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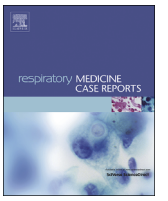

Case Report

# Empyema and bacteremia caused by *Parvimonas micra*: A case report

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ABSTRACT

*Parvimonas micra* is a gram-positive anaerobic coccus (GPAC) that colonizes the oral cavity and gastrointestinal tract. Recent advances in bacterial identification have confirmed the clinical importance of *Parvimonas micra*. Here, we report a case of empyema with bacteremia caused by *Parvimonas micra*. We successfully treated the patient with the appropriate antibiotics and drainage. *Parvimonas micra* can cause respiratory infections, including empyema, which can progress to bacteremia if treatment is delayed. In *Parvimonas micra* infections, not only the oral cavity but also the entire body must be investigated to clarify the entry mechanism.

## 1. Introduction

*Parvimonas micra* is a gram-positive anaerobic coccus (GPAC) that colonizes the oral cavity and gastrointestinal tract [1]. It was originally classified as *Peptostreptococcus micros*, after which it was transferred to the *Micromonas* genus in 1999 and was known as *Micromonas micros* [2]. The *Micromonas* genus was replaced by the *Parvimonas* genus in 2006, with *Parvimonas micra* as its sole species [1].

The clinical characteristics of *Parvimonas micra* have not been well studied owing to historical difficulties in laboratory identification and culture. However, recent advances in bacterial identification, such as microarray, and 16S rRNA sequencing using real-time polymerase chain reaction (PCR), and a new technique, i.e., matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS), have confirmed the clinical importance of *Parvimonas micra* [3].

Clinical studies on infectious diseases caused by *Parvimonas micra* are often based on case reports [4,5]. *Parvimonas micra* has been reported to be most associated with oral pathogens in endoperiodontal lesions, apical abscesses, and periodontitis [6]. However, it has also been implicated in infections in other body parts, such as spondylodiscitis, arthritis, infective endocarditis, deep abscesses, chronic sinusitis, and various other diseases [7–10]. However, there are not so many reports of pulmonary disease, including empyema caused by *Parvimonas micra*, and its detailed clinical course for cases that have further progressed to bacteremia is not well understood [11,12]. Moreover, it is less commonly reported as a cause of monobacterial bacteremia [5]. In addition, the route of infection was thought to be the oral cavity; however, cases involving the gastrointestinal tract and cases with unknown entry have been reported; hence, its entry route is also not well understood [13].

In this article, we report a case of empyema with monobacterial bacteremia caused by *Parvimonas micra* that was successfully treated with antibiotics and source control.

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## 2. Case report

A 70-year-old man with no self-reported specific medical history but without a history of regular hospital visits and check-ups presented to our hospital with progressive dyspnea, cough, and sputum production for the past 2 months. He had a 40 packs/year history of smoking from 20 to 60 years of age. He drank two ounces of whiskey every day. He had 3 cats. He used dentures; however, there was no recent history of dental procedures or dental diseases such as periodontitis or periodontal abscess. He did not undergo regular medical check-ups. He was a retired citizen and a previous public servant who lived with his wife.

The vital signs at the first visit were as follows: body temperature, 37.0 °C; blood pressure, 119/94 mmHg; heart rate, 124 beats/min; respiratory rate, 14 breaths/min; and oxygen saturation level, 90% (room air). The body mass index was 16.2 kg/m$^2$ (height 167.2 cm, weight 45.4 kg). On physical examination, an oral cavity examination revealed that his mucous membranes were slightly dry, and the nasopharynx and oropharynx had no signs of inflammation, such as redness, swelling, tenderness, or heat sensation.

The cardiac examination revealed tachycardia with a regular rhythm without murmurs. In the respiratory system, there were no rales, but there were less sounds in the right lower lung. No clubbing, cyanosis, superficial lymphadenopathy, or edema of the arms, hands, legs, or feet were noted. Abdominal, musculoskeletal, neurological, and cutaneous examinations revealed no abnormalities.

