Case Report

Chryseobacterium indolegenes infection in a patient with chronic obstructive pulmonary disease

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Summary Chryseobacterium indolegenes is a rare pathogen that causes a variety of infections in inviduals who are mostly hospitalized with severe underlying diseases. Here we present a case of C. indolegenes in a 69-year-old male with chronic obstructive pulonary disease (COPD) who was admitted to the chest disease outpatient clinic with symptoms like cough, fever and sputum production and followed up on a suspicion of pneumonia. Despite the fact that our patient did not have any history of hospitalization for at least one year, pneumonia cause was due to C. indolegenes. Clinicians should pay attention to the rare pathogens such as C. indologenes while managing COPD patients without prior hospitalization history.

> Keywords: Chryseobacterium indologenes, chronic obstructive pulmonary disease, penumonia, multidrug resistant

1. Introduction

Chryseobacterium genus belong to Flavobacteriaceae family and it is firstly described in 1994 (1). Chryseobacterium spp. is a catalase positive, indole positive, oxidase positive, non-glucose fermenting, aerobic Gram negative bacilli. C. indologenes is not a part of human microflora (2). It is widely distributed in nature primarily in soil and water sources. It was reported that it can survive even in chlorine-treated water, so can be a good source for healthcare associated infection (3). The infections due to C. indologenes are mostly associated with long term hospitalization, especially in patients who are immunocompromised, using medical devices (respirators, humidifiers,

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intravascular catheters, intubation tubes, etc.) and subject to prolonged exposure to broad spectrum antibiotics (4,5). In this case report, we report a C. indolegenes which was isolated from a 69 year old male with chronic obstructive pulmonary disease (COPD) admitted to the hospital with cough, fever, and sputum production. Authors emphasize that C. indologenes must be kept in mind as a cause of infection in chronic diseases like COPD.

2. Case Report

In this study, a 69-years-old male with COPD was admitted to the chest disease outpatient clinic with symptoms such as cough, fever, and sputum production and followed up on suspicion of pneumonia. The patient had no history of hospitalization at least for one year. First of all laboratory tests indicated a C-reactive protein (CRP) level of 17,71 mg/L (reference range, 0-5 mg/L) and a white blood count (WBC) of 17,600/ mm³ with 77.8% neutrophils. After samples were taken for blood and sputum cultures, emprical treatment was started with imipenem and levofloxacin. Yellowpigmented Gram negative bacilli colonies were isolated

from a sputum sample after 24 hours incubation in a 5% sheep blood agar. The microorganism was found as oxidase positive and non-lactose fermenting. The isolated bacterium was identified as C. indologenes by VITEK2 identification and antibiogram system (bioMerieux, Nürtingen, Germany). The strain was found to have intermediate resistance to levofloxacin 4 µg/mL, cefoperazone-sulbactam 32 µg/mL and resistant to ampicillin $\geq 32 \ \mu g/mL$, trimethoprimsulfamethoxazole $\geq 320 \ \mu g/mL$, cefuroxime ≥ 64 μ g/mL, cefoxitin \geq 64 μ g/mL, tobramycin \geq 16 μ g/ mL, ampicillin/sulbactam $\geq 32 \ \mu g/mL$, piperacillin \geq 128 µg/mL, piperacillin-tazobactam \geq 128 µg/ mL, ceftazidime $\geq 64 \ \mu g/mL$, cefepime $\geq 64 \ \mu g/mL$ mL, imipenem $\geq 16 \ \mu g/mL$, meropenem $\geq 16 \ \mu g/mL$ mL, amikacin $\geq 64 \ \mu g/mL$, ciprofloxacin $\geq 4 \ \mu g/mL$, tetracyline \geq 16 µg/mL, tigecycline \geq 8 µg/mL, colistin \geq 16 µg/mL, amoxicillin/clavulanic acid \geq 32 µg/mL.

The clinical findings, growing of *C. indologenes* in sputum culture, high serum CRP level and increased WBC and neutrophil count lead the clinician to the diagnosis of pneumonia. Imipenem treatment was stopped and treatment was continued with levofloxacin (500 mg/IV). The clinical and laboratory findings of patient improved and there was no growth in control cultures after 14 days of treatment.

3. Discussion

COPD is a progressive lung disease which is characterized by airflow obstruction that is progressive and partly reversible. It is associated with abnormal inflamatory responses which are triggered by noxious particles or gases. A rapid decline in clinical status of COPD occur by exacerbations which are associated with microbial and airway inflammation (6). According to the Guidelines for management of COPD the impact of exacerbations could be minimised by using appropriate treatment with oral steroids and/or antibiotics. Up to now no sufficient evidence is found to begin prophylactic antibiotic therapy for managing stable COPD (7). In the literature Haemophilus influenzae, Moraxella catarrhalis, Streptococcus pneumoniae, Pseudomonas aeruginosa, Klebsiella pneumoniae, Haemophilus parainfluenzae, Serratia marcessens, Acinetobacter spp. are bacterial pathogens isolated from patients experiencing exacerbation of COPD (8,9). In our case we identified an uncommon pathogen, C. indologenes, in our COPD patient.

The natural habitat of *Chryseobacterium* spp. is water, soil, foodstuffs and plants. They are not a part of normal human flora (4). It was reported that *C. indologenes* are responsible from various clinical conditions, such as bacteremia, sepsis, pneumonia, shunt infection, urinary tract infection, infection of the central nervous system (10-15). It was reported that some underlying conditions such as indwelling devices, malignancies, hypertension diabetes mellitus lead to severe infections in hospitalized patients (10). Although it is rising importance in healthcare associated infections, there is no guideline for management of C. *indologenes* infections (3,4).

Although being low-virulent, they may cause serious infections in patients with underlying conditions such as long term hospitalization, being immunocompromised, use of medical devices (respirators, humidifiers, intravascular catheters, incubation tubes, *etc.*) and prolonged exposure to broad spectrum antibiotics (5,10). Despite the fact that our patient had no history of hospitalization for at least one year, pneumonia cause was found to be *C. indolegenes*.

Chryseobacterium is intrinsically resistant to carbapenems and cephalosporins *via* class A beta lactamase and class B carbapenem hydrolyzing beta lactamase activity. According to literature *C. indologenes* is frequently resistant to aminoglycosides, chloramphenicol, linezolid, and glycopeptides and susceptible to levofloxacin, ciprofloxacin, trimethoprim-sulfamethoxazole and piperacillin-tazobactam (5,10). In our case the strain was found to have intermediate resistance to levofloxacin and sefaperazon-sulbactam whereas it was resistant to other tested antibiotics.

In conclusion, surveillance programs are needed to delineate the suitable antimicrobial therapy for rarely isolated pathogens like *C. indologenes* and clinicians should keep in mind the rare pathogens while managing COPD patients without prior hospitalization history.

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