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CASE REPORT

Disseminated Ochroconis in a Lung Transplant Recipient

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Introduction

Dematiaceous molds are increasingly being recognized as opportunistic pathogens in immunocompromised hosts. *Ochroconis gallopava* is a member of genus *Dactylaria* is one such pathogen.

Ochroconis is a darkly pigmented "black mold," which is thermotolerant and found in soil as well as in decaying vegetables, hot springs, cave rocks and Paleolithic paintings. It is known to cause encephalitis in domestic poultry, but there are increasing reports of it being a human pathogen. As it is primarily acquired through the respiratory tract, the lungs are the most common site of infection; it can produce cavitary lung lesions and abscesses. Amongst immunocompromised patients, the usual host includes organ transplant recipients and it typically occurs as a late post-transplant infection; other risk factors include human immunodecifiency virus (HIV), as well as hematological malignancy. The first human case was reported in 1986 in an acute myelogenous leukemic patient with subcutaneous abscesses. There are thirty-four case reports available in the literature. We report a case of disseminated *Ochroconis gallopava* infection in a lung transplant recipient.

Case Presentation

A 65-year-old Hispanic female with a history of Type II Diabetes Mellitus, idiopathic pulmonary fibrosis status postright lung transplantation sixteen months previously whopresentedwith complaints of a non-tender "ball" over the right upper back that developed over a week.

Our patient's post-transplant course was complicated by A1 rejection and hypoxic respiratory failure requiring tracheostomy. She had no renal dysfunction. Her pre-transplant bronchoalveolar lavage (BAL) fluid had grown cladosporium and she had been receiving treatment with voriconazole since that time. Of note her voriconazole level was 0.9 mcg/mL prior to admission and was not at a therapeutic level for treatment. Her immunosuppressive regimen on admission included leflunomide 10 mg daily and prednisone 5 mg bid both at stable doses for ten months. Leflunomide had been chosen as the patient was unable to tolerate cellcept due to neutropenia. She was also on tacrolimus 1.5 mg/1 mg which had been reduced the month prior from 2 mg/2 mg. Her goal tacrolimus level was 8-10 ng/mL. Of note there were no interactions with other medications while she was on tacrolimus and her renal function remained within normal limits.

On admission, she had a respiratory rate of 22, was saturating 94% on 2 liters of oxygen (which was stable at home); all other vitals were within normal limits. Exam revealed decreased breath sounds over right posterior lung field, and crackles throughout left posterior lung field. Also noted was a 1-2 centimeter soft, non-tender mass over the right scapula. Admission labs demonstrated stable leukopenia to 2500 cells/mcL, hemoglobin of 9.4 g/dL (her baseline), and platelets of 174000 cells/mcL. Tacrolimus level was 5.9 ng/mL on admission to the hospital.

Diagnosis

Initial chest radiography showed stable interstitial disease of the native left lung. Contrast computed tomogra-

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phy of the chest revealed a right upper lobe nodule and a new right posterior lateral chest wall fluid-attenuation mass 2.3 × 6.7 × 2.4 centimeters, possibly communicating with the right pleural fluid space.

BAL fluid from the right lower lobe obtained via bronchoscopy revealed pseudohyphal and yeast forms on methenamine silver stain; bacterial culture grew *E. Coli*, stenotrophomonas and mold. Blood cultures grew mold. Biopsy of a right upper lobe nodule revealed necrotizing granulomatous pneumonitis secondary to fungal yeasts and mycelial forms. BAL and pleural fluid, nodule tissue and blood cultures all grew *O. gallopava.* Standard fungal cultures were the method of identification of the fungal organism.

Our patient underwent ophthalmologic evaluation, revealing left-sided endophthalmitis and right-sided subretinal lesions. Subsequent magnetic resonance imaging of the brain and orbit without gadolinium only showed prior cerebellar infarct. She did undergo lumbar puncturewas negative.

