

Original Article

Clinical Characteristics and Treatment Outcomes After First *Achromobacter* Isolation in Children with Cystic Fibrosis (Pre–CFTR Modulator Era)

Şeyda Karabulut¹, Merve Selçuk Balcı¹, Mine Yüksel Kalyoncu¹, Aynur Gulieva¹,
Mürüvvet Yanaz¹, Cansu Yılmaz Yeğit¹, Almala Pınar Ergenekon¹, Burcu Uzunoğlu²,
Gamze Taştan², Özgenur Demirkol³, Ela Erdem Eralp¹, Yasemin Gökdemir¹,
Ayşegül Karahasan³, Fazilet Karakoç¹, Bülent Karadağ¹

¹Division of Pediatric Pulmonology, Marmara University Faculty of Medicine, İstanbul, Türkiye

²Selim Coremen Cystic Fibrosis Center, Marmara University Faculty of Medicine, İstanbul, Türkiye

³Department of Medical Microbiology, Marmara University Faculty of Medicine, İstanbul, Türkiye

Cite this article as: Karabulut Ş, Selçuk Balcı M, Yüksel Kalyoncu M, et al. Clinical characteristics and treatment outcomes after first *Achromobacter* isolation in children with cystic fibrosis (pre-CFTR modulator era). *Thorac Res Pract.* [Epub Ahead of Print]

ABSTRACT

OBJECTIVE: We aimed to describe the clinical and demographic characteristics of children with cystic fibrosis (cwCF) diagnosed with a first *Achromobacter* (Ach) isolation, and to evaluate treatment outcomes at our CF center during the pre-modulator era.

MATERIAL AND METHODS: We retrospectively reviewed children followed at a single CF center (2014–2021) with at least four respiratory cultures per year and ≥12 months of follow-up after first Ach isolation. Eradication was defined as no Ach isolation for 12 months after the first detection; persistent infection was defined as intermittent or chronic isolation according to the Leeds criteria. We compared clinical characteristics and 12-month outcomes between the eradicated and non-eradicated groups.

RESULTS: Forty children were included; mean age at first isolation was 10.9±7.0 years, and eradication was achieved in 19/40 (47.5%). The non-eradicated group was older at first isolation (12.7±8.0 vs. 8.5±5.1 years; $P = 0.04$), had more pulmonary exacerbations (Pex) in the preceding year (median 4 vs. 2; $P = 0.01$), and more frequent prophylactic azithromycin use (67% vs. 21%; $P = 0.01$). Airway colonization patterns changed significantly over 12 months in the non-eradicated group ($P < 0.001$), with increased colonization by *Pseudomonas aeruginosa* (PA); changes were not significant in the eradicated group. Among those with available spirometry, 12-month forced expiratory volume in one second (FEV₁) z-scores did not differ by eradication status after adjustment for the baseline FEV₁ z-score.

CONCLUSION: In this pre–cystic fibrosis transmembrane conductance regulator modulator study, persistence of Ach was associated with older age, higher Pex burden before first isolation, and clinically relevant shifts in airway microbiology, including increased PA colonization. Our findings suggest that cwCF with frequent exacerbations or a worsening clinical course may warrant closer attention to Ach, and eradication of Ach may be considered at first detection.

KEYWORDS: Cystic fibrosis, *Achromobacter*, children, emerging pathogens, pulmonary infection, treatment

Received: 15.01.2026

Revision Requested: 28.04.2026

Last Revision Received: 28.04.2026

Accepted: 14.05.2026

Epub: 26.08.2026

INTRODUCTION

Achromobacter species (Ach) are opportunistic, non-fermentative, Gram-negative pathogens that are increasingly isolated in people with cystic fibrosis (pwCF) and can cause pulmonary exacerbations (Pex).^{1–6} According to the 2022 records of the Cystic Fibrosis Foundation (CFF), it was reported as the fourth most commonly detected pathogen in the respiratory tract of pwCF.⁷ In the 2021 European Cystic Fibrosis Society (ECFS) data, the prevalence of Ach was 3% among cwCF and 6.4% among adults.⁸ In the same registry, the prevalence of Ach in Türkiye was reported to be 1.1% among cwCF and 3.1% among adults.⁸

Corresponding author: Şeyda Karabulut, e-mail: seydakarabulut@yahoo.com.tr



Copyright© 2026 The Author(s). Published by Galenos Publishing House on behalf of Turkish Thoracic Society.
Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

More recent registry data suggest a stabilization of Ach prevalence. According to the 2024 CFF patient registry,⁹ Ach is now the fifth most frequently detected respiratory pathogen, with a prevalence of approximately 2%. The 2023 ECFS registry data report an overall European prevalence of 1.6%, with lower rates observed in Türkiye (0.9%).¹⁰ Taken together, contemporary CFF and ECFS data indicate that, although Ach remains a clinically relevant pathogen in pwCF, its prevalence appears to have stabilized at lower levels in recent years, with notable regional variation.

