IDCases 18 (2019) e00656

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcr

Case report

Staphylococcus pasteuri infective endocarditis: A case report

Jaineel Ramnarain, Jang Yoon, Naomi Runnegar*

Princess Alexandra Hospital, 199 Ipswich Road, Woolloongabba, Queensland, 4102, Australia

ARTICLE INFO

Article history:
Received 26 August 2019
Received in revised form 4 October 2019
Accepted 5 October 2019

Keywords:
Staphylococcus pasteuri
Coagulase negative staphylococci
Infective endocarditis
Native valve endocarditis

ABSTRACT

Staphylococcus pasteuri is a coagulase negative bacterium which although formally described in 1993, has only recently become possible to reliably speciate in diagnostic microbiology laboratories. *S. pasteuri* remains an extremely infrequent cause of human infection to date, namely bacteremia in an individual suffering acute myeloid leukemia, catheter-associated urinary tract infection in a patient receiving chemotherapy and endocarditis within a case series without specific clinical information. As such, our report provides the first detailed account of *Staphylococcus pasteuri* infective endocarditis entailing a subacute community-onset infection involving native aortic and mitral valves, multiple systemic emboli, and ultimately cardiothoracic surgery.

© 2019 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Introduction

Infective endocarditis (IE) is a microbial infection of the endocardial surface of the heart [1]. IE can be a difficult diagnosis due to the diversity and often unclear nature of symptoms. In Australia, IE affects 3–10 per 100,000 individuals [2] with *Staphylococcus aureus* constituting up to 32% of cases [3]. Coagulase negative staphylococci (CoNS) account for 6%–7% of infections of which >85% are attributed to *Staphylococcus epidermidis*; the remainder are due to multiple other variants although not routinely delineated to the species level [4]. This is especially problematic within health-care settings wherein CoNS are responsible for ~40% of native valve [4] and over 50% of prosthetic valve IE [5]. Despite their relative avirulence, native valve CoNS infective endocarditis has a hospital mortality of 19% which approaches *Staphylococcus aureus* IE at 25% [4]. To the best of our knowledge, this is the first prospectively documented case of *S. pasteuri* IE.

Case

A 65 year old male retired builder presented to hospital with left chest pain arising suddenly overnight. His pain was pleuritic and radiated to the ipsilateral shoulder on inspiration. He also described weight loss, night sweats and progressive low back pain over the preceding month. His medical history included amyloidosis, aortic stenosis, chronic back pain and hepatitis C. Methadone constituted his only regular medication and he had no known drug allergies. Family history was insignificant whereas his social situation entailed previous intravenous drug use, current smoking and alcohol misuse.

Examination revealed a man of small stature in reasonable overall health. Vital signs verified apyrexia as well as hemodynamic stability. Cardio-respiratory assessment revealed a constellation of important findings: multiple tattoos across his bilateral upper limbs, injection track marks, extensive dental decay, prominent ejection systolic murmur and left upper quadrant tenderness with guarding. There were no signs of congestive cardiac failure.

Full blood examination showed normocytic anemia (119 g/L [reference interval: 135–180 g/L]) while white cell count, differential and platelets were within normal limits. Urea and electrolyte panel found reduced creatinine (60 g/L [64–108 g/L]) without further abnormalities. Liver function tests revealed elevated aspartate aminotransferase (44 U/L [<35 U/L]) and lactate dehydrogenase (263 U/L [120–250 U/L]), while albumin was low (30 g/L [35–50 g/L]). Coagulation studies were unaffected; lipase was normal. Cardiac enzymes were serially non-elevated. In contrast C-reactive protein (102 mg/L [<5 mg/L]) was raised.

Electrocardiography revealed nil ischemia while computed tomography pulmonary angiography found no pulmonary emboli. Contrast abdominal and pelvic computed tomography showed an enlarged spleen with non-enhancing focus suggestive of infarction (Fig. 1). Lumbosacral magnetic resonance imaging uncovered bilateral psoas abscesses and lumbar vertebral discitis (L1/L2) within a diffusely degenerative spine (Fig. 2).

