



RESEARCH ARTICLE

CHRYSEOBACTERIUM INDOLOGENES INFECTION IN SMALL FOR GESTATIONAL AGE (SGA) NEONATE: A CASE REPORT

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ABSTRACT

C. indologenes is a widespread bacteria in the environment, primarily in soil and water. *Chryseobacterium indologenes* is an aerobic, non-motile, catalase, oxidase & indole positive and non-glucose fermenting Gram negative bacillus. It can survive in chlorine-treated municipal water supplies & can colonize the sink basins and tap waters of the hospitals. Contamination of the medical devices containing water (respirators, intubation tubes, humidifiers, incubators for newborns, etc.) in hospital settings may lead to serious infections. Typically, *C. indologenes* causes major health care-associated infections such as pneumonia, empyema, pyelonephritis, cystitis, peritonitis, septicemia, meningitis. Though *C. indologenes* is not a part of normal microbial flora it is rarely reported as a pathogen. Management of *C. indologenes* infection in neonates is not adequately documented due to underreporting, particularly in India. Because of its intrinsic resistance and the less availability of data from the literature, the effective empirical treatment of *C. indologenes* is challenging. We present an uncommon case of bacteremia caused by *C. indologenes* in a late preterm SGA newborn having moderate respiratory distress syndrome with thrombocytopenia who was successfully treated. Multidrug resistant *C. indologenes* should be considered a health-care associated pathogen in neonates receiving treatments involving invasive equipment use and long-term antibiotic therapy.

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INTRODUCTION

Chryseobacterium indologenes, previously classified under the Flavobacterium CDC group IIb, is a rare human pathogen which is widely distributed in soil, plants, and water (1). In 1994, Vandamme and colleagues described it as a nonmotile, Gram-negative, rod-shaped, aerobic, non-lactose-fermenting, catalase- and oxidase-positive bacillus, which was classified in the genus *Chryseobacterium*. (2) Among the six species of this genus, *Chryseobacterium meningosepticum*, which is called *Elizabethkingia meningosepticum*, is the most virulent while *C. indologenes* is minimally virulent (1). *C. indologenes* is not part of the human microflora (1). The clinical importance of *C. indologenes* has increased in recent years after being identified as the causative agent of bacteremia, pneumonia, indwelling device-associated infection, peritonitis, and ocular infection (2). Bacteremia and pneumonia caused by this bacterium generally result in high mortality, which may partly be attributed to the inherent multidrug-resistant nature of *C. indologenes* (3). *C. indologenes* is typically isolated from

hospital environments such as from basin sinks, indwelling vascular catheters, vials, feeding tubes, other equipment in contact with fluids and water. Thus, *C. indologenes* can grow easily in liquid disinfectants (3). Although there is a big knowledge gap about *Chryseobacterium* spp. epidemiology, it was reported that the highest frequency of *Chryseobacterium* spp. infection occurred among elderly people (≥65 years old) and the lowest frequency occurred among children under 5 years of age. (4) Most cases of infections caused by *C. indologenes* in the pediatric population have been reported from neonate with congenital heart diseases or solid tumors. (5). In this report, we describe a case of blood stream infection due to *C. indologenes* in a low birth weight neonate, small for gestational age with moderate respiratory distress syndrome and thrombocytopenia.

CASE PRESENTATION

A late preterm female infant of a gestational age of 35 weeks and birth weight of 1300 g was delivered through a cesarean

section owing to impending eclampsia in the mother. She was transferred to the neonatal intensive care unit (NICU) because of labored breathing and chest indrawing. No history of amniotic fluid leak or maternal fever was recorded. The heart rate and respiratory rate on admission to the NICU were 164/min and 64/min, respectively. SPO₂ was 90%. APGAR score was 7 at 1min, and 9 at 5 min. Bilateral air entry equal on both sides. A Silverman–Anderson distress score of 6.