In this context, a recent study suggests that highly effective cystic fibrosis transmembrane conductance regulator (CFTR) modulator therapy may have influenced the epidemiology of Ach in CF. This longitudinal comparison between the year preceding initiation and the first year following initiation of elexacaftor/tezacaftor/ivacaftor therapy demonstrated a reduction in Ach positivity from 7.8% to 5.6%.¹¹ However, this apparent decline may not reflect the intrinsic clinical behavior of Ach, as access to CFTR modulators remains inequitable and many patients are either ineligible for them or subject to delayed treatment.¹² In Türkiye, CFTR modulators became available only after 2021 and were not reimbursed by the national health insurance system until June 2025, necessitating individual legal action for access.

It remains unclear whether Ach is a cause of more rapid deterioration in lung function or occurs in severe CF disease.^{3,13,14} More clinical data are needed to understand how Ach affects lung function, mortality, and morbidity in pwCF worldwide. There are no standard treatment protocols.

In this study, we aimed to describe the clinical and demographic characteristics of cwCF in whom Ach was isolated, and to evaluate treatment outcomes at our CF center during the pre-modulator era.

MATERIAL AND METHODS

This single-center retrospective study was conducted at the Marmara University CF Center between January 2014 and June 2021. A total of 400 pwCF medical records were reviewed.

Data Collection

All sputum and deep pharyngeal swab cultures were reviewed, and cwCF in whom Ach was isolated were included in the

study. Gender, age at isolation, forced expiratory volume in one second (FEV₁) z-score, body mass index (BMI) z-score, genotype, pancreatic insufficiency, cystic fibrosis-related diabetes mellitus (CFRD), cystic fibrosis-related liver disease (CFRL), hypertonic saline use, azithromycin use, dornase-alpha use, inhaled corticosteroid use, allergic bronchopulmonary aspergillosis (ABPA), chest physiotherapy techniques, Pex, and eradication regimens were obtained from the medical records. Pex was defined using the criteria modelled by Ng et al.¹⁵ ABPA, CFRD, and CFRL definitions were defined as stated in the relevant literature.^{8,16-18} None of these cwCF were treated with a CFTR modulator or high-dose ibuprofen at the time of the study.

Inclusion Criteria

Forty cwCF were included in the study. The inclusion criteria were as follows: CF diagnosis according to the consensus guidelines from the CFF,¹⁹ regular follow-up in the CF center outpatient clinic, and having at least four sputum or deep pharyngeal swab cultures per year (every 2–4 months). The cwCF were followed up for at least one year after their first isolation of Ach. cwCF with fewer than four respiratory samples in the years before and after Ach isolation were excluded.

Infection State

Initial isolation was defined as the first-ever positive culture or as a new isolation after being Ach-free for the previous 12 months. Ach isolation was determined using the Leeds criteria.²⁰ Leeds criteria define chronic colonization as isolation of the same pathogen from more than 50% of respiratory tract cultures after the first detection of the microorganism, and intermittent colonization as isolation in less than 50% of respiratory tract cultures. Eradication was defined as no isolation for 12 months after the initial isolation.

In our study, cwCF were divided into eradicated and non-eradicated groups. The non-eradicated group included cwCF with intermittent and chronic colonization. CwCF without Ach isolation for 12 months after the initial isolation were considered eradicated.

Lung Function

For children over six years of age, FEV₁ z-score values measured one year before and one year after the first Ach isolation were recorded with the Minispir spirometry device (Medical International Research, Rome, Italy) according to American Thoracic Society/European Respiratory Society technical standards.²¹

Microbiological Studies

Isolates were identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (VITEK MS, BioMérieux, France) with a matrix. However, using this method, species differentiation between *Achromobacter xylosoxidans* and *Achromobacter denitrificans* could not be achieved, and therefore species information was not included in the study. Antimicrobial susceptibility tests were conducted on Mueller Hinton agar (BioMérieux, France) using the disk diffusion method in accordance with European Committee on Antimicrobial Susceptibility Testing (EUCAST)

Main Points

- This study evaluates the clinical features of *Achromobacter* (Ach) in children with cystic fibrosis (cwCF).
- It underscores the risk of opportunistic infections in cwCF populations without cystic fibrosis transmembrane conductance regulator modulators.
- The study emphasizes the need for careful monitoring of cwCF who have frequent pulmonary exacerbations for Ach colonization.
- The findings suggest that eradication may be considered when Ach is identified in cwCF.

recommendations, and zone diameters were recorded in millimeters.²² Because the EUCAST guidelines provide zone diameter values only for *Achromobacter xylosoxidans* and no specific criteria are defined for other species, the recommended cut-off values for this species were used.²²

Antimicrobial Treatment

There was no standard treatment protocol; cwCF received their treatment according to the clinical status and antibiotic susceptibility of their microbial cultures. Treatment was not given to cwCF who had no complaints, even if they were in isolation. The cwCF with fever, low oxygen saturation, and weight loss were hospitalized and treated with intravenous (IV) antibiotics. The cwCF, whose general condition did not require hospitalization, received oral antibiotic treatment.

Among the oral antibiotics, amoxicillin-clavulanic acid, trimethoprim-sulfamethoxazole, and ciprofloxacin were used based on antimicrobial susceptibility testing. Meropenem, meropenem + colistin, and ceftazidime + amikacin were the preferred IV antibiotics. Oral antibiotics were administered for at least 14 days, and IV antibiotics for 14 to 21 days, depending on the clinical condition of the individuals.

