

2013

Renal abscess caused by a *Providencia stuartii* isolate biochemically misidentified as *Pasteurella*

Robin R. Chamberland
Saint Louis University

Erin McElvania TeKippe
Washington University School of Medicine in St. Louis

Carey-Ann D. Burnham
Washington University in St Louis

Donald J. Kennedy
Saint Louis University

Follow this and additional works at: https://digitalcommons.wustl.edu/open_access_pubs

Please let us know how this document benefits you.

Recommended Citation

Chamberland, Robin R.; TeKippe, Erin McElvania; Burnham, Carey-Ann D.; and Kennedy, Donald J., "Renal abscess caused by a *Providencia stuartii* isolate biochemically misidentified as *Pasteurella*." *Journal of Clinical Microbiology*. 51, 8. 2775-2777. (2013).

https://digitalcommons.wustl.edu/open_access_pubs/2172

This Open Access Publication is brought to you for free and open access by Digital Commons@Becker. It has been accepted for inclusion in Open Access Publications by an authorized administrator of Digital Commons@Becker. For more information, please contact vanam@wustl.edu.

Renal Abscess Caused by a *Providencia stuartii* Isolate Biochemically Misidentified as *Pasteurella*

Robin R. Chamberland, Erin McElvania TeKippe, Carey-Ann D. Burnham and Donald J. Kennedy
J. Clin. Microbiol. 2013, 51(8):2775. DOI:
10.1128/JCM.00937-13.
Published Ahead of Print 22 May 2013.

Updated information and services can be found at:
<http://jcm.asm.org/content/51/8/2775>

	<i>These include:</i>
REFERENCES	This article cites 6 articles, 3 of which can be accessed free at: http://jcm.asm.org/content/51/8/2775#ref-list-1
CONTENT ALERTS	Receive: RSS Feeds, eTOCs, free email alerts (when new articles cite this article), more»

Information about commercial reprint orders: <http://journals.asm.org/site/misc/reprints.xhtml>
To subscribe to to another ASM Journal go to: <http://journals.asm.org/site/subscriptions/>

Renal Abscess Caused by a *Providencia stuartii* Isolate Biochemically Misidentified as *Pasteurella*

Robin R. Chamberland,^a Erin McElvania TeKippe,^b Carey-Ann D. Burnham,^b Donald J. Kennedy^c

Department of Pathology, Saint Louis University School of Medicine, St. Louis, Missouri, USA^a; Department of Pathology & Immunology, Washington University School of Medicine, St. Louis, Missouri, USA^b; Department of Infectious Diseases, Saint Louis University School of Medicine, St. Louis, Missouri, USA^c

Providencia stuartii is associated with urinary tract infection (UTI) in catheterized patients. Here we report an abscess containing *P. stuartii* in a patient with a history of UTI, renal stones, and stent placement. This organism was identified by matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) mass spectrometry and 16S rRNA gene sequencing following biochemical identification as *Pasteurella*.

CASE REPORT

An 85-year-old man presented with increasing left-flank swelling and pain of 2 weeks duration. In addition, he had a past medical history of multiple urinary tract infections (UTIs), renal stones, and previous evaluation for left-sided hydronephrosis managed by a percutaneous nephrostomy and left urethral stent placement. The etiologies of his previous UTIs were unknown, as they had been diagnosed and treated at other facilities and those records were not available for examination. He had no other symptoms of note, including absence of fever, nausea, dysuria, or hematuria. A computerized tomography (CT) scan of the abdomen and pelvis revealed the left urethral stent and extensive inflammatory changes of the left kidney with a 3.5-by-5.4-by-6.8-cm loculated, rim-enhancing fluid collection contiguous to the upper lobe of the left kidney (Fig. 1).

On examination, the patient had a temperature of 98.6°F and a 4-by-4-cm fluctuant swelling of the left flank with mild tenderness and erythema. Laboratory values revealed a white blood cell count of $9.7 \times 10^3/\mu\text{l}$ with 66% neutrophils, a creatinine level of 1.5 mg/dl, and a urinalysis with 2,756 white blood cells/high-power field and 84 red blood cells/high-power field. Routine blood and urine cultures were negative. The patient underwent ultrasound-guided percutaneous aspiration and drainage of the left-flank fluid collection, which yielded 30 ml of thick yellow fluid.

