Journal of Microbiology, Immunology and Infection (2016) 49, 452–454

Available online at www.sciencedirect.com

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CASE REPORT

Leclercia adecarboxylata bacteremia in a patient with long-term use of nonsteroidal anti-inflammatory drugs

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Received 1 June 2013; accepted 10 September 2013
Available online 31 October 2013

KEYWORDS
Bacteremia;
Immunocompetent;
Leclercia adecarboxylata;
Nonsteroidal anti-inflammatory drugs;
Peptic ulcer

Leclercia adecarboxylata, a ubiquitous Gram-negative bacillus, is generally viewed as an opportunistic pathogen because it is rarely cultured from clinical samples. Although rare, bacteremia due to *L. adecarboxylata* tends to occur in immunocompromised hosts and patients with systemic comorbidities. Only one case of bacteremia due to *L. adecarboxylata* has been reported in a previously healthy patient. We describe a male patient with an active peptic ulcer who developed *L. adecarboxylata* bacteremia after a long-term use of nonsteroidal anti-inflammatory drugs. The abdomen is believed to have been the most probable portal of entry. After appropriate medical management, the patient recovered without sequela.

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http://dx.doi.org/10.1016/j.jmii.2013.09.004

Introduction

Leclercia adecarboxylata, a motile, aerobic, Gram-negative bacillus, first described by Leclerc in 1962,[1] is ubiquitously distributed in nature and has been isolated from food, water, and other environmental sources.[2] *L. adecarboxylata* is generally viewed as a bystander because of its rare frequency of isolation from clinical samples. *L. adecarboxylata* bacteremia predominantly occurs in patients with some degree of immunosuppression[3–6] and in those with end-stage renal disease.[7,8] However, bacteremia due to *L. adecarboxylata* has never been reported in patients with peptic ulcers. Herein we report a case of *L. adecarboxylata* bacteremia in a patient with a duodenal ulcer after overuse of nonsteroidal anti-inflammatory drugs (NSAIDs) and present a review of relevant literature regarding bacteremia due to *L. adecarboxylata* in healthy patients without a history of trauma.

Case report

A 66-year-old man with a history of hemorrhage in the upper gastrointestinal tract was hospitalized in April 2012 because of epigastralgia, tarry-stool passage, and abdominal fullness for 4 days. The patient did not present with symptoms or signs of sepsis initially, but mentioned that he had been taking indomethacin as well as other NSAIDs for approximately 6 months to relieve joint pain. No immunosuppressive or antimicrobial agents had been administered prior to his admission, and no diarrhea developed prior to the occurrence of tarry-stool passage. Panendoscopy revealed a duodenal ulcer (Forrest classification 1a, sprouting hemorrhage) with exposed vessel in the postbulbar duodenum. Epinephrine was administered in combination with heater probe thermal coagulation treatment. The patient also received a transfusion of packed red blood cells and was administered a parenteral proton-pump inhibitor (omeprazole 40 mg, every 12 hours). Hemogram obtained on Day 1 of hospitalization revealed eosinophilia (1160/mm^3). On Day 4 of hospitalization, the patient presented with chills and spiking fever. No intravenous administration of fluids or drugs was given at that time. A detailed physical examination did not reveal any infectious focus. Findings on chest and abdominal X-rays were normal. Laboratory findings revealed a reduced white blood cell count (1910/mm^3), normal serum creatinine concentration (1.1 mg/dL), and mildly elevated C-reactive protein concentration (2.78 mg/dL). Based on the laboratory findings, the patient was administered cefmetazole at an intravenous dose of 1 g every 8 hours.