Treatment was initiated seven days after sample collection for individuals who did not require hospitalization because the microbiological result was notified within that period.

Official approval for the study was received from the Marmara University Ethics Committee (protocol no: 09.2024.30, decision date: 12.01.2024). Given the retrospective study design and the use of fully anonymized patient data, written informed consent was not required and therefore not obtained in accordance with the ethics committee's decision.

Statistical Analysis

Numerical variables are expressed as mean $\pm$ standard deviation or median (Q1-Q3) as appropriate to the distribution of variables. Continuous data were analyzed using the independent-samples t-test or the Mann-Whitney U test for non-parametric data. Categorical data were analyzed using χ^2 or Fisher's exact tests. $P \leq 0.05$ was considered significant.

Changes in airway colonization status before and after Ach isolation were assessed using the Stuart-Maxwell test for marginal homogeneity, applied separately to the eradicated and non-eradicated groups. Exact P values were reported given sparse transitions across colonization categories.

To examine whether eradication status was associated with lung function, an analysis of covariance was performed to compare follow-up FEV₁ z-scores between groups, adjusting for baseline FEV₁ z-score. Analyses were restricted to individuals who were able to perform spirometry and who had FEV₁ z-score measurements available at both baseline and follow-up time points. Statistical analyses were performed using IBM Statistical Package for the Social Sciences (SPSS) version 31.

RESULTS

A total of 400 cwCF were screened, and Ach was isolated in 40. Of these, 30 received treatment.

The mean age of the cwCF at the first Ach isolation was 10.9 (± 7) years; 8.5 (± 5.1) years in the eradicated group and 12.7 (± 8) years in the non-eradicated group ($P = 0.04$, Table 1).

Baseline FEV₁ z-scores were similar between the eradicated and non-eradicated groups [-4.82 (± 1.61) vs. -5.08 (± 1.56); $P = 0.68$]. Over the 12-month follow-up period, lung function declined in both groups; however, there was no significant difference in 12-month FEV₁ z-scores between eradicated and non-eradicated patients [-5.11 (± 1.42) vs. -5.42 (± 1.35); $P = 0.59$]. After adjustment, eradication status was not independently associated with 12-month FEV₁ z-scores (adjusted means -5.24 vs. -5.32 ; $P = 0.63$), and similar patterns of lung function change were observed in both groups over the 12-month period (Table 2).

No differences in medication use between the groups were observed, except for azithromycin. The use of azithromycin prophylaxis was higher in the non-eradicated group than in the eradication group ($P = 0.01$). The baseline characteristics of the groups are presented in Table 1.

The number of pre-Ach Pex was higher in the non-eradicated group [median (Q1-Q3): 4 (2–5); $P = 0.01$; Table 1].

Of the 40 cwCF with Ach isolation, eradication was achieved in 19 (47.5%). Twenty-one (52.5%) cwCF were considered non-eradicated. Of these, 14 (35%) had intermittent colonization and 7 (17.5%) had chronic colonization. No treatment was administered to 3 (7.5%) cwCF in the eradicated group and to 7 (18%) cwCF in the non-eradicated group ($P = 0.3$).

Colonization patterns remained stable in the eradicated group, with no statistically significant change over time as determined by the two-tailed exact test ($P = 0.063$). In contrast, the non-eradicated group demonstrated a statistically significant shift in colonization status over time ($P < 0.001$). These transition patterns are illustrated in Figure 1.

The treatment modalities for cwCF are shown in Figure 2. Sixteen cwCF ($n = 16/19$) in the eradicated group and 14 cwCF ($n = 14/21$) in the non-eradicated group received IV or oral treatment, according to clinical status and culture-sensitivity results. In the eradicated group, 11 cwCF received only oral treatment, two cwCF received only IV treatment, and three cwCF received combined IV and oral treatment. Spontaneous eradication was observed in three cwCF who did not receive treatment. In the non-eradicated group, six cwCF received oral therapy, five received IV therapy, and three received oral + IV therapy. There was no statistically significant difference in IV and/or oral antibiotic treatment regimens between the groups ($P = 0.6$ in the eradicated group, $P = 0.5$ in the non-eradicated group).

Table 1. The baseline characteristics of the groups (before the first *Achromobacter* isolation) (n = 40)

