Bacteremic cholangitis due to *Raoultella planticola* complicating intrahepatic bile duct stricture 5 years post-laparoscopic cholecystectomy: a case report

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**Abstract**

**Background:** *Raoultella Planticola* is a facultative anaerobic, gram-negative, water- and soil-dwelling rod bacterium rarely reported as a cause of human disease. However, the number of reported *R. planticola* infections is growing, without a concomitant increase in research on the microbe or its pathogenesis. Previous genomic studies demonstrating genetic similarities between *R. planticola* and *Klebsiella pneumoniae* suggest that capsule biosynthesis, mucoid phenotype, biofilm production, and lipopolysaccharide (endotoxin) synthesis may all be potential virulence factors of *R. planticola*. We present a unique case of *R. planticola* infection of the biliary tract 5 years after biliary surgery in a patient with no previously documented risk factors. We also use *in silico* techniques to predict virulence factors of *R. planticola*.

**Case presentation:** This case report is the first to discuss a *R. planticola* infection in the biliary tract of late onset postsurgery (5 years) in a Caucasian patient with no previously documented risk factors.

**Conclusions:** An in-depth search of the current literature did not yield other similar cases of *R. planticola* infections. Moreover, to the best of our knowledge, our case is the first case of *R. planticola* isolated from post-endoscopic retrograde cholangiopancreatography (ERCP) as part of biliary sepsis not associated with gastroenteritis. The late onset of the infection in our patient and the results of the *in silico* analysis suggest that *R. planticola* may have survived exposure to the host immune system through the creation of an intracellular biofilm or in a non-culturable but viable state (NCBV) for the 5-year period. The *in silico* analysis also suggests that biofilms, enterobactin, and mucoid phenotype may play a role in the pathogenesis of *R. planticola*. However, further research is needed to illuminate the significance of pili, capsule biosynthesis, and lipopolysaccharide (LPS) in the virulence of *R. planticola*. Lastly, as our patient did not have any risk factors previously associated with *R. planticola*, we suggest that biliary tract stricture, cholecystitis, and prior surgery may be possible novel risk factors.

**Keywords:** *Raoultella planticola*, Bacteremia, Cholangitis, Intrahepatic bile ducts, Gall bladder resection

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**Background**

The *Raoultella* genus comprises gram-negative, oxidase-negative, facultative anaerobic bacteria within the *Enterobacteriaceae* family. *R. planticola*, earlier known as *Klebsiella planticola* and *Klebsiella trevisanii*, is a gram-negative, rod-like bacterium first described by Ferragut [1] from aquatic and soil isolates and later differentiated from *Klebsiella* after phylogenetic analysis by Drancourt and associates [2]. Matrix-assisted laser desorption/ionization–time of flight (MALDI-TOF) mass spectrometry
is the current method used to identify and differentiate \textit{R. planticola} [3]. \textit{R. planticola} is an emerging pathogen which has been linked to fatal infections. Only 33 cases of \textit{R. planticola} were reported prior to the middle of 2015 [4]. There have been 19 novel cases reported since that time. Pediatric cases, although extremely rare, have also been reported [5]. Further, \textit{R. planticola} may cause bacteremia, pneumonia, intra-abdominal infections, urinary tract infections, soft tissue infections, and conjunctivitis [6–8]. To our knowledge, there have been no reports of \textit{R. planticola} biliary tract infections or a thorough investigation of microbial virulence or immune escape. Herein, we report a case of \textit{R. planticola} biliary tract infection as a long-term postsurgical complication in a 31-year-old woman who initially presented with acute cholecystitis. We also discuss the results of a genome-based comparison between \textit{R. planticola} and \textit{K. pneumoniae} in order to examine possible virulence factors of \textit{R. planticola}.

