


Retrospective Cohort Study on *Delftia acidovorans* Infections in Patients: A Rare and Significant Infection

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Background: In recent years, *Delftia acidovorans* has gained attention for its rare occurrence in patient infections. The literature consists mostly of case reports, necessitating further research to comprehensively understand risk factors, clinical characteristics, and management strategies.

Methods: We conducted a retrospective cohort study involving patients diagnosed with *Delftia acidovorans* infection at a tertiary teaching hospital between January 2014 and December 2022. The data included demographic details, comorbidities, bacterial cultures, antibiotic susceptibility, and treatment outcomes.

Results: There were 26 patients diagnosed with *Delftia acidovorans* infection who were predominantly older with multiple comorbidities. Approximately 76.9% of *Delftia acidovorans* infection patients had polymicrobial infections. Twenty-one patients had received antibiotics within three months before they developed the *Delftia acidovorans* infection, and these antibiotics were primarily third-generation cephalosporins, glycopeptides and fluoroquinolones. Antibiotic susceptibility testing showed resistance to aminoglycosides and susceptibility to imipenem, meropenem, ceftazidime, and piperacillin/tazobactam. Treatment outcome showed a mortality rate of 11.5%, mainly in patients with malignancy and advanced age.

Conclusion: *Delftia acidovorans* infections predominantly affect older patients with multiple comorbidities. In terms of antibiotic therapy, carbapenems, cephalosporins, and piperacillin/tazobactam with antipseudomonal activity could all be considered.

Keywords: *Delftia acidovorans*, antibiotic susceptibility, mortality rate

Introduction

The term “*Delftia*” originates from the city of Delft in the Netherlands, where the type species was first isolated. This nomenclature pays homage to the pioneering contributions of research groups in Delft to the field of bacteriology. In 1987, *Pseudomonas acidovorans* was reclassified as a *Comamonas species* based on phenotypic and chemotaxonomic characters, along with DNA–DNA homology.¹ Later, in 1999, it was renamed *Delftia acidovorans* (*D. acidovorans*) following 16S rRNA gene sequence analysis after being removed from the *Comamonas* genus.² In recent years, the medical community has become increasingly aware of infrequent infections caused by a diverse array of microbial pathogens. Among these pathogens, *D. acidovorans*, characterized as a gram-negative, aerobic, nonfermenting, and nonsporeforming rod-shaped bacterium, is commonly found in soil and water environments. Although initially considered nonpathogenic with limited clinical significance, it has gained attention due to its rare occurrence in patient infections. Beyond its environmental habitat, *D. acidovorans* has been detected in clinical settings, including surgical vacuum manifolds, operating scrub sinks, hemodialysis wall boxes, prime buckets, and reverse osmosis systems. Notably, it exhibits survival within biofilms and displays resistance to various disinfectants, including chlorhexidine.³ The clinical spectrum of these infections spans from ocular, respiratory, or urinary tract infection to bloodstream

infection, peritonitis, and even endocarditis.^{4–9} Despite the relatively low incidence of *D. acidovorans* infections, their impact on affected patients can be substantial.

Currently, the understanding of *D. acidovorans* infection remains limited and is primarily derived from case reports and sporadic retrospective case series studies. Given the paucity of knowledge surrounding the pathogenicity and clinical outcomes associated with *D. acidovorans* infections, further research is imperative to elucidate risk factors, clinical features, and optimal management strategies for affected patients. To bridge this knowledge gap, we embarked on a retrospective cohort study involving patients diagnosed with *D. acidovorans* infection at a tertiary teaching hospital between 2014 and 2022. The aim of the study is to elucidate the clinical characteristics and outcomes of these infections. Through a comprehensive analysis of diverse case data, the populations most susceptible to these infections were identified, and the efficacy of the current treatment modalities was evaluated.

Methods

Study Design

We undertook a retrospective investigation involving individuals diagnosed with *D. acidovorans* infection at a tertiary teaching hospital, during the period from January 2014 to December 2022. The patient cohort included those with confirmed *D. acidovorans* infection based on positive culture reports, coupled with the presence of clinically relevant signs and symptoms associated with the infection.

