

60 mg, and 60 mg. Sixty minutes after the last dose, she developed erythema and pruritus in the palms, although these symptoms were milder, with complete resolution in 10 minutes with methylprednisolone 40 mg, and dexchlorpheniramine 5 mg. On the second day she was given 120 mg and 120 mg at an interval of 30 minutes, which she tolerated well, followed by 240 mg/d for the rest of the cycle.

We present a case of an anaphylactic reaction to temozolomide presumably due to an IgE-mediated reaction or pseudoallergy. To the best of our knowledge, ours is the first case of successful desensitization with temozolomide to be presented. The second protocol described seems safer. The patient was able to complete her chemotherapy treatment successfully.

RUIZ-GARCIA, MÓNICA, MD*

MANUEL DE LAS HERAS GOZALO, MD*

ELENA HERNANDEZ GARCIA DE LA BARRERA, MD*

JOAQUIN SASTRE DOMINGUEZ, MD, PhD*

*Fundación Jimenez Diaz

Madrid, Spain

monicaruizgarcia@hotmail.com, mruizga@fjd.es

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A NOVEL METHOD OF DESENSITIZATION FOR FLUCONAZOLE HYPERSENSITIVITY IN A PATIENT WITH AIDS

Fungal infections are common in human immunodeficiency virus (HIV)-infected patients and range from skin infections to oral or esophageal candidiasis to cryptococcal meningitis. Fluconazole is one of the mainstay therapies for such infections. Adverse effects of fluconazole include nausea, headache, rash, hepatitis, and anaphylaxis.¹⁻⁴ Fluconazole hypersensitivity has been described, and if acceptable alternative therapy is not available, desensitization may be required. Published desensitization protocols have ranged from 3 to 4 hours in non-HIV-infected patients and from 7 to 15 days in HIV-infected patients.^{1,5,6} We describe a case of successful desensitization to fluconazole in 2 days in an HIV-infected patient with cryptococcal meningitis.

The patient is a 29-year-old, Hispanic woman with HIV/AIDS diagnosed 14 years ago (recent CD4 cell count of 14/mm³, viral load of 130,000, and highly active antiretroviral therapy with intermittent compliance), chronic active hepatitis B virus, and *Mycobacterium avium*-intracellulare. She presented with a headache for 1.5 weeks and was found to have a positive serum cryptococcal antigen (1:1,024). Laboratory studies revealed a normal basic metabolic profile, an aspartate aminotransferase level of 54 IU/L (reference range, 7–40 IU/L), an alanine aminotransferase level of 56 IU/L (reference range, 7–40 IU/L), and an alkaline phosphatase level of 252 IU/L (reference range, 70–230 IU/L). Blood cell count revealed leukopenia (leukocyte count of 3,000/mm³ with 6.6% eosinophils, absolute eosinophil count of 200/mm³) and anemia (hemoglobin level of 8.4

g/dL). Outpatient medications included tenofovir/emtricitabine, ritonavir, darunavir, azithromycin, ethambutol, and atovaquone. The patient reported previous allergies from fluconazole (rash), trimethoprim-sulfamethoxazole (itch), dapsone (rash), and shellfish (rash).