Two blood culture sets from this patient grew *L. adecarboxylata*. The biochemical profile for *L. adecarboxylata* included the following: (1) negative reactions for citrate, lysine decarboxylase, ornithine decarboxylase, arginine dihydrolase, DNase, and hydrogen sulfide, and (2) positive reactions for esculin hydrolysis, indole production, Voges–Proskauer test, and malonate assimilation. In addition, manual tube tests for sugar fermentation included positive reactions for adonitol, D-melibiose, sucrose, D-arabitol, raffinose, L-rhammnose, and lactose, but negative reactions for *myo*-inositol and D-sorbitol. For confirmation of the accuracy of *L. adecarboxylata* identification, we used two automated systems including the Microscan (Spectris Company, West Sacramento, CA, USA) and the BD crystal (Becton Dickinson & Company, Sparks, MD, USA). The bio numbers of this strain were 67310230 and 5764427552, respectively, with the probability of *L. adecarboxylata* being 99.0% and 99.4%. In accordance with the guidelines of the Clinical and Laboratory Standards Institute 2012 (*Escherichia coli* ATCC 25922 and *E. coli* ATCC 35218 as quality control strains),[9] the *L. adecarboxylata* isolate was susceptible to all antimicrobial agents tested, including ampicillin, ampicillin–sulbactam, cefazolin, cefmetazole, cefotaxime, ceftazidime, cefepime, piperacillin/tazobactam, ertapenem, gentamicin, amikacin, and ciprofloxacin. After cefmetazole was administered, rapid defervescence and gradual recovery of the patient's leukocyte count were noted. The immunologic survey resulted in the following findings: the ratio of CD4 to CD8 leukocyte count (on Day 6 of hospitalization) was grossly normal (2.05); there was no evidence of hyperglycemia (i.e., serum glucose concentration $\leq$ 140 mg/dL); and the serological results of human immunodeficiency virus and important autoimmune antibodies were all negative. However, persistent eosinophilia (958/mm^3, with a total leukocyte count of 3990/mm^3, on Day 6 of hospitalization) drew our attention. The sole abnormal finding on his abdominal sonographic investigation was fatty liver. Further investigations were performed to determine the cause of persistent eosinophilia. Neither parasites nor ova were detected in stool samples. Although the levels of serum lactic dehydrogenase (164 U/L; normal range, 100–190 U/L) and serum gamma-GT (26 U/L; normal range, 0–48 U/L) were normal, IgE level (771 mg/dL; normal level, $<$100 mg/dL) was elevated markedly. We suspected that the eosinophilia was drug related. The proton-pump inhibitor agent was, therefore, changed to an H_2 antagonist (famotidine) and cephalosporin was switched to oral ciprofloxacin 500 mg twice a day as the maintenance antibiotic agent. Follow-up hemogram on Day 16 of hospitalization showed resolution of eosinophilia, and the patient recovered without sequela.

Discussion

Our patient did not show evidence of any intra-abdominal problems with the exception of a duodenal ulcer. However, he had habitually abused NSAIDs prior to the development of bacteremia due to *L. adecarboxylata*. The majority ($>$67%) of cases of *L. adecarboxylata* bacteremia occur in immunosuppressed patients.[10] Based on a review of the literature, only one immunocompetent patient without a history of chronic systemic comorbidities or recent trauma has been reported to have developed bacteremia due to *L. adecarboxylata*.[11] Studies have shown that overuse of NSAIDs can result in immunosuppression[12]; however, it is not clear whether chronic NSAID usage in our patient resulted in a suppressed immune system.

It is noteworthy that the eosinophil count in our patient was less than 1500/mm^3 (the threshold for hypereosinophilic syndrome, the definition of which also includes its

persistence for more than 6 months and evidence of organ damage).[13] Besides, his clinical condition was clearly not compatible with hypereosinophilic syndrome or Churg—Strauss syndrome (asthma, pulmonary infiltrates, and dermatologic manifestation of necrotizing vasculitis, which were primarily relieved by glucocorticoids).[14] Furthermore, his normal serum lactic dehydrogenase level and end-organ function, and lack of evidence of parasitic infestation suggest that the eosinophilia was possibly caused by drugs. In addition, the subsidence of eosinophilia after our patient's medication was adjusted indicates that the eosinophilia was due to medication prescribed during hospitalization.

To the best of our knowledge, no evidence exists for an association between *L. adecarboxylata* bacteremia and peptic ulcers. Although peptic ulcers have not been reported as an entry portal into the bloodstream for pathogenic organisms, we still suspect that the bacteremic strain in this patient originated in the abdomen. *L. adecarboxylata*, which is generally considered to be a benign bystander in humans, may have a propensity to invade the bloodstream of patients with specific abdominal stress, such as the duodenal ulcer.

In contrast to the drug-resistant *L. adecarboxylata* bacteremic isolates occasionally cultured from immunocompromised patients,[5,15] the isolate obtained from our patient was susceptible to all tested antimicrobial agents. Similar findings have been reported earlier,[2,11] suggesting that most cases of *L. adecarboxylata* are relatively easy to treat.

In conclusion, we have presented a patient with peptic ulcer and a long-term history of NSAID usage who developed bacteremia due to *L. adecarboxylata*. Although this pathogen is usually susceptible to most antibiotics, infection with *L. adecarboxylata* can result in bacteremia in immunocompetent hosts with some degree of abdominal stress.

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