Image no. 1. Yellow pigmented colonies on nutrient agar

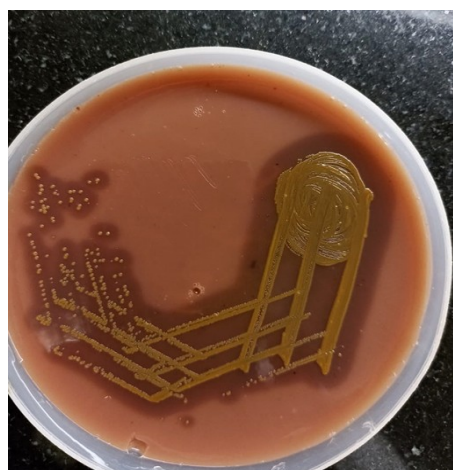


Image no. 2. Beta hemolysis on blood agar



Image no. 3 – Yellow pigmented colonies on blood agar



Image no. 4. MH Agar plate showing sensitivity to vancomycin and Resistance to polymixin B



Image no. 5. Pigmented Production after pouring 10% KOH

indicating moderate respiratory distress. The mask continuous positive airway pressure (CPAP) mode of ventilation was used. At the time of admission, the total leukocyte count was 13,500/mm³ with 49% neutrophils and 39% lymphocytes. Platelet count was 1,55,000/cumm. The neonate showed feed intolerance at 48 h of life and was kept nil orally for the next 72h. The neonate was provided with maintenance intravenous fluids, stomach wash (thrice daily) in view of vomiting, Inj. Vit. K 1mg IV twice daily, INJ. Ampicillin 65 mg IV two divided doses and INJ. Gentamicin 3mg IV BD empirically, as per NICU policy. Fresh Frozen Plasma transfusion was given for thrombocytopenia. Respiratory secretions, oxygen requirements, and CPAP demands decreased after 72h of life. Inj. Ampicillin stopped and INJ. Piperacillin-tazobactam 130 mg IV TDS & Inj. Metronidazole 15 mg IV TDS was started. The neonate maintained saturation on CPAP and was shifted to nasal prong. ABG (Arterial blood gas monitoring) was done. Off O₂ trial was given and started on RT feed. On 12th day of life patient was icteric with total bilirubin 8.7mg/dl and decreased lymphocytic count (1300/ul) in view of which blood culture was sent. The neonate's blood culture on chocolate agar showed 1-2 mm in size smooth, circular yellow pigmented colonies with beta hemolysis after 18-24 hrs. of incubation. Nutrient agar showed the growth of smooth and circular 1-2 mm yellow-pigmented colonies after 24 h. Circular, low-convex, smooth, mucoid colonies of 1-2 mm

with beta-hemolysis developed on 5% sheep blood agar. However, when cultured on MacConkey agar, growth was observed after 96 hrs. of incubation as yellow colored sticky colonies. Biochemical reactions showed the oxidase-positive, catalase-positive, nonmotile, glucose non-fermenting, indole - positive, Methyl red negative, VP-Negative, Urease - negative with bile esculin positive. On Mueller Hinton agar isolate showed sensitivity to vancomycin and resistance to polymyxin B. (See Image no. 4). When 10% KOH was gradually poured on the yellow colonies, they turned reddish orange because of the production of water-insoluble yellow pigment flexirubin (6) (See image no. 5). Using conventional identification methods and the VITEK 2 system (bioMérieux India Pvt. Ltd., New Delhi, India), the isolate was identified as *C. indologenes* (1). Antimicrobial susceptibility testing was performed by both determining the minimal inhibitory concentration (MIC) value using the micro-broth dilution method and measuring the inhibition zone diameter on Mueller–Hinton agar medium aerobically at $35 \pm 2^\circ\text{C}$ for 18–24 h incubation by using Kirby–Bauer's disk diffusion method using Clinical and Laboratory Standards Institute guidelines (CLSI-2024) protocols for other non-enterobacteriaceae, non-fermenters other than *Pseudomonas aeruginosa* and members of the genera *Stenotrophomonas* and *Burkholderia* were used as reference for interpreting the results (7).

Table 1. Antimicrobial susceptibility of *Chryseobacterium indologenes* isolated from the patient's blood culture.

1	Piperacillin/Tazobactam	Resistant
2	Ceftazidime	Resistant
3	Cefoperazone/Sulbactam	Resistant
4	Cefepime	Resistant
5	Imipenem	Resistant
6	Meropenem	Resistant
7	Amikacin	Resistant
8	Gentamicin	Resistant
9	Aztreonam	Resistant
10	Ciprofloxacin	Intermediate
11	Levofloxacin	Sensitive
12	Minocycline	Sensitive
13	Trimethoprim/ Sulfamethoxazole	Sensitive

Patient's antibiotic treatment was changed to inj. levofloxacin 15 mg IV BD according to culture and sensitivity report and continued for 5 days. Patient's condition improved and she was discharged after completion of the antibiotic treatment.