**Case presentation**

A 31-year-old Caucasian woman presented to the emergency department with sudden onset of abdominal pain, fever, chills, and malaise. She had a history of laparoscopic cholecystectomy in 2008 complicated by bile leak requiring biliary stents. In 2011, she developed hepatic cysts, which were surgically extirpated in 2011 and 2012. She remained afebrile and mostly asymptomatic, with only occasional mild right upper quadrant pain until the current presentation which caused her to seek medical attention at the emergency department. Importantly, she denied any history of solid organ transplants, hematologic malignancy, chemotherapy, transplantation neutropenia, cirrhosis, seafood ingestion, or proton pump inhibitor (PPI) use.

Vital signs on presentation were temperature of 98.1 °F, heart rate of 85 beats per minute, respiration of 18 breaths per minute, and blood pressure of 126/83 mmHg. Physical examination revealed an afebrile, anicteric female in moderate, painful distress with slight, diffuse abdominal tenderness on palpation. Laboratory/radiographic tests revealed a white blood cell count of 41.1 cells/µL; elevated liver enzymes (alanine aminotransferase 102 U/L and aspartate aminotransferase 74 U/L); alkaline phosphatase of 318 U/L; and total bilirubin of 2.4 mg/dl. Computed tomography and magnetic resonance cholangiopancreatography (MRCP) were significant for dilated right intrahepatic bile duct with evidence of a surgically absent gall bladder (Figs. 1, 2, 3). During admission in the emergency room, the patient became febrile, and blood cultures (BC) were drawn. In light of the clinical picture and imaging studies, biliary sepsis and bacteremia due to intrahepatic duct stricture were suspected.

The patient was admitted to the hospital and empirically started on piperacillin/tazobactam. The BC was positive for gram-negative rods in two of two peripheral BC after 24 hours. \textit{R. planticola} was reported as the isolate on the third hospital day and was resistant to ampicillin and piperacillin but susceptible to ceftriaxone (microbial resistance and susceptibilities were completed by Quest Diagnostics). Therapy was changed to ceftriaxone 2 g parenterally every 24 hours, and the patient quickly improved clinically, with normalization of liver function within 3 days (hospital day 6). She was discharged on home therapy with referral for subsequent evaluation and treatment of her intrahepatic duct strictures.
Case discussion and methods

Previously reported *R. planticola* cases

*Raoultella planticola* is an emerging bacterial pathogen (see Fig. 4) that has previously been associated with nonclinical environments such as aquatic habitats and therefore has been linked to consumption of seafood [9]. However, case studies since 1985 have indicated an increased number of clinical cases [10] and multiple organ infections [11]. Previously, *R. planticola* was isolated from patients with comorbid leptospirosis [10] and found to be a cause of pneumonia [12]. *R. planticola* was also observed to occur in hematological malignancy when the organism was isolated from the oral ulcers of a patient with chemotherapy-induced oral mucositis [13].

Similarly, other cases have been documented that suggest an increased susceptibility to infection in immunocompromised states [13]. Table 1 summarizes all reported *R. planticola* infections prior to 2018 found during an in-depth literature search.

Analysis of all the documented patients showed that *R. planticola* caused bacteremia in 22% of cases, soft tissue infections in 17%, urinary tract infections in 15%, lower respiratory tract infections in 10%, and eye infections in 10%. Sources of isolation correlated with the infected organ system ($r^2 = 0.72$). The annual timeline frequency of documented infections caused by this pathogen potentially indicate a biannual prevalence (see Fig. 4).

Pathogenesis and virulence factors in *R. planticola* genome

The pathogenesis of *R. planticola* has not yet been established; however, fimbria, biofilm production, encapsulation, lipopolysaccharide (LPS), and siderophores have been observed to be important virulence factors in the closely related species *Klebsiella pneumoniae* species [3, 14].