Baseline characteristics and clinical outcomes	Eradicated (n = 19)	Non-eradicated (n = 21)	P value
Ach isolation age (years), mean $\pm$ SD	8.5 $\pm$ 5.1	12.7 $\pm$ 8	0.04*
Female, n (%)	8 (42%)	9 (43%)	0.9
BMI z-score, median (Q1-Q3)	-1 (-2.1 ~ -0.06)	-1 (-2.2 ~ 0)	0.9
CFTR variants, n (%)			
p.Phe508del/p.Phe508del	7 (37%)	3 (14%)	0.2
p.Phe508del/other	5 (26%)	9 (43%)	
Other/other	7 (37%)	9 (43%)	
Pancreatic insufficiency, n (%)	17 (89%)	19 (90%)	0.9
CFRD, n (%)	3 (16%)	6 (29%)	0.4
CFRL, n (%)	7 (37%)	6 (29%)	0.7
ABPA, n (%)	2 (11%)	1 (5%)	0.5
Pulmonary hypertension, n (%)	0 (0%)	2 (10%)	0.4
Inhaled corticosteroid use, n (%)	8 (42%)	10 (48%)	0.7
Chest physiotherapy, n (%)	18 (95%)	18 (86%)	0.6
7% hypertonic saline use, n (%)	15 (79%)	19 (90%)	0.3
Dornase-alpha use, n (%)	15 (79%)	20 (95%)	0.1
Oxygen use, n (%)	2 (11%)	8 (38%)	0.06
Azithromycin prophylaxis, n (%)	4 (21%)	14 (67%)	0.01*
No prior chronic airway colonization before first Ach isolation, n (%)	12 (63%)	18 (85%)	0.4
Pex in prior 12 months, median (Q1-Q3)	2 (1-4)	4 (2-5)	0.01*

*P < 0.05

Ach: *Achromobacter*, BMI: body mass index, CFTR: cystic fibrosis transmembrane conductance regulator, CFRD: cystic fibrosis-related diabetes mellitus, CFRL: cystic fibrosis-related liver disease; ABPA: allergic bronchopulmonary aspergillosis, Pex: pulmonary exacerbation, SD: standard deviation**Table 2.** Baseline and 12-month FEV₁ z-scores according to *Achromobacter* eradication status

Ach status	Baseline FEV ₁ z-score (mean $\pm$ SD)	12-month FEV ₁ z-score (mean $\pm$ SD)	Adjusted 12-month FEV ₁ z-score (mean; 95% CI)*
Eradicated (n = 10 ^a)	-4.82 ($\pm$ 1.61)	-5.11 ($\pm$ 1.42)	-5.24 (-5.52; -4.97)
Non-eradicated (n = 14 ^a)	-5.08 ($\pm$ 1.56)	-5.42 ($\pm$ 1.35)	-5.32 (-5.56; -5.10)
P value	0.68 [†]	0.59 [†]	0.63 [†]

[†]independent samples t-test, [‡]analysis of covariance (ANCOVA) adjusted for baseline FEV₁ z-score, *adjusted means estimated at a baseline FEV₁ z-score of -4.97,^aanalyses were restricted to patients who were able to perform spirometryFEV₁: forced expiratory volume in one second, SD: standard deviation, CI: confidence interval

DISCUSSION

In this single-center retrospective study, we evaluated the demographic and clinical characteristics of cwCF according to the treatment outcomes after initial Ach isolation. We observed that eradication was less likely among children who were older at the time of the first Ach isolation. Those in whom Ach persisted experienced a higher burden of Pex in the year preceding detection and were more likely to be receiving long-term azithromycin therapy. Persistent (non-eradicated) Ach was associated with changes in lower airway colonization over time.

The prevalence of Ach has been reported to range from 17.9% to 29.3% in previous studies.^{2,23} In our cwCF population, Ach positivity was detected in at least one respiratory culture in 10% of cases.

Kerem et al.²⁴ evaluated ECFS registry data and reported that the age of Ach occurrence peaked in the third decade of life and increased with advancing age. However, the mean age at first Ach isolation in our cohort was 10.9 years. Notably, more Ach isolates were identified in childhood than expected based on reports from low-income countries.^{2,24,25} In such settings, factors, including unfavorable environmental conditions, limited access to adequate nutrition, and suboptimal adherence to treatment, may contribute to earlier lung function decline and increased susceptibility to opportunistic airway pathogens in children.

In our study, consistent with the studies conducted by Lambiase et al.¹³ and De Baets et al.², our results showed a reduction in FEV₁ z-scores at the 12th month following Ach isolation in both groups, without reaching statistical significance. However, a study conducted in our country by Sunman et al.²⁶ reported