The results of the laboratory tests are listed in Table 1. The complete blood count showed a hemoglobin level of 11.2 g/dl; hematocrit level, 37.7%; white blood cell count, 9100 cells/mm$^3$ (neutrophil, 78.0%; lymphocyte, 11.7%); and platelet count, 48.6 × 10$^4$ cells/mm$^3$. Biochemistry results revealed an elevated C-reactive protein (CRP) level of 11.24 mg/dL. Laboratory tests showed negative results for HBsAg, anti-HCV, anti-HIV, β-D-glucan, and interferon-gamma release assay tests.

The posterior–anterior view of the chest radiograph revealed radio-opacity in the right middle and lower zones (Fig. 1). Contrast-enhanced computed tomography (CT) revealed right-sided pleural effusion with air accumulation and pleural thickening. Centrilobular emphysema was observed entirely in both lungs (Fig. 2 A and C). Contrast-enhanced computed tomography (CT) revealed no abscesses throughout the body. Thoracocentesis was then performed, and a yellow purulent fluid was aspirated. Laboratory tests of the pleural effusion revealed an exudated pleural effusion (fluid protein level, 1.2 g/dL; serum protein level, 8.2 g/dL; fluid lactate dehydrogenase (LD) level, 9653 U/L; serum LD level, 95 U/L). Other pleural fluid analyses showed a fluid cell count of 66000/μL; fluid glucose level, 25 mg/dL; and pH, 6.812 (Table 1).

The patient was diagnosed with empyema. We wanted him to be admitted and initiated on intravenous antibiotics. However, beds were not available at the time; therefore, we prescribed oral amoxicillin (1500 mg/day)/clavulanate (375 mg/day), and the patient was admitted 6 days after the first visit.

The results of various cultures submitted at the first visit showed *Parvimonas micra*, *Pasteurella multocida*, *Fusobacterium naviforme*, and *Streptococcus pyogenes* in the pleural fluid culture using blood culture bottles; *Pasteurella multocida* in the sputum culture; and only *Parvimonas micra* in two sets of blood cultures, without using real-time PCR or MALDI-TOF MS in any of the cultures. The blood culture bottles used were BD BACTEC™ plus aerobic and anaerobic medium culture bottles, and the pathogens were identified using standard incubation and monitoring methods according to the product information and instructions. The pleural fluid and blood samples tested positive for pathogens after 2 and 70 hours of inoculation in anaerobic medium culture bottles, respectively.

**Table 1**
Laboratory tests.

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Blood cell count | | | ALT | 14 | U/L | HBs antibody | | negative |
| WBC | 9100 | /μL | LD | 95 | U/L | HBc antibody | | negative |
| Neutro | 78.0 | % | CK | 10 | U/L | HCV antibody | | negative |
| Eosino | 0.1 | % | ALP | 97 | U/L | IgG | 2922 | mg/dL |
| Baso | 0.2 | % | γ-GTP | 25 | U/L | IgA | 539 | mg/dL |
| Mono | 10.0 | % | Na | 136 | mEq/L | IgM | 65 | mg/dL |
| Lymph | 11.7 | % | K | 4.4 | mEq/L | C3c | 125 | mg/dL |
| RBC | 424 × 10$^4$ | /μL | Cl | 99 | mEq/L | C4 | 33.5 | mg/dL |
| Hb | 11.2 | g/dL | BUN | 10 | mg/dL | CH50 | 44.3 | U/mL |
| Ht | 37.7 | % | sCr | 0.66 | mg/dL | ANA titer | <1:40 | |
| Plt | 48.6 × 10$^4$ | /μL | eGFR | 90.30 | mL/min/1.73m$^2$ | | | |
| | | | CRP | 11.24 | mg/dL | Pleural fluid analysis | | |
| Blood coagulation | | | BNP | 61.7 | pg/mL | Appearance | Purulent | |
| PT-INR | 1.16 | | Ferritin | 326.5 | ng/mL | Cell count | 66000 | /mL |
| APTT | 33.5 | sec | Glucose | 137 | mg/dL | Neutrophils | N/A | % |
| | | | | | | Lymphocytes | N/A | % |
| Biochemistry | | | Immunological test | | | Macrophages | N/A | % |
| TP | 8.2 | g/dL | HIV-1/2 antibody | <1.0 | negative | Glucose | 25 | mg/dL |
| Alb | 2.0 | g/dL | IGRA | | negative | pH | 6.812 | |
| T-bil | 0.5 | mg/dL | β-D-glucan | <5 | pg/mL | TP | 1.2 | g/dL |
| AST | 19 | U/L | HBs antigen | 0.00 | negative | LD | 9653 | U/L |

