Acute nephritis by Aspergillus in a patient with AIDS

E. Muñoz-Bustillo¹, D. Tejedor², A. García³ and A. Noguerado¹

Departments of ¹Internal Medicine, ²Radiology and ³Pathology, Hospital de la Princesa, C/Diego de León, 62, Madrid, Spain

Introduction

Aspergillosis is a rare complication of AIDS. It is usually seen in patients with advanced disease who have additional risk factors for infection with Aspergillus, such as neutropenia, steroid use, alcoholism, broad-spectrum antibacterial therapy, or haematological malignancy [1-3]. AIDS itself is not considered to be a full risk factor because the cellular deficit mainly concerns T lymphocytes and to a much lesser extent to neutrophils and macrophages, which are the main defence against Aspergillus [4]. Renal involvement is not common; however, virtually all the opportunistic infections described in AIDS may localize in the renal parenchyma as a manifestation of disseminated disease. Only a few cases of primary renal aspergillosis have been described [5-7].

On the other hand, acute bacterial nephritis represents a form of localized, non-liquefied renal infection that it is easily identified by computed tomography (CT) because of the different radiological density secondary to the subsequent tissue injury. Therefore, acute bacterial nephritis is defined as a clinicoradiological entity situated in a pathological range between acute pyelonephritis and renal abscess.

We report a rare case of an AIDS patients with a CT scan image of acute bacterial nephritis produced by Aspergillus.

Case report

A 38-year-old bisexual man was known to be HIV positive since 1987 when he presented a CD4 absolute count of more than 500/mm³. In September 1990 he received Zidovudine, which was changed to DDI because of haematological intolerance. This drug was withdrawn because of peripheral neuropathy. In October 1993 the patient was admitted at the hospital with Pneumocystis carinii pneumonia, showing an excellent response to a week’s therapy of intravenous trimethoprim-sulphamethoxazole at the maximum dose. The patient was discharged to finish the treatment with oral antibiotic therapy. However, 1 week later he was again admitted to the hospital because of a clinical pneumocystis carinii pneumonia relapse. Intravenous co-trimoxazole was again initiated with excellent outcome. Three days later the patient complained of low back pain unresponsive to diclofenac, intermittent fever of 38°C, night sweats, and occasional dysuria. On physical examination he appeared febrile, presenting oral Candida and tenderness to percussion over the right costovertebral angle. Laboratory data showed a peripheral white blood cell (WBC) count of 2.1 x 10⁹, with an absolute CD4 count of 69/mm³; haemoglobin was 9.5 mg/dl and C-reactive protein (CRP) was 42 mg/l. Serum creatinine was 0.7 mg/dl and the blood urea nitrogen was 12 mg/dl. Several sets of blood and urine cultures proved negative. Urinalysis revealed intermittent haematuria and pyuria. Chest and abdominal radiographs were unremarkable. Abdominal echography was performed, demonstrating a hyperechogenic image at the upper pole of the right kidney without evidence of hydronephrosis. A CT postcontrast scan demonstrated a mass-like radiolucent lesion, with enlargement of the right kidney consistent with the diagnosis of acute bacterial nephritis (Figure 1). Empirical treatment with intravenous ciprofloxacin 200 mg/12 h was initiated with only slight improvement of the patient’s clinical status. A new CT postcontrast scan showing ill-defined mass-like areas of low attenuation on the right kidney, interpreted as acute bacterial nephritis (group II).
monary involvement have been described. Among them is only three cases of isolated renal aspergillosis [5-7]. Some cases of extrapulmonary disease without pulmonary involvement have been described, as in our patient, in subjects with advanced disease, with a CD4 absolute count of less than 100/mm$^3$, and recent history of pneumocystis carinii pneumonia treated with trimethoprim-sulphamethoxazole. The clinical onset of this opportunistic infection in AIDS. However, Aspergillus seems to be an increasingly prevalent pathogen during the advanced stages of AIDS, as recently reported [7].

The relative high frequency of aspergillosis in immunocompromized hosts contrast with the low incidence of this opportunistic infection in AIDS. However, Aspergillus seems to be an increasingly prevalent pathogen during the advanced stages of AIDS, as recently reported [7].

Most of the cases of aspergillosis in AIDS described in literature concern pulmonary invasive aspergillosis [1,7,8]. Extrapulmonary infection usually appears as a result of disseminated disease from a pulmonary focus. Some cases of extrapulmonary disease without pulmonary involvement have been described. Among them are only three cases of isolated renal aspergillosis [5-7]. In all, primary renal aspergillosis was described, as in our patient, in subjects with advanced disease, with a CD4 absolute count of less than 100/mm$^3$, and recent history of pneumocystis carinii pneumonia treated with trimethoprim-sulphamethoxazole. The clinical onset was similar in these cases and ours: fever, haematuria and gross flank pain, and so the laboratory data and the absence of microbiologic documentation.

Acute bacterial nephritis is a recently described entity based on computed tomography [9]. This newly introduced entity is intermediate between acute pyelonephritis and renal abscess and reflects different degrees of severity of renal infection. It comprises three groups depending on the parenchymal alteration: group I refers to wedge-shaped lesions of decreased enhancement that could be focal or diffuse and unilateral or bilateral; group II comprises ill-defined mass-like areas of low attenuation or decreased enhancement. The lesion is always single (focal) but may be unilateral or bilateral; group III refers to multifocal or diffuse ill-defined mass-like areas of low attenuation or decreased enhancement, unilateral or bilateral. Every group presents different prognostic and therapeutic implications.

The previously described cases of renal aspergillosis appeared as frank abscesses or aspergillomas. Our patient presented a lesion with a CT postcontrast scan appearance of group II acute bacterial nephritis that progressed to group III. The ultrasonic examination of the renal biopsy denoted an intense inflammatory interstitial infiltration that deformed the normal renal architecture, as is described in the pathological correlation of group II acute bacterial nephritis. We agree with the importance of this correlation in the clinical management of renal infection, but we would like to suggest that perhaps the term is confusing because not every CT image of acute bacterial nephritis is secondary to a bacterial infection, as our experience confirms.

Recently an expert committee from the Society of Uroradiology has suggested a return to the terminology based on the simple term 'acute pyelonephritis' referring to the radiological findings encountered in renal infection, because they consider that terms like acute bacterial nephritis or lobar nephronia induce substantial confusion in practice and literature [10].

In summary, we have presented the case of an AIDS patient who developed isolated renal aspergillosis with fatal outcome. Aspergillosis seems to be an emergent complication of advanced stages of AIDS, and other atypical forms of appearance different from pulmonary aspergillosis are described, including renal aspergillosis without evidence of disseminated disease.

References


Received for publication: 24.7.95
Accepted. 26.7.95