In order to investigate possible virulence factors for *R. planticola*, in silico analysis was conducted using the National Center for Biotechnology Information (NCBI) Basic Local Alignment Search Tool (BLAST) (NCBI.gov) [15], Universal Protein Resource (UniProt; uniprot.com) [16], and Integrated Microbial Genomes (IMG; img.jgi.gov) system [17]. In the analysis, previously documented virulence factor gene sequences, including known *K. pneumoniae* virulence factor gene sequences, were used to generate queries to blast against the *R. planticola* genome. Using these databases, gene sequences and
accession numbers for virulence factors in *K. pneumoniae* were found for over 40 genes pertaining to pili components, pili chaperone proteins, biofilm synthesis and initiation, capsule assembly, capsule biosynthesis, capsule initiation, LPS synthesis, outer membrane surface protein chaperones, and expression of mucoid phenotype (genes present, see Tables 2, 3). Using the gene function on IMG [17], permanent drafts of *K. pneumoniae* pKP469IL Plasmid (B) [P], *K. pneumoniae* pKP531IL (B) [P], *K. pneumoniae* 1162281 (B) [P], and *K. pneumoniae* 1191100241 (B) [P] were searched for gene sequences and accession numbers for known virulence factors [18]. The genome for *R. planticola* strain GODA was selected as a target for querying the virulence factors relevant for endotoxins, capsules, fimbria, pili production, and biofilm production using the BLASTn and BLASTp tools.

| Reported case | Clinical manifestation | Culture site | Age | Sex (M/F) | Region | Outcome |
|---------------|------------------------|--------------|-----|-----------|--------|---------|
| [35]          | Bacteremia             | Blood        | 69  | Unknown   | France | Recovered |
| [35]          | Pneumonia              | Blood, sputum| 57  | Unknown   | France | Recovered |
| [36]          | Pancreatitis           | Peritoneal fluid | 45  | M         | Brazil | Recovered |
| [37]          | Pneumonia              | Blood        | 83  | F         | Ohio, USA | Died |
| [37]          | Soft tissue            | Blood        | 64  | M         | New Jersey, USA | Died |
| [38]          | Cellulitis             | Wound        | 30  | M         | Ireland  | Recovered |
| [39]          | Soft tissue            | Unknown      | 66  | M         | Texas, USA | Unknown |
| [40]          | Cholangitis            | Blood        | 65  | M         | Japan   | Improved, transferred |
| [41]          | Necrotizing fasciitis  | Abdominal fluid | 66  | M         | South Korea | Recovered |
| [42]          | Cholecystitis          | Gallbladder fluid | 62  | F         | UK      | Recovered |
| [43]          | Cholangitis            | Blood        | 59  | M         | Ontario, Canada | Recovered |
| [44]          | UTI                    | Urine        | 89  | M         | New Mexico, USA | Recovered |
| [45]          | Bacteremia             | Blood        | 63  | M         | Spain   | Recovered |
| [46]          | Prostatitis            | Urine        | 67  | M         | Greece  | Recovered |
| [47]          | Bacteremia from seafood| Blood        | 56  | F         | Ontario, Canada | Recovered |
| [7]           | Consecutiveisit        | Conjunctival swab | 58  | F         | UK      | Recovered |
| [48]          | Cholangitis            | Blood        | 70  | M         | Italy   | Recovered |
| [49]          | Cholecystitis          | Biliary fluid | 49  | M         | Connecticut, USA | Recovered |
| [50]          | Cholangitis            | Unknown      | Unknown | Unknown | Unknown | Unknown |
| [6]           | Pneumonia              | Sputum       | 60  | M         | China   | Died |
| [51]          | UTI                    | Urine        | 92  | F         | Connecticut, USA | Recovered |
| [52]          | Cystitis               | Urine        | 1   | M         | South Korea | Recovered |
| [53]          | Peritonitis            | Peritoneal fluid | 65  | M         | South Korea | Recovered |
| [12]          | Pneumonia              | Sputum       | 58  | M         | South Korea | Recovered |
| [8]           | Consecutiveisit        | Conjunctival swab | 88  | F         | Malta   | Recovered |
| [8]           | Consecutiveisit        | Conjunctival swab | 71  | M         | Malta   | Unknown |
| [8]           | Consecutiveisit        | Conjunctival swab | 15  | F         | Malta   | Unknown |
| [8]           | Consecutiveisit        | Conjunctival swab | 69  | F         | Malta   | Recovered |
| [54]          | Prostatitis            | Prostatic fluid | 53  | M         | New York, USA | Recovered |
| [55]          | UTI                    | Urine        | 57  | M         | Unknown | Recovered |
| [56]          | Implantation site infection | Pus from site | 79  | M         | Unknown | Recovered |
| [57]          | UTI                    | Urine        | 73  | M         | Florida | Recovered |
| [58]          | UTI                    | Urine        | 2 months | F       | Unknown | Recovered |
| [59]          | UTI                    | Urine        | 57  | M         | Unknown | Recovered |
| [60]          | Cirrhosis              | Blood        | 66  | M         | Unknown | Recovered |
| [13]          | Oral mucositis         | Oral ulcers  | 16  | M         | Unknown | Recovered |
| [61]          | Spinal epidural abscess| Unknown      | Unknown | Unknown | Unknown album | Recovered |
| [62]          | Wound infection        | Unknown      | 73  | F         | Unknown | Recovered |
| [5]           | Consecutiveisit        | Conjunctival swab | 28 weeks | F       | Unknown | Recovered |