This patient was initially prescribed flucytosine and amphotericin and received subsequent lumbar punctures to decrease intracranial pressure. On day 9 of therapy, the patient experienced a neck rash and a low-grade fever (temperature of 100.3°C); flucytosine and amphotericin therapy was discontinued and her rash improved. Five days later, the patient was prescribed oral fluconazole (400 mg/d), and within 10 minutes of the first dose, the patient became febrile (temperature of 100.3°C) and developed a pruritic, maculopapular rash on her face, neck, upper chest, and back with no mucosal involvement. The patient had a similar rash 1 year before admission when treated with fluconazole for oral candidiasis. At that time, the rash had appeared within 3 minutes of the first dose and then improved within the course of 1 day; fluconazole therapy was not restarted after the first dose and subsequent development of symptoms. Our differential diagnosis in light of the symptoms after fluconazole administration included the following: immediate-type hypersensitivity, cytotoxic (ie, type II) hypersensitivity, immune complex-mediated (ie, type III) hypersensitivity, hypersensitivity caused by the other medications used by this patient, and drug fever. Because the patient similarly reacted within minutes of the most recent dose of fluconazole, we presumed immediate-type hypersensitivity with this recent dose. Of note, a previously reported HIV-infected patient was successfully desensitized with fluconazole after also experiencing fever and rash following medication administration.⁶ Skin prick testing was not performed in our patient because of the recent antihistamines received by the patient after the rash induced by fluconazole and because of the lack of available standardized fluconazole skin testing methods. Given the necessity for maintenance therapy for cryptococcal meningitis with fluconazole, the patient was advised of the risks of desensitization and agreed to the procedure.

The patient underwent oral fluconazole desensitization starting with 200 µg of fluconazole. A 2-mg/mL fluconazole solution was made by dissolving 200 mg of fluconazole in 100 mL of sterile water, and the starting dose was 2 drops of the solution (200 µg). The starting dose was gradually increased every 6 hours (Table 1) until a dose of 200 mg was reached. The interval of 6 hours is shorter than in available protocols regarding HIV patients. This case represents the most rapid oral fluconazole desensitization regimen that

Table 1. Oral Fluconazole Desensitization Protocol^a

Hour	Concentration, mg/mL	Volume administered	Dose administered
0	2	100 µL	200 µg
6	2	1 mL	2 mg
12	2	2 mL	4 mg
18	2	4 mL	8 mg
24	2	8 mL	16 mg
30	2	15 mL	30 mg
36	2	30 mL	60 mg
42	2	60 mL	120 mg
48	NA	NA	200 mg (tablet)
60	NA	NA	200 mg (tablet)

Abbreviation: NA, not applicable.

^a Premedication with diphenhydramine, 25 mg, and famotidine, 20 mg, 30 minutes before the first dose. Continue the diphenhydramine, 25 mg, 3 times daily and famotidine, 20 mg, twice daily during the entire desensitization.

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has been successfully performed in a patient with HIV/AIDS, thereby representing a novel time course for desensitization. Our starting dose was higher than the 0.02-mg starting dose administered to a non-HIV patient during fluconazole desensitization; however, our initial dose was equal to or less than other protocols involving HIV-infected patients.^{1,5,6} Given this patient's HIV status, we used a cautious desensitization approach (eg, dose escalation every 6 hours) because of the evidence that HIV-infected individuals may experience possibly more severe drug-induced reactions, which may result from enhanced T_H2-type responses in the setting of HIV.^{7,8}

Within 2 days, the patient was able to tolerate the full, therapeutic dose of 200 mg twice per day of oral fluconazole. This protocol was completed without any adverse reactions. Premedication with diphenhydramine, 25 mg, and famotidine, 20 mg, was administered 30 minutes before starting desensitization. Diphenhydramine, 25 mg, was then given every 8 hours during the protocol, and famotidine was given every 12 hours. The patient was not taking systemic steroids before or during the desensitization. This premedication regimen was chosen because the patient was determined to have a high likelihood of drug-induced allergic reactions.

This case demonstrates that a more rapid oral fluconazole desensitization may be possible in HIV-infected patients with a history of hypersensitivity to this medication. Further studies with more patients are needed to better characterize rapid desensitization protocols in patients with HIV/AIDS with fluconazole hypersensitivity.

