CASE REPORT

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A Case of Pneumonia Caused by *Raoultella planticola*

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Raoultella species are gram-negative, non-motile, aerobic bacilli that are primarily considered as environmental bacteria. *Raoultella planticola* is reportedly a rare cause of human infections. Also, the definite pathological mechanism of *Raoultella planticola* is currently unknown. We report a case of pneumonia caused by *Raoultella planticola*.

Keywords: Environment; Pneumonia

Introduction

Raoultella planticola is a gram-negative, non-motile, aerobic bacillus that is primarily considered as environmental bacteria[1]. *R. planticola* was previously described as *Klebsiella planticola* and *K. trevisanii*, were combined into one species in 1986, i.e., *K. planticola*, based on DNA-DNA homology. In 2001, *K. planticola* was renamed *R. planticola* based on 16S rRNA and rpoB gene sequencing[2].

R. planticola does not typically cause invasive infections in humans; furthermore, current literature suggests that the bacteria is a rare cause of human infections. In the present report, we describe a patient with pneumonia due to primary infection by *R. planticola*.

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Case Report

A 58-year-old man visited our hospital with cough, purulent sputum, and dyspnea since a month prior. The patient presented no medical history except a 30 pack-year smoking history.

On admission, the patient's vital signs were as follows: blood pressure 100/70 mm Hg, pulse rate 92 beats per minute, body temperature 37.4°C, and respiratory rate 35 breaths per minute. Auscultation of lungs revealed coarse breathing sounds with crackle and wheezing on both lower lung fields. The oxygen saturation was 94%, pH 7.48, PaO_2 63.0 mm Hg, and $PaCO_2$ 32.0 mm Hg while the patient was breathing ambient air. A complete blood count showed a white blood cell (WBC) count of 6,920/mm^3 (neutrophils 70.9%, lymphocytes 21.9%, monocytes 20.9%, and eosinophils 9.8%), a hemoglobin of 12.4 g/dL, and platelets 198,000/mm^3. Serum chemistry showed blood urea nitrogen 18.6 mg/dL, creatinine 1.0 mg/dL, aspartate aminotransferase 158 IU/L, alanine aminotransferase 439 IU/L, alkaline phosphatase level of 386 IU/L, and a C-reactive protein 7.0 mg/dL. The following day, WBC increased to 19,220/mm^3 with neutrophil 91.4%; and C-reactive protein increased to 8.5 mg/dL. An electrocardiogram revealed normal sinus rhythm and cardiac enzyme within was normal range.

Plain chest radiography on admission showed cardiomegaly and consolidation on both lung fields (Figure 1). Contrast-enhanced chest computed tomography demonstrated diffuse ground glass opacity and consolidation in both lungs. Underlying emphysematous and bullous changes were also noted predominantly in the lower lobes (Figure 2). An echocardiography showed an ejection fraction of 10% and severe left ventricular systolic dysfunction with no regional wall motion

abnormality.

Initially, we suspected bacterial pneumonia and heart failure consequent to pulmonary edema. Empirically, levofloxacin, piperacillin-tazobactam, and diuretics were started intravenously. At 7 days follow-up after admission, 2 sputum cultures showed *R. planticola* with showed sensitivity to most antibiotics except for ampicillin and ciprofloxacin; no different bacteria were detected. Blood cultures were sterile. We diagnosed a pneumonia caused by *R. planticola*. We stopped levofloxacin based on the sputum result and continued piperacillin-tazobactam treatment.

Seven days after hospitalization, dyspnea had improved and cough and sputum production was reduced. Fifteen days after admission, laboratory analysis was as follows: a WBC count of 5,420/mm^3 (neutrophils 55.9%) and C-reactive protein 0.26 mg/dL. At 3 weeks follow-up after admission, chest radiography showed decrease in both ground glass opacity and consolidation in both lung fields; in addition, contrast-enhanced chest computed tomography showed improvement of pneumonia (Figure 3). The patient continued antibiotic therapy for 4 weeks and was discharged. Since discharge, he has been followed up at an outpatient clinic without evidence of recurrence.

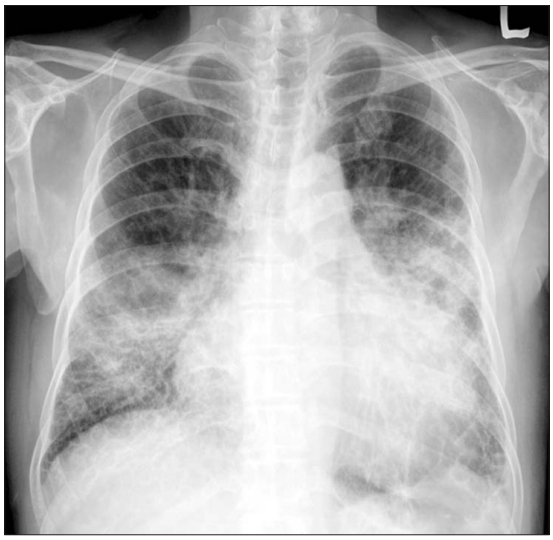

Figure 1. Chest radiography showed cardiomegaly and consolidation on both lung fields.