*UTI* urinary tract infection, *M* male, *F* female
In order to investigate the clinical relevance, the genome of ATCC 33531 was compared against GODA and found to be 99% identical (see Table 4).

For functional gene annotation, greater than 60% query coverage, 70% nucleotide identity, and Expect (E)-value below 0.001 were used as the minimum similarity criteria between functionally documented genes of *K. pneumoniae* and unknown *R. planticola* genes (see Tables 2, 3).

### Results and discussion

The results of blasting for capsule and mucoid production genes are shown in Tables 2 and 3.

#### Endotoxin production

WzzE [20] and O-acetyltransferase [21], genes known to be involved in the synthesis of LPS, showed 93% and 82%

| Species   | Strain       | Accession no. | Ecosystem             | Chromosomal cassette gene % | In silico genome hybridization |
|-----------|--------------|---------------|-----------------------|-----------------------------|-------------------------------|
| *R. planticola* | GODA        | CP019899.1    | Soil and ground water | Not previously documented   | Max score 5.12E+05             |

### Table 2 Genomic identification of virulence factors in the genome of *R. planticola* ATCC 33531

| Gene name                              | Gene ID                | Max score | Total score | Query cover (%) | E-value | Identity (%) |
|----------------------------------------|------------------------|-----------|-------------|-----------------|---------|--------------|
| LPS biosynthesis protein WzzE          | NC_016845.1            | 28696     | 4.24E+06    | 71              | 0.0     | 93           |
| O-acetyltransferase                    | NC_016845.1            | 2143      | 2143        | 67              | 0.0     | 82           |
| Major type 1 subunit fimA              | 2546382621             | No        | No          |                 |         |              |
| Pilin (type 1 fimbra component protein) | 2546385281             | No        | No          |                 |         |              |
| KpsS (capsule synthesis)               | NC_025184.1            | 2146      | 2.01E+04    | 5               | 0.0     | 99           |
| Uncharacterized protein related to capsule biosynthesis enzymes | NC_010870.1 | 2100 | 10206 | 1 | 0.0 | 99 |
| Capsule assembly protein Wzi            | 2549022389             | 950       | 950         | 94              | 0.0     | 79           |
| Periplasmic chaperone for outer membrane proteins Skp | 2546385958 | 693 | 693 | 100 | 0.0 | 92 |
| Periplasmic chaperone for outer membrane proteins SurfA | 2546385592 | 1474 | 1474 | 100 | 0.0 | 87 |
| Regulator of mucoid phenotype rmpA      | 2657583                | No        | No          |                 |         |              |
| Regulator of mucoid phenotype rmpA2     | 2657677                | No        | No          |                 |         |              |
| Putative negative regulator of RcsB-dependent stress response | 2549023015 | 538 | 538 | 100 | 3.00E-153 | 82 |
| Regulator of capsule synthesis rcsA     | CIG23_03380            | 299       | 299         | 100             | 1.00E-93| 69          |

