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Case Report Open Access

Kocuria rhizophila Intracerebral Abscess in Diabetic Ketoacidosis

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Abstract

Intracerebral abscesses are encapsulated intraparenchymal infections associated with a high mortality rate. These abscesses can be caused by a number of different organisms ranging from bacteria to fungi, parasites, and even malignancies. The most common causative agent depends on pre-disposing risk factors. In this article, we report a case of a brain abscess caused by *Kocuria rhizophila*, a pathogen that has not been previously reported to cause cerebral intraparenchymal abscesses.

Keywords: Intracerebral abscess; *Kocuria rhizophila*; Diabetic ketoacidosis

Introduction

Intracerebral abscess development begins as a necrotic focus of cerebritis, edema, and inflammation. Over time the necrotic zone becomes more discrete from granulation tissue that surrounds it [1-3]. A capsule begins to form as fibroblasts proliferate and neovascularization proceed before the capsule is fully completed and thickened [3]. Cerebral abscesses originate via contiguous spread of infection, hematogenous dissemination, or metastasis [4]. Intracerebral abscesses can be caused by bacteria, fungi, parasites, other microorganisms, or malignancies, with each etiologic agent tending to appear in different cases. Quick suspicion and identification of causative pathogens is essential to prompt and effective treatment as mortality (9.5%) and morbidity (29%) of these conditions remain high [2]. Here we report a case of a patient with diabetic ketoacidosis who developed a brain abscess that after culture was determined to be *Kocuria rhizophila*. A literature review was performed, determining that cerebral abscesses caused by *K. rosea* and *K. varians* have been reported, as have two cases of hematogenous infection by *K. rhizophila* [5-8]. However, there have been no reports of *K. rhizophila* causing intracerebral abcesses.

Case Report

A 52-year-old male with no known past medical history was found unresponsive with Kussmaul breathing at his hotel. Emergency response personnel found him to be severely hyperglycemic and administered 500 mL of normal saline en route to the emergency department.

On physical examination, his vital signs were notable for a respiratory rate of 36 breaths per minute. Blood work revealed that his serum osmolality was 341 and a random blood glucose was 678 mg/dL. An arterial blood gas was done revealing a pH of 6.79, partial pressure of CO_2 of 22 mmHg, partial pressure of O_2 of 47 mmHg, and a bicarbonate concentration of 3 mEq/L. The patient was found to be hyponatremic, hyperkalemic, hypocalcemic, and azotemic. A complete blood count revealed a leukocyte count of 21.79×10^3 cells/μL. He was subsequently diagnosed with diabetic ketoacidosis and treated with fluids, electrolytes, and insulin per protocol. He was HIV negative.

A subsequent CT scan revealed an age indeterminant hypodensity in the right basal ganglia, confounded by motion artifact. An MRI subsequently revealed 2 lesions in the right basal ganglia causing mild effacement of the right lateral ventricle and no midline shift. Differential diagnoses included abscess, tumor, and subacute hemorrhage. A repeat MRI several days later demonstrated evolution of the lesion with severe mass effect on the right lateral ventricle and a 3 mm right to left midline shift. Stereotactic biopsy of the lesion was carried out, confirming the presence of an abscess. Initial Gram and Grocott methenamine silver stains were done, but no organisms could be identified. Routine aerobic, anaerobic, and fungal cultures initially came back negative. He was empirically started on Vancomycin, Metronidazole, and Ceftriaxone but developed a rash, leading to discontinuation of Ceftriaxone. Midline shift progressed to 6 mm before stabilizing, and he was treated with IV antibiotics as cultures continued, a 6-week course of Vancomycin, Metronidazole, and Meropenem. He returned for a follow-up, and one colony of *K. rhizophila* was isolated. Although the abscess was unresolved and unchanged in size, the recommendation was to finish the course of antibiotics. The patient left the long-term acute care facility against medical advice 2 days prior to finishing the course of antibiotics and has failed to return for any scheduled follow-up visits.

Discussion

The genus Kocuria belongs to the family *Micrococcaceae* and consists of nonencapsulated, non-endospore-forming, gram positive cocci. *Kocuria* species are catalase positive, coagulase negative, and nonhalophilic. They lack mycolic acid and teichoic acid [9,10]. Reports of *Kocuria* infections have been few and mostly involved immunocompromised hosts. Both of the case reports on *K. rhizophila* involved contamination of catheters. *K. rhizophila* is known to live in soil and food, and Becker et al. hypothesized that their patient may have been colonized following contact with an environmental source such as dust, food, or freshwater but did not expand upon this hypothesis with any data [8,11,12]. Becker et al. reported that their patient had lactic acidosis and methylmalonic aciduria, creating the possibility that *K. rhizophila* growth is promoted by acidity [8].

Interestingly, the *K. rhizophila* isolated from food in a previous study came from chicken meat treated with oxalic acid [11].

It is unclear how our patient developed a *K. rhizophila*-infected abscess. Motion artifacts made it is unclear whether or not the patient had had an abscess prior to initial CT imaging or if the original lesion was indeed an area of ischemia that then developed into an abscess. The latter is more likely, but this scenario makes it likely that the patient was inoculated by *K. rhizophila*, which benefited from the patient's ketoacidotic state, during his hospital stay.

Sensitivity data of Kocuria species are limited. Becker et al. reported that *K. rhizophila* was sensitive to aminoglycosides, β-lactams, glycopeptides, macrolides, and quinolones, rifampin [8]. Moissenet et al. treated their patient with combination of gentamicin (3 mg/kg/day) and vancomycin (40 mg/kg/day) for 2 and 10 days respectively. Montoya et al. treated brain abscess due to *K. rosea* with Cefepime for 4 weeks, inducing complete remission of abscesses. Tsai et al. administered Ceftazidime and Metronidazole prior to surgical excision of a *K. varians*-caused brain abscess. Ceftazidime was continued post-operatively while Metronidazole was ceased due to sensitivity testing showing resistance. After 4 weeks, the patient was switched to Ceftibuten for 2 weeks. While cephalosporins appear to be the drug-of-choice for Kocuria-caused brain abscesses based on these studies, our patient was allergic. However, Vancomycin and Meropenem exhibit moderately high blood-brain-barrier penetrance [13].

Of note, Micrococcus luteus has been noted in the literature to cause intracranial abscesses [14]. *K. rhizophila* was originally a particular strain (ATCC 9341) of *M. luteus* but was reclassified in 2003 due to significant differences in phenotype and riboprints.15 Although it is unlikely that the case reported by Selladurai et al. was M. luteus ATCC 9341, this possibility cannot be ruled out [15].

Conclusion

Infections caused by the genera Kocuria are rare. Here we report the first confirmed case of *Kocuria rhizophila* associated with intraparenchymal brain abscess. The mechanism by which the patient procured The *K. rhizophila* is unclear, but there is evidence in literature that acidic environments may help *K. rhizophila* outcompete other flora. Our patient was ketoacidotic, which may have contributed to the development of his condition.

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