Transthoracic and transesophageal echocardiography both demonstrated retracted aortic leaflets associated with lack of

* Corresponding author.
E-mail address: naomi.runnegar@health.qld.gov.au (N. Runnegar).

https://doi.org/10.1016/j.idcr.2019.e00656
2214-2509/© 2019 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

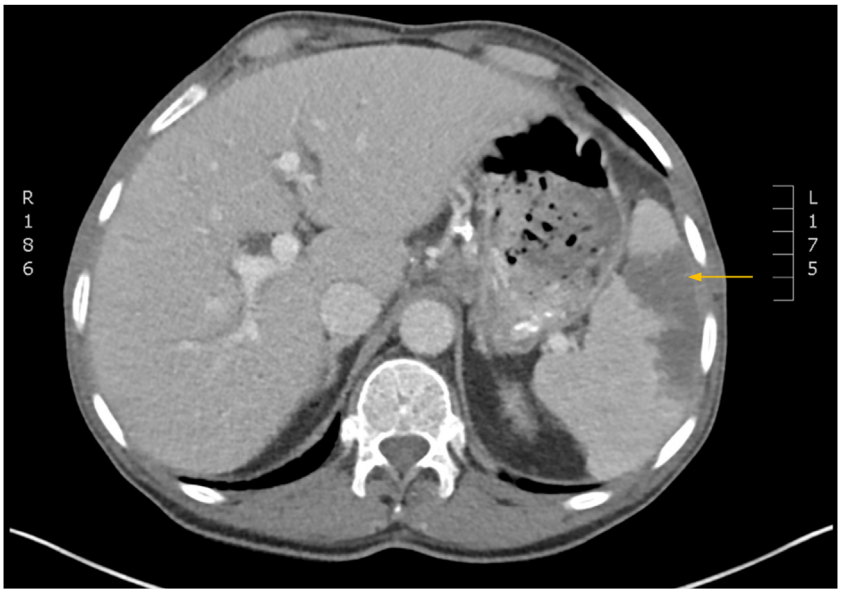

Fig. 1. Computed tomography abdomen and pelvis demonstrating hypointense lesion suggesting splenic infarction (arrow).

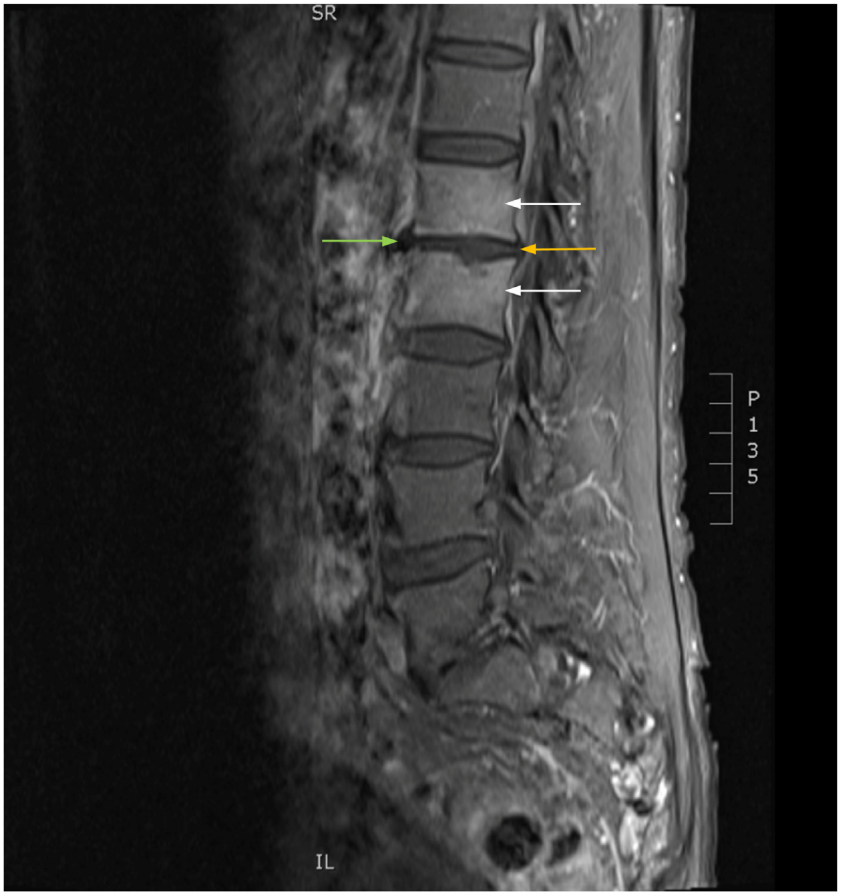

Fig. 2. Lumbosacral magnetic resonance imaging with multiple foci depicting L1/L2 discitis (orange arrow), osteomyelitis (white arrows) and psoas abscess (green arrow).

