Clinical Vignettes

Staphylococcus Lugdunensis Native Tricuspid Valve Endocarditis: a Case Report and Review of Literature

Ritesh Patil, MD, MPH[1], Trupti Patil, MD, MPH[2], and K. M. Anwar Hussain, MD, FACP, FACC[3]

[1]Millard Fillmore Gates Circle Hospital, Buffalo, NY, USA; [2]Infectious Disease, State University of New York at Buffalo, Buffalo, NY, USA; [3]Temple University School of Medicine, Philadelphia, PA, & Attending Cardiologist, Memorial Medical Center, Johnstown, PA, USA.

Coagulase negative staphylococci are skin commensals and are generally disregarded as contaminants in clinical specimens. Repeated isolation of coagulase negative staphylococci in blood cultures should warrant a species identification to recognize unusually virulent organisms that demand aggressive treatment, such as *Staphylococcus lugdunensis. Staphylococcus lugdunensis* is known to cause a wide variety of infections, including a predominant left-sided endocarditis. We report a rare case of native tricuspid valve *Staphylococcus lugdunensis* endocarditis in a non-intravenous drug user and include a brief literature review.

KEY WORDS: Staphylococcus lugdunensis; coagulase negative staphylococci; skin commensals; left-sided endocarditis.
J Gen Intern Med 26(10):1209–11
DOI: 10.1007/s11606-011-1718-5

INTRODUCTION

Coagulase negative staphylococci (CNS) are normal skin flora that cause a wide variety of clinical infections. They are generally disregarded as contaminants in clinical specimens. Repeated isolation of coagulase negative staphylococci in blood cultures should warrant a species identification to recognize unusually virulent organisms that demand aggressive treatment, such as *Staphylococcus lugdunensis* (*S. lugdunensis*). *S. lugdunensis* is has been reported to cause central nervous system infections, abscesses, wound infections, joint infections, and endocarditis.[1–3] We report a rare case of native tricuspid valve *S. lugdunensis* endocarditis in a non-intravenous drug user and include a brief literature review.

CASE PRESENTATION

A 46-year-old white man was transferred to our hospital after presenting to an outside facility with nausea, vomiting, loss of appetite, bilateral leg swelling and intermittent low-grade fevers for two weeks. He had several previous hospitalizations at our facility, with multiple exploratory laparotomies for blunt abdominal trauma sustained during a motor vehicle accident. His clinical course was complicated by a ventral hernia mesh infection that led to an enterocutaneous fistula. He became dependent on parenteral nutrition administered via a peripherally inserted central catheter (PICC). From his previous hospital admissions, he had a total of eight (four sets of two) blood cultures positive for CNS. Two of the positive cultures were drawn from the PICC line. He was treated with vancomycin and PICC lines were removed on three separate occasions. Once the blood cultures identified CNS, the organisms were considered contaminants and antibiotics were discontinued.

At the outside hospital, he was febrile (104.7 F), tachycardic (152 beats per minute) and hypotensive (95/56 mmHg). He was hydrated with intravenous fluids and was transferred to our facility. Upon arrival to our ED for his current admission, he was hypothermic at 95.4 F and persistently hypotensive (86/59 mmHg). Physical exam revealed a well healing abdominal incision and bilateral lower extremity pitting edema. White blood cell count was 6,000/mm^3 with 6% immature neutrophils. Vancomycin was started empirically because of the prior knowledge of blood cultures containing CNS. The number of previous positive cultures raised the concern that this was not a contaminant. Shortly after admission (day 2), blood cultures returned positive for CNS. This prompted a transthoracic echocardiogram. Echocardiogram showed multiple vegetations on the tricuspid valve with the largest being 2.6 cm X 1.9 cm (Fig. 1). Vancomycin was continued with a consideration for surgical intervention of the tricuspid valve in the near future.

While being treated, he developed a new onset dry cough. A chest x-ray was performed that showed right lung perihilar and lower lobe infiltrate. In addition a left upper lobe nodule measuring 14 mm was noted. To further delineate these lesions, a computerized tomography scan of the chest was performed that showed multiple, bilateral, ill-defined, 1.0-to-2.0 cm pulmonary opacities suggestive of septic emboli (Fig. 2a and b).

Species identification was performed at this time, and the organism was identified as *S. lugdunensis* (day 9). Based on sensitivities, vancomycin was switched to nafcillin, after which he started to defervesce. Repeat blood cultures showed no growth. Tricuspid valve replacement was subsequently performed. Nafcillin was administered via a PICC line for a total of 4 weeks with good clinical response.

DISCUSSION

S. lugdunensis is an unusually virulent coagulase-negative staphylococcus that was first reported by Frenet et al. in 1988. It was isolated in a Lyon, France, where it derives its name.

Received November 12, 2009
Revised March 24, 2010
Accepted March 29, 2011
Published online May 3, 2011

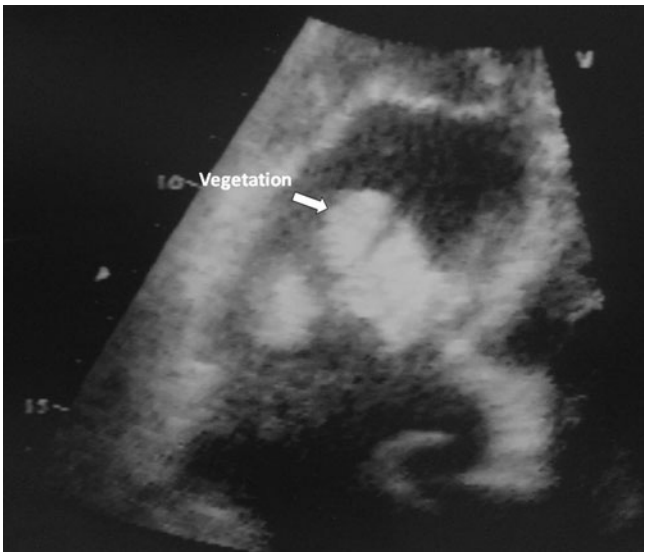

Figure 1. 2D-Echo demonstrating large vegetations on tricuspid valve leaflets. RV=right ventricle, RA=right atrium.

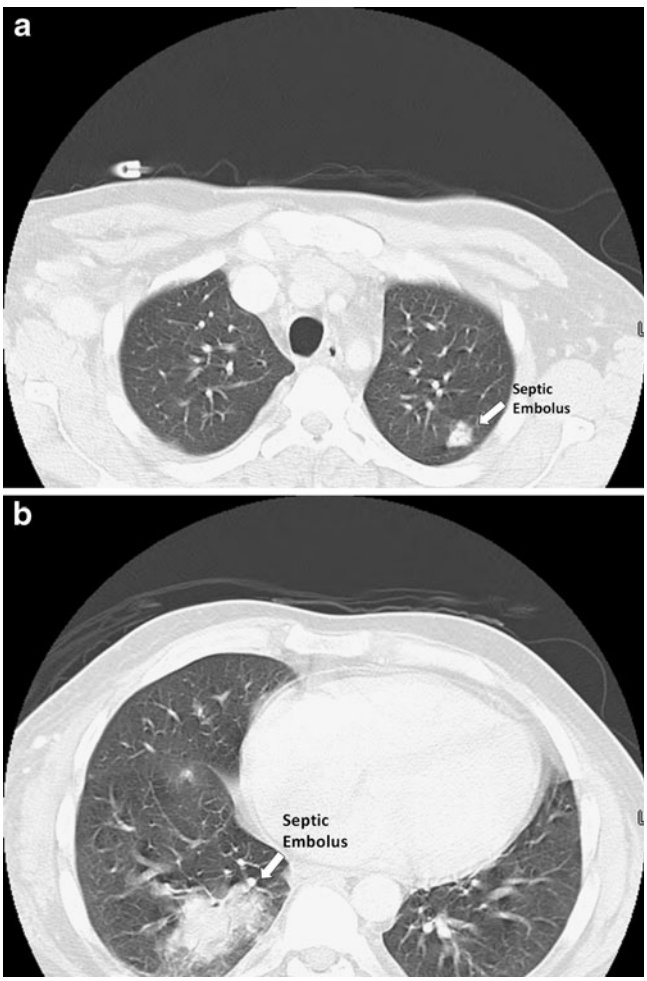

Figure 2. a, b CT chest demonstrating pulmonary septic emboli.

Lyon is the Latin adjective of Lugdunum.[2,3] Since its isolation, *S. lugdunensis* has been increasingly recognized as an emerging infection, causing both community-acquired and nosocomial infections.[1]

CNS are considered normal inhabitants of the skin.[4] The most common CNS species identified to cause human infections include *S. epidermidis, S. hemolyticus, S. saprophyticus,* and *S. lugdunensis*. CNS are generally deemed as contaminants in clinical specimens, but recurrent blood cultures for CNS should raise the suspicion of a pathogenic organism such as *S. lugdunensis*. Unlike other CNS, *S. lugdunensis* infections resemble Staphylococcus aureus infections in terms of virulence, tissue destruction and clinical course. Early identification and treatment of *S. lugdunensis* is important because of its unusual virulence.[5] In addition, *S. lugdunensis* is rarely a clinical specimen contaminant, and its isolation warrants further investigation.[6]

CLINICAL FEATURES OF *S. LUGDUNENSIS* INFECTION

S. lugdunensis is a part of the resident flora of the human skin, particularly in the perineum and the breast.[7] It has been associated with a wide variety of infections, including skin and soft tissue infections,[3] lymphadenitis,[8] osteomyelitis and prosthetic joint infection,[5] endophthalmitis,[9] peritonitis, meningitis,[1] brain abscesses, urinary tract infections,[10] pacemaker infections,[1] and endocarditis.[2,7,11]

Endocarditis is the one of the most devastating complications of an *S. lugdunensis* infection. In contrast to other CNS having a subacute or indolent presentation, *S. lugdunensis* has a fulminant course similar to *S. aureus* endocarditis with frequent complications (60%). Anguera et al. in their review of 69 patients noted heart failure (45%), periannular abscess (19%), and systemic embolization (30%) to be the common complications.[3]

S. lugdunensis predominantly causes native valve endocarditis (77%), as opposed to other CNS that tend to infect prosthetic valves.[3,7] It has a predilection to involve the left-sided valves in the following order: mitral valve (49%), aortic valves (26%) and both mitral and aortic valves (17%).[7] Ours is an unusual case of tricuspid valve endocarditis. Very few cases involving the right side of the heart have been described in the literature. Vegetations caused by *S. lugdunensis* tend to be large[11] and prone to embolization.

Van Hoovels et al. in their literature review of 48 cases of *S. lugdunensis endocarditis* found that most patients (74%) have an acute presentation with symptoms of less than 3 weeks' duration.[11] Seenivasan and Yu[7] in their review of 47 patients with *S. lugdunensis endocarditis* in 2003 report 54 years as the mean age at diagnosis with 66% being males.

Among the patients with a known portal of entry, perineal skin is one of the common sites of entry. Procedures that involve a femoral approach, such as cardiac catheterization, femoral popliteal bypass, and pacemaker insertion, may be at a higher risk of *S. lugdunensis* bacteremia and endocarditis.[7,12,13]

Although *S. lugdunensis* is susceptible to most antibiotics, including beta-lactams, in-vitro, medical therapy alone is rarely successful. Most patients require some form of surgical intervention. In a review of 69 patients, 51% with native valve endocarditis and 55% with prosthetic valve

infection required surgical intervention.[3] In addition, the mortality risk from medical therapy alone is higher compared to combined medical-surgical therapy. A recent analysis of the treatment strategies for native valve *S. lugdunensis* endocarditis revealed that the mortality risk was 3.1 times higher for those patients that received only medical therapy.[14]

With the implementation of combined medical-surgical therapy, the mortality rate associated with *S. lugdunensis* endocarditis appears to be on the decline. Vandenesch et al. reviewed 20 patients of *S. lugdunensis* endocarditis in 1993 and reported a mortality rate of 70%.[15] However, in the 28 cases identified by Seenivasan and Yu in 2003, they reported a lower mortality of 18%.[7] It has been speculated that this decline could also be due to early detection of complicated endocarditis as a result of higher sensitivity of modern noninvasive imaging techniques, there by prompting early surgical intervention.[12]

CONCLUSION

Infections due to CNS are common and are on the rise with increasing use of intravenous catheters. They are generally regarded as contaminants in clinical specimens. Our case emphasizes the fact that repeated positive cultures for CNS demand attention and further investigation. It should prompt a species level identification of the organism to recognize virulent strains such as *S. lugdunensis*. Identification of *S. lugdunensis* together with knowledge of the organism's natural history are extremely important preconditions for successful management of affected patients.

Key Points

- Repeated positive cultures of coagulase negative staphylococcus (CNS) demand attention and species identification
- *S.lugdunensis* is an aggressive CNS, with virulence similar to *S.aureus*.
- *S.lugdunensis* causes a predominant left sided endocarditis
- Combined medical-surgical therapy is effective

Acknowledgements: *None.*

Funding Sources: *None.*

Conflict of Interest: *None disclosed.*

Corresponding Author: *Ritesh Patil, MD, MPH; Millard Fillmore Gates Circle Hospital, 3 Gates Circle, Buffalo, NY 14209, USA (e-mail: drriteshpatil@yahoo.co.in).*

REFERENCES

1. **Seifert H, Oltmanns D, Becker K, Wisplinghoff H, von Eiff C.** Staphylococcus lugdunensis pacemaker-related infection. Emerg Infect Dis. 2005;11(8):1283–6.
2. **Hellbacher C, Törnqvist E, Söderquist B.** Staphylococcus lugdunensis: clinical spectrum, antibiotic susceptibility, and phenotypic and genotypic patterns of 39 isolates. Clin Microbiol Infect. 2006;12(1):43–9.
3. **Anguera I, Del Río A, Miró JM, Matínez-Lacasa X, Marco F, Gumá JR, Quaglio G, Claramonte X, Moreno A, Mestres CA, Mauri E, Azqueta M, Benito N, García-de la María C, Almela M, Jiménez-Expósito MJ, Sued O, De Lazzari E, Gatell JM, Hospital Clinic Endocarditis Study Group.** Staphylococcus lugdunensis infective endocarditis: description of 10 cases and analysis of native valve, prosthetic valve, and pacemaker lead endocarditis clinical profiles. Heart. 2005;91(2):e10.
4. **Tan TY, Ng SY, Ng WX.** Clinical significance of coagulase-negative staphylococci recovered from nonsterile sites. J Clin Microbiol. 2006;44(9):3413–4.
5. **Greig JM, Wood MJ.** Staphylococcus lugdunensis vertebral osteomyelitis. Clin Microbiol Infect. 2003;9(11):1139–41.
6. **Herchline TE, Ayers LW.** Occurrence of Staphylococcus lugdunensis in consecutive clinical cultures and relationship of isolation to infection. J Clin Microbiol. 1991;29(3):419–21.
7. **Seenivasan MH, Yu VL.** Staphylococcus lugdunensis Endocarditis—the hidden peril. Eur J Clin Microbiol Infect Dis. 2003;22(8):489–91.
8. **Kim JH, Lee JY, Kim HR, Heo KW, Park SK, Lee JN, Yu SM, Shin JH.** Acute lymphadenitis with cellulitis caused by Staphylococcus lugdunensis. Korean J Lab Med. 2008;28(3):196–200.
9. **Chiquet C, Pechinot A, Creuzot-Garcher C, Benito Y, Croize J, Boisset S, Romanet JP, Lina G, Vandenesch F, French Institutional Endophthalmitis Study Group.** Acute postoperative endophthalmitis caused by Staphylococcus lugdunensis. J Clin Microbiol. 2007;45(6):1673–8.
10. **Spanu T, Rigante D, Tamburrini G, Fiori B, D'Inzeo T, Posteraro B, Policicchio D, Sanguinetti M, Fadda G.** Ventriculitis due to Staphylococcus lugdunensis: two case reports. J Med Case Reports. 2008;11(2):267.
11. **Van Hoovels L, De Munter P, Colaert J, Surmont I, Van Wijngaerden E, Peetermans WE, Verhaegen J.** Three cases of destructive native valve endocarditis caused by Staphylococcus lugdunensis. Eur J Clin Microbiol Infect Dis. 2005;24(2):149–52.
12. **Zinkernagel AS, Zinkernagel MS, Elzi MV, Genoni M, Gubler J, Zbinden R, Mueller NJ.** Significance of Staphylococcus lugdunensis bacteremia: Report of 28 cases and review of literature. Infection. 2008;36(4):314–21.
13. **Takahashi N, Shimada T, Ishibashi Y, Yoshitomi H, Sugamori T, Sakane T, Sato H, Oyake N, Murakami Y.** The pitfall of coagulase-negative staphylococci A case of Staphylococcus lugdunensis endocarditis. Int J Cardiol. 2009;137(1):e15–7.
14. **Jung JJ, Yoo D, Law K, Wang YF, Higgins S.** Recurrent Staphylococcus lugdunensis Infective Endocarditis and Review of the Literature. Infect Dis Clin Pract. 2010;18:16–22.
15. **Vandenesch F, Etienne J, Reverdy ME, Eykyn SJ.** Endocarditis due to Staphylococcus lugdunensis: report of 11 cases and review. Clin Infect Dis. 1993;17(5):871–6.