CASE REPORT

Endocarditis of the native aortic valve caused by *Lactobacillus jensenii*

Soumya Patnaik,¹ Carlos Daniel Davila,¹ Anupama Chennupati,² Alexander Rubin³

¹Department of Internal Medicine, Albert Einstein Medical Center, Philadelphia, Pennsylvania, USA

²Department of Medicine, Albert Einstein Medical Center, Philadelphia, Pennsylvania, USA

³Division of Cardiology, Albert Einstein Medical Center, Philadelphia, Pennsylvania, USA

Correspondence to

Dr Soumya Patnaik,
patnaiks@einstein.edu

Accepted 18 February 2015

SUMMARY

Lactobacilli are Gram-positive anaerobic rods or coccobacilli, commonly found as commensals in human mucosa. Rarely, they can cause serious infections such as infective endocarditis (IE), and the most frequently implicated species causing serious infections are *L. casei* and *L. rhamnosus*. IE caused by *Lactobacillus jensenii* is very rare, with only six reported cases so far, to the best of our knowledge. We present a case of native aortic valve endocarditis caused by *L. jensenii*, complicated by root abscess and complete heart block, and requiring emergent surgical intervention.

BACKGROUND

The epidemiology and microbiology of infective endocarditis (IE) are changing.¹ Lactobacilli are pleomorphic, Gram-positive, facultative anaerobic or microaerophilic, or strictly anaerobic rods or coccobacilli, commonly found as commensals in human mucosa. They can cause serious infections such as meningitis, pneumonia, IE and sepsis, as well as other suppurative infections, particularly in immunocompromised patients. Mortality from *Lactobacillus septicaemia* is reported to be as high as 30%.²

Most reported cases of *Lactobacillus* IE have been caused by *L. casei* and *L. rhamnosus*.³ The presented case demonstrates IE caused by *Lactobacillus jensenii* in the setting of a high-degree atrioventricular (AV) block.

CASE PRESENTATION

A 56-year-old African-American man with diabetes with a history of paraplegia, and neurogenic bladder necessitating chronic indwelling urinary catheterisation, presented to the emergency room with a 3-day history of chest pain, shortness of breath and extreme generalised weakness. He denied any fever, chills, headache or palpitations. He had a history of recurrent urinary tract infections with *Escherichia coli* and *Pseudomonas*. The patient was a non-smoker, non-alcoholic and denied any drug misuse. There was no history suggestive of dietary supplements, probiotic or vitamin use or excessive intake of dairy products.

On admission, the patient's vitals were unstable, with a blood pressure of 83/42 mm Hg, pulse rate 49/min, respiratory rate 24/min and hypoxia. His temperature was 36.7°C. He was drowsy and disoriented to time and place. Head and neck examination revealed poor dentition, broken teeth and no oral thrush. Cardiovascular examination revealed normal, regular heart sounds and a grade 3/6

systolic murmur that was best auscultated in the second right intercostal space. Respiratory system examination revealed tachypnoea and diffuse bilateral rales.

INVESTIGATIONS

Chest radiograph showed cardiomegaly and features of pulmonary oedema (figure 1). An ECG revealed a third-degree AV block requiring an emergent temporary transvenous pacemaker (figure 2). Further investigations revealed anaemia (haemoglobin 8.7 mg/dL, leucocytosis (WCCs 22 400/mm³ with 86% neutrophils), worsening renal function, troponin-I of 0.17 (normal <0.03) and a brain natriuretic peptide of 1260 pg/mL. The patient was started on intravenous empiric broad spectrum antibiotics, using a combination of vancomycin and piperacillin-tazobactam.

A transthoracic echocardiogram was performed, which revealed an echodense mass suggestive of a vegetation attached to the coronary cusp. There was prolapse of the non-coronary cusp, with associated mild aortic regurgitation and moderate aortic stenosis. The aortic valve peak gradient was 73 mm Hg, and the mean gradient was 37 mm Hg. Left and right ventricular size and systolic functions were normal. A transoesophageal echocardiogram (Philips iE33 xMATRIX, Philips Electronics, Massachusetts, USA) revealed moderate aortic stenosis, with a freely mobile 2.8×1.6 cm vegetation attached to the non-coronary cusp; the vegetation was prolapsing, resulting in mild regurgitation. The aortic root was thickened up to 0.5 cm, suggestive of an aortic root abscess (figure 3).

Figure 1 Chest radiograph showing cardiomegaly, left ventricle contour and increased bronchovascular markings, peribronchial cuffing and prominent upper lobe veins suggesting pulmonary oedema.

To cite: Patnaik S, Davila CD, Chennupati A, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-206288

Figure 2 Initial ECG showing complete heart block; complete right bundle branch block with junctional escape and ventricular rate of 60 bpm.