coaptation and severe regurgitation in addition to a linear, mobile subvalvular mitral echodensity (Fig. 3) without significant regurgitation. Taken together these findings established infective endocarditis as per Modified Duke Criteria [6] in conjunction with prior clinical features. Three sets of blood cultures grew a single microorganism speciated by matrix-assisted laser desorption ionisation time-of-flight mass spectrometry (MALDI-TOF MS) [VITEK® MS, bioMérieux] as *Staphylococcus pasteuri*. This beta-lactamase negative organism was susceptible to penicillin, flucloxacillin and cephalexin [VITEK® 2, bioMérieux].

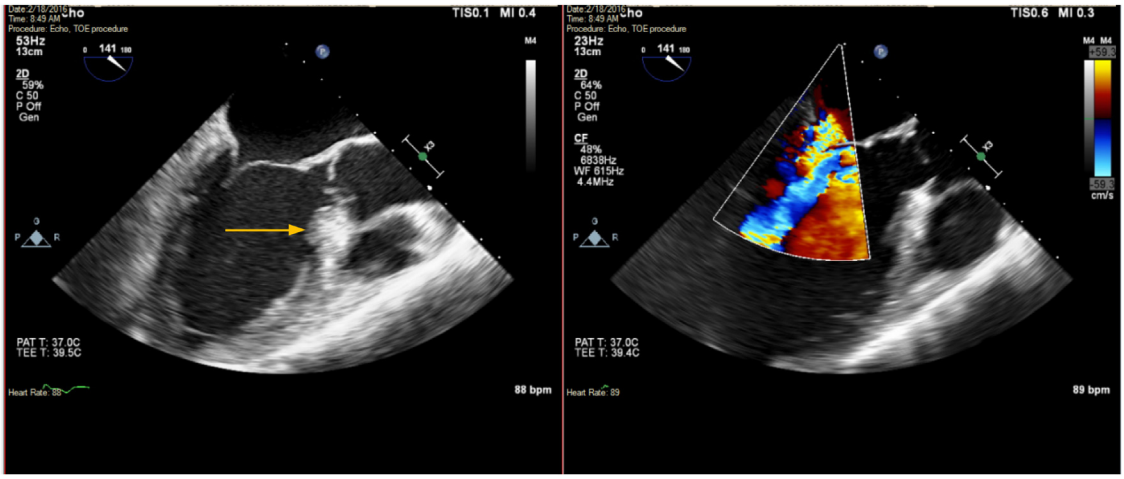

Fig. 3. Transesophageal echocardiography depicting mitral subvalvular chordae echodensity (14 mm) in keeping with vegetation (arrow) along with severe aortic regurgitation on color-flow Doppler (blue jet).

Intravenous benzylpenicillin was accordingly commenced – 2.4 g (4 million units) every four hours – however progressive valvular dysfunction necessitated cardiothoracic surgery. Fragile vegetations involving the left coronary cusp with complete valve destruction were noted intra-operatively for which a bioprosthetic Carpentier-Edwards Perimount Magna Ease aortic valve replacement was performed. In contrast, a solitary lesion affixed to the anterior mitral leaflet was effectively managed by surgical debridement. The explanted native aortic valve was culture-negative after five days; the mitral vegetation was not successfully harvested for microbial analysis due to friability.

Despite critical complications of post-operative tamponade and systolic heart failure, the patient responded well to multidisciplinary intervention by cardiothoracic surgeons, cardiologists and infectious disease physicians comprising open pericardial clot evacuation, anti-failure pharmaceuticals namely ramipril, bisoprolol, frusemide and spironolactone, cardiac rehabilitation as well as extended antimicrobials totaling 12 weeks of penicillin respectively. He presently retains pre-morbid functioning beyond three years since diagnosis.

