have evidence of acute respiratory distress syndrome with diffuse alveolar and interstitial edema, capillary congestion, intra-alveolar hemorrhage, and hyaline membrane formation [1]. It is postulated that TNF and other cytokines released from activated macrophages damage the pulmonary microcirculation [1, 2]. Nonetheless, high levels of TNF have been documented in infection with Plasmodium vivax, which rarely causes pulmonary complications and, like P. ovale, causes benign disease [4, 5]. Other factors, such as sequestration of parasitized erythrocytes in small blood vessels, may be important in respiratory syndromes caused by P. falciparum [2]. Sequestration has not been demonstrated in any species other than P. falciparum, although rosette formation by erythrocytes, a phenomenon associated with cerebral malaria in P. falciparum infections, may occur with P. ovale infection [6].

Ovale malaria is the rarest type of malaria; it is associated with the lowest levels of parasitemia and is the mildest form of the disease [7]. Its geographic distribution is limited to sub-Saharan Africa and to a lesser extent Southeast Asia, countries in the western Pacific, and a few countries elsewhere. In 1994, only 3% of reported cases of imported malaria in the United States were due to P. ovale [8]. Recent studies suggest, however, that rates of infection in areas of endemicity and among travelers have been underestimated. P. ovale infection was found in 63% of cases of malaria among travelers returning to Portugal from São Tomé e Príncipe [7], and 49% of children in a Senegalese village became infected with this species during a 4-month period of observation [9]. Such data, along with a report of a traveler to Ghana who died of a ruptured spleen [10] and our experience in the present case, indicate that the importance of ovale malaria as a health problem should not be underrated.

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First Case of Pasteurella gallinarum Isolation from Blood of a Patient with Symptoms of Acute Gastroenteritis in Japan

Pasteurella gallinarum was first described as a new species by Hall et al. [1] and is generally known as an indigenous bacterium in poultry [2]. There has been only one human case of infectious disease caused by P. gallinarum, which was isolated from the blood of a patient with endocarditis who was described by Al Fadel Saleh et al. [3] in 1995. Herein, we report the first case of P. gallinarum isolation from the blood of a patient with symptoms of acute gastroenteritis in Japan.

A 34-year-old man (weight, 79 kg; height, 172 cm) with an alcohol intake of 700 mL of beer/d presented with malaise in March 1998. A diagnosis of fatty liver was made on the basis of changes in the liver that were detected on an abdominal ultrasound scan. Thereafter, the patient received treatment as an outpatient. On 18 May 1998 at about 12:30 P.M., the patient developed fever, and he presented to the hospital at 9:00 A.M. complaining of sharp lower abdominal pain, diarrhea, and nausea. An increased WBC count (16.5 × 10^9/L) was detected; therefore, infectious enteritis was suspected. Cefazolin (1 g intravenously) and, for severe abdominal pain, pentaazocine (7.5 mg intramuscularly) were administered. At 6:00 P.M., his body temperature was 39.1°C, and rigors, abdominal pain, and nausea were present. Thus, scopolamine butylbromide (20 mg) was given orally, and the abdominal pain subsided; however, since lower abdominal tympanism and nausea persisted and rigors were worse at 7:30 P.M., the patient was hospitalized.

On the same day at 9:00 P.M., his body temperature was 40.6°C, and lower abdominal hyperesthesia and nausea were present. A diclofenac suppository (50 mg) and famotidine (20 mg intravenously b.i.d.) were administered, and blood and urine were cultured. On the second day after disease onset, the abdominal pain subsided. His body temperature was 38.4°C, and soft stools were passed four times. Laboratory studies disclosed the following values: WBCs, 12.7 × 10^9/L (32% band forms, 64% segmented granulocytes, and 4% eosinophils, and Hb, 14.3 g/dL; Hct, 43.6%); Na, 140 mEq/L; K, 4.2 mEq/L; Cl, 101 mEq/L; CO2, 22 mEq/L; glutamic-oxaloacetic transaminase (GOT), 56 U/L; glutamic-pyruvic transaminase (GPT), 36 U/L; total protein, 6.2 g/dL; albumin, 3.2 g/dL; total protein, 9.0 mg/dL; and C-reactive protein, 0.4 mg/L.

