

CASE REPORT

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A case of hospital-acquired pneumonia associated with *Chryseobacterium indologenes* infection in a patient with HIV infection and review of the literature

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Abstract

Background *Chryseobacterium indologenes* is an opportunistic, multidrug-resistant Gram-negative bacillus increasingly recognized as a cause of hospital-acquired infections, particularly in immunocompromised patients. Although rare, its intrinsic resistance to beta-lactams and its ability to colonize medical devices pose significant therapeutic challenges.

Case Presentation We describe a case of *C. indologenes* hospital-acquired pneumonia in a 43-year-old HIV-positive patient with multiple comorbidities, including Kaposi sarcoma, diabetes mellitus, and chronic kidney disease requiring hemodialysis. The patient was initially admitted with fever and elevated inflammatory markers, and empirical broad-spectrum antibiotic therapy was initiated. Despite initial improvement, the patient developed respiratory failure, requiring oxygen therapy. A respiratory panel identified *Rhinovirus*, while sputum culture revealed *C. indologenes*, resistant to multiple antibiotics but susceptible to levofloxacin. Targeted therapy led to clinical improvement. However, the course was complicated by *Clostridioides difficile*-associated diarrhea, followed by fatal sepsis due to *Klebsiella pneumoniae*. Our review of the literature identified 71 reported cases, with bacteremia (51%) and pneumonia (29%) as the most common clinical presentations. Medical devices and prolonged antibiotic exposure were key risk factors. While *C. indologenes* is intrinsically resistant to beta-lactams and carbapenems, fluoroquinolones and trimethoprim-sulfamethoxazole demonstrated efficacy in most cases. Emerging therapies, such as cefiderocol, may provide additional options for multidrug-resistant strains. This case highlights the critical need for accurate microbial identification, targeted therapy, and vigilant antimicrobial stewardship to improve outcomes in vulnerable patient populations.

Conclusion *C. indologenes* infections remain rare but clinically significant in hospitalized patients with immune dysfunction. The pathogen's multidrug resistance profile complicates treatment, necessitating early identification

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and targeted antimicrobial therapy. Fluoroquinolones, trimethoprim-sulfamethoxazole, and cefiderocol may serve as effective treatment options, emphasizing the importance of susceptibility-guided management.

Keywords *Chryseobacterium indologenes*, Hospital-acquired pneumonia, Immunocompromised, Multidrug resistance, HIV

Introduction

Chryseobacterium spp are non-motile, oxidase-positive, glucose non-fermentative, or slowly fermentative Gram-negative bacilli. The genus *Chryseobacterium* includes several species formerly classified as *Flavobacterium* species. Among those, *Chryseobacterium indologenes* (*C. indologenes*, formerly classified as *Flavobacterium indologenes*, belonging to CDC group IIb) is the most common. Colonies are smooth, convex, circular, mucous, 1–2 mm in diameter. The yellow culture, due to the production of flexirubin, turns red after being poured into a 10% KOH solution [1–5]. *C. indologenes* has been identified in various environmental sources, including soil, foodstuffs, plants, salt water, fresh water, and drinking water (demonstrating resistance to chlorination). Though widely distributed in nature, it is not part of the normal human microflora and is an uncommon human pathogen. Infections have been reported in pediatric patients, as well as in individuals who are immunocompromised, critically ill, or have indwelling medical devices. This includes patients with conditions such as cancer, diabetes mellitus, or neutropenia, and those receiving long-term treatment with broad-spectrum antibiotics. Within medical institutions, *C. indologenes* has been identified as a contaminant on various types of devices, including indwelling vascular catheters, vials, sink traps, feeding tubes, and other fluid-associated equipment [6–10]. It has also been demonstrated that disinfectants can act as reservoirs for *C. indologenes* [6–10]. Reported infections include bacteremia, meningitis, pneumonia, and device-associated infections. The management of *C. indologenes* infections presents a significant challenge due to the bacterium's unpredictable and rapidly evolving antimicrobial resistance [11–15]. Although it exhibits low virulence, it may cause life-threatening infections due to its multidrug resistance. Antimicrobial susceptibility is a matter of concern due to the high rate of intrinsic resistance to broad-spectrum antibiotics and the lack of standard guidelines for the management and treatment of infections, worsened by the ability to produce biofilm on foreign materials [16]. It produces molecular class A β -lactamase and class B carbapenem hydrolyzing metallo- β -lactamase, providing resistance to cephalosporins and carbapenems. The newer quinolones and trimethoprim-sulfamethoxazole are the most effective agents. *C. indologenes* harbors resistance to many agents typically used for empiric treatment of Gram-negative infections. Furthermore, no standardized guidelines exist for its treatment [16–20].