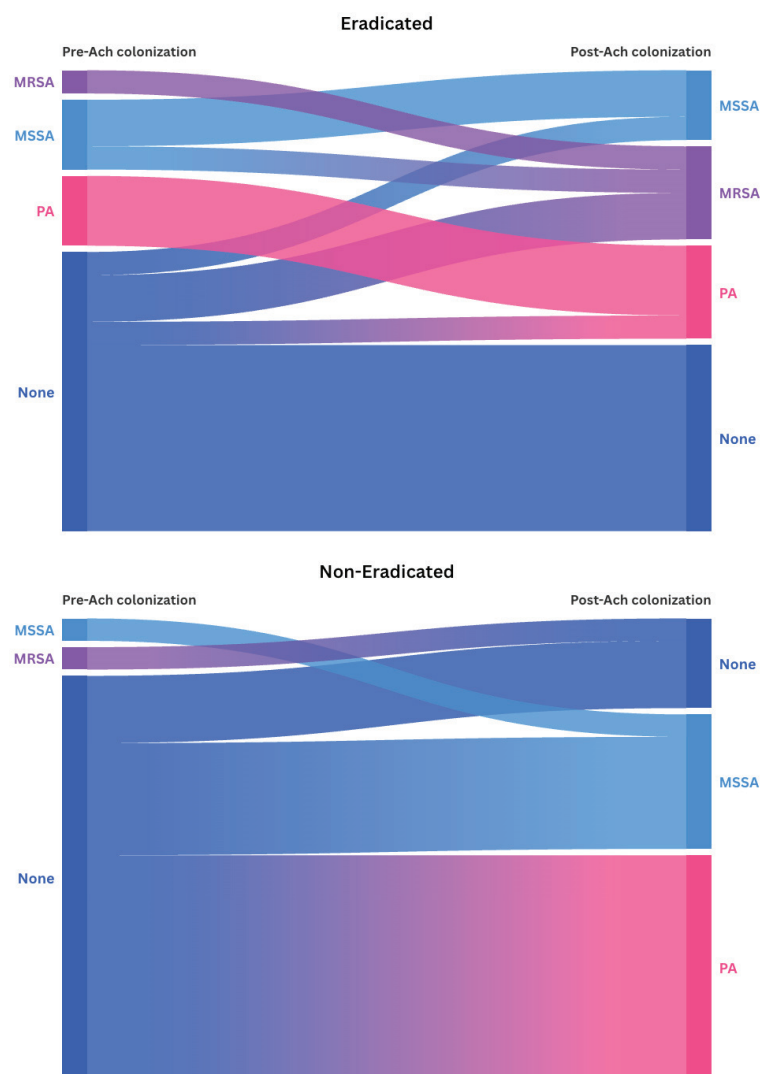


Figure 1. Transitions in airway colonization before and after *Achromobacter* (Ach) isolation stratified by eradication status. Sankey diagrams illustrate changes in airway colonization patterns from pre- to post-Ach isolation in the eradicated (top panel) and non-eradicated (bottom panel) groups. Colonization categories include no bacterial isolation (none), methicillin-susceptible *Staphylococcus aureus* (MSSA), methicillin-resistant *Staphylococcus aureus* (MRSA), and *Pseudomonas aeruginosa* (PA). In the eradicated group, the pre-Ach categories were none (n = 12), MSSA (n = 3), MRSA (n = 1), and PA (n = 3), while the post-Ach categories were none (n = 8), MSSA (n = 3), MRSA (n = 4), and PA (n = 4). In the non-eradicated group, the pre-Ach categories were none (n = 18), MSSA (n = 1), and MRSA (n = 1), while the post-Ach categories were none (n = 4), MSSA (n = 6), and PA (n = 10). The width of each flow represents the number of individuals transitioning between colonization states

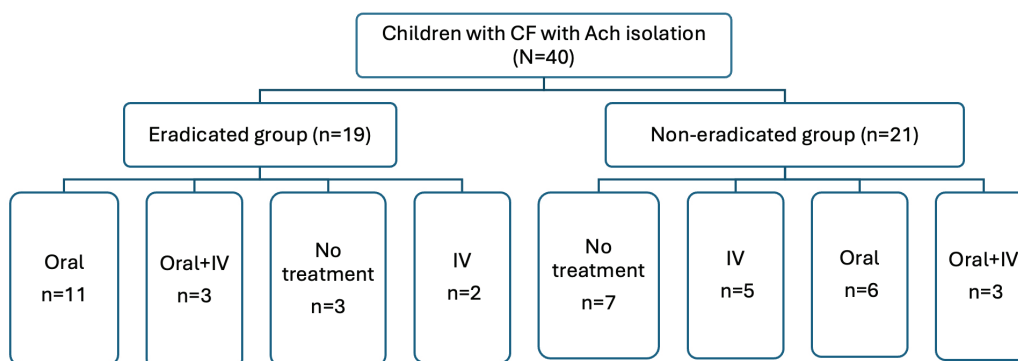


Figure 2. Antibiotic treatment regimens in eradicated and non-eradicated *Achromobacter* infection

CF: cystic fibrosis, Ach: *Achromobacter*, IV: intravenous

that Ach isolation is associated with a more rapid decline in lung function in cwCF. Although baseline pulmonary function tests and decreases in FEV₁ were not different between the groups, the pre-Ach Pex number was higher in the non-eradicated group, similar to the pediatric study by Marsac et al.²⁷ They observed that before the initial Ach isolation, patients had more Pex, hospitalizations, IV, and oral antibiotic courses than controls. In our study, FEV₁ z-scores declined over the 12 months following Ach detection in both groups; however, these changes did not reach statistical significance. While this short-term follow-up did not demonstrate a clear difference in lung function trajectories by eradication status, the higher burden of Pex observed in the non-eradicated group may indicate a subgroup at risk for more pronounced functional decline over longer observation periods.