Data Collection

We procured patient data and pertinent clinical information regarding confirmed *D. acidovorans* infections through the utilization of microbiology laboratory reporting system, clinical information and medical record repository. The data collection encompassed a spectrum of aspects, including demographics, existing comorbidities, antibiotic treatments administered within the preceding 3 months, results of bacterial cultures and antibiotic susceptibility, the trajectory of hospitalization, and ultimate clinical outcomes.

Antibiotics Susceptibility

Using the Clinical & Laboratory Standards Institute's M100–25 interpretative criteria, antibiotic susceptibility was assessed using the VITEK[®] II system and VITEK[®] II Gram Negative Susceptibility cards (bioMérieux, Marcy-l'Étoile, France). The transfer tube and matching suspension tube were positioned in the adjacent slots, together with one Gram-negative identification card and another VITEK II AST-N322 card (for susceptibility testing of aerobic Gram-negative bacilli against selected antimicrobials).¹⁰ Our microbiology laboratory susceptibility test report for *D. acidovorans* included amikacin, gentamicin, ciprofloxacin, ceftazidime, cefepime, imipenem, meropenem, and piperacillin/tazobactam.

Ethics and Consent Protocol of the Study

This study was conducted in accordance with the Declaration of Helsinki. Ethical considerations were rigorously evaluated and approved by the Dalin Tzu Chi Hospital Research Ethics Committee (Approval IRB No. B11203013). The study was a retrospective medical records data collection and analysis. The risk and degree of harm or discomfort were no greater than in everyday life, a normal physical examination, or a psychiatric assessment. The legal biological database cannot identify a specific person. It is unable to identify specific individuals' data, files, information, or samples for research purposes. The study had the lowest risk, and the potential harm to the research subject was not greater than that of the nonparticipating researchers, and the researcher's rights were not affected by the exemption from prior consent. The research risk did not surpass the minimum risk. The subject's rights and well-being were unaffected by the exempt informed consent. Research could not be performed without exempting informed consent. Informed consent statement is not applicable. No individual patient cases are described in the manuscript. No written informed consent is required.

Results

Our study focused on *D. acidovorans* infection and involved a total of 26 patients between January 2014 and December 2022. The patient characteristics and comorbidities are presented in Tables 1 and S1–S5. Notably, the mean age of the patients at the time of infection was 73 years, indicating that this infection primarily affected older individuals. Out of the 26 patients, 16 were male and 10 were female. The average number of comorbidities, 2.3, indicates that each patient with a *D. acidovorans* infection had on average two or more concurrent medical conditions. These comorbidities encompassed a wide range of conditions, including cardiovascular diseases, malignancies, diabetes mellitus, cerebrovascular diseases, chronic kidney disease or end-stage renal disease, chronic obstructive pulmonary disease, autoimmune rheumatic diseases, and hepatitis B or C.

The distribution of *D. acidovorans* infections among the patient cohort is presented in Table 2. Among the varied clinical presentations, pneumonia was the most prevalent condition, impacting 10 patients. Urinary tract infections were the second most prevalent, with nine cases, followed by primary bacteremia, which was four cases. Furthermore, a noteworthy pattern emerged, as 76.9% of *D. acidovorans* infections were accompanied by coexisting microorganisms, as shown in Table S6. *Escherichia coli* (five cases), *Pseudomonas aeruginosa* (four cases), and *Stenotrophomonas maltophilia* (three cases) were the predominant companions. Out of these cases, 21 individuals (80.8%) had undergone antibiotic therapy preceding 3 months, as indicated in Table 3. The comprehensive analysis of the antibiotic susceptibility patterns is presented in Table 4. Our findings reveal significant resistance rates, particularly within the aminoglycosides

Table 1 Clinical Characteristics and Comorbidities in Twenty-Six Cases of *Delftia acidovorans* Infection

	Male	Female	Total Patients
Case number (%)	16 (61.5%)	10 (38.5%)	26
Age at the time of infection, Mean (min, max)	71.4 (51, 89)	76.1 (37, 90)	73.2 (37, 90)
Intensive care unit hospitalization, Case number (%)	4 (25.0%)	4 (40.0%)	8 (30.7%)
Cured, Case number (%)	14 (87.5%)	9 (90.0%)	23 (88.5%)
Deceased, Case number (%)	2 (12.5%)	1 (10.0%)	3 (11.5%)
Comorbidities, Case number (%)	15 (93.7%)	9 (90.0%)	24 (92.3%)
Cardiovascular diseases	9	7	16
Malignancies	8	3	11
Diabetes mellitus	7	3	10
Cerebrovascular diseases	5	2	7
Chronic obstructive pulmonary disease	5	1	6
Chronic kidney disease or End-stage renal disease	2	2	4
Autoimmune rheumatic diseases	0	3	3
Hepatitis B or C	3	0	3
Total Comorbidity number, (average)	39 (2.4)	21 (2.1)	60 (2.30) *

Notes: *Patients may have more than one comorbidity. Additionally, among the patients, there is one male and one female without any comorbidity.