Treatment

Intravenous voriconazole 200 mg q12 h was started on admission due to concern for invasive fungal infection in this transplant patient, and Ambisome 3.5 mg/kg q24 h was added when the result of mold returned in BAL and blood cultures. Sensitivity testing was performed on the *O. gallopava* and the minimum inhibitory concentrations (MICs), in micrograms per milliliter were as follows: posaconazole (0.125), voriconazole (1), micafungin (0.015), Ambisome (1). Literature reports the best *in vitro* activity of posaconazole, but after discussion with ophthalmology, voriconazole was chosen as the primary azole. She received bilateral intravitreal amphotericin and voriconazole [1]. Once the retinal lesions stabilized, Ambisome was discontinued and she received micafungin and voriconazole for the rest of her inpatient stay which was 4 weeks of therapy and she tolerated those medications well. She was discharged on one month of micafungin and a year's course of voriconazole. Since that time she has been followed for an additional 2 years and has no evidence of active fungal infection and has remained clinically stable with no new radiographic abnormalities.

Discussion

With regards to invasive mold infections in transplant recipients, Aspergillus causes the large majority. In a single center retrospective analysis from 1990-2012 at Stanford Hospital, 14% of 637 patients with heart or heart-lung transplants were diagnosed with invasive mold infections; of these, 72% were *Aspergillus* species and 28% were other molds (*Scedosporium, Fusarium, Rhizopus).* Disease due to non-*Aspergillus* molds tended to occur more than ninety days post-transplant, with disseminated disease and increased one-year mortality. In the largest prospective surveillance study of invasive mold infections in lung transplant recipients, 143 of 1172 patients developed invasive mold infections [2]. 72.7% were due to *Aspergillus,* 5.6% due to *Zygomycetes/Scedosporium,* and 16.8% were classified as 'other mold'. Nine of those classified as "other" were dematiaceous molds or phaeohyphomycosis; of these, one was an *Ochroconis spp*.

A review of thirty-four reported *Ochroconis* cases revealed that twenty five were in organ transplant recipients, predominantly being lung or liver transplants, followed by heart and kidney. Of the remaining patients, one had advanced HIV, five had hematologic malignancy, and two were immunocompetent. Ochroconis is known to be neurotropic and thirteen of the reported cases had central nervous system involvement [3]. The only case with ocular involvement reported was one of *O*. *gallopava* endophthalmitis in a 69 year old male with underlying chronic lymphocytic leukemia; he received intravitreal amphotericin and oral itraconazole therapy but died two months after diagnosis [4]. In a separate review of nine cases of *O. gallopava* infection in organ transplant recipients, the median time to infection post-transplant was seventeen months, and three of those patients had been on voriconazole prophylaxis [5].

Treatment is not well defined, with regards to the best antifungal as well as duration. Availability of *in vitro* susceptibility profiles is scant and interpretive break points for antifungal susceptibilities have not been established. However, *in vitro* studies indicate the best activity in echinocandins and posaconazole. In our patient, a reference lab did susceptibility testing, demonstrating low MICs to micafungin, posaconazole and voriconazole. In addition to micafungin, voriconazole was chosen as the foundation for our patient's regimen in light of her endophthalmitis, as voriconazole is known to achieve therapeutic intraocular levels and has been clinically successful when administered systemically for *Candida* endophthalmitis [6].

Conclusions

Invasive mold infections are a major source of mortality amongst organ transplant recipients. *O. gallopava* is an emerging cause of infection in transplant recipients, but should be considered as a potential pathogen with other dematiaceous molds in any immunosuppressed population. Given its neurotropism, brain imaging and lumbar puncture may be warranted. Given the rarity of reported cases of *Ochroconis* infection, there is minimal data on choice of antifungal and interpretive guidelines for susceptibilities. Echinocandins and posaconzole seem to have the best *in vitro* activity per literature, but treatment should be based onantifungal susceptibilities because clinical data is limited. Our case is the first case with reported ophthalmologic involvement of *Ochroconis* in a lung transplant patient and shows resolution on antifungal therapy.

As with many transplant related infections, treatment of *Ochroconis* is an art that will require accounting for the patient's clinical context, and referencing the limited number of case reports of successfully treated infections.

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