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Case Report

Tracheostomy co-colonization in autoimmune disease: beyond empiric treatment

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ABSTRACT

We report an unusual case of mixed *Proteus mirabilis* and *Candida glabrata* colonization of a tracheostomy tube in a patient with rheumatoid arthritis and Sjögren syndrome. Despite the striking appearance of blackish deposits on the tube surface, the patient was asymptomatic and endoscopic evaluation revealed no local pathology. The patient's comorbidities likely contributed to altered airway microbial ecology, favoring biofilm persistence. Understanding biofilm behavior and differentiating colonization from infection prevented unnecessary antimicrobial therapy. This case highlights the importance of correlating microbiological findings with clinical and endoscopic assessments to guide management in chronic tracheostomy patients.

Keywords: Autoimmune disease, Colonisation, *Candida glabrata*, *Proteus mirabilis*, Sjogren, Tracheostomy

INTRODUCTION

Tracheostomy tubes in long-term use are prone to biofilm formation and microbial colonization, which can harbor both bacterial and fungal organisms.[1,2] Differentiating colonization from infection remains a clinical challenge, as positive cultures alone may not warrant treatment. In immunocompromised or autoimmune patients, altered mucosal defense mechanisms can influence colonization patterns, making interpretation even more complex.[3-5] This case describes a rare silent co-colonization by *Proteus mirabilis* and *Candida glabrata* in a patient with autoimmune disease, emphasizing biofilm ecology in guiding management decisions.

CASE REPORT

A 66-years-old woman with rheumatoid arthritis and Sjögren syndrome underwent tracheostomy in 2017 for bilateral vocal cord palsy. She attended regular follow-up with scheduled tube changes every six months. During a routine change, the outer cannula was found to be entirely covered with blackish mold-like deposits. The tube was sent for culture, which grew *Proteus mirabilis* and *Candida glabrata*.

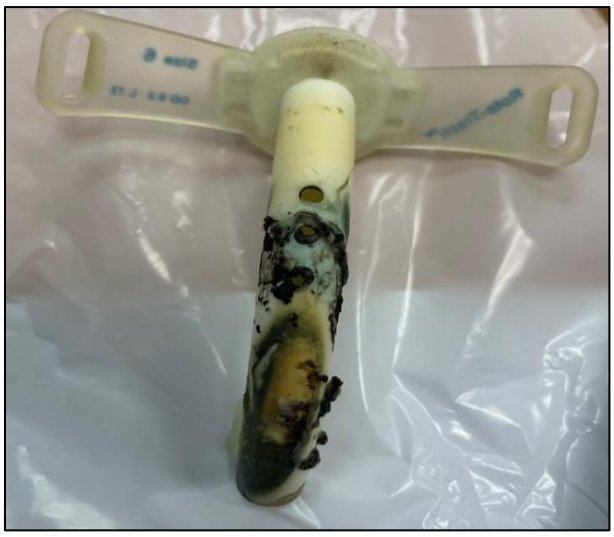

Figure 1: Tracheostomy tube with mold-like deposits observed during routine tube change.

Airway endoscopy revealed no lesions or mucosal changes. The patient remained asymptomatic, with no respiratory distress, fever or increased secretions. Given the absence of clinical and endoscopic evidence of infection, no antimicrobial therapy was initiated. She continued regular follow-up without adverse outcomes.

DISCUSSION

Long-term tracheostomy tubes serve as ideal substrates for biofilm formation, which can harbor mixed microbial communities, including bacteria and fungi.[1,2] In this case, both *P. mirabilis* and *C. glabrata* were isolated. *P. mirabilis is* a motile, urease-producing Gram-negative bacillus well known for biofilm formation on medical devices.[8] *C. glabrata*, although a commensal of the human mucosa, can behave opportunistically in immunocompromised settings and is intrinsically less susceptible to azoles.[3,4,6] Their coexistence likely reflects synergistic biofilm ecology, where bacterial and fungal communities protect each other from host defenses and environmental stress.[7]

The patient's rheumatoid arthritis and Sjögren syndrome may have predisposed her to this colonization through several mechanisms. Autoimmune-mediated mucosal dryness reduces mucociliary clearance, allowing microbes to persist. Immunomodulatory therapies commonly used in these conditions can further impair local and systemic immune responses, favoring colonization by opportunistic organisms.[3,4] In addition, long-term indwelling foreign material such as a tracheostomy tube creates a surface conducive to biofilm establishment, where microbial communities are shielded from host immunity and antimicrobial agents.[2,7]

Despite the unusual and visually concerning colonization, the absence of local or systemic signs of infection supported a conservative approach. This aligns with literature showing that respiratory isolation of Candida species rarely indicates invasive disease in the absence of compatible symptoms or radiological findings.[5,6] Similarly, colonization by Gram-negative bacilli such as P. mirabilis in tracheostomy tubes does not always mandate treatment unless there is clinical deterioration.[7] Treating such findings empirically risks unnecessary antimicrobial exposure, selection of resistant organisms and disturbance of the airway microbiome.

Prevention of such colonization relies on meticulous tracheostomy care, including regular tube changes, thorough cleaning of inner cannulas and patient or caregiver education on hygiene.[1] In patients with autoimmune disease, optimizing mucosal hydration, such as using humidification systems or saline nebulization, may reduce biofilm development. Clinical judgment should balance microbiological findings with the patient's presentation to guide management, reserving antimicrobial therapy for confirmed infection.

Looking ahead, novel anti-biofilm strategies such as quorum sensing inhibitors, bacteriophage therapy and enzymatic disruption of extracellular matrices may offer promising avenues for non-antibiotic biofilm control. Additionally, antimicrobial-coated tracheostomy tubes and silver-impregnated materials are under investigation as preventive interventions.[8,9] Incorporating these innovations could further improve outcomes in patients susceptible to complex biofilm-associated colonization.

CONCLUSION

This case illustrates that in long-term tracheostomy patients with autoimmune-related mucosal dryness and altered immunity, visually striking mixed bacterial–fungal colonization may represent benign biofilm rather than active infection. Careful clinical and endoscopic correlation, coupled with an understanding of biofilm ecology, can safely prevent unnecessary antimicrobial therapy. Emphasizing patient-specific risk factors, preventive care and vigilant follow-up ensures optimal outcomes while supporting antimicrobial stewardship.

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