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Case report

Chryseobacterium gleum pneumonia in an infant with nephrotic syndrome

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ABSTRACT

Introduction: Chryseobacterium gleum is commonly distributed in the environment. It can cause a wide variety of infections in immunocompromised patients in hospital setting.
Case presentation: A 6 month old infant with nephrotic syndrome was admitted to the emergency room for an acute onset of fever, difficulty breathing, cyanosis, and low oral intake. Cultures of endotracheal tube specimens were positive for *Chryseobacterium gleum* which was confirmed by ribosomal sequencing. The organism was susceptible to trimethoprim-sulfamethoxazole, minocycline, and levofloxacin. The patient clinically improved on levofloxacin treatment.
Conclusion: To the best of our knowledge, this is the first case of pneumonia caused by *Chryseobacterium gleum* in an infant with nephrotic syndrome. It is also the first report of *C. gleum* causing respiratory tract infection in Saudi Arabia.

Introduction

Chryseobacterium species are widely distributed in the environment. They cause infections in hospitalized patients with underlying conditions including immunocompromised patients [1–3]. Age (infants, elderly) and medical devices such as mechanical ventilators or indwelling intravascular devices are also common risk factors [1,4].

Chryseobacterium species, formerly known as *Flavobacterium*, belong to the family of Flavobacteriaceae [5]. CDC group IIb comprises *Chryseobacterium indolgenes* and *C. gleum* and other strains [5]. They are aerobic, yellow pigmented on blood agar, catalase positive, oxidase positive, non motile, non glucose fermenting Gram negative bacilli on MacConkey agar. These strains can be differentiated based on DNA sequencing, several phenotypic testing, and MALDI-TOF MS [5,6].

Although less common than *C. indolgenes*,*C. gleum* has been reported to cause wide variety of infections including respiratory tract infections, urinary tract infections, pyonephrosis, septicemia, meningitis, wound infections, and peritonitis [1,3,7–9]. Infections caused by *C. gleum* had been reported in several countries including India, Hungary, Croatia, Qatar, and Taiwan [1,6–9]. *Chryseobacterium* spp are resistant to several antibiotics such as aminoglycosides, chloramphenicol, tetracyclines, clindamycin, teicoplanin, and erythromycin [1,10,11]. In addition, these strains chromosomally encode class A carbapenemases and class B metallo beta lactamases which confer resistance to all β-lactams [12].

Case report

A 6 month old infant brought to the emergency room in the King Fahad Specialist Hospital-Dammam, Saudi Arabia with cough, fever, difficulty breathing, sneezing, irritation, excessive crying, decreased oral intake, and cyanosis. He had been diagnosed with infantile nephrotic syndrome 5 months earlier. In his past medical history, he had several episodes of septicemia caused mainly by *Pseudomonas aeruginosa*. In addition, he was previously on multiple courses of antibiotics including meropenem, ceftriaxone, and vancomycin. He was intubated and ventilated. Blood specimens revealed mild neutrophilia and elevated CRP. Blood culture and urine culture were negative. Chest X rays revealed left sided pleural effusion with left lower lobe opacities. Several respiratory specimens from endotracheal tube (ETT) were submitted to the microbiology laboratory for culture. Round yellow pigmented non hemolytic colonies grew on blood agar plates. Gram stain revealed Gram negative bacilli. The organism was catalase and oxidase positive. Organism identification and antimicrobial susceptibility