WBC: white blood cells, Neutro: neutrophils, Eosino: eosinophils, Baso: basophils, Mono: monocytes, Lymph: lymphocytes, RBC: red blood cells, Hb: hemoglobin, Ht: hematocrit, Plt: platelets, PT-INR: prothrombin time-international normalized ratio, APTT: activated partial thromboplastin time, TP: total protein, Alb: Albumin, T-bil: total-bilirubin, AST: aspartate transaminase, ALT: alanine transaminase, LD: lactate dehydrogenase, CK: creatine kinase, ALP: alkaline phosphatase, γ-GTP: γ-glutamyltransferase, BUN: blood urine nitrogen, sCr: serum creatinine, eGFR: estimated glomerular filtration rate, CRP: C-reactive protein, BNP: brain natriuretic peptide, HIV-1/2 antibody: human immunodeficiency virus 1/2 antibody, IGRA: interferon gamma releasing assay (QuantiFERON®-TB Gold Plus), HBs antigen: hepatitis B surface antigen, HBs antibody: hepatitis B surface antibody, HBc antibody: hepatitis B core antibody, HCV antibody: hepatitis C virus antibody, IgG: immunoglobulin G, IgA: immunoglobulin A, IgM: immunoglobulin M, C3c: complement 3c, C4: complement 4, CH50: 50% hemolytic complement, ANA titer: antinuclear antibodies titer.

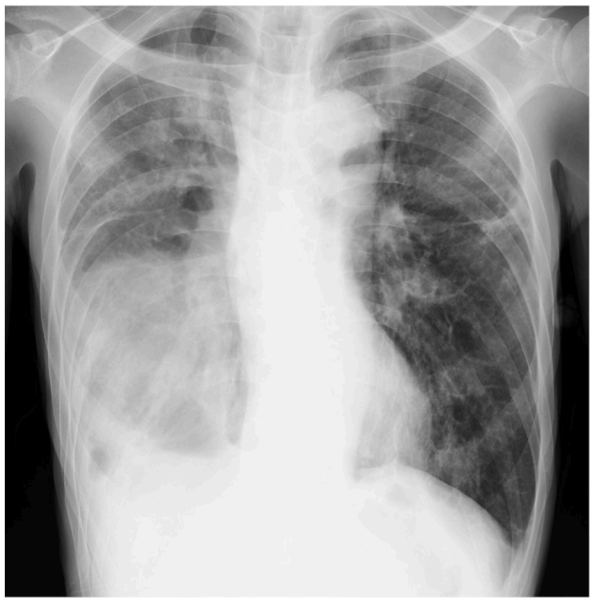

**Fig. 1.** Posterior anterior erect chest radiograph showing opacifications in the right middle to lower zone. The right costophrenic angle is obliterated, suggestive of a right-sided pleural effusion.

