Case of Moxifloxacin-Induced Black Hairy Tongue

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Patient: Female, 69-year-old
Final Diagnosis: Black hairy tongue
Symptoms: Brown discoloration of the tongue
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases • General and Internal Medicine

Objective: Rare coexistence of disease or pathology
Background: Black hairy tongue (BHT) is a relatively uncommon acquired benign condition, with a prevalence ranging from 0.6% to 11.3%. It presents as a superficial black hairy carpet-like lingual growth. The exact etiology of BHT remains unclear, and both extrinsic and intrinsic factors are potentially contributive. Several types of antibiotics are also associated with BHT, but no English reports of moxifloxacin-induced BHT exist. Here, we report the first case of moxifloxacin-induced BHT.

Case Report: A 69-year-old woman presented with a brown and hairy tongue. She was taking prednisolone for mixed connective tissue disease and developed right finger flexor tenosynovitis, which was complicated by osteomyelitis due to Mycobacterium chelonae. Based on the susceptibility results, she was treated with tobramycin, imipenem, and clarithromycin for 6 weeks, and then switched to moxifloxacin and clarithromycin. Within 10 days, she developed brown discoloration on the dorsum of the tongue, with carpet-like elongated filiform lingual papillae. The diagnosis of BHT was made. After stopping moxifloxacin, improvement was seen within 2 days, and her right finger has shown no signs of recurrence for 12 months.

Conclusions: Clinicians should be vigilant against agents and lifestyles that can precipitate BHT, especially moxifloxacin. It is essential to counsel patients before such treatments to avoid patient anxiety or treatment changes.

Keywords: Antibiotics M-4365 • Moxifloxacin

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Background

Black hairy tongue (BHT) is a relatively uncommon benign condition with a reported prevalence ranging from 0.6% to 11.3%, depending on age, sex, ethnicity, and study population [1]. Both intrinsic and extrinsic factors are known to induce BHT, and, while several types of antibiotics are also associated with BHT, no English reports of moxifloxacin-induced BHT have been published. To the best of our knowledge, this is the first report of moxifloxacin-induced BHT.

Case Report

A 69-year-old woman with a past medical history of mixed connective tissue disease (MCTD), hypertension, hyperlipidemia, diabetes mellitus, and osteoporosis presented to our clinic with brown discoloration of the tongue. She was taking oral prednisolone 5 mg once daily for the MCTD. About 3 months prior to her presentation, she underwent debridement and reconstruction surgery for magnetic resonance imaging-proven right index and middle finger flexor tenosynovitis, which was complicated by osteomyelitis due to Mycobacterium chelonae. Based on the susceptibility results, a regimen of intravenous tobramycin and imipenem and oral clarithromycin was started for induction therapy. She was treated with 6 total weeks of antibiotics before switching to oral moxifloxacin and clarithromycin. Within 10 days after the switch, she noticed changes in her tongue color and presented to the clinic without fever, nausea, and taste alteration. She denied any medication changes, had taken no herbal/traditional Chinese medicine, did not smoke, and had no alcohol use. She also denied using oxidizing/irritant mouthwashes, tongue cleaners, or tongue scrapers.

Upon examination, she was found to be in good general health with a blood pressure of 135/76 mmHg, pulse of 80 beats per min, respiration rate of 15 breaths per min, and body temperature of 36.7°C. The oral mucosa was dry and there was little pooled saliva on the floor of the mouth. The brown discoloration was seen on the dorsum of the tongue, with carpet-like elongated filiform lingual papillae (Figure 1). There were no dental caries, halitosis, discharge, bleeding, or exudates. Her gingiva was clear without any trauma. She did not use dentures. Her right finger was healed and all other clinical examinations were unremarkable. Laboratory results showed mild anemia (hemoglobin, 11.5 g/dL), a white blood cell count of 9800/mm³, erythrocyte sedimentation rate of 20 mm/h, and C-reactive protein level of 1.0 mg/dL. No tongue swab culture was performed as she did not have significant symptoms. The tentative diagnosis was stained tongue from foods or BHT from antibiotics. Other differential diagnoses such as oral hairy leukoplakia or acanthosis nigricans were ruled out based on the relatively rapid presentation of her tongue discoloration. She was observed for 2 weeks with daily oral hygiene and tongue scraping; however, the discoloration and elongated filiform lingual papillae of her tongue did not improve. Thus, in accordance with her wishes and lower susceptibility of moxifloxacin than clarithromycin for isolated Mycobacterium chelonae, we discontinued moxifloxacin but continued treatment solely with clarithromycin. After stopping moxifloxacin, her tongue improved to normal within 2 days (Figure 2), and her right finger has shown no signs of recurrence for 12 months.