Table 2 Distribution of *Delftia acidovorans* Infections Cases

Clinical Presentation	Case Number (%)
Pneumonia	10 (38.5%)
Urinary tract infection	9 (34.6%)
Bacteremia	4 (15.4%)
Peritonitis	2 (7.7%)
Liver abscess	1 (3.8%)
Total patients	26 (100%)

Table 3 Prior Intravenous Antibiotic Use Within 3 Months in Twenty-One Cases of *Delftia acidovorans* Infection

Types of Antibiotics	Case Number*
Third generation cephalosporin [#]	16
Glycopeptides (Vancomycin, Teicoplanin)	10
Quinolones (Levofloxacin, Ciprofloxacin)	10
Carbapenems (Ertapenem, Imipenem, Meropenem)	5
Piperacillin/tazobactam	2
Colistin	2
Aminoglycoside (Amikacin)	1

Notes: *Patients had received one or more types of antibiotics within the 3 months before *Delftia acidovorans* infection was identified. [#]Third generation cephalosporin included Flomoxef, Ceftriaxone, and Cefazidime.

Table 4 Antimicrobial Susceptibility to *Delftia acidovorans* Among Three Cohorts Patients

	AMK	GEN	CIP	CAZ	FEP	IMI	MEM	TZP
Our Cohort								
Susceptible number	1	1	7	22	18	23	22	21
Resistant number	24	24	17	3	6	1	2	3
Susceptible rate (%)	4.0%	4.0%	29.2%	88.0%	75.0%	95.8%	91.7%	87.5%
Højgaard et al								
Susceptible rate (%)	N	7.7%	90.7%	94.4%	N	94.2%	94.6%	94.0%
Other cases cohort								
Susceptible rate (%)	17.2%	13.8%	80.0%	76.0%	25.0%	100.0%	90.9%	84.6%

Abbreviations: N, No data; AMK, Amikacin; GEN, Gentamycin; CIP, Ciprofloxacin; CAZ, Ceftazidime; FEP, Cefepime; IMI, Imipenem; MEM, Meropenem; TZP, Piperacillin/tazobactam.

and ciprofloxacin. In contrast, our study found that carbapenems, specifically imipenem and meropenem, demonstrated susceptibility rates of 95.8% and 91.7%, respectively. Furthermore, the isolates were susceptible to ceftazidime (88.0%), piperacillin/tazobactam (87.5%), and cefepime (75.0%).

The treatment outcomes are presented in Table 1, revealing that 23 of the 26 patients (88.5%) achieved a successful cure. Notably, among these patients, four were treated with imipenem, while 12 received 3rd- or 4th-generation cephalosporins (including eight patients treated with ceftazidime, three with cefepime, and one with ceftibuten). Additionally, four patients were treated with penicillin agents, comprising one piperacillin/tazobactam, two with ampicillin/sulbactam, and one with amoxicillin/clavulanate. An intriguing observation is the successful recovery of three outpatients with positive urine cultures of *D. acidovorans* without documented antibiotic therapy.

Regrettably, three patients succumbed to the infection despite receiving antibiotic therapy, including one case of urinary tract infection, one case of pneumonia, and one case of primary bacteremia. All of the three patients had underlying malignant disease (one case of pancreatic cancer, one case of colon cancer, and one case of rectal cancer). A distinctive observation within our cohort pertains to a hepatocellular carcinoma patient developed a polymicrobial liver abscess, including *D. acidovorans*, *Aeromonas caviae* and *Escherichia coli*. This underscores the potential for this microorganism to be involved in unique and atypical clinical scenarios.