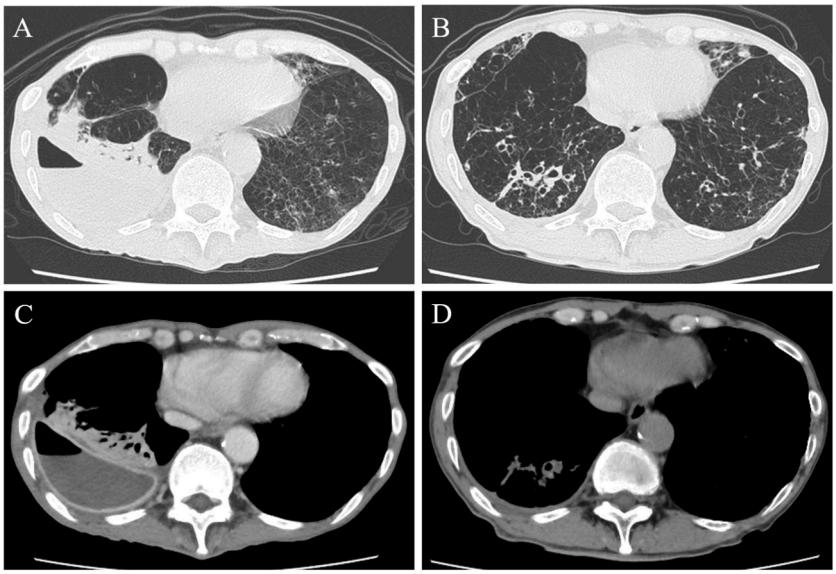


**Fig. 2. A, C)** Contrast-enhanced computed tomography in axial view on the initial examination showing loculated pleural effusion and consolidation on the right lower back side with centrilobular emphysema. **B, D)** Non-contrast enhanced CT in axial view on the 5-month follow-up exhibiting the disappearance of both loculated pleural effusion and consolidation on the right lower back side.
CT: computed tomography.

On admission, we switched from oral antibiotics to intravenous antibiotics and initiated ampicillin (8 g/day)/sulbactam (4 g/day), based on drug susceptibility tests. Additionally, a chest drain was placed in the thoracic cavity. Both clinical manifestations (fever, cough, and sputum) and high CRP levels improved soon after treatment. Blood cultures collected four days after admission showed negative results. The radiological findings assessed using chest radiography and chest CT gradually improved. Therefore, he was discharged from our hospital 17 days after admission and switched to oral amoxicillin (1500 mg/day)/clavulanate (375 mg/day) again.

During admission, we thoroughly investigated the entry site of *Parvimonas micra*. The transthoracic echocardiograph revealed no vegetation and a normal ejection fraction. The dental examination revealed a slightly dry oral mucous membrane and dental plaque, and no periodontitis or periodontal abscess. Upper gastrointestinal endoscopy revealed no abnormal findings. Furthermore, lower

gastrointestinal endoscopy revealed a 2-cm pedunculated sigmoidal polyp. Polypectomy was performed for the sigmoid polyp after discharge, and a pathological diagnosis of a low-grade adenoma was made.

In the follow-up after discharge from the hospital, the patient was clinically stable. However, considering the poor adherence to treatment and poor radiographic response, antibiotic therapy was prolonged for relapse prevention. After it was confirmed that the chest CT (without contrast) findings (Fig. 2 B and D) and CRP (0.27 mg/dL) had completely improved, antibiotic therapy was completed after 6 months of treatment.

## 3. Discussion

We report a case of empyema with bacteremia caused by *Parvimonas micra* that was successfully treated with the appropriate antibiotics and drainage. This case report highlights two important clinical points.

First, we should consider that *Parvimonas micra* can cause respiratory infections, including empyema, and progress to bacteremia. *Parvimonas micra* is recognized as an important causative agent of oropharyngeal infections, deep abscesses, spondylitis, and infectious endocarditis [3]. However, few case reports have suggested the involvement of respiratory disease, and there are even fewer cases of bacteremia [5,11]. A literature review of available case reports of pleural infection with *Parvimonas micra* revealed five previously reported cases [4,11,14–16]. Four were male and only one was female. The mean age of the patients was 66.6 years (standard deviation: ±10.3 years). The mean time from onset of illness to diagnosis of infection was 32 days (one case was not reported), although there was one case that took 90 days (range: 10–90 days) [11]. All cases originated from oral disease (dental caries, periodontitis, etc.). In every case, *Parvimonas micra* was identified by pleural fluid culture; however, no cases had positive blood cultures. Molecular biology techniques (real-time PCR, MALDI-TOF MS) had not been used for bacterial identification in any of the cases. Antimicrobial therapy was administered in all cases, with the addition of surgery in one case [16]. All cases reported favorable outcomes. A systematic review of monobacterial bacteremia caused by *Parvimonas micra*, such as this case, has reported only one respiratory case of septic pulmonary embolism of unknown entry out of a total of 26 cases [5]. However, a retrospective observational study of bacteremia with *Parvimonas micra* reported 4–10% of cases in which respiratory disease was implicated as a cause of bacteremia, and there may be cases of *Parvimonas micra* involvement in respiratory disease not identified by culture [3,13].