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testing were carried out using the Vitek 2 automatic system (bioMerieux, Paris, France) according the manufacturer's instructions. *E. coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 strains were used as controls for the antimicrobial susceptibility testing. There are no Clinical and Laboratory Standards Institute (CLSI) guidelines specific for *Chryseobacterium* species. However, minimum inhibitory concentrations (MIC) and breakpoints were determined according to the CLSI recommendation for other Non-Enterobacteriaceae. The organism was identified as *Chryseobacterium gleum* and it was confirmed using API20NE kit and 16 S rRNA sequencing. The organism was resistant to ceftazidime (MIC $\geq$ 64 μg/mL), cefepime (MIC $\geq$ 64 μg/mL), meropenem (MIC $\geq$ 16 μg/mL), piperacillin-tazobactam (MIC $\geq$ 128 μg/mL), colistin (MIC $\geq$ 8 μg/mL), gentamicin (MIC $\geq$ 16 μg/mL), and amikacin (MIC $\geq$ 64 μg/mL). It was intermediate to imipenem (MIC = 8 μg/mL), ciprofloxacin (MIC = 2 μg/mL), and tigecycline (MIC = 4 μg/mL). The organism was susceptible to trimethoprim-sulfamethoxazole (MIC $\leq$ 20 μg/mL), minocycline (MIC $\leq$ 1 μg/mL), and levofloxacin (MIC = 0.5 μg/mL). In addition, the isolate was resistant to vancomycin. Environmental samples from the mechanical ventilator and the patient room did not grow any *Chryseobacterium* isolate. *Chryseobacterium* was tested for the presence of carbapenem resistant genes (OXA48, NDM1, IMP, VIM, CTX-14, CTX-M15, and KPC) by multiplex PCR methodology using ARM-D for β-Lactamase ID kit (Streck, Omaha, NE, USA) as instructed by the manufacturer. Positive controls and an internal control are included in the kit. In addition, molecular grade water (Promega, WI, USA) was used as a negative control to detect contamination. None of the tested genes was detected by PCR. The patient improved on levofloxacin for a period of 16 days and subsequent respiratory specimens did not grow *C. gleum*. Neither trimethoprim-sulfamethoxazole nor minocycline was used since they were not available in hospital at time of treatment.

Discussion

A case of pneumonia caused by *C. gleum* in an infant with nephrotic syndrome is reported in this study. To the best of our knowledge, this is the first report of respiratory tract infection due to *C. gleum* in Saudi Arabia. It is also the first report of association between *Chryseobacterium* infection and nephrotic syndrome. This is a noteworthy case since *Chryseobacterium* species are ubiquitous in nature, are not part of human flora, and can cause infections in hospitalized patients with underlying disease [1,2,4]. In addition, they are resistant to chlorination and are found on wet services of medical devices and water systems. Therefore, medical devices such as humidifiers, intubation tubes, and ventilators are important sources for nosocomial infections caused by *Chryseobacterium* spp [1,13]. VAP incidence is estimated to be 10–25% in Saudi Arabia and is associated with 25–50% mortality rates of these cases [14]. Environmental and mechanical ventilator samples did not grow any *C. gleum* in this case. However, intubation is a well known risk factor for VAP caused by *Chryseobacterium*. Therefore, it should be considered in the differential diagnosis as a causing agent whenever a VAP case is encountered.

Patients with nephrotic syndrome suffer from infections due to the loss of proteins including immunoglobulins in urine [15]. This may explain in part why the patient developed pneumonia and previous multiple episodes of septicemia. This study suggests that nephrotic syndrome can be a risk factor for *C. gleum* infections. *C. gleum* has the ability of biofilm formation which can play a significant role in the establishment of infections and resistance to wide variety of antimicrobial agents [7].

The choice of antibiotic agent for treatment is not well established since there are insufficient data regarding the minimum inhibitory concentration profile. In addition, there are no CLSI guidelines for *Chryseobacterium* [10,11]. According to report from the SENTRY antimicrobial surveillance program (1997–2001), *Chryseobacterium* represented 0.27% of the studied non fermentative Gram-negative rods and 0.03% of all bacterial isolates [10]. Trimethoprim-sulfamethoxazole and newer generations of quinolones (levofloxacin, garenoxacin, and gatifloxacin) were the most active agents against *Chryseobacterium* [10]. Our data correlated with this study where *C. gleum* strain was susceptible to levofloxacin and trimethoprim-sulfamethoxazole. However, resistance to piperacillin-tazobactam and susceptibility to minocycline in our study contradict data reported in the SENTRY study regarding these two antibiotics [10].

There are discrepant data regarding *Chryseobacterium* susceptibility to vancomycin. Several reports suggested susceptibility of *Chryseobacterium* to vancomycin while other reports revealed resistance to vancomycin [1,4,13]. In this study, the organism was resistant to vancomycin in vitro.

