found with liver[6] and stem cell transplant.[7] Our case is the first reported in a kidney transplant patient.

In conclusion, immunosuppression associated to transplant and underlying diseases predispose to uncommon infections. A first description of an *H. alvei* infection in a kidney transplant patient whose main clinical manifestation was septicemia is given.

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Segmental renal artery stenosis causing renovascular hypertension

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To the editor: Renovascular hypertension (RVH) is the most common form of secondary hypertension, with a prevalence ranging from 5%-15%.[1] RVH is caused by stenosis of the renal artery of its branches that induces renal hypoperfusion leading to activation of the renin-angiotensin system and subsequent occurrence of hypertension.[2] Atherosclerotic