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Case Report

Primary atrophic rhinitis associated with *Klebsiella pneumoniae* subsp. *ozaenae*: a case report with microbiological correlation

Emmanuel Jabakumar*, Samit Sen, Trisha Tarunita, Shweta Satija

Department of Microbiology, Lady Hardinge Medical College, New Delhi, India

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***Correspondence:**
Dr. Emmanuel Jabakumar,
E-mail: dremanjrpeter@gmail.com

ABSTRACT

Primary atrophic rhinitis (ozena) is a chronic, debilitating nasal disorder characterized by progressive mucosal atrophy, crust formation, foul smell, and widening of the nasal cavity. Although its etiology is multifactorial, *K. pneumoniae* subsp. *ozaenae* has been frequently associated with this condition, although its exact pathogenic role remains debated. Early microbiological diagnosis and confirmation is essential for targeted antimicrobial therapy and prevention of chronic complications. A 32-year-old male presented with chronic nasal crusting, foul-smelling discharge, and recurrent upper respiratory tract infections. Computed tomography (CT) of the paranasal sinuses revealed pansinusitis with bilateral osteomeatal complex obstruction. Direct microscopy showed no acid-fast bacilli or fungal elements. Culture of nasal crust specimens yielded mucoid, non-lactose fermenting colonies identified as *K. pneumoniae* subsp. *ozaenae* using conventional biochemical tests and also confirmed by automated VITEK 2 (bioMérieux). Antimicrobial susceptibility testing (Kirby-Bauer method, CLSI guidelines) demonstrated susceptibility to amoxicillin-clavulanate, piperacillin-tazobactam, cefepime, aminoglycosides, ciprofloxacin, cotrimoxazole, and carbapenems, with resistance to ceftriaxone. The patient improved following culture-directed oral ciprofloxacin therapy combined with local nasal care. This case highlights the diagnostic challenge in distinguishing colonization from pathogenic involvement and emphasizes the importance of clinicomicrobiological correlation. Culture-guided therapy ensures favourable outcomes and supports antimicrobial stewardship.

Keywords: Atrophic rhinitis, Ozena, *Klebsiella ozaenae*, Pansinusitis, Antimicrobial susceptibility

INTRODUCTION

Primary atrophic rhinitis (ozena) is a chronic nasal disorder characterized by progressive atrophy of the nasal mucosa and turbinates, formation of thick crusts, anosmia, and a characteristic fetor.[1] This condition in developing regions remains sporadically reported and continues to pose diagnostic and therapeutic challenges.[2] Although the exact pathogenesis is multifactorial, infectious, vascular, nutritional, and immunological mechanisms have been implicated.[3] Among infectious causes, *K. pneumoniae* subsp. *ozaenae* has been consistently associated with primary atrophic rhinitis and is regarded as a significant contributory pathogen.[4] Early microbiological confirmation is crucial for guiding targeted antimicrobial therapy and preventing chronic sinonasal complications.[5] Despite its historical association with ozena, the exact role of *K. pneumoniae* subsp. *ozaenae* as a true pathogen versus a colonizer in chronically atrophic nasal mucosa remains uncertain. This distinction has important clinical implications, particularly in guiding antimicrobial therapy. The present case aims to highlight this clinicomicrobiological correlation and the need for cautious interpretation of culture results.

CASE REPORT

A 32-year-old male presented to the outpatient department with complaints of chronic nasal obstruction,

persistent foul-smelling nasal discharge, and progressive nasal crusting for several months. He also reported recurrent episodes of upper respiratory tract infections. There was no history of fever, epistaxis, facial trauma, previous nasal surgery, or known contact with tuberculosis cases. The patient had no significant comorbidities.

On clinical examination, widening of the nasal cavity with thick, adherent crusts and mucosal dryness was noted. Removal of crusts revealed atrophic mucosa. A characteristic fetor was present. Based on clinical findings, chronic rhinosinusitis with atrophic changes was suspected, and microbiological evaluation was undertaken as recommended in chronic infective nasal conditions.[6]

Non-contrast computed tomography (CT) of the paranasal sinuses demonstrated partial opacification of the frontal sinuses (right more than left), ethmoid, maxillary, and sphenoid sinuses. Bilateral osteomeatal complex obstruction and mild right-sided deviation of the nasal septum were observed. These findings were consistent with pansinusitis and correlated with previous CT imaging findings, supporting chronic sinonasal involvement.[7]

Nasal crust specimens were collected under aseptic precautions prior to initiation of antibiotic therapy and transported promptly to the microbiology laboratory. Direct Gram staining revealed 1-2 pus cells per high-power field without visible organisms. Ziehl-Neelsen staining was negative for acid-fast bacilli, and potassium hydroxide mount did not demonstrate fungal elements, thereby excluding mycobacterial and fungal etiologies.[8]