In this study, we found that colonization status changed over time among patients in whom Ach could not be eradicated. The observed changes in Pa colonization patterns following detection of Ach highlight a potential interaction between Ach and other airway pathogens in the CF airway. This result is similar to that of a case-control study conducted at two pediatric CF centers in France, which observed an increased colonization with Pa within 2 years following Ach colonization.²⁷ Studies have shown that co-infection with Pa + Ach is significantly associated with lower lung function and BMI across all age groups.²⁴ Additionally, Ach, after its initial colonization of the respiratory tract, induces neutrophilia, cytokine production, and inflammatory responses. We speculate that Ach may create a microenvironment conducive to opportunistic pathogens.²⁸

In our study, the non-eradicated group had a higher percentage of azithromycin use in the year prior to initial Ach isolation. The mean age and the number of Pex were higher in this group. Although not statistically significant, the FEV₁ z-score was lower. These clinical factors were indicative of a worse health status that led to treatment with azithromycin. Tan et al.²⁹ argued that intensive antibiotic usage, such as azithromycin, may be a risk factor for the development of opportunistic infections. In contrast, Cogen et al.³⁰ stated that long-term use of azithromycin does not increase opportunistic respiratory pathogens in pwCF.

Poor nutritional status in pwCF leads to declines in pulmonary function, more frequent Pex, and increased mortality.^{31,32} Studies have demonstrated that pwCF with Ach colonization have lower BMI z-scores than those without Ach colonization.^{24,33} We compared the baseline BMI z-scores between the eradicated and non-eradicated groups and found that both groups had low scores. This result may be attributed to challenges accessing adequate nutritional resources for cwCF in our country, independent of microbiological isolation.³⁴

Considering the lack of a significant difference in BMI z-scores at the 12th month follow-up between the eradicated and non-eradicated groups, our findings suggest that, in the short-term, Ach alone does not appear to adversely affect the nutritional status. It is crucial to improve the nutritional status of cwCF, regardless of the presence of a specific pathogen.

There are no standardized eradication treatment protocols. Clinicians evaluate each pwCF individually, considering *in vitro*

antimicrobial susceptibility reports and post-treatment clinical outcomes.³⁵ To our knowledge, two studies have evaluated treatment outcomes after Ach isolation in pwCF.^{36,37} In a study conducted by Wang et al.³⁶, meropenem, piperacillin-tazobactam, and trimethoprim-sulfamethoxazole were highly effective against Ach. Additionally, it was concluded that ceftazidime, colistin, and tobramycin were sufficient for inhalation therapy. In a study conducted in the microbiology laboratory of our hospital that presented antimicrobial susceptibility data for all isolates of Ach, we observed high resistance rates to piperacillin-tazobactam, meropenem, and trimethoprim-sulfamethoxazole.³⁸ In our study, eradication treatments were planned based on antibiogram results. Although the number of uses of meropenem + colistin and trimethoprim-sulfamethoxazole was higher in our study, this difference was not statistically significant. Nebulized antibiotics could not be used for Ach due to reimbursement issues.

Some argue that chronic use of inhaled antibiotics in pwCF may lead to the development of multidrug resistance; however, adding inhaled antibiotics to the eradication protocol can prevent chronic colonization by pathogens, including Ach.³⁹ Therefore, the likelihood of Ach colonization may decrease, thereby potentially reducing chronic nebulized antibiotics. The traditional approach to eradicating Ach in pwCF involves the use of oral and IV agents, depending on the individuals' clinical status, and our study also included the administration of oral and/or IV antibiotics for Ach.⁴⁰ No significant differences were found in oral and/or IV antibiotic use. In the eradicated group, spontaneous eradication of Ach in three cwCF suggested that these cases might involve non-pathogenic strains of Ach.

This study is significant because it assesses the clinical features of Ach in cwCF. The absence of cwCF using CFTR modulators in this cohort underscores the relevance of this research for countries where access to these therapies remains limited, highlighting the risk of opportunistic infections. Furthermore, the study advocates heightened clinical awareness of Ach in cwCF experiencing frequent Pex and suggests that eradication should be considered when Ach is identified.

Study Limitations

Our study has several limitations. This single-center retrospective study included a limited number of cwCF. Although microbial species differentiation of Ach could not be performed, the eradication success rate was high.

CONCLUSION

In this pediatric CF cohort from the pre-CFTR modulator era, non-eradicated Ach was associated with older age at first isolation, a higher Pex burden in the preceding year, and significant shifts in airway microbiology over 12 months, including increased Pa colonization. Although 12-month lung function did not differ by eradication status among those with available spirometry, the observed microbiological transitions underscore the need for careful clinical and microbiological follow-up after the first Ach isolation, especially in children with frequent exacerbations.

Ethics

Ethics Committee Approval: Official approval for the study was received from the Marmara University Ethics Committee (protocol no: 09.2024.30, decision date: 12.01.2024).

Informed Consent: Given the retrospective study design and the use of fully anonymized patient data, written informed consent was not required and therefore not obtained in accordance with the ethics committee's decision.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ş.K., M.S.B., M.Y.K., A.G., M.Y., C.Y.Y., A.P.E., Ö.D., E.E.E., Y.G., A.K., F.K., B.K., Concept: Ş.K., M.S.B., A.P.E., B.U., G.T., Ö.D., E.E.E., Y.G., A.K., F.K., B.K., Design: Ş.K., M.S.B., A.P.E., Ö.D., E.E.E., Y.G., A.K., F.K., B.K., Data Collection or Processing: Ş.K., M.S.B., M.Y.K., A.G., M.Y., C.Y.Y., A.P.E., B.U., G.T., Ö.D., Analysis or Interpretation: Ş.K., M.S.B., A.P.E., E.E.E., Y.G., A.K., Literature Search: Ş.K., M.S.B., M.Y.K., A.G., M.Y., C.Y.Y., A.P.E., Y.G., A.K., F.K., B.K., Writing: Ş.K., Y.G., A.K., F.K., B.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