Here, we describe a case of hospital-acquired pneumonia caused by *C. indologenes* in a patient with HIV infection, and we review the previous cases described in the literature [21–81].

Case presentation

A 43-year-old male patient with a four-year history of HIV infection presented to the infectious disease clinic complaining of a high fever (self-recorded maximum temperature: 39 °C) associated with shaking chills for a few days. He was admitted because of the suspicion of bacteremia. His medical history was notable for AIDS-related Kaposi sarcoma on treatment with paclitaxel, insulin-dependent diabetes, hypertension, and chronic kidney disease (CKD) requiring hemodialysis via a central line for 1 year. Furthermore, the patient had been hospitalized on multiple occasions over the preceding two years for *Staphylococcus aureus* endocarditis, pneumonia, and cellulitis. HIV infection was diagnosed four years before and he had a CD4 nadir of 139 cells/mm³ (13%), with an HIV viral load of 1,200,000 copies/ml. The patient was on an antiretroviral therapy regimen comprising raltegravir, darunavir, and ritonavir and he has maintained an undetectable viral load since February 2021.

Regarding Kaposi sarcoma, the patient previously received liposomal doxorubicin for a period of three years, until six months prior to the initiation of hemodialysis, due to a deterioration in renal function. In light of renal impairment, therapy was switched to paclitaxel, a drug that has been shown to improve tumour-related symptoms and is particularly suitable for patients who have rapid progression after initial treatment with a liposomal anthracycline.

On admission, the patient's body temperature was 36.0 °C, blood pressure was 155/84 mmHg, pulse rate was 80 beats per minute, oxygen saturation was 97% on room air, and respiratory rate was 22 breaths per minute. The HIV viral load was undetectable (<20 copies/ml), and the CD4+ T cell absolute count of 271 cells/mm³, CD4% of 22%.

Biochemical examination (Table 1) highlighted a marked elevation of procalcitonin (PCT) and C-reactive protein (CRP) levels (48 mcg/L and 268 mg/l, respectively). Blood cultures from the central venous catheter and peripheral veins and urine culture were obtained, and empiric broad-spectrum antibiotic therapy was initiated with meropenem and daptomycin. Blood and urine

Table 1 Exams of the patient on admission, performed on Beckman uniceL DxH 900 analyzer (VCS 360 technology). Abbreviations: WBC: white blood count; PLT: platelets; hb: hemoglobin; CRP: C-reactive protein; PCT: procalcitonin (CLIA technique, biological matrix: serum); MDW: monocyte distribution width

Laboratory analysis	Patient's result	Reference range
WBC (cells/ μ L)	10,800	400–11,000
Neutrophils (%)	81.2	40–74
Lymphocytes (%)	11.6	20–48
Monocytes (%)	6.3	3–11
PLT (n/ μ L)	259,000	150,000–500,000
Hematocrit (%)	29.1	37–52
Hb (g/dL)	9.5	12–18
CRP (mg/L)	268	< 5
PCT (μ g/mL)	48.7	< 0.5
MDW	23.9	< 23



Fig. 1 CT chest imaging with contrast, parenchymal window, shows the following: presence of multiple consolidative areas with air-bronchogram, tending to confluence. Centimetric areola with a ground-glass appearance at the border between the anterior and posterior segments of the left upper lobe, in subpleural location. Left and mild right pleural effusion, with minimal atelectasis of contiguous lung parenchyma

cultures were negative; despite this there was a marked reduction of CRP and PCT levels over five days with antibiotic therapy. Computed tomography (CT) of the thorax was unremarkable, and transthoracic echocardiography was negative for endocarditis. On the tenth day of hospitalization, despite antibiotic administration, the patient exhibited a recurrence of fever and elevation of CRP and PCT. He developed respiratory failure, and within three days, oxygen saturation measured by pulse oximetry was 84% in room air. A new chest CT revealed multiple bilateral lung infiltrates with an air-bronchogram (Fig. 1).