Discussion

Characteristics of the Patients with *Delftia acidovorans* Infections

We conducted a systematic review of *D. acidovorans* infection cases based on previously reported cases in the literature from January 1990 to June 2023. To ensure the integrity of our analysis and to prevent the inclusion of potentially misleading information, we excluded papers that were unavailable, not in English, or lacked patient data. Our effort

yielded a collection of 40 sporadically reported cases of *D. acidovorans* infections (excluding Højgaard et al's study). Among these cases, the spectrum of infections included 16 cases of bacteremia,^{7,11–23} eleven ocular infections (including three patients with positive contact lens cultures),^{4,24–27} two pneumonia cases,^{5,28} three empyema cases,^{29–31} one urinary tract infection,⁶ one combined bacteremia and urinary tract infection,³² three cases of peritonitis in patients undergoing continuous ambulatory peritoneal dialysis^{8,33,34}, two endocarditis,^{9,35} and one skin infection.³⁶ Regrettably, our analysis also revealed three cases resulting in mortality. The first case was in 1990. Horowitz et al reported a 42-year-old patient with alcoholic liver disease and intravenous drug abuse. Two weeks prior, she received intravenous ampicillin therapy for viridans streptococcal endocarditis. The chest radiograph demonstrated ill-defined densities in the right lobe, and the blood culture was positive for *Pseudomonas species*. Further biochemical testing revealed the organism to be *D. acidovorans*, which was susceptible to ceftazidime and resistant to amikacin and gentamicin. The clinical course progressively deteriorated despite antibiotic therapy. She died on hospital day 25 with progressive multisystem failure.⁹ The second case was reported by Singh et al a 29-year-old patient underwent chemotherapy for breast cancer. After 10 days of the last dose of chemotherapy, she presented with pancytopenia, pneumonia, and bacteremia. A blood sample was obtained from peripheral and central lines, and the isolates were identified as *D. acidovorans*. The isolates were susceptible to ceftazidime, piperacillin-tazobactam, meropenem, levofloxacin and aztreonam but resistant to amikacin and colistin. Antibiotic therapy with meropenem was continued for 4 days. She developed respiratory failure and bradycardia. She was declared dead.²¹ The third case was reported by Khan et al, involving a 4-year-old child who presented with fever, cough, and respiratory distress. The patient had no preexisting comorbidities. A chest X-ray revealed fluid accumulation on the right side of the chest. Antibiotic treatment with ceftazidime and amikacin was initiated. An intercostal drainage tube was inserted, and cultures of pleural effusion and sputum identified *D. acidovorans*. Notably, the isolate showed resistance to gentamicin and ceftazidime. Although the antibiotic regimen was subsequently changed to cefoperazone-sulbactam based on a susceptible profile, the patient's condition deteriorated, leading to septic shock and eventual death on the 12th day of admission.³⁰ This observation resonates with our cohort, where the deceased patient also had a preexisting comorbidity. Notably, Bilgin et al have previously emphasized the rarity of *D. acidovorans* infections, highlighting their potential to cause severe and even fatal infections in individuals with compromised immune systems, malignancies, or those undergoing immunosuppressive therapy.⁵

The most recent and comprehensive cohort study, conducted by Højgaard et al, has significantly contributed to our understanding of *D. acidovorans* infections.³⁷ This study encompassed 59 patients between 2002 and 2020 at a tertiary referral center in Denmark, representing the largest cohort to date. Within these patients, a sex distribution of 35 male patients (59%) and a median age of 47 years at the time of infection were observed. Notably, the high prevalence of preexisting comorbidities was evident in 57 cases (97%), including conditions such as malignancies (N=25) and cardiovascular diseases (N=12). The study suggested that blood (N=17) and airway secretions (N=20) constituted the predominant sources of infected specimens. Among these specimens, a substantial proportion (70%) exhibited polymicrobial cultures, frequently involving *Pseudomonas spp.* (N=12) and *Stenotrophomonas maltophilia* (N=10). Four cases resulted in mortality within 30 days. An important observation is the correlation between the fatal cases and polymicrobial cultures, implicating the potential impact of this aspect on clinical outcomes. It is crucial to acknowledge that only 35 patients out of the total 59 provided comprehensive data on prior antibiotic therapy, revealing that 25 of them had undergone antibiotic treatment within three months prior to their initial positive *D. acidovorans* culture. Penicillins and fluoroquinolones were the predominant antibiotics employed.

