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CASE REPORT

Cytomegalovirus and *Aspergillus* spp. coinfection in organ transplantation: a case report and review of the literature

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Abstract With the advent of potent immunosuppressive options, acute rejection episodes have decreased at the expense of increased incidence of opportunistic infections in solid organ recipients. In the absence of any preventive therapy, 30–75 % of transplant recipients develop cytomegalovirus (CMV) infection. *Candida* spp. and *Aspergillus* spp. account for more than 80 % of invasive fungal infections in solid organ recipients. This co-occurrence of two commonly seen opportunistic infections may end up in fatality. Here, we present a case of concomitant *Aspergillus* spp. and CMV infection and discuss the relevant literature. A 54-year-old male patient presented with fever, shortness of breath, and chest pain on the 9th posttransplant week after renal transplantation. CMV-DNA by polymerase chain reaction (PCR) was 1,680,000 copies/ml, thus, valganciclovir dose was increased. There were inspiratory crackles at both lung bases, and chest computed tomography (CT) revealed multiple fungal balls throughout the right lung. Galactomannan antigen was positive, and voriconazole and other antimicrobials were subsequently added to the treatment. At the end of the therapy, on control CT, pneumonic consolidation had disappeared, sputum cultures didn't show *Aspergillus* spp., and CMV-DNA reduced to 700 copies/ml. The patient showed a favorable clinical response to combined treatment; fever, dyspnea, and pleuritic chest pain disappeared. Both CMV disease and aspergillosis may present as pulmonary disease; thus, the characterization of one may not preclude the search for the other and the timely initiation of treatment is of paramount importance for good outcomes.

Keywords Aspergillosis · Concomitant infection · Cytomegalovirus · Renal transplant recipient

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Introduction

Although advances in immunosuppressive therapy have led to an increased survival rate of renal transplant recipients, there are greater risks of developing infectious complications [1]. Although cytomegalovirus (CMV) rarely develops as a primary infection, reactivation of a previous CMV infection or the development of a superinfection often occur in transplant patients. It has been shown that CMV can lead to the development of bacterial and fungal superinfections by trigger cellular immunosuppression in solid organ transplant patients with a history of CMV infection [2, 3]. However, concomitant CMV infection and *Aspergillus* spp. pneumonia have rarely been documented [4]. There are only 7 cases to date in the literature. The diagnosis of coexisting infections is difficult and, thus, may

delay the initiation of appropriate antimicrobial treatment. We report the successful treatment of this coinfection in a renal transplant recipient.

Case report

A 54-year-old male patient was on a regular hemodialysis program for 17 years due to renal failure secondary to nephrolithiasis. He had undergone parathyroidectomy for severe secondary hyperparathyroidism 1 year earlier. The patient underwent deceased donor renal transplantation in April 2010. The immediate postoperative period was unremarkable and he did not experience delayed graft function. He was administered antithymocyte globulin (ATG Fresenius, rabbit antiglobulin) 2 mg/kg for 10 days and methylprednisolone. His maintenance immunosuppressive regimen included tacrolimus (doses adjusted to maintain trough drug levels between 10 and 15 ng/ml), mycophenolate mofetil (1000 mg twice a day), and prednisone (tapered to 10 mg at the end of the second week). He was on valacyclovir 500 mg thrice a day for CMV prophylaxis. He was seropositive for CMV; however, the CMV status of the donor was not known at the time of transplantation. The serum creatinine of the patient at discharge was 0.8 mg/dl. He underwent uneventful transurethral resection for benign prostatic hyperplasia at on the 7th posttransplant week.