SUNIT JARIWALA, MD*
NATALIA VERNON, MD*
GABRIELE DE VOS, MD*

*Allergy/Immunology Division
Albert Einstein College of Medicine
Bronx, New York
nvernon@montefiore.org

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IDENTIFICATION OF MOLD ON SEASONAL INDOOR CONIFEROUS TREES

Epidemic peaks of respiratory illnesses in all age groups are observed around December 25, specifically 1 week before and 1 week after for school-aged children and adults, respectively,¹ often raising

suspicion that a live, indoor, coniferous Christmas tree may be playing a role. The sole 1970 investigation² of Christmas tree allergy concluded that aerosolized ragweed, grass, and tree pollen on the tree bark or oleoresin were likely causes. In 2007, levels of airborne mold spores in an apartment increased from 800 spores per cubic meter before the introduction of a live conifer to 5,000 spores per cubic meter after its presence of 14 days, but no spore identification was made.³ Because allergy testing can identify a patient's mold sensitivity, it would be useful to know what molds may be found on conifers used as Christmas trees.

During December 2007, faculty and staff of the Department of Pediatrics at Upstate Medical University, Syracuse, New York, were solicited to provide clippings of their home Christmas tree that had been placed in unused commercial zip-lock bags; 28 samples were submitted to the Clinical Microbiology Laboratory at University Hospital. A 1-in piece of each sample, including needles and stem, was placed into a sterile, plastic screw-cap tube containing 1.0 mL of sterile water. The sample was vortexed for 30 seconds, and 0.1 mL of the water was removed and plated onto each of the following media: Sabouraud dextrose agar (Emmon's modification), inhibitory mold agar, and brain heart infusion agar with sheep blood, gentamicin, chloramphenicol, and cycloheximide (Remel, Lenexa, Kansas). Cultures were incubated at 30°C in ambient air for 4 weeks. Phenotypic identification of molds was made by observance of macroscopic and microscopic characteristics using tape preparations stained with lactophenol cotton blue. Molds that could not be identified phenotypically were subcultured on Sabouraud dextrose agar (Emmon's modification) and incubated at 30°C in ambient air until visible growth was seen.

Nucleic acid was extracted using PrepMan Ultra Sample Preparation Reagent (Applied Biosystems, Foster City, California) according to the manufacturer's instructions. When visible growth was observed, 1 to 2 mm was removed with a sterile applicator stick and placed in 100 μ L of PrepMan reagent, 50 μ L of the supernatant was stored at –20°C until polymerase chain reaction (PCR) was performed, and 2.5 μ L of supernatant was used as template for each amplification reaction.

Real-time PCR was performed targeting the internal transcribed spacer (ITS) region, which is present in all fungi.^{4,7} Forward and reverse primers ITS1 (5'-TCCGTAGGTGAACCTGCGG-3') and ITS4 (5'-TCCTCCGCTTATTGATATGC-3') were used, which encompass ITS1, 5.8S ribosomal DNA, and ITS2. Each 25- μ L reaction contained 2.5 μ L of prepared template, 3 μ L of magnesium chloride (4 mM), 2.5 μ L of Light Cycler Fast Start DNA Master SYBR Green I (Roche Diagnostics, Indianapolis, Indiana), and 1.25 μ L of each primer (0.5 μ M). Thermal cycling (Smart Cycler II, Cepheid, Sunnyvale, California) parameters were 95°C for 10 minutes, followed by 50 cycles of 95°C for 5 seconds, 60°C for 20 seconds, and 76°C for 30 seconds with a final extension at 72°C for 2 minutes. The PCR products were prepared using the DNA Clean and Concentrator-5 kit (Zymo Research, Orange, California). Cycle sequencing was performed using BIG DYE Version 3.1 (Applied Biosystems), and the products were separated on an ABI 3100 Genetic Analyzer (Applied Biosystems).

The BLAST function of the National Center for Biotechnology Information database was used to generate sequence alignments. Only entries showing 100% query coverage were considered. Species identification was acceptable when matches were 99.0% or greater with 0.9% or greater difference among species. Genus identification was limited if matches were 97% to 98.9% or if less than 0.9% differences were observed among species with a 99.0% or greater match.

Fifty-three individual molds were cultured from 26 specimens; 23 were identified by phenotypic methods, and 24 required ITS se-

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