Comparing our cohort study to Højgaard et al's findings, our patient population demonstrated an older age distribution, with a median age at infection onset of 77 years; 76% were aged over 65 years; and no pediatric cases were included. Similarly, both cohorts exhibited a high prevalence of preexisting comorbidities, especially malignancies. The concurrent presence of multiple comorbidities highlights the considerable burden of preexisting health conditions carried by infected patients. This situation could impact their health and potentially affect the outcomes of their treatment. The dominant infected specimens in our study were airway secretions and urine. We observed a comparable ratio of polymicrobial cultures in our cohort, with 76.9% of the specimens indicating polymicrobial involvement, often including *Escherichia coli*, *Pseudomonas aeruginosa*, and *Stenotrophomonas maltophilia*. Differing from the Højgaard et al study, our investigation encompassed comprehensive antibiotic therapy histories for all cases. Among the 26 patients within our

cohort, 80.8% had undergone antibiotic treatment within the three months preceding the *D. acidovorans* infection, predominantly involving 3rd generation cephalosporins, followed by glycopeptides and fluoroquinolones. This observation suggests a possible connection between exposure to these antibiotics beforehand and the development or continuation of *D. acidovorans* infections in the group of patients being studied. Looking closely at the specific patterns of antibiotic usage before *D. acidovorans* was detected can provide valuable insights into potential factors that contribute to the risk, thus guiding future treatment strategies.

Antimicrobial Susceptibility Pattern of *Delftia acidovorans*

In the context of antibiotic susceptibility, a comprehensive analysis of 40 sporadically cases was conducted, and it has been consistently observed that *D. acidovorans* infections exhibit susceptibility to certain antibiotics, such as ciprofloxacin, ceftazidime, carbapenems (imipenem and meropenem), and piperacillin/tazobactam. Notably, a significant pattern emerges, with most case reports indicating resistance to aminoglycosides such as amikacin, and gentamicin (Table 4). The analysis of antibiotic susceptibility in Højgaard et al's study revealed that a substantial proportion of the isolates exhibited susceptibility to meropenem (94.6%), imipenem (94.2%), ceftazidime (94.4%), ciprofloxacin (90.7%), and piperacillin/tazobactam (94.0%). Conversely, higher resistance rates were observed for gentamicin (92.3%).³⁷

Our observations pertaining to antibiotic susceptibility closely paralleled those outlined in Højgaard et al's work, with over 85% of isolates exhibiting susceptibility to meropenem, imipenem, ceftazidime, and piperacillin-tazobactam and a 75% susceptibility rate for cefepime. Our study showed that the isolates had a high rate of resistance to aminoglycosides (96%). The susceptibility rate of ciprofloxacin was 29.2% in our study, and the susceptibility rate of ciprofloxacin was 90.7% in Højgaard et al's study, which was the major difference between the two studies. Camargo et al demonstrated their findings by studying *D. acidovorans* isolates isolated from tracheal aspirate samples of 21 adult inpatients in a Brazilian hospital. The susceptibility rate of ciprofloxacin was 16.7%. The isolates showed resistance to polymyxin B and aminoglycosides (such as amikacin, gentamicin, and tobramycin), but no β -lactam resistance was found.³⁸ These researches revealed both parallels and variations in the clinical and microbiological features of *D. acidovorans* infections across countries and patient groups.

Regarding the phenomenon of β -lactam antibiotic resistance, Ravaoarinoro et al conducted an insightful investigation into the underlying mechanisms within *D. acidovorans* strains. Their study elucidated a significant factor contributing to the resistance of *D. acidovorans* to β -lactam antibiotics, namely, the synergistic interplay between the production of β -lactamase enzymes and structural alterations in the outer membrane proteins. Furthermore, their findings highlighted the correlation between the relative hydrophilicity of *D. acidovorans* strains and their susceptibility to β -lactam antibiotics. Notably, strains exhibiting higher hydrophilicity were discernibly more resistant to β -lactam antibiotics than those with higher hydrophobicity.³⁹

Kang et al made a significant observation, noting the intrinsic resistance of *Delftia* species to aminoglycoside and quinolone antibiotics. This intrinsic resistance could signify an evolutionary adaptation toward resistance against a broader spectrum of antibiotics, further emphasizing the dynamic nature of bacterial antibiotic resistance mechanisms.⁴⁰ These scientific insights collectively contribute to a deeper understanding of the intricate factors governing antibiotic resistance patterns and mechanisms within *D. acidovorans* and related species.