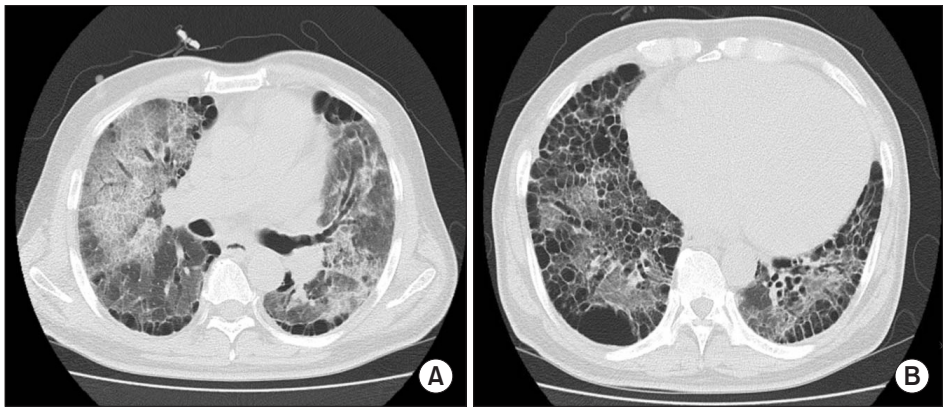

Figure 2. Chest computed tomography demonstrated diffuse ground glass opacity and consolidation in both lungs (A). Underlying emphysematous and bullous changes are also noted predominantly in both lower lobes (B).

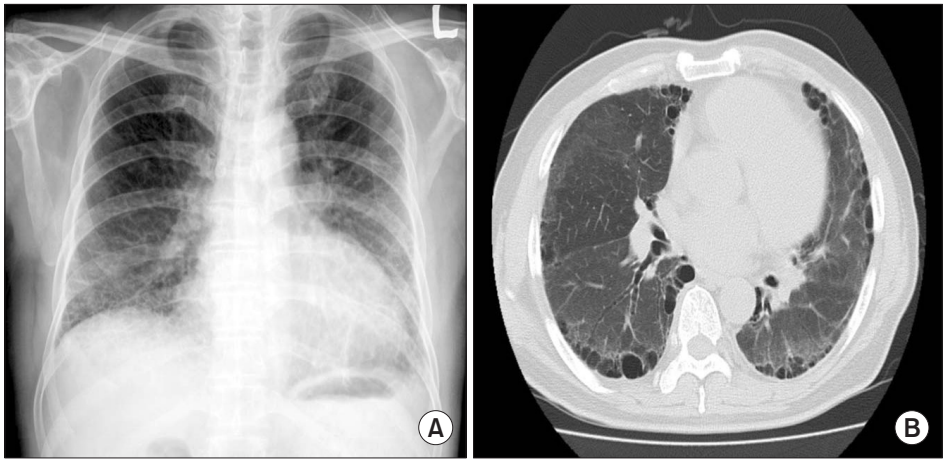

Figure 3. Follow-up, chest radiography (A) and computed tomography (B) showed improvement of ground glass opacity and consolidation in both lung fields.

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Discussion

R. planticola is an aquatic, botanical and soil organism that does not typically cause invasive infections in humans. *Raoultella* species produce histidine decarboxylase and have been implicated in scombroid (histamine) fish poisoning, but the clinical significance of this organism in humans has not been characterized[3].

R. planticola is found to have similar pathogenetic features as *Klebsiella* species. *Klebsiella* spp. are associated with severe infections in hospitalized and immunocompromised patients, including bacteremias, pneumonias, and urinary tract infections, and have been estimated to cause between 3% and 7% of all nosocomial infections[2]. In 2000, Podschun et al. evaluated the common virulence factors between *R. planticola* and the *Klebsiella* spp.[2] Specifically, both *K. pneumoniae* and *R. planticola* exhibit type 1 fimbriae and mannose-sensitive hemagglutination. *Klebsiella* spp. have the ability to resist the bactericidal effects of human serum, a feature that is shared by *R. planticola*[2].

R. planticola is a rare cause of human infections and only 18 cases have been reported worldwide. The first case report was described by Freney et al.[4] in Lyon, France, of 69-year-old patient with *R. planticola* bacteremia who was admitted to an intensive care unit 9 days following a mitral valve replacement.