The patient was administered oxygen therapy initially using a Venturi mask and subsequently high-flow nasal cannula oxygen, but no improvement was observed. Three sets of surveillance blood cultures were negative, and the patient continued to exhibit fever and a persistent increase in inflammatory indices. Considering the patient's rectal colonization with

carbapenemase-producing organisms, antibiotic therapy was adjusted to include meropenem-vaborbactam and ceftobiprole. BIOFIRE® FILMARRAY® Pneumonia Panel plus tests for 27 bacteria and viruses that cause pneumonia and other lower respiratory tract infections, as well as for 7 genetic markers of antibiotic resistance. This molecular panel performed on sputum detected only the presence of *Rhinovirus*. However, sputum culture revealed the growth of *C. indologenes* susceptible to levofloxacin.

Sputum culture isolate identification was obtained by means of matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI Biotyper, Bruker Daltonics, Billerica, MA, USA). Antimicrobial susceptibility testing (AST) for antibiotics other than cefiderocol, imipenem-relebactam, and meropenem-vaborbactam was performed by means of an automated system (Phoenix, Becton Dickinson Diagnostics, Sparks, MD, USA) following the manufacturer's instructions. Minimum inhibitory concentrations (MICs) were interpreted according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) clinical breakpoints, version 14.0 (<http://www.eucast.org/>). Cefiderocol, imipenem-relebactam, and meropenem-vaborbactam AST were performed by means of a gradient test (Liofilchem S.r.L., Italy) with a concentration range of 0.016–256 μ g/mL, 0.002/4–32/4 μ g/mL, 0.016/8–256/8 μ g/mL, respectively. The breakpoints of MICs for susceptibility were determined by applying EUCAST standards for susceptibility for *Pseudomonas aeruginosa* and non-*Enterobacteriaceae* [82]. The presence of carbapenemase-encoding gene/s was detected using a commercial genotypic assay (Xpert® Carba-R; Cepheid, Sunnyvale, CA, USA) which rapidly detects and qualitatively differentiates five common carbapenemase genes (*bla*_{KPC}, *bla*_{NDM}, *bla*_{VIM}, *bla*_{OXA-48}, and *bla*_{IMP}) directly from purified colonies. The analysis of carbapenemase-encoding genes showed a negative result for all the tested genes. The isolate showed susceptibilities to levofloxacin, ciprofloxacin, and piperacillin/tazobactam

and resistance to amikacin, cefepime, ceftazidime, colistin, imipenem, meropenem, piperacillin, and tobramycin, as shown in Table 2. Furthermore, it was susceptible to cefiderocol (MIC 0,38 µg/ml) and resistant to meropenem/vaborbactam (MIC 24 µg/ml) and to imipenem/relebactam (no inhibition halo). In view of these findings, the previous antibiotic regimen was discontinued, and levofloxacin 750 mg/day was initiated, leading to progressive clinical improvement and weaning off oxygen therapy within 15 days. Unfortunately, the clinical course was complicated by the development of *Clostridioides difficile*-associated diarrhea, treated with fidaxomicin, and finally by fulminant sepsis caused by KPC-producing *K. pneumoniae*, which resulted in the patient's death.