In alignment with our cohort study, we identified analogous findings, revealing a notable trend of elevated resistance to amikacin and gentamicin. Furthermore, resistance to ciprofloxacin was also observed, while a consistent and substantial susceptibility was noted toward ceftazidime, meropenem, imipenem, and piperacillin/tazobactam. This complexity emphasizes the necessity for an in-depth exploration into the intricate genetic, molecular, and environmental determinants that contribute to these noticeable patterns. Such investigations hold the potential to enhance our comprehension of the underlying mechanisms and further illuminate avenues for comprehensive research in this domain.

Furthermore, Højgaard et al identified four patients (including 2 children) who met the criteria for persistent infection or colonization. The specimens in these cases included wound discharge, airway secretions, and blood, with one patient ultimately succumbing to the infection.³⁷ Notably, in our cohort, observation emerged involving three outpatients who exhibited positive polymicrobial urine cultures but spontaneously recovered without any documented antibiotic therapy.

These observations reveal the potential for spontaneous resolution or bacterial colonization without eliciting clinical symptom.

Limitation

Only limited clinical experience can be obtained from our tiny case series retrospective study of *D. acidovorans* infections. Clinical characteristics and treatment strategies for *D. acidovorans* infections will be improved by an additional 40 sporadically reported cases and Højgaard et al's study of *D. acidovorans* infections.

Conclusions

Our retrospective analysis showed that *D. acidovorans* infections predominantly affect older patients with multiple comorbidities, such as cardiovascular diseases, and malignancies. Most *Delftia acidovorans* infection patients had polymicrobial infections and had received antibiotics within three months before they developed the *Delftia acidovorans* infection. In terms of antibiotic therapy, carbapenems, cephalosporins, and piperacillin/tazobactam with antipseudomonal activity could all be considered. These findings have the potential to significantly enhance our understanding of *D. acidovorans* infections.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests in this work.

References

1. Tamaoka J, Ha D-M, Komagata K. Reclassification of *Pseudomonas acidovorans* den Dooren de Jong 1926 and *Pseudomonas testosteroni* Marcus and Talalay 1956 as *Comamonas acidovorans* comb. nov. and *Comamonas testosteroni* comb. nov. with an Emended Description of the Genus *Comamonas*. *Int J Syst Evol*. 1987;37:52–59. doi:10.1099/00207713-37-1-52
2. Wen A, Fegan M, Hayward C, et al. Phylogenetic relationships among members of the Comamonadaceae, and description of *Delftia acidovorans* (den Dooren de Jong 1926 and Tamaoka et al. 1987) gen. nov. comb. nov. *Int J Syst Bacteriol*. 1999;49(Pt 2):567–576. doi:10.1099/00207713-49-2-567
3. Rema T, Lawrence JR, Dynes JJ, et al. Microscopic and spectroscopic analyses of chlorhexidine tolerance in *Delftia acidovorans* biofilms. *Antimicrob Agents Chemother*. 2014;58(10):5673–5686. doi:10.1128/AAC.02984-14
4. Stonecipher KG, Jensen HG, Kastl PR, Faulkner A, Rowsey JJ. Ocular infections associated with *Comamonas acidovorans*. *Am J Ophthalmol*. 1991;112(1):46–49. doi:10.1016/s0002-9394(14)76211-7
5. Bilgin H, Sarmis A, Tigen E, et al. *Delftia acidovorans*: a rare pathogen in immunocompetent and immunocompromised patients. *Can J Infect Dis Med Microbiol*. 2015;26(5):277–279. doi:10.1155/2015/973284
6. Del Mar Ojeda-Vargas M, Suárez-Alonso A, de Los Angeles Pérez-Cervantes M, et al. Urinary tract infection associated with *Comamonas acidovorans*. *Clin Microbiol Infect*. 1999;5(7):443–444. doi:10.1111/j.1469-0691.1999.tb00170.x
7. Ender PT, Dooley DP, Moore RH. Vascular catheter-related *Comamonas acidovorans* bacteremia managed with preservation of the catheter. *Pediatr Infect Dis J*. 1996;15(10):918–920. doi:10.1097/00006454-199610000-00021
8. López-Mencherero R, Sigüenza F, Caridad A, et al. Peritonitis due to *comamonas acidovorans* in a CAPD patient. *Perit Dial Int*. 1998;18(4):445–446. doi:10.1177/089686089801800420
9. Horowitz H, Gilroy S, Feinstein S, et al. Endocarditis associated with *Comamonas acidovorans*. *J Clin Microbiol*. 1990;28(1):143–145. doi:10.1128/jcm.28.1.143-145.1990
10. Huang C. Extensively drug-resistant *Alcaligenes faecalis* infection. *BMC Infect Dis*. 2020;20(1):833. doi:10.1186/s12879-020-05557-8