Discussion

Bacterial endocarditis is predominately caused by staphyloccocal species which are classified according to biochemical profile, principally coagulase reaction. CoNS however are not routinely subtyped given they lack reliable methods of species differentiation [7,8], often represent contamination and generally convey low pathogenicity [8]. In addition *Staphylococcus pasteuri* has frequently been misidentified as *Staphylococcus warneri* due to high phenotypic similarity [9] which potentially limits data on its role in infectious disease. Nevertheless, increasing availability of newer modalities such as MALDI-TOF MS or ribonucleic acid polymerase β subunit (*rpoB*) sequencing [7] improve routine identification of CoNS subspecies as well as other uncommon pathogens.

Staphylococcus pasteuri was first described as a unique species in 1993 [9]. While its preferred ecological niche is uncertain, the microbe has been isolated from an array of sources including vegetables, goat milk, naturally fermented Italian sausages, vacuum-packed lamprey [8], drinking water supplies [10] and stratospheric air samples [11]. Clinically, it is over-represented in the gastrointestinal microbiota of children with active celiac disease [12]. It is a recognized contaminant of platelet transfusions [13–15] and has also been isolated from human vomit, urine, blood, peri-prosthetic tissue and bones without clearly precipitating disease [8,9].

The few case reports strongly suggestive of *S. pasteuri* human infection however include infective endocarditis retrospectively identified from a collection of CoNS isolates without specific clinical details [7], bacteremia in a patient suffering acute myeloid leukemia [16] and a catheter-associated urinary tract infection in an individual receiving cervical cancer chemotherapy [17]. Prognosis was favorable where described.

In our patient, *S. pasteuri* was repeatedly and exclusively isolated on serial blood cultures. Most importantly, MALDI-TOF MS was utilized to facilitate accurate microbial diagnosis [18]. Unfortunately negative aortic valve cultures did not reaffirm these findings likely due to antecedent antimicrobials administered prior to surgery [19].

In summary, this case not only highlights *Staphylococcus pasteuri* as a possible pathogenic species, but also demonstrates the serious sequelae it may precipitate in the setting of IE. On this occasion the microorganism exhibited no resistance against routinely therapeutic antimicrobials unlike other isolates which have displayed variable microbial sensitivities [20]. Given the availability of modern molecular methods, this case supports routine speciation of CoNS to characterize the epidemiology, clinical significance and implications of this emerging microbe in human diseases such as IE.

Sources of funding

None.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution

Jaineel Ramnarain – Conceptualisation, Writing – Original Draft, Writing – Editing and Reviewing, Visualisation, Project Administration.

Jang Yoon – Methodology, Investigation, Writing – Editing and Reviewing, Visualisation.

Naomi Runnegar – Writing – Editing and Reviewing, Supervision.

Declaration of Competing Interest

None.

Acknowledgements

None.