References
neutrophils, 2% lymphocytes, 1% monocytes, 1% bone marrow cells); C-reactive protein, 127 mg/L; total protein, 66 g/L; alanine aminotransferase, 20 U/L; aspartate aminotransferase, 53 U/L; γ-glutamyltransferase, 138 U/L; lactate dehydrogenase, 406 U/L; alkaline phosphatase, 251 U/L; total cholesterol, 1,510 mg/L; triglycerides, 1,270 mg/L; high-density lipoprotein cholesterol, 360 mg/L; creatine kinase, 110 U/L; amylase, 28 U/L; blood urea nitrogen, 142 mg/L; and creatinine, 10 mg/L. Serological tests for hepatitis B surface antigen and antibody to hepatitis C virus as well as the rapid plasma reagin test and Treponema pallidum hemagglutination assay were negative. On the third day, both abdominal pain and nausea resolved; his body temperature decreased to 36.8°C, and soft stools were passed four times.

The patient was discharged. Stool collected on this day was cultured. Blood culture yielded gram-negative coccobacilli, which were susceptible to ampicillin, piperacillin, cefazolin, cefmetazole, cefotaxime, azithromycin, imipenem, gentamicin, amikacin, norfloxacin, and streptomycin. On the basis of their biochemical properties, the bacteria were identified as *P. gallinarum*, which is indigenous in poultry.

This patient had held a barbecue 2 days before the onset of disease (16 May 1998) and had eaten chicken. Since there were no other recollections relating to birds, information was obtained through questionnaires sent to all the guests. It was found that seven of eight guests had eaten chicken, and two (29%; including our patient) of them had developed gastrointestinal symptoms. The patient seemed to have eaten more chicken than the six other persons. The other guest with gastrointestinal symptoms ate only a small amount of chicken, but diarrhea and anorexia in this person lasted for 20 hours. Further, this person thought that the chicken was spoiled. None of the eight guests had eaten food causing gastrointestinal symptoms around the time of the party, and they were in good physical condition.

*P. gallinarum* is an indigenous gram-negative coccobacillus mainly inhabiting the upper airway in domestic fowl [2]. There have been a few reports of chicken endocarditis and fowl cholera caused by *P. gallinarum* [4–6]. To our knowledge, the first report of *P. gallinarum* infection in humans was in 1995 by Al Fadel Saleh et al. [3] who isolated this bacterium from a patient’s blood; *P. gallinarum* isolation from our patient’s blood may be the second such case. Furthermore, this is the first case of *P. gallinarum* isolation from the blood of a patient with symptoms of acute gastroenteritis. *P. gallinarum* was considered to be involved in the gastrointestinal symptoms because of the following reasons: *P. gallinarum* is mainly indigenous in domestic fowl [2], and the only contact that the patient had with domestic fowl was the chicken he ate; although *P. gallinarum* was not isolated by stool culture, the bacterium was isolated from blood; two (29%) of the eight guests developed gastrointestinal symptoms after eating chicken; and of the seven guests who ate chicken, our patient ate the largest amount, suggesting that the gastrointestinal symptoms were caused by ingestion of chicken contaminated with *P. gallinarum* (although it was difficult to identify the source of infection).

Since the route of transmission was not clarified in either our case or the one reported by Al Fadel Saleh et al. [3], further investigation is necessary, including establishment of a method for measuring titers of serum antibody to *P. gallinarum*.

It has been reported that liver dysfunction is a major factor associated with *Pasteurella multocida* sepsis [7]. Our patient had fatty liver, which was considered to be due to alcohol intake and obesity, associated with mild liver dysfunction and had a temperature of >38°C and a WBC count of >12 × 10⁹/L, thereby fulfilling the criteria of systemic inflammatory response syndrome [8]. Thus, it is important to treat cases of *P. gallinarum* infection and observe its course as a disease that progresses to systemic inflammation.

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