TREATMENT

The patient was taken to the operating room urgently for valve debridement, abscess drainage, aortic valve replacement and pericardial patch reconstructive annuloplasty (figure 4A, B).

Multiple blood cultures from anaerobic bottles grew Gram-positive rods. Valve tissue also grew Gram-positive rods and coccobacilli in anaerobic medium. Vancomycin and piperacillin-tazobactam were discontinued at this time and therapy was narrowed down to ampicillin-sulbactam. This was a directed antimicrobial therapy based on the above culture results. Most oral Gram-positive anaerobes are covered by ampicillin. However, anaerobes from intra-abdominal sources (particularly *Bacillus fragilis*) can produce β -lactamase and are β -lactam resistant unless an inhibitor is administered. An alternative agent such as metronidazole or carbapenem may be used. Although the preliminary results were highly suspicious for *Lactobacillus* infection, sulbactam provided additional anaerobic coverage, and the ampicillin-sulbactam combination was continued until further culture and sensitivity results were available. Clinical improvement was noted.

The organisms from valve tissue cultures were later identified as *L. jensenii* by 16S ribosomal RNA gene sequencing. Final culture and susceptibility results revealed sensitivity to ampicillin, penicillin, erythromycin, clindamycin and vancomycin.

Figures 3 Transoesophageal echocardiogram in a 120° view showing large vegetation on the native aortic valve and a lucent area around the root suggestive of a para-aortic abscess.

Antibiotic therapy with penicillin was administered for a total of 6 weeks from the time of negative blood cultures.

OUTCOME AND FOLLOW-UP

The patient's condition promptly improved on antibiotics and a permanent pacemaker was implanted after the blood cultures were sterile. Early surgery and appropriate antibiotics, followed by permanent pacemaker implantation, led to a satisfactory clinical outcome.

DISCUSSION

Gram-positive bacteria that can cause IE include *Listeria monocytogenes*, *Nocardia* spp, *Erysipelothrix rhusiopathiae*, *Clostridium* spp, non-toxicogenic corynebacteria and *Lactobacillus* spp.⁴ Lactobacilli are ubiquitous, pleomorphic, rod-shaped or coccobacilli, lactose-fermenting, catalase-negative, non-spore forming, microaerophilic or facultatively anaerobic bacteria. They are an important component of mucosal microbiota. Immunocompromised patients are particularly vulnerable to infection with lactobacilli, especially in the setting of poor oral hygiene, recurrent dental infections, recent procedures or continuous ambulatory peritoneal dialysis. Bacteraemia, IE, liver or splenic abscess, urinary tract infections, endometritis and meningitis have been known to occur after iatrogenic mucosal trauma, such as that can occur during oesophagogastroduodenoscopy, colonoscopy, appendectomy, and dilation and curettage of the uterus. Although probiotic use has been identified as a risk factor for *Lactobacillus* infections in some studies, a causal association remains controversial.⁵

Lactobacillus is estimated to represent 0.05–0.4% of all IE cases,⁶ especially if there is known structural heart disease,⁷ and following invasive procedures on the heart such as implantation of prosthetic valves or heart transplantation. The most common sources are systemic emboli (42%) and dental procedures (75%).⁸ Some of the most frequently implicated species are thought to possess a greater ability to bind collagen and fibrinogen, aggregate platelets, and produce enzymes such as glycosidases and proteases, resulting in colonisation of the vascular endothelial surfaces.

To the best of our knowledge, there have been only seven reported cases of serious infection caused by *L. jensenii*.^{2 8–12}

Figure 4 (A) Intraoperative photograph showing large vegetation; (B) newly replaced valve after aortic annuloplasty.

Six of these cases involved IE. These cases showed a female preponderance, which may be explained by the fact that *L. jensenii* is a common member of the vaginal flora.

Lactobacilli may be difficult to culture, and the use of specific culture media has been suggested. Conventional methods identify only 30–50% of the isolates.¹³ Molecular analysis by 16S ribosomal RNA sequencing is a reliable method for the identification of lactobacilli, as has been proven in several studies.¹⁴

The treatment of severe lactobacilli infection can be challenging. Even with appropriate antimicrobial treatment, *Lactobacillus* endocarditis may lead to recurrent infection or death. Mortality due to *Lactobacillus* endocarditis can be as high as 25%.¹⁵ High-dose penicillin, sometimes combined with aminoglycoside, is recommended for endovascular infections. Vancomycin resistance is not uncommon and clindamycin is recommended for patients allergic to penicillin.² Cephalosporins are not recommended given their wide variation in susceptibility.¹¹

Similar to the case reported by Fradiani *et al*,⁹ our patient required valve surgery, suggesting that *L. jensenii* can cause severe IE, requiring surgical intervention.

To conclude, lactobacilli can sometimes behave like true pathogens and cause serious, life-threatening infections. With rising numbers of immunocompromised survivors in the present era, there may be an increasing number of *Lactobacillus* opportunistic infections. This awareness will help physicians, cardiologists, as well as microbiologists, in deciding an appropriate management strategy.