#### Table 3 The results of queries previously functionally annotated in *R. planticola*

| Functionally annotated genes in *R. planticola* (Accession CP 019899.1) | Gene name          | Gene ID |
|-------------------------------------------------------------------------|--------------------|---------|
| Biofilm protein TabA                                                     | EG12530 tabA       |         |
| Biofilm regulator BssS                                                   | UA70_04275         |         |
| Biofilm formation protein BssR                                           | UA70_18665         |         |
| Type 1 fimbria regulatory protein FimB                                   | 2588758217         |         |
| Type 1 fimbria regulatory protein FimE                                   | 2588758216         |         |
| Fimbrial, FimD or usher-like                                             | 2588757070         |         |
| Surface assembly of capsule Wzi                                          | IPRO26950          |         |
| ybdA enterobactin exporter                                              | 2588761180         |         |
| Enterobactin synthetase component D                                     | 2588761188         |         |
| Enterobactin synthetase component F                                     | 2588761184         |         |

TabA is a gene responsible for initiation of biofilm synthesis [63]. BssS and BssR are genes associated with biofilm stress response induction and upregulation of motility transcription [64]. FimB, FimD, and FimE are genes encoding chaperone proteins for pili components [65]. Wzi is a gene associated with the synthesis of the capsule [24]. Enterobactin synthetase components D and F and ybdA enterobactin exporter are genes involved in the production of the siderophores enterobactin which allow the pathogen to outcompete the host iron-acquisition system [14].

### Table 4 Information for the host-associated isolated ATCC 33531 strain and the environment-isolated GODA strain

| Species   | Strain       | Accession no. | Ecosystem             | Chromosomal cassette gene % | In silico genome hybridization |
|-----------|--------------|---------------|-----------------------|-----------------------------|-------------------------------|
| *R. planticola* | GODA        | CP019899.1    | Soil and ground water | Not previously documented   | Max score 98.23 5.12E+05     |

Available from NCBI.gov [15]. In order to investigate the clinical relevance, the genome of ATCC 33531 was compared against GODA and found to be 99% identical (see Table 4).

For functional gene annotation, greater than 60% query coverage, 70% nucleotide identity, and Expect (E)-value below 0.001 were used as the minimum similarity criteria between functionally documented genes of *K. pneumoniae* and unknown *R. planticola* genes (see Tables 2, 3). Novel queries identified by BLASTn analysis of *R. planticola* GODA against *K. pneumoniae* are shown in Table 2, and previously functionally annotated queries are shown in Table 3.

#### Results and discussion

The results of blasting for capsule and mucoid production genes are shown in Tables 2 and 3.

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A adenine, T thymine, C cytosine, G guanine
Capsule and mucoid production
We observed the following virulence factor relationships between the target genomes: Wzi (99% identity in 94% of the gene fragments); rcsA (69% identity in 100% of the gene fragments); rcsB (82% identity of 100% of the gene fragments); KpsS (99% identity in 5% of the gene fragments) suggesting a shared prosthetic group; uncharacterized protein (99% identity in 1% of the gene fragments) suggesting a shared prosthetic group. The presence of such genetic similarities indicates that R. planticola has the potential to synthesize, regulate, and assemble a capsule and express a mucoid phenotype that could help it escape the host immune system [22–26]. Mucoid phenotype regulators rmpA and rmpA2 did not show significant matching, indicating that capsular mucoid composition in R. planticola may be expressed through a different mechanism from that in K. pneumoniae. The results of blasting for capsule and mucoid production genes are shown in Tables 2 and 3.

Adhesins, pili, and fimbria
Skp and SurfA queries were found to be 92% and 87% identical, respectively, between R. planticola and K. pneumoniae, suggesting that R. planticola codes for periplasmic chaperoning of outer membrane protein assembly [27, 28]. Type 1 fimbria regulatory proteins FimB and FimE and fimbria FimD queries have previously been documented in R. planticola (see Table 3). No other similarities for different fimbrial components were found. This variability that R. planticola possesses in its fimbria may contribute to its host cell attachment and opsonization prevention in ways that differ from those mechanisms in K. pneumoniae.