On the 9th posttransplant week, the patient presented with fever, shortness of breath, chest pain, and malaise in the emergency department of our hospital. He was hospitalized with an initial diagnosis of community-acquired pneumonia. Physical examination findings at admission were as follows: body temperature 38.8 °C, blood pressure 130/80 mmHg, heart rate 100 beats per min, regular. His heart sounds were rhythmic, and no murmurs were heard. There were inspiratory crackles at both lung bases. Abdomen examination was unremarkable, organomegaly was not detected. There was no pedal edema. Laboratory parameters at admission were as follows: blood glucose: 154 mg/dl, urea: 41 mg/dl, creatinine: 0.66 mg/dl, sodium: 135 mEq/l, potassium: 4.3 mEq/l, calcium: 7.3 mg/dl, phosphorus: 1.3 mg/dl, magnesium: 2 mg/dl, total cholesterol: 96 mg/dl, triglycerides: 83 mg/dl and LDL cholesterol: 55 mg/dl, AST: 21 U/l, ALT: 13 U/l, hsCRP: 37 mg/l, erythrocyte sedimentation rate: 27 mm/h, white blood cell count: 2180/mm^3, hemoglobin: 10.2 g/dl, and platelet count: $74 \times 10^3/mm^3$. Urinalysis revealed +WBC, ++++RBC, protein (−), and nitrites (−). Once blood cultures had been taken, the patient was administered imipenem on an empirical basis. High-resolution computed tomography (HRCT) of the chest revealed increased ground-glass opacities at both lung bases, scattered cavitary lesions in both lungs, the largest of which measured 19 × 15 mm in diameter, consolidation with air bronchogram on superior segment of the right lower lung, and minimal pleural effusion on the right side (Fig. 1). Galactomannan antigen was found to be positive. Voriconazole (400 mg twice daily loading and 200 mg twice daily maintenance doses) was added to his treatment. During the course of the hospitalization, he experienced unilateral leg swelling and pain. Doppler ultrasound revealed acute deep vein thrombosis and the patient was administered low-molecular-weight heparin.

CT-guided transthoracic lung biopsy showed branched hyphae on a necrobiotic background under histochemical PAS staining (Fig. 1). Acidoresistant bacillus (ARB) was found to be negative. After the procedure, the patient developed pneumothorax, and a chest tube was inserted. On subsequent HRCT, there was reduction in the number

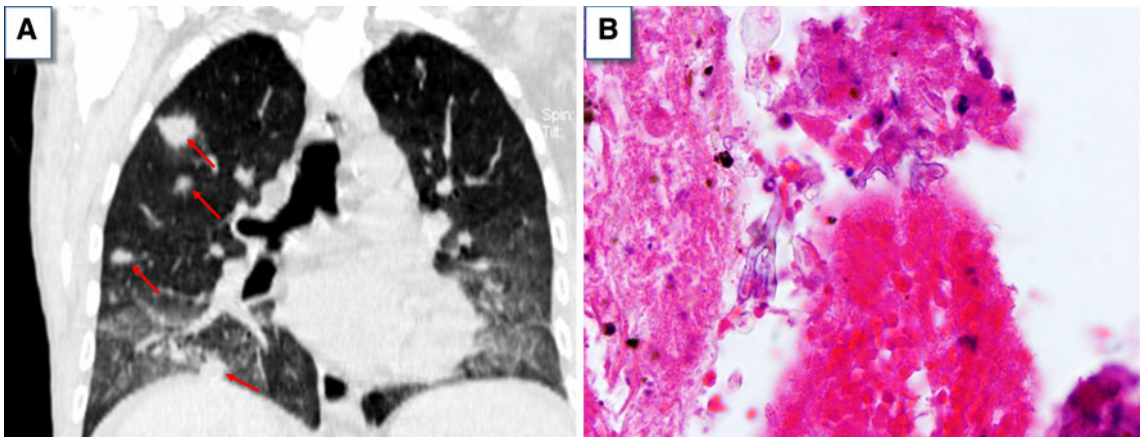

Fig. 1 **a** Coronal section of computed tomography (CT) of the chest showing multiple fungal balls (*arrows*) throughout the right lung and bibasilar ground-glass opacities due to pneumonia. **b** Lung biopsy showing branched hyphae on a necrobiotic background, histochemical PAS staining

of nodular opacities and size of the cavitations. Pneumonic consolidation disappeared with the ongoing treatment. During the course of the treatment, imipenem was changed to piperacillin–tazobactam based on the sputum culture that yielded *Enterobacter* spp. Urine culture also yielded *Enterococcus gallinarum*. Subsequently, his antibiotic treatment was changed to tigecycline. Sputum cultures did not show *Aspergillus* spp. or any other fungal pathogen.

CMV-DNA by polymerase chain reaction (PCR) at admission was 1,680,000 copies/ml, thus, the valganciclovir dose was increased to 900 mg twice daily. Owing to transient neutropenia, the valganciclovir dose was reduced. At the end of the hospitalization, CMV-DNA reduced to 700 copies/ml. The patient showed a favorable clinical response to combined treatment; fever, dyspnea, and pleuritic chest pain disappeared. He did not experience an acute rejection episode during the course of the hospitalization. The highest recorded serum creatinine value was 1.49 mg/dl, which subsequently returned to the baseline value. The patient was discharged with voriconazole 200 mg twice daily per oral and valganciclovir 450 mg twice daily per oral in addition to his maintenance immunosuppressive regimen. The patient is still free of fever, chest pain, and dyspnea, and his serum creatinine is 0.9 mg/dl as of February 2011. His latest CMV-DNA value was 0 copies/ml.