Figure 1. After starting moxifloxacin, the patient developed brown discoloration on the dorsum of the tongue with carpet-like elongated filiform lingual papillae.

Figure 2. After stopping moxifloxacin, the black hairy tongue improved.
Discussion

BHT (also called Lingua villosa nigra) [2] is an acquired benign condition classically presented as a superficial black and hairy carpet-like lingual growth. Since originally described by Amatus Lusitanus in 1557 as hairs on the tongue that would regrow upon being removed, several cases have been described in the literature [3]. The pathophysiology of BHT has not been fully elucidated but it is thought to arise from defective desquamation of the dorsal tongue surface, which then prevents normal debridement, leading to the accumulation of keratinized layers. The resulting hypertrophy and elongation of the filiform papillae appear superficially hair-like and these elongated papillae then collect fungi, bacteria, and debris. This collection can include residue from tobacco, coffee, tea, and other foods as well as porphyrin-producing chromogenic organisms in the oral flora that impart a characteristic hue. Although recognized for its distinctive black color, these hues can range from blackish-brown to yellow-green to unpigmented [1]. A diagnosis of BHT is made by visual examination, but microscopic examination is useful to detect the presence of elongated hypertrophied filiform papillae [4]. It is important to differentiate BHT from a discolored (or black) tongue which does not exhibit long, thick filiform papillae.

The etiology of BHT remains unclear and is likely multifactorial. Both extrinsic and intrinsic factors potentially contribute to BHT. Extrinsic factors include heavy use of tobacco, alcohol, or intravenous drug use, excessive coffee or black tea consumption, prolonged oxidizing/irritant mouthwash use, or recent radiation therapy to the head and neck region. Intrinsic factors include poor oral hygiene, HIV, or trigeminal neuralgia which lead to limited tongue movement [1]. In addition, xerostomia is known to be a strong risk factor for BHT [3]. In our case, the patient had xerostomia from MCTD and developed BHT after starting moxifloxacin, but this improved rapidly after discontinuing moxifloxacin. We calculated the Naranjo adverse drug reaction probability scale to evaluate the association between the moxifloxacin and BHT. The score was 5, which was higher than other drugs taken by the patient.

Antibiotic-induced BHT is uncommon, and several cases have been reported after administration of penicillins, cephalosporins, erythromycin, minocycline, doxycycline, metronidazole, imipenem/cilastatin, and linezolid [3-6]. However, to the best of our knowledge, our report is the first to describe a patient with moxifloxacin-induced BHT.

The hypothesized mechanism of antibiotic-induced BHT is dysbiosis in the mouth that gives rise to chromogenic bacteria, especially Porphyromonas gingivalis. Secondary fungal infections or discoloration from antibiotics themselves are also reported as causative hypotheses [3]. Another mechanism, inhibition of the intestinal nicotinamide-producing flora, was reported specifically for penicillin therapy [7]. It is unclear if these hypotheses are adaptable to all antibiotics, because only limited antibiotics have been found causative for BHT. As BHT is generally a self-limiting disease and rarely gives subjective symptoms, it may be underdiagnosed outside of dental clinics [1]. In our case, the patient did not have significant symptoms, but wanted to discontinue antibiotics anyway. The discontinuation of potentially causative agents and maintenance of good oral hygiene are the standard treatments [1] and usually improve symptoms within a few days, as seen in our case.

Conclusions

Clinicians should be aware of agents or lifestyles that may cause BHT, and it is essential to counsel on the possibility of developing BHT before starting treatment to avoid patient anxiety or premature treatment cessation.

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Declaration of Figures’ Authenticity

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