DISCUSSION

We summarized the published (reported) clinical cases in the neonatal age group Table 2.

Age/Sex	Underlying condition	Medical Device	Infection Type	Treatment	Outcome	Reference
6days/F	SGA	None	Meningitis, Bacteremia	Ciprofloxacin, TMP-SMX	Survived	[8]
18 days/M	Congenital Diaphragmatic Hernia	Oscillatory Ventilator	VAP	Levofloxacin, TMP-SMX	Survived	[9]
36 weeksNB/M	SGA	Ventilator	Bacteremia	Cefoperazone-sulbactam	Survived	[10]
20 days/M	Complex CHD	Ventilator	VAP	Piperacillin-tazobactam	Survived	[5]
Full term NB/F	Respiratory Distress	Ventilator	VAP	Cefepime	Survived	[11]
10 days/F	Complex CHD	Central catheter	Bacteremia	Ciprofloxacin, Imipenem	Survived	[12]
8 days/F	None	None	Meningitis	Cefepime	Survived	[13]
32 week NB/M	Prematurity /RDS	CPAP	Bacteremia	Ciprofloxacin, Cefoperazone-Sulbactam	Survived	[14]
35 weeks NB	IUGR/RDS/SGA/Thrombocytopenia	CPAP and Ryle's Tube	Bacteremia	Levofloxacin	Survived	Present case

Abbreviations:

SGA: small for gestational age, **CPAP:** continuous positive airway pressure, **VAP:** ventilator-associated pneumonia

TMP–SMX: Trimethoprim–Sulfamethoxazole, **RDS:** Respiratory Distress Syndrome.

C. indologenes although uncommon, is an important pathogen causing infection in hospitalized patients. *C. indologenes* is resistant to chlorination and can survive in the water supplied from municipal sources (3). The pathogenicity of *C. indologenes* is not well established, however, biofilm formation and proteases production are important mechanisms involved in its pathogenesis (15).

Chryseobacterium grows on wet and humid surfaces in hospitals and in catheters containing fluids, such as central venous catheter, tracheostomy tubes, and feeding tubes (14). Production of β -lactamases makes this organism resistant to most β -lactam drugs, including the carbapenems, aztreonam, aminoglycosides, chloramphenicol, linezolid, and glycopeptides (16). *C. indologenes* shows maximum susceptibility to the newer quinolones like levofloxacin, and cotrimoxazole (95%). (14). *C. indologenes* infections were generally nosocomial and frequently associated with invasive devices, immune compromised status and prolonged treatment with broad-spectrum antibiotics (4). The isolates were consistently resistant to β -lactam antibiotics, aminoglycosides, erythromycin, clindamycin or teicoplanin. Susceptibility to trimethoprim-sulfamethoxazole (TMP-SMX), ofloxacin and ciprofloxacin was variable (3).

In our case, the isolate was resistant to piperacillin/tazobactam and cefoperazone -sulbactam, Imipenem. Intermediate for ciprofloxacin but was sensitive to other antibiotics, such as levofloxacin, Minocycline, TMP-SXM as shown in Table 1.

Our patient responded well to Levofloxacin. It is therefore recommended that clinicians should rely on the MIC values in the culture report to treat patients with *C. indologenes* infections, and microbiology laboratories should report the MIC values of ciprofloxacin along with those of newer quinolones like levofloxacin (14). In hospitals where colistin and tigecycline use is high for coverage of carbapenem-resistant pathogens, *C. indologenes* is emerging as a health care-associated pathogen (HCAP). (17)

CONCLUSION

In conclusion, this case shows that *C. indologenes* is one of the causative agent of hospital acquired infections causing neonatal sepsis in neonates who receive long-term broad-spectrum antibiotics and in those where invasive medical equipment used. The management of this infection needs better identification, susceptibility testing and monitoring of patients with long hospital stays. Selecting an effective drug for the empirical therapy of the infections caused by *C. indologenes* is difficult owing to its narrow antibiotic spectrum susceptibility. The spread of this Health Care Associated Pathogen can be controlled by strict compliance with hand hygiene and infection control practices. This report showed the importance of survey of environmental bacteria which can cause hospital-acquired infection.

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