Discussion

As a prevalent pathogen among transplant patients, CMV affects up to 75 % of all solid organ transplant recipients [5]. In the absence of any preventive therapy, 30–75 % of transplant recipients develop CMV infection, and the reported incidence of CMV disease is 8–30 % [6]. CMV disease is defined as "CMV infection manifesting with signs and symptoms of fever, malaise, leukopenia, and/or documented CMV invasive disease into organs". The most frequent presentations of CMV disease are fever (58 %), pneumonitis (26.3 %), and enterocolitis (15.8 %) [7]. Most infections are associated with the reactivation of latent CMV [8]. Most complications of this infection occur in the first 6 month after engraftment [9]. The virus can influence the production of various cytokines and chemokines that can inhibit natural killer and T cell responses, as well as target humoral immune responses; in fact, it is these immunomodulatory properties that may be responsible for the indirect consequences of CMV infection [6]. The increased incidence of concurrent opportunistic viral, fungal, and bacterial infections in the setting of CMV infection has been well characterized in the literature. The immunomodulatory properties of CMV are attributed to solid organ transplant recipients' increased vulnerability to these infections [5]. To our knowledge, coinfection of *Aspergillus* spp. and CMV has been reported in 7 cases in the literature to date. The patient and disease characteristics of these cases are depicted in Table 1. Some common features of reported cases were as follows: most of the cases were middle-aged males who received a deceased donor renal transplantation. Seven out of 8 cases presented within 3 months after transplantation. Six of 8 cases were treated with amphotericin B and 7 out of 8 cases were treated with i.v. ganciclovir (Table 1). The intensity of maintenance immunosuppression was reduced in all cases and 3 out of 8 patients died, while 1 patient lost his renal graft and returned to dialysis. Interestingly, in 4 of these 7 cases, there was no mention with regards to CMV prophylaxis, while 2 patients were not given prophylaxis against CMV. In our patient, CMV infection developed despite appropriate prophylactic treatment with oral valganciclovir.

Fungal infections are aggressive and associated with high morbidity and mortality in patients undergoing organ transplantation. The occurrence of invasive fungal infections is highest in the early posttransplant period, when immunosuppression is greatest. The prevalence of invasive fungal infections has decreased over the last decade, due, in a large part, to improvements in transplant surgical methods [10]. Invasive fungal infections following kidney transplantation are rare, occurring in only 1–14 % of kidney transplants [11]. *Candida* spp. and *Aspergillus* spp. account for more than 80 % of invasive fungal infections in solid organ recipients [12]. Invasive *Aspergillus* spp. infection is a devastating complication following solid organ transplantation, with associated high mortality. It typically develops within the first 12 months and is most often seen within the first 3 months after transplantation [2]. Presenting symptoms are usually nonspecific, including low-grade fever, weight loss, fatigue, and dry cough [13]. A recent study reported an incidence of aspergillosis after solid organ transplant of 0.65 %, and the 12-month survival after infection was 59 % for patients with invasive aspergillosis [14]. Another study revealed autopsy findings and clinical associates of patients with invasive pulmonary aspergillosis [15]. Forty-one percent of the patients had no respiratory symptoms. Fungal etiology was not entertained clinically in any of the patients. The major associated conditions were prolonged antibiotic therapy, steroid therapy, renal transplantation, and underlying lung disease

Our case is the second in which voriconazole was used for invasive pulmonary aspergillosis. The patient is currently still on maintenance antifungal treatment with voriconazole and control HRCT showed significant improvements in pulmonary findings.

An interesting aspect of our patient was the development of deep vein thrombosis. Previously, it has been reported that, in a renal transplant recipient, invasive

Table 1 Patient and disease characteristics of reported cases of coinfection of *Aspergillus* spp. and cytomegalovirus (CMV)