Extensive literature search, indicated a total of eighteen cases of *R. planticola* infections, including infections with *K. planticola* and *K. trevisanii* (Table 1). This case reports included one cholecystitis[1], two urinary tract infections[2,5], seven bacteremias[2,4,6-8], one central line infection[3], three soft tissue infections[9-11], one pancreatitis[12], two cholangitis[13,14], and one pneumonia[15]. Of the eighteen cases, four case patients died and the others reportedly recovered. This is the first case in Korea of community-acquired pneumonia with *R. planticola*.

Previous reports, suggested the potential risk factors as invasive medical procedures, and trauma with potential soil contamination, with significant comorbidities except for 1 case[1]. A rare case of cholecystitis caused by *R. planticola* was reported in 2012. Interestingly, the patient in this report did not have any of these risk factors. In our case, distinguishing between pathogen and colonization was difficult; however, except for *R. planticola*, no any other bacteria grew in sputum culture. Furthermore, the sputum grade was group 5 (leukocyte >25/low power field [LPF], epithelial cell <10/LPF) and a predominant gram-negative bacilli by Gram stain, and pathogen quantity was moderate to heavy. Hence, it is likely that *R. planticola* developed pneumonia due to pathogenic action.

The natural susceptibility of 221 *Klebsiella* strains to 71 an-

Table 1. Summary of the reported cases of Raoultella planticola infections

Authors	Age/Sex	Clinical	Treatment	Outcome
Teo et al.[1]	62 yr/F	Cholecystitis	Amoxicillin/clavulanic acid	Recovered
Olson et al.[2]	89 yr/M	Cystitis	Ciprofloxacin	Recovered
Olson et al.[2]	57 yr/M	Bacteremia	Ceftriaxone	Recovered
Olson et al.[2]	69 yr/M	Bacteremia	Ceftriaxone, tobramycin	Recovered
Nada and Areej[3]	15 mo/F	Central line infection	Ampicillin, gentamicin, metronidazole	Recovered
Freney et al.[4]	47 yr/M	Bacteremia	Cefotaxime, tobramycin	Recovered
Gangcuangco et al.[5]	92 yr/F	UTI	Ceftriaxone	Recovered
Castanheira et al.[6]	83 yr/F	Pneumonia, bacteremia	Carbapenem	Died
Castanheira et al.[6]	64 yr/M	Bacteremia	Imipenem/doxycycline	Died
Hu et al.[7]	59 yr/M	Bacteremia	Piperacillin-tazobactam	Recovered
Puerta-Fernandez et al.[8]	63 yr/M	Bacteremia	Cefotaxime	Recovered
Kim et al.[9]	66 yr/M	Necrotizing fasciitis	Ceftriaxone, levofloxacin	Recovered
Wolcott et al.[10]	66 yr/M	Surgical site infection	Etrapenem	Recovered
O'Connell et al.[11]	30 yr/F	Cellulitis	Clindamycin, ciprofloxacin	Recovered
Alves et al.[12]	45 yr/M	Pancreatitis	Imipemen, amikacin	Recovered
Lee et al.[13]	75 yr/M	Cholangitis	Cefotaxime, metronidazole	Died
Yokota et al.[14]	65 yr/M	Cholangitis, bacteremia	Meropenem	Recovered
Xu et al.[15]	60 yr/M	Pneumonia	Imipenem/cilastatin	Died
Present case	58 yr/M	Pneumonia	Piperacillin-tazobactam, levofloxacin	Recovered

UTI: urinary tract infection.

tibiotics was examined. *R. planticola* were sensitive to different penicillins except oxacillin, amoxicillin. Also, *R. planticola* were sensitive to all cephalosporins and carbapenems[9]. Recently, the emergence of carbapenem resistance *R. planticola* has been sporadically reported[5,15]. The known mechanism of carbapenem resistance in *R. planticola* is production of carbapenemases, including class A β-lactamase (KPC), class B metal-β-lactamase (IMP-8, NDM-1), and class D β-lactamase (OXA-48). It is notable that bla_{KPC}, bla_{IMP-8}, and bla_{NDM-1} were usually located on plasmids or transposons, suggesting possible gene exchange between *R. planticola* and other Enterobacteriaceae such as *K. pneumoniae*[15]. Although most of the *R. planticola* isolates published in literature are susceptible to carbapenems[5], a study on the antimicrobial resistance of *R. planticola* is also required.

In conclusion, *R. planticola* is an environmental bacterium that can cause serious infections in humans. Commonly, it does not cause human infections in the normal state, but human infections are in invasive medical procedures, trauma with potential soil contamination, significant comorbidities or with the decline of immunization state. Our case was meaningful in that community-acquired *R. planticola* infection was developed in a normal state. Elucidating the mechanism of action of *R. planticola* in human infections is difficult. However, as new laboratory techniques become more common, the mechanism of action of *R. planticola* in human infections is likely to be revealed.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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