

Title: NOTHING TO SAVOR, THIS FOOD HAS NO FLAVOR: A CASE OF PROFOUND DYSGEUSIA IN DISSEMINATED NOCARDIA

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Introduction: Nocardia has a known predilection for brain tissue and may present with unique symptoms. We share a case of significant dysgeusia in a presumptively immunocompetent patient as a complication of disseminated nocardiosis.

Case: A 70-year-old previously healthy male with a history of diabetes, presented with three months of fatigue, productive cough, loss of appetite, and weight loss. Chest CT demonstrated a large mass involving the right upper and middle lobes. Bronchoscopically obtained cultures were positive for branching gram-positive bacilli ultimately identified as *Nocardia bscensus* complex.

Intravenous (IV) trimethoprim-sulfamethoxazole (TMP-SMX) was started as initial therapy. A brain MRI revealed two peripherally enhancing lesions in the right temporal lobe and cerebellar vermis prompting the addition of IV imipenem and amikacin. He subsequently developed severe, progressive dysgeusia, reporting that “everything tastes horrible,” accompanied by frequent episodes of nausea. Initially postulated to be side effects of amikacin, he was switched to IV linezolid, and started on scheduled haloperidol and ondansetron. Despite these changes, his symptoms persisted. Otolaryngology noted that the temporal lobe brain abscess was located within the insular cortex in close proximity to the gustatory center and the potential source of his symptoms.

Susceptibilities of the Nocardia ultimately showed sensitivity to TMP-SMX, linezolid, and ceftriaxone, but resistance to imipenem. Before discharge, the patient was transitioned to IV ceftriaxone and high dose oral TMP-SMX. At one month infectious disease follow-up, the patient reported marked improvement in his dysgeusia and repeat MRI demonstrated significant reduction in size of his brain abscesses. TMP-SMX was discontinued after one month due to persistent hyperkalemia; ceftriaxone is planned for a full year.

Discussion:

Nocardia species has been found to have a predilection for neural tissue, with one study showing up to 44% of cases involving the CNS. There does not seem to be further specification as to what CNS regions Nocardia prefers. The resolution of symptoms coinciding with the shrinking of the abscesses suggests that the location of the lesions was the source of the patient's prolonged dysgeusia. The insular cortex is well documented to be implicated in the feelings of disgust, nausea, emesis and taste.

Nocardia infection is more common in frankly immunocompromised individuals. While this patient has a history of diabetes (Hgb A1C range 6.7-8.6 mg/dL), he had no other risk factors suggesting immunosuppression. The patient previously worked in manufacturing and farming and continues to engage in yardwork on his rural property. Nocardia can be commonly found in soil and dead vegetation, which seems to be the most likely source of infection.

Immunosuppressed patients diagnosed with disseminated Nocardia usually require one year of antibiotics comprised of 2-3 agents. There are no specific guidelines for the immunocompetent; this patient is currently planned for one year of ceftriaxone.