11. Castagnola E, Tasso L, Conte M, et al. Central venous catheter-related infection due to *Comamonas acidovorans* in a child with non-Hodgkin's lymphoma. *Clin Infect Dis*. 1994;19(3):559–560. doi:10.1093/clinids/19.3.559-a
12. Lair MI, Bentolila S, Grenet D, et al. *Oerskovia turbata* and *Comamonas acidovorans* bacteremia in a patient with AIDS. *Eur J Clin Microbiol Infect Dis*. 1996;15(5):424–426. doi:10.1007/BF01690106
13. Perla RJ, Knutson EL. *Delftia acidovorans* bacteremia in an intravenous drug abuser. *Am J Infect Dis*. 2005;1(2):73–74. doi:10.3844/ajidsp.2005.73.74
14. Oliver JW, Stapenhorst D, Warraich I, et al. *Ochrobactrum anthropi* and *Delftia acidovorans* to bacteremia in a patient with a gunshot wound. *Infect Dis Clin Pract*. 2005;13(2):78–81. doi:10.1097/01.idc.0000155840.49633.f6
15. Kawamura I, Yagi T, Hatakeyama K, et al. Recurrent vascular catheter-related bacteremia caused by *Delftia acidovorans* with different antimicrobial susceptibility profiles. *J Infect Chemother*. 2011;17(1):111–113. doi:10.1007/s10156-010-0089-x
16. Chotikanatis K, Bäcker M, Rosas-Garcia G, et al. Recurrent intravascular-catheter-related bacteremia caused by *Delftia acidovorans* in a hemodialysis patient. *J Clin Microbiol*. 2011;49(9):3418–3421. doi:10.1128/JCM.00625-11
17. Lang KJ, Chinzowu T, Cann KJ. *Delftia acidovorans* as an unusual causative organism in line-related sepsis. *Indian J Microbiol*. 2012;52(1):102–103. doi:10.1007/s12088-011-0221-3
18. Hagiya H, Murase T, Sugiyama J, et al. *Delftia acidovorans* bacteremia caused by bacterial translocation after organophosphorus poisoning in an immunocompetent adult patient. *J Infect Chemother*. 2013;19(2):338–341. doi:10.1007/s10156-012-0472-x
19. Sharma Y, Chumber SK, Bahl P. *Delftia acidovorans*-isolated from umbilical venous tip of a neonate: a case study and mini-review. *National J Med Res*. 2015;5:166–168.
20. Patel D, Iqbal AM, Mubarik A, et al. *Delftia acidovorans*: a rare cause of septic pulmonary embolism from catheter-related infection: case report and literature review. *Respir Med Case Rep*. 2019;27:100835. doi:10.1016/j.rmcr.2019.100835
21. Singh R, Chaudhary R, Sahu C, et al. A case of sepsis by a rare pathogen *Delftia acidovorans* in a patient undergoing chemotherapy. *J Clin Diagn Res*. 2022;16(12):1–2.
22. Backman L, Dumigan DG, Oleksiw M, et al. A cluster of gram-negative bloodstream infections in Connecticut hemodialysis patients associated with contaminated wall boxes and prime buckets. *Am J Infect Control*. 2023;51(6):638–643. doi:10.1016/j.ajic.2022.08.007
23. Agarwal N, Jindal A, Bhargava A. *Delftia acidovorans*: rarely a pathogen: a case report. *Pediatr Infect Dis J*. 2023;42(4):e130–e131. doi:10.1097/INF.0000000000003818
24. Cho BJ, Lee YB. Infectious keratitis manifesting as a white plaque on the cornea. *Arch Ophthalmol*. 2002;120(8):1091–1093.
25. Lee SM, Kim MK, Lee JL, et al. Experience of *Comamonas acidovorans* keratitis with delayed onset and treatment response in immunocompromised cornea. *Korean J Ophthalmol*. 2008;22(1):49–52. doi:10.3341/kjo.2008.22.1.49
