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ATYPICAL MICROBIOLOGICAL PROFILE IN PEDIATRIC CYSTIC FIBROSIS: PREDOMINANCE OF STAPHYLOCOCCUS AUREUS OVER PSEUDOMONAS AERUGINOSA

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ABSTRACT

Background: Cystic Fibrosis (CF) is a multi-organ genetic disorder characterized by chronic airway infections. While *Pseudomonas aeruginosa* typically becomes the dominant pathogen as the disease progresses, some pediatric cases exhibit unusual colonization patterns. **Case Presentation:** We report the case of a 5-year-old male patient (M.R.) presenting with recurrent respiratory distress and growth failure. Diagnosis was confirmed by a positive sweat chloride test (>60 mmol/L). **Microbiological Findings:** Contrary to the expected transition towards *Pseudomonas*-dominant lungs, repeated cultures and antibiograms consistently showed a heavy and exclusive colonization of *Staphylococcus aureus*. This persistent "Staph-dominant" profile, despite clinical exacerbations, is an atypical finding in symptomatic pediatric patients. **Conclusion:** This case highlights the necessity of personalized, culture-guided antibiotic therapy and challenges the systematic assumption of *Pseudomonas* emergence in early childhood CF management.

KEYWORDS: Cystic Fibrosis, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, Bacterial Competition, Pediatrics.

INTRODUCTION

Cystic Fibrosis (CF) is caused by mutations in the CFTR gene, leading to impaired mucociliary clearance. The microbiological landscape of CF lungs is typically age-dependent: *Staphylococcus aureus* and *Haemophilus influenzae*predominate in early childhood, while *Pseudomonas aeruginosa* becomes the primary pathogen in adolescence and adulthood, driving lung function decline.[1,2]

Recent studies suggest that while *P. aeruginosa* often outcompetes *S. aureus* through the secretion of virulence factors, certain patients maintain a stable, long-term colonization of *S. aureus*.[4] In North Africa, diagnostic delays and specific environmental factors may influence these microbial dynamics.[5] This report analyzes the case of patient M.R., whose clinical course is characterized by a persistent staphylococcal carriage without the common transition to *Pseudomonas*.

CASE PRESENTATION

Patient M.R., a 5-year-old boy, presented with a history of chronic productive cough and failure to thrive. Physical examination showed digital clubbing and diffuse pulmonary rales. The sweat chloride test was diagnostic (68 mmol/L).

Microbiological monitoring via sputum cultures and bronchial secretions revealed a consistent and high bacterial load of *Staphylococcus aureus*. Notably, despite multiple pulmonary exacerbations that usually herald the arrival of Gram-negative opportunistic pathogens, *Pseudomonas aeruginosa* remained absent or below detectable levels. Antibiogram results indicated, necessitating a specialized antibiotic regimen focused on Gram-positive eradication rather than standard anti-pseudomonal protocols.

RESULTS AND DISCUSSION

The clinical evolution of patient M.R. confirms a CF phenotype with a "Staph-heavy" microbiological profile. In the classical "replacement model," *P. aeruginosa* is expected to suppress *S. aureus* growth through 2-heptyl-4-hydroxyquinoline N-oxide (HQNO) and siderophores.[6,7]

Discussion of Atypical Dominance The absence of *Pseudomonas* transition in this symptomatic 5-year-old patient is unusual. Literature suggests that *S. aureus* can adapt to the CF lung by forming Small Colony Variants (SCVs) or biofilms, which provide protection against both host immunity and inter-species competition.[8,9]

The persistence of *S. aureus* in this case may be explained by

1. **Bacterial Interference:** Recent research shows that certain strains of *S. aureus* can coexist with or even inhibit *P. aeruginosa* colonization through specific metabolic niches.[7,10]
2. **Early Therapeutic Selection:** Continuous anti-pseudomonal prophylaxis or environmental factors in the North African context might have delayed the acquisition of *Pseudomonas*, allowing *Staphylococcus* to establish a robust, dominant niche.[5]

This atypical carriage is critical for clinicians: assuming a *Pseudomonas* infection based on age or symptoms without culture confirmation could lead to inappropriate antibiotic use, further selecting for resistant *Staphylococcus* strains.[10]

CONCLUSION

The case of patient M.R. demonstrates that the "Staph-to-Pseudomonas" switch is not an absolute rule. The persistent dominance of *Staphylococcus aureus* in this pediatric patient highlights the diversity of the CF microbiome. Precision medicine, based on frequent and accurate microbiological cultures, remains the gold standard for managing airway infections in cystic fibrosis.

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