- Amoureux L, Sauge J, Sarret B, et al. Study of 109 *Achromobacter* spp. isolates from 9 French CF centres reveals the circulation of a multiresistant clone of *A. xylosoxidans* belonging to ST 137. *J Cyst Fibros*. 2019;18(6):804-807. [\[Crossref\]](#)
- De Baets F, Schelstraete P, Van Daele S, Haerynck F, Vanechoutte M. *Achromobacter xylosoxidans* in cystic fibrosis: prevalence and clinical relevance. *J Cyst Fibros*. 2007;6(1):75-78. [\[Crossref\]](#)
- Esposito S, Pisi G, Fainardi V, Principi N. What is the role of *Achromobacter* species in patients with cystic fibrosis? *Front Biosci (Landmark Ed)*. 2021;26(12):1613-1620. [\[Crossref\]](#)
- Saiman L, Siegel JD, LiPuma JJ, et al. Infection prevention and control guideline for cystic fibrosis: 2013 update. *Infect Control Hosp Epidemiol*. 2014;35 (Suppl 1):S1-S67. [\[Crossref\]](#)
- LiPuma JJ. The changing microbial epidemiology in cystic fibrosis. *Clin Microbiol Rev*. 2010;23(2):299-323. [\[Crossref\]](#)
- Wormser GP, Stratton C. Manual of Clinical Microbiology, 9th Edition. *Clin Infect Dis*. 2008;46(1):153-154. [\[Crossref\]](#)
- Cystic Fibrosis Foundation Patient Registry Highlights. Bethesda, Maryland: Cystic Fibrosis Foundation; 2023. Accessed August 13, 2026. [\[Crossref\]](#)
- Zolin A, Jung A, van Rens J. ECFSPR Annual Report 2021. European Cystic Fibrosis Society Patient Registry; 2023. Accessed August 13, 2026. [\[Crossref\]](#)
- Foundation CF. 2024 Cystic Fibrosis Foundation Patient Registry Highlights. Bethesda, Maryland, 2025. Accessed August 13, 2026. [\[Crossref\]](#)
- Zolin A, Anagnostopoulou A, Bakkeheim E, et al. ECFSPR Annual Report 2023. European Cystic Fibrosis Society Patient Registry; 2025. Accessed August 13, 2026. [\[Crossref\]](#)
- Pollak M, Gambazza S, Orenti A, et al. Respiratory infections after elexacaftor/tezacaftor/ivacaftor treatment in people with cystic fibrosis: analysis of the European Cystic Fibrosis Society Patient Registry. *ERJ Open Res*. 2025;11(4):01248-02024. [\[Crossref\]](#)
- Ergenekon AP, Selcuk M, Unal G, et al. Comparison of interrupted and continuous modulator therapy in cystic fibrosis: a real life experience in Turkey. *Front Pediatr*. 2025;13:1659633. [\[Crossref\]](#)
- Lambiase A, Catania MR, Del Pezzo M, et al. *Achromobacter xylosoxidans* respiratory tract infection in cystic fibrosis patients. *Eur J Clin Microbiol Infect Dis*. 2011;30(8):973-980. [\[Crossref\]](#)
- Magni A, Trancassini M, Varesi P, et al. *Achromobacter xylosoxidans* genomic characterization and correlation of randomly amplified polymorphic DNA profiles with relevant clinical features [corrected] of cystic fibrosis patients. *J Clin Microbiol*. 2010;48(4):1035-1039. [\[Crossref\]](#)
- Ng C, Nadig T, Smyth AR, Flume P. Treatment of pulmonary exacerbations in cystic fibrosis. *Curr Opin Pulm Med*. 2020;26(6):679-684. [\[Crossref\]](#)
- Stevens DA, Moss RB, Kurup VP, et al. Allergic bronchopulmonary aspergillosis in cystic fibrosis--state of the art: Cystic Fibrosis Foundation Consensus Conference. *Clin Infect Dis*. 2003;37 (Suppl 3):S225-S264. [\[Crossref\]](#)
- Moran A, Hardin D, Rodman D, et al. Diagnosis, screening and management of cystic fibrosis related diabetes mellitus: a consensus conference report. *Diabetes Res Clin Pract*. 1999;45(1):61-73. [\[Crossref\]](#)
- Naito Y, Cystic Fibrosis Trust, Clarke S. UK Cystic Fibrosis Registry 2022 Annual Data Report. Cystic Fibrosis Trust; 2023. Accessed August 13, 2026. [\[Crossref\]](#)
- Farrell PM, White TB, Ren CL, et al. Diagnosis of Cystic Fibrosis: Consensus Guidelines from the Cystic Fibrosis Foundation. *J Pediatr*. 2017;181S:S4-S15.e1. [\[Crossref\]](#)
- Lee TW, Brownlee KG, Conway SP, Denton M, Littlewood JM. Evaluation of a new definition for chronic *Pseudomonas aeruginosa* infection in cystic fibrosis patients. *J Cyst Fibros*. 2003;2(1):29-34. [\[Crossref\]](#)
- Stanojevic S, Kaminsky DA, Miller MR, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. *Eur Respir J*. 2022;60(1):2101499. [\[Crossref\]](#)
- European Committee on Antimicrobial Susceptibility Testing (EUCAST). Accessed August 13, 2026. [\[Crossref\]](#)
- Raso T, Bianco O, Grosso B, Zucca M, Savoia D. *Achromobacter xylosoxidans* respiratory tract infections in cystic fibrosis patients. *APMIS*. 2008;116(9):837-841. [\[Crossref\]](#)
- Kerem E, Orenti A, Zolin A, Annicchiarico L, Drevinek P; ECFSPR with the list of contributing authors. Clinical outcomes associated with *Achromobacter* species infection in people with cystic fibrosis. *J Cyst Fibros*. 2023;22(2):334-343. [\[Crossref\]](#)
- Firmida MC, Pereira RH, Silva EA, Marques EA, Lopes AJ. Clinical impact of *Achromobacter xylosoxidans* colonization/infection in patients with cystic fibrosis. *Braz J Med Biol Res*. 2016;49(4):e5097. [\[Crossref\]](#)
- Sunman B, Emiralioglu N, Hazirolan G, et al. Impact of *Achromobacter* spp. isolation on clinical outcomes in children with cystic fibrosis. *Pediatr Pulmonol*. 2022;57(3):658-666. [\[Crossref\]](#)
- Marsac C, Berdah L, Thouvenin G, Sermet-Gaudelus I, Corvol H. *Achromobacter xylosoxidans* airway infection is associated with lung disease severity in children with cystic fibrosis. *ERJ Open Res*. 2021;7(2):00076-02021. [\[Crossref\]](#)