Discussion and conclusions

In addition to the described case, we conducted a literature search on *C. indologenes* infections using the PubMed database. We employed the search strings “(*C. indologenes*) AND (infection) AND (case report OR case series OR clinical cases)”. No language and temporal restrictions were applied during the research. We yielded 61 relevant articles [21–81]. A comprehensive collection of pertinent data concerning the clinical presentation of the patient in question, as well as relevant literature, was meticulously collected and subjected to rigorous analysis. Only case reports and case series involving infections in humans were considered, resulting in a total number of 71 patients. Of these, 59% were male, with ages ranging from 12 h to 94 years (median: 43 years, interquartile range: 9–66 years). Comorbidities were reported in 43 patients (60%), including hematological diseases (22%) [22, 23, 29, 35, 37, 39, 53, 69, 70], cardiovascular diseases (21%) [25–27, 31, 40, 44, 46, 51, 52, 57, 60, 61, 65, 77], neurological diseases (11%) [23, 34, 42, 46–48, 50, 71], oncological diseases (11%) [33, 40, 41, 43, 54, 73, 79,

81], diabetes mellitus (11%) [24, 26, 31, 38, 57, 58, 74, 77], chronic kidney disease (CKD) (8%) [27, 31, 57, 61, 65, 66], pulmonary disease with radiological findings (7%) [24, 25, 45, 61, 77], history of transplantation (6%) [39, 45, 66, 70], ophthalmic diseases (4%) [55, 58, 72], preterm delivery (4%) [48, 62, 68], endocrine disorders (3%) [45, 58], urinary tract disease (1%) [24], gastroenterological disease (1%) [45], and other miscellaneous conditions (11%) [23, 28, 35, 36, 74, 75, 80]. HIV infection was reported in only one case, in a patient with controlled viral load and medical history remarkable for end-stage renal disease on peritoneal dialysis [27]. Consistent with most cases reported in the literature, our patient presented with multiple comorbidities, including chronic kidney disease, diabetes mellitus, and Kaposi sarcoma. All these conditions contribute to immune system dysfunction. Additionally, he had an HIV infection that was well-controlled with antiretroviral therapy. The respiratory panel also detected rhinovirus, which, according to some studies, is associated with an increased risk of pneumonia due to impairment of upper airway defenses [83].

Our review revealed that 21% of previously reported *C. indologenes* infections were diagnosed during ICU stays. In 39% of patients, medical devices were involved, including central venous catheters (CVCs) (11%) [22, 23, 29, 33, 54, 79], Port-A-Cath systems (8%) [23, 41, 45, 73], ventriculoperitoneal shunts (4%) [47, 48, 71], peritoneal dialysis catheters (4%) [27, 57, 61], Hickman catheters (2%) [37], contact lenses (1%) [55], lumboperitoneal shunts (1%) [42], lumbar external drainage (1%) [46], indwelling Foley catheters (1%) [24], and Malecot catheters (1%) [32]. Device removal was performed in 17 cases (61%), particularly when persistent high fever occurred despite targeted antibiotic therapy, and was associated with clinical improvement and negative cultures [29, 42]. An antibiotic-lock approach combined with systemic therapy was effective in rescuing colonized vascular access devices in an 11-year-old boy with non-metastatic Ewing sarcoma [79]. However, source control was pivotal in achieving resolution, particularly in cases of relapse [42, 73]. As in our case, twenty-seven patients (38%) had a history of prior broad-spectrum antibiotic therapy targeting Gram-negative pathogens before bacterial isolation. This may have contributed to the selection of multidrug-resistant pathogens, including metallo-β-lactamase-producing strains [32, 74, 78]. Two cases involved strains harboring bla_{NDM-1} [69] or co-expressing bla_{IND-2}, bla_{CIA}, and bla_{CcrA} [63]. A notable case involved a 77-year-old male who developed cellulitis and bacteremia caused by *C. indologenes*. While the strain isolated from a skin swab was susceptible to piperacillin-tazobactam, the bloodstream isolate exhibited resistance to the same antibiotic, likely due to the bacterium's adaptation under antibiotic pressure [40].

Table 2 Antibiogram of *C. indologenes* isolate

Antimicrobial agent	MIC	Susceptibility
Amikacin	> 16	R
Amoxycillin / clavulanic acid	> 32/2	R
Ampicillin	> 8	R
Cefepime	> 8	R
Cefotaxime	> 4	R
Ceftazidime	> 8	R
Ciprofloxacin	≤ 0,25	S
Colistin	> 4	R
Imipenem	> 8	R
Levofloxacin	≤ 0,5	S
Meropenem	> 8	R
Piperacillin	> 16	R
Piperacillin-tazobactam	≤ 4/4	S
Tobramycin	> 4	R
Trimethoprim-sulfamethoxazole	≤ 1/19	S

Table 3 Summary of susceptibility results from 64 antibiograms from cases reported in the literature

Antibiotics	Susceptibility	Increased exposure	Resistance
Imipenem	7/39 (18%)	0	32/39 (82%)
Meropenem	3/32 (9%)	0	29/32 (91%)
Trimethoprim-sulfamethoxazole	37/39 (95%)	0	2/39 (5%)
Aminoglycosides	4/49 (8%)	0	45/49 (92%)
Fluoroquinolones	31/48 (65%)	2/48 (4%)	15/48 (31%)
Cefazolin	0	0	9/9 (100%)
Cefoxitin	0	1/3 (33%)	2/3 (67%)
Cefotaxime	1/3 (33%)	0	2/3 (67%)
Ceftriaxone	1/24 (4%)	0	23/24 (96%)
Ceftazidime	11/40 (27%)	2/40 (5%)	27/40 (68%)
Cefoperazone	1/4 (25%)	1/4 (25%)	2/4 (50%)
Cefoperazone-sulbactam	5/14 (36%)	1/14 (7%)	8/14 (57%)
Cefepime	12/35 (34%)	0	23/35 (66%)
Aztreonam	1/30 (3%)	0	29/30 (97%)
Moxalactam	0	0	7/7 (100%)
Ampicillin	0	0	10/10 (100%)
Ampicillin-sulbactam	0	0	4/4 (100%)
Amoxicillin-clavulanic acid	1/9 (12%)	0	8/9 (88%)
Piperacillin	6/13 (46%)	0	7/13 (54%)
Piperacillin-tazobactam	21/39 (54%)	3/39 (8%)	15/39 (38%)
Tigecycline	3/12 (25%)	2/12 (17%)	7/12 (58%)
Minocycline	7/8 (87%)	0	1/8 (13%)
Polymyxin	2/17 (12%)	0	15/17 (88%)
Vancomycin	3/12 (25%)	0	9/12 (75%)
Teicoplanin	1/8 (12%)	0	7/8 (88%)

Our findings align with previous reports indicating intrinsic resistance of *C. indologenes* to cephalosporins and carbapenems, although no carbapenemase genes (e.g., bla_{KPC}, bla_{NDM}, bla_{VIM}, bla_{OXA-48}, bla_{IMP}) were detected in our analysis.

Regarding clinical features, our case presented as severe pneumonia. Bacteremia (51%) [22, 23, 28, 29, 34, 35, 37–41, 43, 45, 49, 54, 59, 62–65, 67–69, 73, 74, 79, 81] and pneumonia (29%) were the most clinical pictures reported in the previous cases [21–23, 26, 36, 44, 50, 52, 53, 59, 60, 64, 68–70, 77, 80]. Among cases of pneumonia, 47.6% were ventilator-associated pneumonia. Other clinical pictures included meningitis (11%) [28, 30, 34, 42, 46–48, 71], urinary tract infections (8%) [24, 31, 32, 66, 69, 76], peritonitis (7%) [27, 33, 61, 75], keratitis (4%) [55, 56, 72], cellulitis (4%) [40, 78], pleural effusion (1%) [51], and peritonitis associated with dialysis (1%) [57]. Notably, most meningitis cases (62%) occurred in newborns (<1 year), primarily in those with predisposing factors such as hydrocephalus, ventriculoperitoneal shunt placement, prematurity, and low gestational age. However, one case was reported in a healthy 8-day-old female with no identifiable risk factors. These findings highlight the need to consider *C. indologenes* in cases of neonatal meningitis that do not respond to empirical antibiotic therapy and lack other microbiological findings [30].