Authors	Age (years) and gender	Underlying disease, serologic status of CMV, induction immunosuppression	Type of transplant and time of presentation	Clinical presentation	Treatment	Tests used for the diagnosis of CMV and *Aspergillus* spp. infections	Changes made in immunosuppressive treatment	Outcome
Wong et al. [18]	63, male	End-stage alcoholic cirrhosis (R? D?) Prophylaxis against CMV? Postop medications included tacrolimus, ATG, methylprednisolone, and prednisone	Orthotopic liver transplant Seven weeks after transplantation	CMV infection of the duodenum Hypertrophic plaques of *Aspergillus* spp. on both forearms	Ganciclovir 5 mg/kg/day for 14 days Amphotericin B: topical terbinafine and surgical excision	Skin biopsy specimen: subcorneal pustule with branching, saptate hyphae, and ill-defined granulomas and microabscesses in the dermis Occasional stromal cells had enlarged nuclei with intranuclear inclusion bodies An immunoperoxidase stain with CMV antibody was positive Fungal culture from the skin biopsy specimen grew *Aspergillus fumigatus*	Postop medications included tacrolimus, ATG, methylprednisolone, and prednisone	Deceased
Siu et al. [4]	63, male	Diabetic nephropathy (R+ D+) Prophylaxis against CMV not reported No antilymphocyte antibody induction	Deceased donor renal transplant Six weeks after transplantation	Hepatic dysfunction dyspnea, dry cough, hypoxemia CMV pneumonitis and hepatitis Invasive pulmonary aspergillosis	Ganciclovir 5 mg/kg 12 hourly	CMV pp65 antigen >700 Ag+ cells/2 $\times$ 10^5 peripheral blood leucocytes (PBL) Repeated sputum culture: negative Transbronchial biopsy subsequently showed fungal hyphae with acute-angle branching consistent with *Aspergillus* spp. Transbronchial biopsy immunohistochemistry study for CMV was positive Culture of the BAL fluid grew *Aspergillus fumigatus*	MMF was stopped Tacrolimus and prednisolone were continued	Deceased

Table 1 continued

Authors	Age (years) and gender	Underlying disease, serologic status of CMV, induction immunosuppression	Type of transplant and time of presentation	Clinical presentation	Treatment	Tests used for the diagnosis of CMV and *Aspergillus* spp. infections	Changes made in immunosuppressive treatment	Outcome
Tigen et al. [19]	47, male	Dilated cardiomyopathy CMV seronegative before transplantation (R− D?) Prophylaxis against CMV for 1 month ganciclovir and oral valganciclovir for 2 months Induction therapy not reported	Orthotopic heart transplant Third posttransplant month	Pneumonia (dyspnea, cough, purulent sputum, fever), bicytopenia, elevated liver enzymes, cerebral symptoms CMV infection and invasive pulmonary aspergillosis	Ganciclovir 2 × 5 mg/kg Voriconazole (2 × 6 mg/kg loading dose, 2 × 4 mg/kg maintenance dose)	PCR for serum CMV: 31245 copies/ml CMV pp65 antigen >8 Ag+ cells/2 × 10^5 PBL Chest CT: bilateral nodular lesions, cavitation in the superior lobe of the right lung Galactomannan antigen index: 2.3 Sputum culture: *Klebsiella pneumonia*	Cyclosporine and MMF were continued	Survived
Sung et al. [20]	46, male	? CMV seronegative before transplantation (R− D?) Prophylaxis against CMV not reported Induction therapy not reported	Deceased donor renal transplant Ninth posttransplant year	Increased blood glucose, urea, and creatinine initially, then dyspnea, fever, hypoxemia Desquamative interstitial pneumonia	Ganciclovir 1.5 mg/kg/day Liposomal amphotericin B 1 mg/kg/day Oral itraconazole 5 mg/kg/day and corticosteroids	HRCT: bilateral ground-glass attenuation and several cavities in both lower lobes with bilateral pleural effusions CMV pp65 antigen 65 Ag+ cells/2 × 10^6 PBL Bronchoscopic specimen… Sputum culture: *Aspergillus* spp. Thoracoscopic lung biopsy: acute-angle multiseptate hyphae in terminal bronchioles and many multinucleated giant cells containing degenerated fungal material in the interstitium	All immunosuppressants were discontinued, except for low doses of steroids	Survived He became dependent on hemodialysis 9 months after discontinuing the immunosuppressants