Second, when we encounter patients with bacteremia caused by *Parvimonas micra*, not only the oral cavity but also the entire body must be investigated, including the gastrointestinal tract. In previous reports of bacteremia caused by *Parvimonas micra*, entry from the gastrointestinal tract was reported to be similar to entry from the oral cavity [13]. In this case, there was no history of dental disease or dental procedure. Therefore, as it was still unclear whether the oral cavity was an entry site for the bacteria that subsequently led to empyema and bacteremia, we additionally performed upper and lower endoscopies and contrast CT of the whole body. We found only a 2-cm-large pedunculated sigmoid adenoma in the large intestine, which was unlikely to have been involved in entry. However, it is important to investigate the entire body for *Parvimonas micra* bacteremia to investigate the entry when we encounter a patient with *Parvimonas micra* bacteremia as there are some reports suggesting an association between colorectal cancer and *Parvimonas micra*, and a review on *Parvimonas micra* bacteremia reported that over 35% of patients had some type of cancer as a background disease, although a detailed breakdown was not available [13,17–20].

Although anaerobic bacteremia accounts for a minority of all bacteremia (0.5%–11.8%), the mortality rate of patients with anaerobic bacteremia is somewhat higher, ranging from 14% to 27% [21,22]. This may be because GPAC bacteremia is primarily observed in older patients with multiple comorbidities or in immunocompromised patients [3]. However, in a retrospective case series of bloodstream infection by *Parvimonas micra*, it has been suggested that bacteremia caused by *Parvimonas micra* has a lower mortality rate than that caused by other types of anaerobic bacteremia [13]. The reason is yet unclear; however, one possibility is that *Parvimonas micra* is more susceptible to antibiotics than other anaerobic bacteria; this high susceptibility to antibiotics might be one of the responsible factors for the good therapeutic outcome of *Pavimonas micra* [23,24]. One week elapsed from the hospital visit to the start of intravenous antibiotics; however, we had a favorable outcome with appropriate antibiotic treatment and source control without serious outcomes such as septic shock or death. However, since few cases have been isolated and identified, and there are few reports of monitoring through continuous antimicrobial susceptibility testing, further studies and accumulation of cases are needed to determine whether *Parvimonas micra* infection has a more favorable outcome than other anaerobic bacteremia.

This case has a limitation. It was unclear whether the bacteremia was caused by the empyema or whether the empyema caused the bacteremia. However, as respiratory symptoms preceded the systemic symptoms, and as the oral cavity was somewhat unsanitary due to tooth loss, although there was no dental disease or dental procedure, moreover other oral bacteria flora (*Fusobacterium naviforme* and *Streptococcus pyogenes*) were detected in the pleural fluid culture, *Parvimonas micra* may have been aspirated into the lungs from the oral cavity, causing empyema and then entering the bloodstream. Tooth loss itself is associated with respiratory complications and abnormalities of the oral flora, which also seems to support our hypothesis [25].

## 4. Conclusions

*Parvimonas micra* can cause pulmonary infections, including empyema, which can progress to bacteremia if treatment is delayed. Because of the increasing number of reports suggesting an association between the gastrointestinal tract and malignancy, it may be important to search not only the oral cavity but also the entire body including the gastrointestinal tract to clarify the entry route. Further studies are needed to clarify the characteristics of *Parvimonas micra* infections.

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## Declaration of competing interest

The authors have no conflicts of interest declare.

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