Learning points

- ▶ Although usually commensals of the mucosa, lactobacilli can sometimes behave like true pathogens and cause serious life-threatening infection and should be included in the differential diagnoses in an appropriate clinical setting.
- ▶ Molecular analysis by 16S ribosomal RNA sequencing is a reliable method for the identification of lactobacilli.
- ▶ Vancomycin resistance is common.
- ▶ High-dose penicillin, combined with an aminoglycoside, is recommended for endovascular infections. Clindamycin is recommended for patients allergic to penicillin.
- ▶ Cephalosporins are not recommended because of variable susceptibility.
- ▶ *Lactobacillus jensenii* can potentially cause severe infective endocarditis (IE), sometimes requiring valve surgery.

Twitter Follow Carlos Davila at @Carlos_DavilaMD

Acknowledgements The authors would like to acknowledge the contributions and inputs from the following physicians in the preparation of this report: Manju Balasubramanian, MD, Chief, Division of Clinical Pathology, Albert Einstein Medical Center, Philadelphia; Pablo Rengifo-Moreno, MD, Fellow, Division of Cardiology, Albert Einstein Medical Center, Philadelphia; and Sarah Perloff, DO, Division of Infectious Diseases, Albert Einstein Medical Center, Philadelphia.

Contributors SP was responsible for drafting the work, substantial contributions to the conception/design of the work, the acquisition, analysis, interpretation of data for the work and revising it critically for important intellectual content. CDD and AC were responsible for substantial contributions to the conception/design of the work, the acquisition, analysis, interpretation of data for the work and revising it critically for important intellectual content. AR was responsible for revising the work critically for important intellectual content and final approval of the version to be published.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Slipczuk L, Codolosa JN, Davila CD, *et al*. Infective endocarditis epidemiology over five decades: a systematic review. *PLoS ONE* 2013;8:e82665.
- 2 Cannon JP, Lee TA, Bolanos JT, *et al*. Pathogenic relevance of *Lactobacillus*: a retrospective review of over 200 cases. *Eur J Clin Microbiol Infect Dis* 2005;24:31–40.
- 3 Asahara T, Takahashi M, Nomoto K, *et al*. Assessment of safety of lactobacillus strains based on resistance to host innate defense mechanisms. *Clin Diagn Lab Immunol* 2003;10:169–73.
- 4 Brouqui P, Raoult D. Endocarditis due to rare and fastidious bacteria. *Clin Microbiol Rev* 2001;14:177–207.
- 5 Joseph RH, Czachor JS, Brown GR. *Lactobacillus* endocarditis: case report and literature review. *Infect Dis Clin Pract* 2006;14:134–6.
- 6 Borriello SP, Hammes WP, Holzapfel W, *et al*. Safety of probiotics that contain lactobacilli or bifidobacteria. *Clin Infect Dis* 2003;36:775–80.
- 7 Sussman JJ, Baron EJ, Goldberg SM, *et al*. Clinical manifestations and therapy of *Lactobacillus* endocarditis: report of a case and review of the literature. *Rev Infect Dis* 1986;8:771–6.
- 8 Atkins MC, Nicolson L, Harrison GA, *et al*. *Lactobacillus jensenii* prosthetic valve endocarditis. *J Infect* 1990;21:322–4.
- 9 Fradiani PA, Petrucca A, Ascenzioni F, *et al*. Endocarditis caused by *Lactobacillus jensenii* in an immunocompetent patient. *J Med Microbiol* 2010;59:607–9.

- 10 Salminen MK, Tynkkynen S, Rautelin H, *et al.* Lactobacillus bacteremia during a rapid increase in probiotic use of Lactobacillus rhamnosus GG in Finland. *Clin Infect Dis* 2002;35:1155–60.
- 11 Salvana EM, Frank M. Lactobacillus endocarditis: case report and review of cases reported since 1992. *J Infect* 2006;53:e5–e10.
- 12 Suarez-Garcia I, Sanchez-Garcia A, Soler L, *et al.* Lactobacillus jensenii bacteremia and endocarditis after dilatation and curettage: case report and literature review. *Infection* 2012;40:219–22.
- 13 Felten A, Barreau C, Bizet C, *et al.* Lactobacillus species identification, H₂O₂ production, and antibiotic resistance and correlation with human clinical status. *J Clin Microbiol* 1999;37:729–33.
- 14 Justesen US, Skov MN, Knudsen E, *et al.* 16S rRNA gene sequencing in routine identification of anaerobic bacteria isolated from blood cultures. *J Clin Microbiol* 2010;48:946–8.
- 15 Yagi S, Akaike M, Fujimura M, *et al.* Infective endocarditis caused by Lactobacillus. *Intern Med* 2008;47:1113–16.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow