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Cedecea lapagei pneumonia: the first reported case in Italy and literature review

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SUMMARY

Cedecea lapagei is a rare Gram-negative bacillus of the Enterobacteriaceae family, typically identified as an opportunistic pathogen in immunocompromised or critically ill patients. Less than twenty clinically significant infections have been described worldwide, many displaying concerning multidrug-resistance profiles. We report the first documented case of *C. lapagei* pneumonia in Italy, occurring in a 73-year-old man with hypogammaglobulinemia and post-stroke dysphagia who presented with a recurrence of pneumonia. Bronchoalveolar Lavage Fluid (BALF) obtained for diagnostic clarification yielded *C. lapagei* as the causative organism. A literature review on *C. lapagei* pneumonia has been conducted as well. This case underscores the pathogenic potential of *C. lapagei* in vulnerable hosts and highlights the importance of thorough microbiological investigation and targeted antimicrobial therapy, given its unpredictable and often extensive antimicrobial resistance pattern.

Keywords: Cedecea lapagei, pneumonia, opportunistic infection, Gram-negative bacterial infection, emerging pathogen.

INTRODUCTION

Cedecea lapagei (*C. lapagei*) is a short, motile, catalase-positive, oxidase-negative, Gram-negative and facultatively anaerobic bacillus belonging to the *Enterobacteriaceae* family [1, 2].
The genus name Cedecea derives from the acronym CDC, referring to the Centers for Disease Control and Prevention in Atlanta, where the organism was first identified. The species epithet lapagei honors Stephen Lapage, a British bacteriologist [1, 3]
Phenotypically, *C. lapagei* shares several characteristics with members of the genus Serratia, including lipase positivity and intrinsic resistance to cephalothin and colistin [2, 3]. In addition, both genera exhibit similar growth patterns on blood agar and MacConkey agar following aerobic incubation at 37 °C.
C. lapagei is generally regarded as an opportunistic pathogen, most often isolated in immunocompromised or critically ill patients and commonly associated with bloodstream, urinary tract, or wound infections. Nevertheless, clinically significant infections remain exceedingly rare, with approximately twenty cases reported worldwide to date [4, 5].
Of particular concern, some reports have described multidrug-resistant isolates, highlighting the potential clinical relevance of this emerging organism, particularly in vulnerable or hospitalized patients [6-8].
The clinical presentation and organ involvement associated with *C. lapagei* infections are heteroge-

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nous, with no clear tissue tropism identified. It has been implicated in a wide range of infections, including pneumonia, sepsis, and urinary tract infections [5, 9, 10]. *C. lapagei* has also been implicated in cutaneous and mucosal infections, further supporting its opportunistic nature and its ability to colonize damaged or immunologically impaired tissues [3, 11-13].

In parallel, environmental sources of Cedecea constituting potential reservoirs for infection are numerous. Similar to other genera in the Enterobacteriaceae family, Cedecea species have been detected in diverse ecological niches, including aquatic habitats, soil or agricultural dusts, plants, retail food, insect vectors, human gut microbiome, and non-human animals [6].

We report the first microbiologically confirmed case of *C. lapagei* pneumonia in Italy and provide a narrative review of previously reported cases of pneumonia due to this pathogen.

CASE REPORT

A 73-year-old man with a medical history of IgG hypogammaglobulinemia, Chronic Obstructive Pulmonary Disease (COPD) and a recent ischemic stroke with residual dysphagia in the context of Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) was admitted for recurrent lower respiratory tract infection. Approximately two months before the index hospitalization, the patient had experienced a first episode of left lower lobe pneumonia, documented by chest radiography and treated empirically with antibiotics and corticosteroids (*Figure 1a*). Partial radiological resolution was subsequently demonstrated on a follow-up chest Computed Tomography (CT) scan performed one month later. At readmission, the patient presented with fever, sore throat and worsening dyspnea. Blood gas analysis showed a mild hypoxemic respiratory failure. Laboratory findings revealed leukocytosis with increase inflammatory markers while immunological work-up confirmed the known humoral immunodeficiency. Microbiological screening for common urinary respiratory pathogens was negative. Chest CT demonstrated a left mid-basal pleural effusion with adjacent parenchymal thickening and focal calcifications, patchy ground-glass opacities with fibrotic streaks in the left lower lobe, and a small pericardial effusion (*Figure 1b, c, d*).

The main clinical, laboratory and radiological findings at presentation are summarized in *Table 1*. Low-flow oxygen therapy (1 L/min via nasal cannula) for mild respiratory failure and empiric therapy with piperacillin–tazobactam (4.5 g IV every 6 hours) was initiated to provide broad-spectrum coverage, particularly targeting possible aspiration-related and hospital-acquired pathogens. Given the recurrence of ground-glass opacities documented on chest CT, flexible bronchoscopy with bronchoalveolar lavage fluid (BALF) culture was performed to clarify the underlying etiology. Microbiological analysis yielded *Cedecea lapagei* at a concentration of 10^5–10^6 CFU/mL, with a fully susceptible antimicrobial profile (*Table 2*).

The specimen was processed using the automated WASP® (Walk-Away Specimen Processor, Copan Diagnostics) system. Briefly, the BALF sample was aliquoted and treated with SLSolution™ (Copan) in a 1:1 ratio, vortexed for 30 seconds, and incubat-

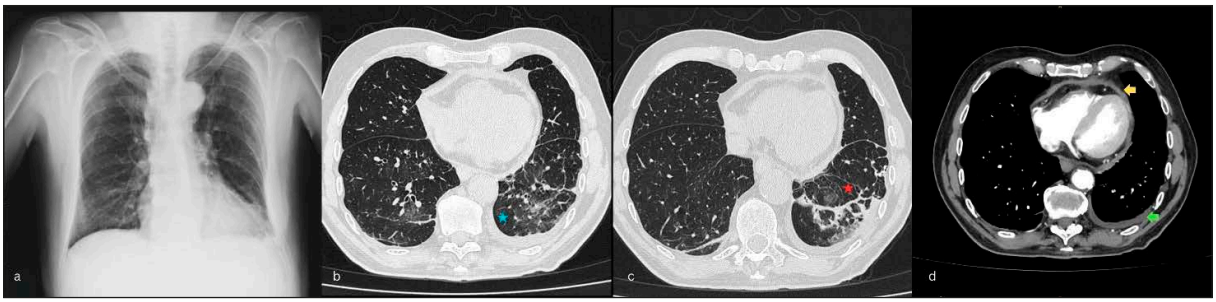

Figure 1 - **a)** Chest X-ray showing a small left basal consolidation associated with blunting of the costophrenic angle and bibasal interstitial thickening. **b)** and **c)** Chest CT (lung window): bibasal ground-glass opacities, more pronounced in the left lower lobe (blue asterisk), associated with consolidative bands and bronchiolar mucoid impaction (red asterisk). **d)** Minimal pericardial effusion (yellow arrow) and left basal pleural effusion (green arrow).

Table 1 - Relevant findings at admission.

	Parameter	*Findings*
Vital signs	Body temperature	36.8 °C
	Blood pressure	125/75 mmHg
	Heart rate	100 beats/min
	Respiratory rate	16 breaths/min
	Oxygen saturation (room air)	91%
Arterial blood gas	PaO_2	54.5 mmHg
Laboratory tests	Hemoglobin	11.5 g/dL
	White blood cell count	10.4×10^9/L
	Neutrophils	9.85×10^9/L
	Platelets	182×10^9/L
	C-reactive protein	15.4 mg/dL
Immunological evaluation	IgG	420 mg/dL (ref. 800–1600)
	IgA	71 mg/dL (ref. 60–400)
	IgM	32 mg/dL (ref. 40–230)
Microbiology	Urinary antigen Legionella pneumophila	Negative
	Urinary antigen Streptococcus pneumoniae	Negative
	Urine culture	Negative
Chest CT		Left mid-basal pleural effusion with adjacent parenchymal thickening and focal calcifications, patchy ground-glass opacities with fibrotic streaks in the left lower lobe, and a small pericardial effusion.
Echocardiography		Small, non-hemodynamically significant pericardial effusion

ed at room temperature for 15 minutes to ensure complete liquefaction. Automated inoculation was subsequently performed using a calibrated 10 µL metal loop according to a standard 4-Quadrant Dilution Pattern. The specimen was plated onto selective and non-selective media, including chromogenic agar, MacConkey agar, blood agar, Chocolate Agar with Bacitracin, and Sabouraud agar. Plates were incubated at 35–37°C under aerobic or microaerophilic conditions and continuously monitored using the WASPLab® high-resolution digital imaging system (Copan Diagnostics). The culture yielded *Cedecea lapagei* in pure culture (monomicrobial growth) at a significant concentration of 10^5–10^6 CFU/mL. No other bacterial or fungal pathogens were isolated. Presumptive identification was initially based on colony morphology, and confirmatory species identification was performed by Matrix-Assisted Laser Desorption Ionization–Time of Flight Mass Spectrometry (MALDI-TOF MS, Bruker Daltonics). The isolate demonstrated a high-confidence identification log score of 2.3, which was validated in triplicate. Antimicrobial Susceptibility Testing (AST) was performed using Etest (bioMérieux) gradient strips on Mueller-Hinton agar to determine precise Minimum Inhibitory Concentrations (MICs). Due to the lack of species-specific breakpoints for *Cedecea* spp., MIC values were primarily interpreted according to European Committee on Antimicrobial Susceptibility Testing (EUCAST) Pharmacokinetic/Pharmacodynamic (PK/PD) non-species related breakpoints. Notably, a comparison using standard *Enterobacterales* breakpoints confirmed an identical susceptibility profile (Susceptible) for all tested antibiotics, reinforcing the clinical validity of the AST results (*Table 2*).

After five days of empiric therapy, clinical improvement was limited; therefore, antibiotic treatment was switched to levofloxacin 750 mg daily

Table 2 - Antimicrobial susceptibility profile of *Cedecea lapagei* isolate (MIC Method: Etest).

Antibiotic	*MIC (mg/L)*	*Interpretation (EUCAST PK/PD)*
Cefotaxime	0.125	S
Ceftazidime	0.38	S
Ceftriaxone	0.125	S
Imipenem	0.38	S
Meropenem	0.064	S
Levofloxacin	0.064	S
Moxifloxacin	0.125	S
Piperacillin/Tazobactam	1.0	S
Trimethoprim/ Sulfamethoxazole	0.125	S

Abbreviations: MIC, Minimum Inhibitory Concentration; S, Susceptible.

for 10 days according to susceptibility testing. Concomitantly, after hematological consultation, intravenous immunoglobulin replacement therapy was initiated as supportive treatment (IVIg 0.4 g/kg). Following treatment modification, inflammatory markers progressively decreased, gas exchange normalized and oxygen supplementation was discontinued, with complete clinical recovery. In light of the patient's clinical background, the recurrence of pneumonia, and the isolation of an unusual pathogen, a potential aspiration-related mechanism was subsequently explored. A dynamic pharyngo-esophageal fluoroscopic study did not demonstrate overt aspiration or structural abnormalities. However, bedside speech therapy assessment documented coughing episodes after large fluid boluses, suggesting subtle oropharyngeal dysfunction as a plausible contributing factor to the recurrent infectious episodes.

At the 3-month follow-up visit after hospital discharge, the patient showed a complete recovery of activities of daily living and was in spontaneous eupneic breathing on room air. Pulmonary function tests demonstrated a moderate obstructive ventilatory defect according to ERS/ATS 2022 criteria, without a significant worsening compared with pre-hospitalization values.

LITERATURE REVIEW

A comprehensive narrative review was conducted on PubMed/MEDLINE and Scopus using search strategies based on the same conceptual framework and including publications indexed from database inception to October 2025. To better characterize this uncommon infection, we focused our analysis exclusively on previously reported cases of pneumonia caused by *Cedecea lapagei*, aiming to identify possible shared predisposing factors or clinical patterns. Search strategy included the following keywords as title/abstract: "*Cedecea lapagei*" AND "pneumonia". Although articles other than case reports and case series were not considered primary sources for the purposes of this analysis, they were extensively examined to identify additional relevant publications that could be eligible for inclusion. A few additional reports published in non-English languages were excluded from the analysis, as the limited availability of complete clinical data and methodological details made it difficult to ensure a consistent and comparable evaluation across cases.

RESULTS AND DISCUSSION

Only six such cases have been documented in the literature, underscoring the extreme rarity of this presentation (*Table 3*).

Most reported patients were male adults, though one case involved a premature neonate, suggesting that infection may occur across a broad age spectrum when favorable conditions exist. In five out of six cases, an underlying predisposing factor was identified, most frequently chronic respiratory disease (e.g., COPD) or systemic immunocompromise (diabetes, hematologic malignancy, or recent viral pneumonia such as COVID-19) [14, 17, 18]. The clinical manifestations varied from relatively mild lower respiratory tract symptoms, such as productive cough and hemoptysis, to acute hypoxemic respiratory failure requiring ventilatory support. In particular, two of the reported cases described patients who developed severe respiratory failure requiring endotracheal intubation emphasizing the potential of this microorganism to cause life-threatening pneumonia in fragile hosts [4, 16]. Radiological patterns were also heterogeneous. Most patients exhibited unilateral or bilateral consolidations, consistent with bacterial pneumonia, whereas one case showed cavitary lesions on chest CT and a coinfection with *Aspergillus sidowii* [15]. This observation suggests that *C. lapagei* may coexist with other pathogens,

Table 3 - Summary of reported cases of *Cedecea lapagei* pneumonia.

Author	*Area*	*Comordbidity*	*Clinical Presentation*	*Respiratory Status on admission*	*Findings*	*Diagnosis*	*Biological Sample*	*Susceptibility Profile*
Hai *et al.* [14]	Vietnam	Man. Type II Diabetes	Recent onset of fatigue, dyspnea, cough with bronchial hypersecretion	Acute hypoxemic hypocapnic respiratory failure.	Bilateral lower lobe infiltration and consolidations on Chest-CT.	Pneumonia	Sputum culture	S: PIP/TAZ, FEP, CRO, MEM, CIP R to: TMP/SMX
Yang *et al.* [15]	China	Man. None	Severe and productive cough with hemoptysis	No respiratory failure.	Lung cavities on Chest-CT	Coinfection Cedecea lapagei and Aspergillus sidowii	BALF	S to: PIP/TAZ, FEP, LFX, Amox-Clav R to: AMP/SUL
Ramaswamy *et al.* [4]	India	36 weeks preterm male infant	Sepsis	need for endotracheal intubation (ETI)	Right-sided consolidation on Chest-XRay	Nosocomial Pneumonia	Blood culture	S to: Pip/Taz, TMP/SMX, CIP and LFX. R to: MEM, AK, FEP.
Hong *et al.* [16]	Korea	COPD	Respiratory Failure	Need for ETI	Left-sided consolidation on Chest-XRay	Pneumonia	Blood culture	S to: PIP/TAZ, CRO, CIP R to: Amox-Clav
Lopez *et al.* [17]	Mexico	None	Fever, multiple painful oral lesions and gum bleeding.	Not known.	Bilateral alveolar infiltration on Chest-XRay	Acute promyelocytic leukemia	Sputum culture	I to: AMP/SUL, GM R to: PIP/TAZ, TMP/SMX, FEP, CRO,MEP, AK, CIP
Deveci *et al.* [18]	Turkey	COPD. Recent COVID-19 Pneumonia	Fever and dyspnea	Not known.	Bilateral consolidation on Chest-XRay	Pneumonia	Sputum culture	I to: MEP, TMP/SMX R to: PIP/TAZ, Amox-Clav, FEP, CRO, CIP.

Abbreviations: (S: susceptible; I: increase dose; R: resistant; PIP/TAZ: Piperacillin-Tazobactam;Amox-Clav: Amoxicilline-Clavulanate; AMP/SUL: Ampicilline/Sulbactam; TMP/SMX: Trimethoprim- Sulfamethoxazole; CIP: Ciprofloxacine; LFX: Levofloxacine MEM: Meropenem; FEP: Cefepime; CRO: Ceftriaxone; AK: Amikacine; GM: Gentamicine).

possibly reflecting colonization or synergistic interactions within compromised airways. The sources of microbiological isolation included sputum, BALF and blood cultures. Positive blood cultures in two cases indicate that *C. lapagei* can breach mucosal barriers and cause bacteremia, thereby behaving as an invasive organism rather than a mere colonizer [4, 16]. In contrast, isolation of Gram-negative bacilli, from non-sterile samples such as sputum or BALF should be interpreted with caution. On the other hand, a pathogen that colonizes the proximal airways and progresses towards the deeper airways, when local defenses are weakened alveolar damage and consequent infection can occur [19].

In this context, the present clinical case is consist-

ent with the patterns emerging from the reviewed literature, particularly regarding host vulnerability. Indeed, it can be hypothesized that, in our case, the coexistence of hypogammaglobulinemia and oropharyngeal dysfunction following a recent ischemic stroke may have contributed to an increased susceptibility to infection.
However, although our isolate was susceptible to all tested antibiotics, including β-lactams – in contrast to previously reported resistance patterns – the clinical response to piperacillin–tazobactam was suboptimal despite a low MIC value (1 mg/L, categorized as susceptible according to PK/PD EUCAST criteria). This discrepancy may be explained by limited pulmonary tissue penetration, or host-related factors such as recurrent microaspiration, which can all reduce the clinical efficacy of β-lactam agents despite in vitro susceptibility. In addition, the empiric antibiotic course was probably too short to achieve a measurable clinical effect, and the switch to levofloxacin was guided by the few available reports in the literature describing marked susceptibility of *Cedecea lapagei* to fluoroquinolones. Furthermore, an inducible β-lactamase activity has also been hypothesized. The extent of drug resistance phenotypes among clinical isolates of Cedecea and the molecular mechanisms underlying an inducible β-lactamase activity in these pathogens have received little attention. Variable resistance patterns have been documented for antibiotics representing the major β lactam classes of bacterial cell wall synthesis inhibitors, included β-lactam/β lactamase inhibitor combinations like piperacillin–tazobactam [6].
However, these observations must be interpreted with extreme caution, as they are based on isolated case reports and lack support from robust clinical studies or high-quality evidence. Therefore, no definitive conclusions regarding the relative efficacy or superiority of specific antimicrobial agents, including fluoroquinolones, can be drawn at present.

CONCLUSIONS

To our best knowledge, this is the first documented case of *Cedecea lapagei* associated pneumonia in Italy with isolation from BALF.
Cedecea lapagei should be recognized as a potential pathogen, particularly in immunocompromised or critically ill patients presenting with severe infections, irrespective of the affected organ system. Its isolation should not be dismissed as a contaminant, as underestimation may lead to delayed diagnosis and inappropriate therapy, with possible negative impact on clinical outcomes. Given its variable resistance profile and capacity for multidrug resistance, accurate microbiological identification and prompt initiation of targeted antimicrobial therapy are crucial to optimize patient management and prognosis.

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Author contributions
All authors contributed equally to the design and writing of the study.

Consent to participate
Written informed consent for the manuscript publication, information and images management was obtained from the patient.

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