The specimen was cultured on 5% sheep blood agar and MacConkey agar and incubated aerobically at 35-37°C for 18-24 hours. Smooth, greyish-white, non-hemolytic mucoid colonies were observed on blood agar, and large, pale, mucoid non-lactose fermenting colonies grew on MacConkey agar. The isolate was non-motile, indole negative, citrate positive, and weakly urease positive. On triple sugar iron agar, an alkaline slant and acidic butt (K/A) without gas or hydrogen sulfide production was noted. These biochemical characteristics were consistent with *Klebsiella* species.[9] Definitive identification using the automated VITEK 2 system (bioMérieux) confirmed *Klebsiella pneumoniae* subsp. *ozaenae*.[10]

Antimicrobial susceptibility testing was performed using the Kirby-Bauer disk diffusion method in accordance with Clinical and Laboratory Standards Institute CLSI) guidelines.[11]

The isolate demonstrated susceptibility to amoxicillin-clavulanate, piperacillin-tazobactam, cefepime, aminoglycosides, ciprofloxacin, cotrimoxazole, and carbapenems, while resistance was observed to ceftriaxone.

Table 1: Antimicrobial susceptibility profile of the isolate.

Antibiotics	Results
Amoxicillin-clavulanate	Sensitive
Piperacillin-tazobactam	Sensitive
Cefepime	Sensitive
Ceftriaxone	Resistant
Amikacin	Sensitive
Gentamicin	Sensitive
Ciprofloxacin	Sensitive
Cotrimoxazole	Sensitive
Imipenem/Meropenem	Sensitive

susceptibility testing was performed using the Kirby-Bauer disk diffusion method in accordance with Clinical and Laboratory Standards Institute CLSI) guidelines.[11] The isolate demonstrated susceptibility to amoxicillin-clavulanate, piperacillin-tazobactam, cefepime, aminoglycosides, ciprofloxacin, cotrimoxazole, and carbapenems, while resistance was observed to ceftriaxone.

Based on susceptibility results, oral ciprofloxacin (500 mg twice daily for 14 days) was initiated along with regular alkaline nasal douching and topical nasal lubrication. At 2-week follow-up, the patient demonstrated marked reduction in nasal crusting and fetor, with significant improvement in nasal obstruction. No recurrence was noted during short-term follow-up. Repeat culture was not performed post-treatment, which limits confirmation of microbiological clearance.

Although species-level identification was strengthened using MALDI-TOF mass spectrometry, repeat cultures to demonstrate persistence or clearance were not performed. Additionally, the inability to definitively differentiate colonization from infection limits causal inference.

DISCUSSION

Primary atrophic rhinitis is a chronic progressive condition characterized by mucosal atrophy and secondary bacterial colonization, which contributes to persistent crusting and fetor.[12] Although the etiopathogenesis remains incompletely understood, colonization or infection with *K. pneumoniae* subsp. *ozaenae* has been repeatedly documented in affected patients.[13] The isolation of this organism in our case reinforces its recognized association with ozena and suggests a possible contributory role; however, definitive causation cannot be established.

Microbiological confirmation plays a pivotal role in differentiating ozena from other chronic nasal conditions, including granulomatous diseases such as tuberculosis and fungal sinusitis.[14] In tuberculosis-endemic settings, exclusion of mycobacterial infection through Ziehl-Neelsen staining and appropriate microbiological workup

is particularly important to avoid misdiagnosis and inappropriate therapy.

A major challenge in interpreting this case is distinguishing colonization from true infection. Chronic atrophic rhinitis results in mucosal damage and altered local immunity, creating a favorable environment for colonization by opportunistic organisms such as *K. pneumoniae* subsp. *ozaenae*. Therefore, isolation of the organism from nasal crusts should be interpreted with caution and correlated with clinical findings rather than assumed to be causative. Radiological evaluation plays a complementary role in disease assessment.

The antimicrobial susceptibility pattern in this case demonstrated preserved sensitivity to β-lactam/β-lactamase inhibitor combinations, aminoglycosides, fluoroquinolones, cotrimoxazole, and carbapenems, with resistance to ceftriaxone. Increasing resistance among *Enterobacterales* highlights the importance of culture-guided therapy rather than empirical prolonged antibiotic use.[15] The favorable response observed with ciprofloxacin therapy underscores the value of laboratory-directed management and adherence to antimicrobial stewardship principles.

Although less common today than in previous decades, sporadic cases of ozena continue to be reported, particularly in resource-limited regions. Careful documentation of microbiological findings and resistance patterns remains important for guiding regional therapeutic strategies and improving patient outcomes.

CONCLUSION

This case underscores the diagnostic complexity of primary atrophic rhinitis and highlights the importance of cautious interpretation of microbiological isolates. While *K. a pneumoniae* subsp. *ozaenae* is frequently associated with ozena, its role as a pathogen versus colonizer remains uncertain. Integration of clinical, radiological, and microbiological findings is essential for appropriate management, and culture-guided therapy can improve patient outcomes while supporting antimicrobial stewardship.

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