References

[1] Cahill TJ, Prendergast BD. Infective endocarditis. Lancet 2016;387 (10021):882–93, doi:http://dx.doi.org/10.1016/S0140-6736(15)00067-7.
[2] Cahill TJ, Baddour LM, Habib G, et al. Challenges in infective endocarditis. J Am Coll Cardiol 2017;69(3):325–44, doi:http://dx.doi.org/10.1016/j.jacc.2016.10.066.
[3] Sy RW, Kritharides L. Health care exposure and age in infective endocarditis: results of a contemporary population-based profile of 1536 patients in Australia. Eur Heart J 2010;31(15):1890–7, doi:http://dx.doi.org/10.1093/eurheartj/ehq110.
[4] Chu VH, Cabell CH, Abrutyn E, et al. Native valve endocarditis due to coagulase-negative staphylococci: report of 99 episodes from the International Collaboration on Endocarditis Merged Database. Clin Infect Dis 2004;39 (10):1527–30, doi:http://dx.doi.org/10.1086/424878.
[5] Lalani T, Kanafani ZA, Chu VH, et al. Prosthetic valve endocarditis due to coagulase-negative staphylococci: findings from the International Collaboration on Endocarditis Merged Database. Eur J Clin Microbiol Infect Dis 2006;25(6):365–8, doi:http://dx.doi.org/10.1007/s10096-006-0141-z.
[6] Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke Criteria for the diagnosis of infective endocarditis. Clin Infect Dis 2000;30(4):633–8, doi:http://dx.doi.org/10.1086/313753.
[7] Petti CA, Simmon KE, Miro JM, et al. Genotypic diversity of coagulase-negative staphylococci causing endocarditis: a global perspective. J Clin Microbiol 2008;46(5):1780–4, doi:http://dx.doi.org/10.1128/JCM.02405-07.
[8] Savini V, Catavitello C, Bianco A, Balbinot A, Antonio DD, D'Antonio D. Epidemiology, pathogenicity and emerging resistances in Staphylococcus pasteuri: from mammals and lampreys, to man. Recent Pat Antiinfect Drug Discov 2009;4(2):123–9.
[9] Chesneau O, Morvan A, Grimont F, Labischinski H, El Solh N. Staphylococcus pasteuri sp. nov., isolated from human, animal, and food specimens. Int J Syst Bacteriol 1993;43(2):237–44, doi:http://dx.doi.org/10.1099/00207713-43-2-237.
[10] Faria C, Vaz-Moreira I, Serapicos E, Nunes OC, Manaia CM. Antibiotic resistance in coagulase negative staphylococci isolated from wastewater and drinking water. Sci Total Environ 2009;407(12):3876–82, doi:http://dx.doi.org/10.1016/j.scitotenv.2009.02.034.
[11] Wainwright M, Wickramasinghe NC, Narlikar JV, Rajaratnam P. Microorganisms cultured from stratospheric air samples obtained at 41 km. FEMS Microbiol Lett 2003;218(1):161–5, doi:http://dx.doi.org/10.1111/j.1574-6968.2003.tb11513.x.
[12] Sánchez E, Donat E, Ribes-Koninckx C, Fernández-Murga ML, Sanz Y. Duodenal-mucosal bacteria associated with celiac disease in children. Appl Environ Microbiol 2013;79(18):5472–9, doi:http://dx.doi.org/10.1128/AEM.00869-13.
[13] Savini V, Catavitello C, Pompetti F, et al. Contamination of a donated platelet unit by Staphylococcus pasteuri. J Infect 2008;57(6):494–6, doi:http://dx.doi.org/10.1016/j.jinf.2008.10.006.
[14] Bianco A, Pompilio A, Savini V, et al. Meticillin-heteroresistant Staphylococcus pasteuri from an apheresis platelet product. J Med Microbiol 2009;58 (11):1527–8, doi:http://dx.doi.org/10.1099/jmm.0.008193-0.
[15] Rood IGH, de Korte D, Ramírez-Arcos S, Savelkoul PHM, Pettersson A. Distribution, origin and contamination risk of coagulase-negative staphylococci from platelet concentrates. J Med Microbiol 2011;60(5):592–9, doi:http://dx.doi.org/10.1099/jmm.0.023176-0.
[16] Savini V, Catavitello C, Carlino D, et al. Staphylococcus pasteuri bacteraemia in a patient with leukaemia. J Clin Pathol 2009;62(10):957–8, doi:http://dx.doi.org/10.1136/jcp.2009.067041.
[17] Morfin-Otero R, Martínez-Vázquez MA, López D, Rodríguez-Noriega E, Garza-González E. Isolation of rare coagulase-negative isolates in immunocompromised patients: staphylococcus gallinarum, Staphylococcus pettenkoferi and Staphylococcus pasteuri. Ann Clin Lab Sci 2012;42(2):182–5.
[18] Croxatto A, Prod'hom G, Greub G. Applications of MALDI-TOF mass spectrometry in clinical diagnostic microbiology. FEMS Microbiol Rev 2012;36(2):380–407, doi:http://dx.doi.org/10.1111/j.1574-6976.2011.00298.x.
[19] Hoen B. Epidemiology and antibiotic treatment of infective endocarditis: an update. Heart 2006;92(11):1694–700, doi:http://dx.doi.org/10.1136/hrt.2005.072595.
[20] Simeoni D, Rizzotti L, Cocconcelli P, Gazzola S, Dellaglio F, Torriani S. Antibiotic resistance genes and identification of staphylococci collected from the production chain of swine meat commodities. Food Microbiol 2008;25 (1):196–201, doi:http://dx.doi.org/10.1016/j.fm.2007.09.004.