C. indologenes infection presents a significant challenge due to its intrinsic antimicrobial resistance. Susceptibility testing was documented in 43 cases, most of which were performed using the bioMérieux VITEK[®]2 System (41%) and the Kirby-Bauer disc diffusion method (39%), while the Becton Dickinson Phoenix[™] 100 Automated Microbiology System was used in 7% of cases [34, 48, 79]. Trimethoprim/sulfamethoxazole was the most effective agent (52%), followed by fluoroquinolones (44%), piperacillin-tazobactam (30%), and cefepime (17%) (Table 3). Our results, which demonstrated the susceptibility of the isolate to ciprofloxacin, levofloxacin, and trimethoprim/sulfamethoxazole, align with the above data. Additionally, our isolate was sensitive to cefiderocol. One case of pan drug resistant *C. indologenes* has been reported [81], in which the patient had received prior broad-spectrum antibiotic therapy followed by trimethoprim/sulfamethoxazole but ultimately succumbed to sepsis. In similar cases, cefiderocol could be considered a potential treatment option following susceptibility testing.

Combination therapy was administered to 26 patients [22–24, 28, 29, 34–37, 41, 42, 44, 45, 47, 48, 51, 53, 54, 57, 58, 61, 62, 70, 76–78]. *Pseudomonas aeruginosa* was isolated as a common co-pathogen in three patients [21, 22], *Cytomegalovirus* [70], and *Acinetobacter baumannii* [71] in one patient each, respectively. Patients received appropriate antibiotic therapy according to susceptibility also

to coexisting pathogens for an average treatment duration of 15 days. A total of thirteen patients (18%) succumbed, eight of them due to sepsis, four to respiratory failure, and one to liver failure. Nevertheless, we must point out that two patients died later due to an underlying disease other than *C. indologenes* infection [35, 53].

Notably, a case of a 25-year-old woman with *C. indologenes* bacteremia who empirically started piperacillin/tazobactam therapy and even though the antibiogram yielded sensitivity to trimethoprim/sulfamethoxazole and minocycline only, empiric therapy was continued because clinic gradually improved, without any recurrence [22]. Therefore, the pathogenetic role of *C. indologenes* in some cases remains doubtful and distinguishing between simple colonization or contamination of a sample rather than the pathogenic role of the microorganism is a challenge. In our case, the patient showed a progressive worsening of respiratory function, and despite several broad-spectrum empirical therapies started, clinical improvement was objective only after the initiation of levofloxacin.

In conclusion, *C. indologenes* is an uncommon but significant pathogen in hospitalized patients with multiple comorbidities. Prolonged antibiotic exposure and immune dysfunction are critical risk factors for infection. Bacteremia and pneumonia are the predominant clinical manifestations. The microorganism is not identified with modern molecular panels, and cultures remain the diagnostic gold standard. Its pathogenic role is not always clear, but suspicion should be heightened in patients unresponsive to broad-spectrum empiric therapy. Intrinsic resistance to beta-lactams limits treatment options; however, trimethoprim-sulfamethoxazole and fluoroquinolones offer therapeutic potential for susceptible strains. Cefiderocol may serve as a potential therapeutic option for pan-drug-resistant strains, confirming its efficacy via susceptibility testing.

Author contributions

Conceptualization A.C.; methodology, L.P., S.C., C.B. and A.C.; validation, L.P. and A.C.; investigation, C.V.M., E.B., L.P., B.R., A.A. and C.G.; data curation, C.V.M., E.B., L.P.; writing—original draft preparation, C.V.M., E.B., L.P., B.R., S.C., R.V., C.B.; writing—review and editing, L.P. and A.C.; visualization, A.A., C.G., S.C., R.V., C.B. and A.C.; supervision, L.P. and A.C. All authors have read and agreed to the published version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee “Palermo 1”, Palermo, Italy (SPP-Study-Verbal n. 25 30/10/2024).

Consent for publication

Written informed consent was obtained from the patient for the publication of this Case Report.

Competing interests

The authors declare no competing interests.

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