26. Ray M, Lim DK. A rare polymicrobial keratitis involving *Chryseobacterium meningosepticum* and *Delftia acidovorans* in a cosmetic contact lens wearer. *Eye Contact Lens*. 2013;39(2):192–193. doi:10.1097/ICL.0b013e3182448881
27. Deb AK, Chavhan P, Chowdhury SS, et al. Endophthalmitis due to *Delftia acidovorans*: an unusual ocular pathogen. *Indian J Ophthalmol*. 2020;68(11):2591–2594. doi:10.4103/ijo.IJO_373_20
28. Yildiz H, Sünnetçioğlu A, Ekin S, et al. *Delftia acidovorans* pneumonia with lung cavities formation. *Colomb Med*. 2019;50(3):215–221. doi:10.25100/cm.v50i3.4025
29. Perumal PG, Appalaraju B, Thiruvengadam P. *Delftia acidovorans*: a rare pathogen isolated from pleural fluid in an immunocompromised host. *J Clin Diagn Res*. 2022;16(10):1–2.
30. Chun J, Lee J, Bae J, et al. *Delftia acidovorans* isolated from the drainage in an immunocompetent patient with empyema. *Tuberc Respir Dis*. 2009;67(3):239–243. doi:10.4046/trd.2009.67.3.239
31. Khan S, Sistla S, Dhodapkar R, et al. Fatal *Delftia acidovorans* infection in an immunocompetent patient with empyema. *Asian Pac J Trop Biomed*. 2012;2(11):923–924. doi:10.1016/S2221-1691(12)60254-8
32. Kam SK, Lee WS, Ou T, et al. *Delftia acidovorans* bacteremia associated with ascending urinary tract infections proved by molecular method. *J Exper Clin Med*. 2012;4(3):180–182. doi:10.1016/j.jecm.2012.04.010
33. Park CK, Kang BL, Chung EJ, et al. *Comamonas acidovorans* peritonitis in a CAPD patient managed with preservation of the catheter. *Korean J Nephrol*. 2002;21:175–177.
34. Artan A, Gursu M, Elcioglu O, et al. *Delftia acidovorans* peritonitis in a patient undergoing peritoneal dialysis. *Turkish J Nephrol*. 2020;29(4):326–328. doi:10.5152/turkjnephrol.2020.4204
35. Mahmood S, Taylor KE, Overman TL, et al. Acute infective endocarditis caused by *Delftia acidovorans*, a rare pathogen complicating intravenous drug use. *J Clin Microbiol*. 2012;50(11):3799–3800. doi:10.1128/JCM.00553-12
36. Khan S, Krishnan T. *Delftia acidovorans* skin infection occurring in a diabetic with peripheral edema. *J Case Rep*. 2014;4(1):127–131. doi:10.17659/01.2014.0032
37. Højgaard SMM, Rezahosseini O, Knudsen JD, et al. Characteristics and outcomes of patients with *Delftia acidovorans* infections: a retrospective cohort study. *Microbiol Spectr*. 2022;10(4):e0032622. doi:10.1128/spectrum.00326-22
38. Camargo CH, Ferreira AM, Javaroni E, et al. Microbiological characterization of *Delftia acidovorans* clinical isolates from patients in an intensive care unit in Brazil. *Diagn Microbiol Infect Dis*. 2014;80(4):330–333. doi:10.1016/j.diagmicrobio.2014.09.001
39. Ravvaorinoro M, Therrien C. Beta-lactamases and outer membrane investigations in beta-lactam-resistant *Comamonas acidovorans* strains. *Int J Antimicrob Agents*. 1999;12(1):27–31. doi:10.1016/S0924-8579(98)00095-8
40. Kang H, Xu X, Fu K, et al. Characterization and genomic analysis of quinolone-resistant *Delftia* sp. 670 isolated from a patient who died from severe pneumonia. *Curr Microbiol*. 2015;71(1):54–61. doi:10.1007/s00284-015-0818-6

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