The fluid was submitted to the laboratory for aerobic and anaerobic culture. The specimen was cultured on 5% sheep blood agar (BBL, Becton, Dickinson, Sparks, MD) and chocolate agar incubated at 37°C in 5% CO₂ and MacConkey agar incubated at 37°C in air. A Gram stain of the specimen revealed many polymorphonuclear cells and rare Gram-negative bacilli (Fig. 2). Anaerobic culture yielded moderate growth of *Fusobacterium necrophorum*. Routine culture yielded small colonies on 5% sheep blood agar and chocolate agar at 24 h, with no growth on MacConkey agar. At 48 h, there was moderate growth of plump Gram-negative bacilli with a mucoid phenotype on blood agar and chocolate agar. This organism did not grow on subculture to MacConkey agar. Biochemical identification using the Vitek2 GN identification (ID) card (bioMérieux, Durham, NC) provided an excellent identification (99% probability) of *Pasteurella canis*. Because the organism was oxidase negative (which is not accounted for by Vitek2 identification) and the Gram stain morphology was not consistent

with *Pasteurella*, a second biochemical identification system was employed. API 20 E (bioMérieux) provided a very good identification to the genus level (biocode 004402010) of *Pasteurella multocida* (86.1%) versus *Pasteurella pneumotropica*/*Mannheimia haemolytica* (13.5%). Antimicrobial susceptibility testing was performed using a combination of Etest (bioMérieux) and Kirby-Bauer disk diffusion on Mueller-Hinton agar (BBL), with the organism testing resistant to ampicillin, ceftriaxone, levofloxacin, and trimethoprim-sulfamethoxazole using CLSI interpretive criteria for *Pasteurella* (1), an antimicrobial susceptibility pattern not typical of *Pasteurella* spp. The organism was sent to a commercial reference laboratory and was identified by biochemical testing as *Pasteurella* spp.

The isolate was analyzed by matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) mass spectrometry (MS) and identified as *Providencia stuartii* using the Biotyper system (software version 3.0; Bruker-Daltonics, Billerica, MA) and Vitek MS (database version 2.0; bioMérieux). Due to the mucoid nature of the isolate, a cotton swab was lightly rolled over the colony and the residual material left on the plate was analyzed by MALDI-TOF MS. Biotyper scores of 2.013 and 2.107 (excellent identification to the species level) were obtained with and without a formic acid overlay, respectively (2). Vitek MS analysis was performed according to the manufacturer's specifications and identified the isolate as *Providencia stuartii* with a confidence value of 99.9%.

The organism was submitted to a commercial reference laboratory for 16S rRNA gene sequencing, yielding a definitive result of *Providencia stuartii*. A total of 905 bases were queried with a 99.6% match using the SmartGene database. Additional testing showed that the organism was susceptible to cefepime, imipenem, and meropenem using Etest and CLSI interpretive standards for *Enterobacteriaceae* (3).

The patient underwent a Technetium-99m renal scan, which

Received 8 April 2013 Returned for modification 5 May 2013

Accepted 14 May 2013

Published ahead of print 22 May 2013

Address correspondence to Robin R. Chamberland, rchambe6@slu.edu.

Copyright © 2013, American Society for Microbiology. All Rights Reserved.

doi:10.1128/JCM.00937-13



FIG 1 CT scan demonstrating extensive inflammatory changes in the superior portion of the left kidney (*) with evidence of a stent (arrow).

demonstrated absent perfusion, lack of function of the left kidney, and mild delay of uptake and clearance by the right kidney without evidence of obstruction. This was followed by a left total nephrectomy with pathological findings of hydronephrosis and almost-complete replacement of the renal cortex by acute and chronic inflammation. The patient was admitted 10 days after the nephrectomy with drainage from the surgical incision site. Repeat aspiration of the left-flank fluid collection revealed thick yellow fluid with many polymorphonuclear cells and growth of aerobic Gram-negative bacilli with growth characteristics and biochemical identification identical to those of the original isolate. The external drain was left in place, and the patient was treated with imipenem. A repeat CT after 18 days demonstrated a dramatic decrease in the abscess size and reduction of inflammation in the left flank.

Human infections with *Pasteurella* spp. are most commonly related to animal bites or exposures and usually present as wound, bone and joint, or respiratory tract infections. Rarely, systemic disease, such as endocarditis, and central nervous system and abdominal infections with this organism occur. *Pasteurella* is a facultatively anaerobic, pleomorphic, Gram-negative coccobacillus that does not grow on MacConkey agar. Clinically relevant species are oxidase, indole, and alkaline phosphatase positive. *Pasteurella* spp. are typically susceptible to penicillin, cephalosporins, tetracyclines, quinolones, and trimethoprim-sulfamethoxazole.

A reliable history was difficult to obtain from this patient because he is an immigrant from Eastern Europe who does not speak English. However, there is no report of any exposure to cats or other animals. Our isolate did not grow on MacConkey, was indole and alkaline phosphatase positive but oxidase negative, and demonstrated a Gram stain more typical of *Enterobacteriaceae*. Additionally, the susceptibility pattern of this isolate was not consistent with *Pasteurella*.

Complicated UTI is associated with abnormalities of the genitourinary tract, including obstruction and instrumentation (4). Causative organisms include *Enterobacteriaceae*, enterococci, and coagulase-negative staphylococci (4). Indwelling devices, such as

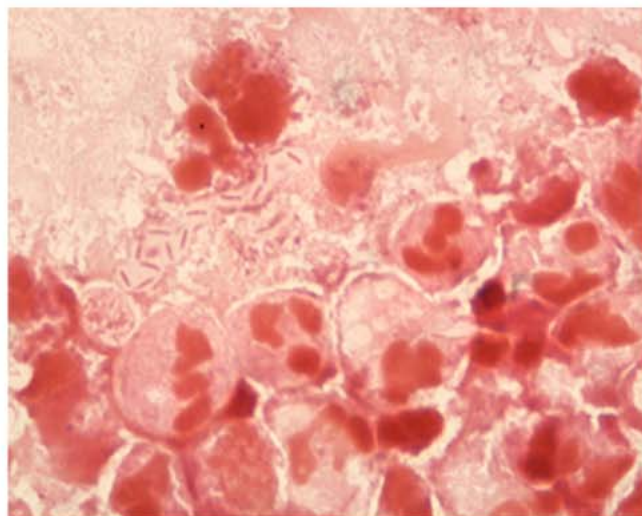


FIG 2 Gram stain of a left-flank aspirate demonstrating polymorphonuclear cells and Gram-negative bacilli surrounded by capsular material.

catheters, stents, and nephrostomy tubes, provide ideal environments for biofilm formation, leading to posttreatment relapses and increasing antimicrobial resistance (4). Urease-producing *Proteaceae* have been implicated in the formation of crystalline biofilms via their ability to generate ammonia from urea, raise the pH of urine, and form calcium and magnesium phosphate crystals (5). About 30% of *P. stuartii* isolates are urease positive; however, our patient's isolate was not. Additionally, biofilm formation appears to be indole dependent in some indole-positive organisms implicated in UTI, including *P. stuartii* (6). While *P. stuartii* is not a common cause of uncomplicated UTI, it has long been associated with persistent infections of indwelling catheters in nursing home settings (7, 8).

Identification in this case was complicated by atypical phenotypic characteristics, including the mucoid phenotype of *P. stuartii*, the fact that it was biochemically inert, and its failure to grow on MacConkey agar. Initial susceptibility results indicating resistance to ampicillin, ceftriaxone, levofloxacin, and trimethoprim-sulfamethoxazole strongly suggested that this organism was not a *Pasteurella* species (1). However, this pattern was not unexpected for *P. stuartii*, as this organism carries a chromosomal inducible AmpC β -lactamase, and the patient had been treated for several UTIs prior to presentation. Cefepime and carbapenems remained susceptible. This case demonstrates the limitation of biochemical identification for even common organisms showing unusual phenotypes. If empirical therapy had been based on the identification of *Pasteurella*, the patient likely would not have received the appropriate antimicrobial agents. Neither 16S rRNA gene sequencing nor MALDI-TOF MS relies on the biochemical reactivity pattern of an organism, and both have the capability to provide identification when conventional means fail. The rapidity and marginal cost of identification by MALDI-TOF make it the preferable method for routine organism identification in many cases.

REFERENCES

1. Clinical and Laboratory Standards Institute. 2010. Methods for antimicrobial dilution and disk susceptibility testing of infrequently isolated or

- fastidious bacteria; approved guideline—second edition. CLSI document M45-A2. Clinical and Laboratory Standards Institute, Wayne, PA.
2. Ford BA, Burnham CA. 2013. Optimization of routine identification of clinically relevant Gram-negative bacteria using MALDI-TOF MS and the Bruker Biotyper. *J. Clin. Microbiol.* **51**:1412–1420.
 3. Clinical and Laboratory Standards Institute. 2013. Performance standards for antimicrobial susceptibility testing; 23rd informational supplement. CLSI document M100-S23. Clinical and Laboratory Standards Institute, Wayne, PA.
 4. Nicolle LE, AMMI Canada Guidelines Committee. 2005. Complicated urinary tract infection in adults. *Can. J. Infect. Dis. Med. Microbiol.* **16**:349–360.
 5. Broomfield RJ, Morgan SD, Khan A, Stickler DJ. 2009. Crystalline bacterial biofilm formation on urinary catheters by urease-producing urinary tract pathogens: a simple method of control. *J. Med. Microbiol.* **58**:1367–1375.
 6. Martino PD, Fursy R, Bret L, Sundararaju B, Phillips RS. 2003. Indole can act as an extracellular signal to regulate biofilm formation of *Escherichia coli* and other indole-producing bacteria. *Can. J. Microbiol.* **49**:443–449.
 7. Warren JW, Tenney JH, Hoopes JM, Muncie HL, Anthony WC. 1982. A prospective microbiologic study of bacteriuria in patients with chronic indwelling urethral catheters. *J. Infect. Dis.* **146**:719–723.
 8. Warren JW. 1986. *Providencia stuartii*: a common cause of antibiotic-resistant bacteriuria in patients with long-term indwelling catheters. *Rev. Infect. Dis.* **8**:61–67.