Biofilm production
Raoultella planticola was found to possess genes similar to K. pneumoniae that have previously been shown to cause infection via biofilm production, upregulation of motility factors, outcompeting host cells for iron, and mucous production (see Table 3).

Gene cassettes
Whole genome blasting analysis between host-associated ATCC 33531 and GODA was conducted to identify chromosomal cassettes. We observed identity of 99% in 93% of gene fragments, suggesting high gene density that could allow efficient regulation of gene expression in mechanisms of antibiotic resistance, host immune system evasion, host cell attachment and invasion, and intracellular survivability. R. planticola may contain pathogenicity islands, a potential result of transduction that would also be involved in bacterial adaption, but further research is needed to confirm this.

Organ systems affected, virulence factors, and potential latent infection in our case
An in-depth search of the current literature did not yield other case studies with a similar isolation of R. planticola. We were also unable to identify another case of R. planticola isolated after endoscopic retrograde cholangiopancreatography (ERCP) as part of biliary sepsis not associated with gastroenteritis. Further, a detailed history and chart review of our patient did not show any of the previously reported risk factors associated with R. planticola including bacteremia/sepsis of the gastrointestinal tract (GI), biliary malignancy, chemotherapy, transplantation, neutropenia, cirrhosis, seafood ingestion, or PPI usage. It is possible that our patient had recently become infected with R. planticola rather than during the time of her laparoscopic procedure; however, as discussed above, an extensive attempt to document any previously associated risk factors failed to illuminate any. It has been reported that patients with chronic biliary strictures are at increased risk of cholangitis, possibly due to static biliary fluid in the stenotic biliary system or because of abnormal anatomic morphology that facilitates bacterial adhesion and colonization [30–32]. Therefore, we speculate that our patient’s bacteremia, which developed 5 years postoperatively, may be due to possible latency of the pathogen. The in silico results might also indicate that this organism survived exposure to the host immune system through the employment of an intracellular biofilm or in a non-culturable but viable (NCBV) state for the 5-year period [33, 34]. Our results also suggest that biofilms, enterobactin, and mucoid phenotype are likely virulence factors in the pathogenesis of R. planticola’s ability to cause infection. Additionally, we identified a conservation of genes involved in pili synthesis regulation, fimbrial protein chaperoning, capsule biosynthesis, and endotoxin production; however, the genetic variation of genes coding for pili, fimbria, and capsule polysaccharide composition may indicate that these genes are subject to antigenic variation or reductive evolution in an attempt to avoid the host immune system. Multiple genomes of newly isolated clinical P. planticola should be sequenced in order to evaluate its level of
evolutionary conservation of the extracellular and surface gangliosides.

**Conclusions**

This unique case adds to the literature on the GI affinity of *R. planticola* and, with the results of the in silico analysis, suggests that potential novel risk factors for infection may be biliary tract stricture, cholecystitis, and prior surgery.

**Abbreviations**

BC: Blood cultures; ERCP: Endoscopic retrograde cholangiopancreatography; Gl: Gastrointestinal tract; LPS: Lipopolysaccharide; MALDI-TOF: Matrix-assisted laser desorption/ionization–time of flight; MRCP: Magnetic resonance cholangiopancreatography; NCVR: Non-culturable but viable state; PPI: Proton pump inhibitor.

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**Authors’ contributions**

All authors contributed to the in silico blast analysis, writing of the manuscript, and approval of the manuscript; EJ was the treating physician. All authors read and approved the final manuscript.

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**Availability of data and materials**

No data or samples from the patient will be made available due to patient privacy.

**Declarations**

Ethics approval and consent to participate

Ethical approval was waived for this case report by St George’s University Institutional Review Board.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors report no competing interests.

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