Table 1 continued

Authors	Age (years) and gender	Underlying disease, serologic status of CMV, induction immunosuppression	Type of transplant and time of presentation	Clinical presentation	Treatment	Tests used for the diagnosis of CMV and *Aspergillus* spp. infections	Changes made in immunosuppressive treatment	Outcome
Matevossian et al. [21]	66, male	Diabetes nephropathy (R− D−) Prophylaxis against CMV not reported Induction therapy not reported (short-term high-dose cortisone)	Deceased donor renal transplant Six weeks after transplantation	Anuria due to urinary tract infection, bilateral pleural effusion, and distal hypostatic atelectasis Invasive necrotizing aspergillosis, CMV pneumonia, acute renal failure, ARDS, multiple organ failure, persistent septic state	Ganciclovir 2.5 mg/kg/day	ELISA for *Aspergillus* spp.: negative CMV-PCR: 5,000,000 copies per mm Autopsy: lung immunohistochemistry staining for CMV shows focal positive cells Autopsy: invasive necrotizing aspergillosis lung infection	Immunosuppressive therapy was discontinued	Deceased
Our case	54, male	Nephrolithiasis (R+ D?) Valacyclovir 500 mg thrice a day for CMV prophylaxis Induction therapy with ATG	Deceased donor renal transplant Nine weeks after transplantation	Dyspnea, fever, chest pain, generalized weakness	Valganciclovir 2 × 900 mg/day Voriconazole 2 × 400 mg kg loading dose and 2 × 200 mg maintenance dose	PCR for serum CMV : 1,680,000 copies/ml Galactomannan enzyme immunoassay: positive HRCT: multiple fungal balls (arrows) throughout right lung and bibasilar ground-glass opacities due to pneumonia Lung biopsy: branching hyphae on a necrobiotic background with PAS stain Sputum culture: *Enterobacter* spp.	MMF dose was reduced, tacrolimus and low-dose steroids were continued	Survived

Table 1 continued

Authors	Age (years) and gender	Underlying disease, serologic status of CMV, induction immunosuppression	Type of transplant and time of presentation	Clinical presentation	Treatment	Tests used for the diagnosis of CMV and *Aspergillus* spp. infections	Changes made in immunosuppressive treatment	Outcome
Kim et al. [22]	58, female	Etiology? (R+ D?) Prophylaxis for CMV was not administered Induction therapy with basiliximab	Deceased donor renal transplant Two months after transplantation	Hematochezia, fever, consolidation on the right upper lobe	I.v. ganciclovir 5 mg/kg/day q 12 h Conventional amphotericin B 1 mg/kg	Chest CT: lobular nodular opacity with minimal pleural effusion Sputum culture and tissue culture: negative Galactomannan enzyme immunoassay: negative Lung biopsy: organizing pneumonia Lobectomy of the lung Pathologic findings: CMV inclusion bodies and fungus showing acute-angle branching and septate hyphae *Aspergillus fumigatus* grew on subsequent tissue culture PCR for serum CMV: 2,265,000 copies/ml	Tacrolimus replaced cyclosporine, other immunosuppressive agents were terminated	Survived

Table 1 continued

Authors	Age (years) and gender	Underlying disease, serologic status of CMV, induction immunosuppression	Type of transplant and time of presentation	Clinical presentation	Treatment	Tests used for the diagnosis of CMV and *Aspergillus* spp. infections	Changes made in immunosuppressive treatment	Outcome
Kim et al. [22]	57, male	Diabetes nephropathy (R+ D?) Prophylaxis for CMV was not administered Induction therapy not reported	Deceased donor renal transplant Four weeks after transplantation	Gross hematuria, renal failure, thrombocytopenia After 12 days, fever, nonproductive cough	I.v. ganciclovir 5 mg/kg/day q 12 h Conventional 1 mg/kg and liposomal amphotericin B 3 mg/kg	PCR for serum CMV: 1890 copies/ml Chest CT: nodular opacity Sputum culture and tissue culture: negative Galactomannan enzyme immunoassay: negative Lung biopsy: organizing pneumonia Lobectomy of the lung Pathologic findings: immunohistochemistry for CMV showing positive, round cells and fungus showing multiseptate, branching hyphae *Aspergillus fumigatus* grew on subsequent tissue culture	MMF was discontinued, prednisolone and cyclosporine were continued	Survived

CMV cytomegalovirus, *R* status of recipient seropositivity for CMV, *D* status of donor seropositivity for CMV, *HRCT* high-resolution computed tomography

Aspergillus spp. infection was complicated by an aortic thrombus [16]. Autopsy studies in patients with invasive pulmonary aspergillosis revealed angioinvasion [15]. The mechanism of arterial obstruction during invasive aspergillosis remains hypothetical. It was suggested that aggregation of the fungus cells in the intima stimulates endothelial cells to become prothrombotic by expressing thromboplastin that activates factor II and initiates the extrinsic coagulation cascade [17]. We think that the reason behind why aspergillosis facilitated the development of thrombosis was the low risk profile of our patient for deep vein thrombosis development. He had none of the risk factors apart from age and recent hospitalization for medical illness. He was fully ambulatory during the hospitalization.

In conclusion, the early posttransplant period is critical for the development of opportunistic viral and invasive fungal infections. Both CMV disease and aspergillosis may present as pulmonary disease; thus, the characterization of one may not preclude the search for the other. Eight cases, together with ours, have been reported in the literature to date and they prompt that recognition and timely initiation of treatment are of paramount importance for good outcomes.

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