28. Billiot CE, McDaniel MS, Lindgren NR, Swords WE. Pathogenesis of *Achromobacter xylosoxidans* respiratory infections: colonization and persistence of airway epithelia and differential gene expression in synthetic cystic fibrosis sputum medium. *bioRxiv* [Preprint]. 2023;2023.04.04.535650. [\[Crossref\]](#)
29. Tan K, Conway SP, Brownlee KG, Etherington C, Peckham DG. Alcaligenes infection in cystic fibrosis. *Pediatr Pulmonol*. 2002;34(2):101-104. [\[Crossref\]](#)
30. Cogen JD, Onchiri F, Emerson J, et al. Chronic azithromycin use in cystic fibrosis and risk of treatment-emergent respiratory pathogens. *Ann Am Thorac Soc*. 2018;15(6):702-709. [\[Crossref\]](#)
31. De Boeck K. Cystic fibrosis in the year 2020: a disease with a new face. *Acta Paediatr*. 2020;109(5):893-899. [\[Crossref\]](#)
32. Kerem E, Viviani L, Zolin A, et al. Factors associated with FEV1 decline in cystic fibrosis: analysis of the ECFS patient registry. *Eur Respir J*. 2014;43(1):125-133. [\[Crossref\]](#)
33. Somayaji R, Stanojevic S, Tullis DE, Stephenson AL, Ratjen F, Waters V. Clinical outcomes associated with achromobacter species infection in patients with cystic fibrosis. *Ann Am Thorac Soc*. 2017;14(9):1412-1418. [\[Crossref\]](#)
34. Kocaman D, Yıldız CA, Metin Çakar N, et al. Feeding the need: a study on food security among people with cystic fibrosis in Turkey. *Pediatr Pulmonol*. 2025;60(5):e71101. [\[Crossref\]](#)
35. Díez-Aguilar M, Ekkelenkamp M, Morosini MI, et al. Antimicrobial susceptibility of non-fermenting Gram-negative pathogens isolated from cystic fibrosis patients. *Int J Antimicrob Agents*. 2019;53(1):84-88. [\[Crossref\]](#)
36. Wang M, Ridderberg W, Hansen CR, et al. Early treatment with inhaled antibiotics postpones next occurrence of *Achromobacter* in cystic fibrosis. *J Cyst Fibros*. 2013;12(6):638-643. [\[Crossref\]](#)
37. Llorca Otero L, Girón Moreno R, Buendía Moreno B, Valenzuela C, Guiu Martínez A, Alarcón Caverio T. *Achromobacter xylosoxidans* infection in an adult cystic fibrosis unit in Madrid. *Enferm Infecc Microbiol Clin*. 2016;34(3):184-187. [\[Crossref\]](#)
38. Demirkol Ö, Alçi G, Karadağ B, et al. Retrospective analysis of *Achromobacter* species isolated from cystic fibrosis and non-cystic fibrosis patients. *ANKEM Derg*. 2022;36(3):125-132. [\[Crossref\]](#)
39. Fainardi V, Neglia C, Muscarà M, et al. Multidrug-resistant bacteria in children and adolescents with cystic fibrosis. *Children (Basel)*. 2022;9(9):1330. [\[Crossref\]](#)
40. Rajan S, Saiman L. Pulmonary infections in patients with cystic fibrosis. *Semin Respir Infect*. 2002;17(1):47-56. [\[Crossref\]](#)