This report highlights the importance of *Chryseobacterium* species as a causing agent of the wide variety of infections. In addition, it emphasizes the significance of establishing antimicrobial susceptibility testing guidelines for the genus *Chryseobacterium*.

Conflict of interests

The authors declare that they have no competing interests.

Authors' contribution

Baha Abdalhamid and Reem Aljindan reviewed the literature and prepared the manuscript. Nasreldin Elhadi and Khaldoon Alsamman performed the technical work. All authors approved the final version of the manuscript.

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References

[1] Virok DP, Abrok M, Szel B, Tajti Z, Mader K, Urban E, et al. *Chryseobacterium gleum*—a novel bacterium species detected in neonatal respiratory tract infections. J Matern-Fetal Neonatal Med 2014;27:1926–9.
[2] Christakis GB, Perlorentzou SP, Chalkiopoulou I, Athanasiou A, Legakis NJ. Chryseobacterium indologenes non-catheter-related bacteremia in a patient with a solid tumor. J Clin Microbiol 2005;43:2021–3.
[3] Garg S, Appannanavar SB, Mohan B, Taneja N. Pyonephrosis due to *Chryseobacterium gleum*: a first case report. Indian J Med Microbiol 2015;33:311–3.
[4] Chen FL, Wang GC, Teng SO, Ou TY, Yu FL, Lee WS. Clinical and epidemiological features of *Chryseobacterium indologenes* infections: analysis of 215 cases. J Microbiol Immunol Infect = Wei mian yu gan ran za zhi 2013;46:425–32.
[5] Bernardet JF, Nakagawa Y, Holmes B. Subcommittee on the taxonomy of F, Cytophaga-like bacteria of the International Committee on Systematics of P. Proposed minimal standards for describing new taxa of the family Flavobacteriaceae and emended description of the family. Int J Syst Evol Microbiol 2002;52:1049–70.
[6] AbdulWahab A, Taj-Aldeen SJ, Ibrahim EB, Talaq E, Abu-Madi M, Fotedar R. Discrepancy in MALDI-TOF MS identification of uncommon Gram-negative bacteria from lower respiratory secretions in patients with cystic fibrosis. Infect Drug Resist 2015;8:83–8.
[7] Lo HH, Chang SM. Identification, characterization, and biofilm formation of clinical *Chryseobacterium gleum* isolates. Diagn Microbiol Infect Dis 2014;79:298–302.
[8] Brkic DV, Zlopaša O, Bedenic B, Plecko V. *Chryseobacterium gleum* infection in patient with extreme malnutrition and hepatic lesion-case report. Signa Vitae 2015;10:50–2.
[9] Ramya TG, Baby S, Das P, Geetha RK. *Chryseobacterium gleum* urinary trasct infection. Genes Rev 2015;1:1–5.
[10] Kirby JT, Sader HS, Walsh TR, Jones RN. Antimicrobial susceptibility and epidemiology of a worldwide collection of *Chryseobacterium* spp: report from

the SENTRY Antimicrobial Surveillance Program (1997–2001). J Clin Microbiol 2004;42:445–8.
[11] Fraser SL, Jorgensen JH. Reappraisal of the antimicrobial susceptibilities of Chryseobacterium and Flavobacterium species and methods for reliable susceptibility testing. Antimicrob Agents Chemother 1997;41:2738–27341.
[12] Bellais S, Naas T, Nordmann P. Molecular and biochemical characterization of Ambler class A extended-spectrum beta-lactamase CGA-1 from Chryseobacterium gleum. Antimicrob Agents Chemother 2002;46:966–70.
[13] Calderon G, Garcia E, Rojas P, Garcia E, Rosso M, Losada A<it&gt. Chryseobacterium indologenes infection in a newborn: a case report. Journal of medical case reports 2011;5:10.
[14] Al-Omari A, Mohammed M, Alhazzani W, Al-Dorzi HM, Belal MS, Albshabshe AO, et al. Treatment of ventilator-associated pneumonia and ventilator-associated tracheobronchitis in the intensive care unit. A national survey of clinicians and pharmacists in Saudi Arabia. Saudi Med J 2015;36:1453–62.
[15] Orth SR, Ritz E. The nephrotic syndrome. N Engl J Med 1998;338:1202–11.