Zoonotic Endocarditis: Is the feather mightier than the sword?

A. Esmail[1], A.F. Doubell[1], H. Piek[2], J.T. Janson[3] and A.J.K. Pecoraro[1]

[1]Division of Cardiology, Department of Medicine, Stellenbosch University and Tygerberg Hospital, Bellville, South Africa
[2]Department of Medicine, Paarl Hospital, Paarl, South Africa
[3]Division of Cardiothoracic Surgery, Department of Surgery, Stellenbosch University and Tygerberg Hospital, Bellville, South Africa

Address for correspondence:
Dr A. Esmail
Division of Cardiology
Department of Medicine
Stellenbosch University and Tygerberg Hospital
Francie Van Zijl Drive
Bellville
7505
South Africa

Email:
ahmed.arg@gmail.com

ABSTRACT

The common causes of infective endocarditis (IE) in South Africa are Staphylococcus aureus, the viridans streptococci and Bartonella species. Infection with Erysipelothrix rhusiopathiae is a rare cause of IE that is usually associated with occupational exposure and can be acquired from humans and animals. It was first reported in 1912 by Gunthar, and thereafter fewer than 60 cases have been reported. We describe a case of Erysipelothrix rhusiopathiae induced endocarditis in Cape Town, South Africa. SAHeart 2022;19:24-27

CASE REPORT

A 37-year-old male who worked on a chicken farm presented to the emergency unit with a 3 week history of fever, night sweats and weight loss. The patient denied any shortness of breath or other cardiac symptoms. He reported the development of a red papule on his lower leg 1 month ago after an accidental skin injury with a pen feather (quill feather) of a chicken. This papule healed with a scar (Figure 1). The patient had a history of alcohol abuse, but no history of intravenous drug abuse.

On examination he was normotensive without a tachycardia. The general examination revealed no peripheral stigmata of infective endocarditis. On cardiac examination his apex was displaced with a 3/6 pansystolic murmur heard over the mitral area. In addition, a parasternal heave suggestive of right ventricular hypertrophy secondary to pulmonary hypertension was palpated. Electrocardiogram demonstrated left ventricular hypertrophy with a dilated left atrium in keeping with chronic severe mitral regurgitation. Microscopic hematuria was present on urine dipstix analysis. Further special investigations revealed a raised C-reactive protein (CRP) of 38mg/L, with normal white cell count, complement levels (C3/C4) and kidney function. He had a mild normocytic anemia with a haemoglobin of 11.2g/dl. A clinical diagnosis of chronic severe mitral regurgitation was made, given his symptoms and the elevated CRP, infective endocarditis was considered. Echocardiography confirmed severe mitral regurgitation due to mitral valve prolapse with flail posterior leaflet and cord rupture (Figure 2). Independent mobile structures were noticed on the atrial surface of the anterior mitral valve leaflet suggestive of vegetations (Figure 2; Video 1).

The patient was initiated on empirical therapy for IE after 4 sets of blood cultures were obtained. All blood cultures were posi-

FIGURE 1: Scar on right lower leg.

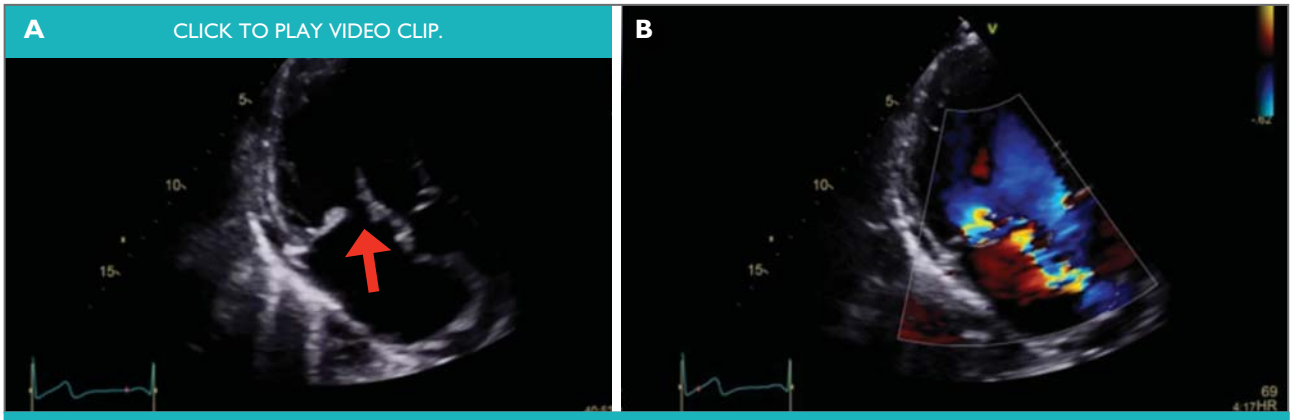

FIGURE 2: (A) Apical long axis view of the mitral valve demonstrating vegetations (arrow) on the atrial surface of anterior mitral valve leaflet. (B) Apical long axis view with colour demonstrating anterior jet of severe MR due to posterior valve prolapse with a flail P2 segment.

tive for Erysipelotrix rhusiopathiae. The patient was treated with penicillin for 27 days before undergoing a mitral valve repair. A quadrangular resection of the P2 segment of the posterior leaflet was performed and the vegetation was resected from the anterior leaflet, leaving a small defect in the anterior leaflet. This was closed with a pericardial patch and 5 - 0 Prolene suture. A 30mm CG Future ring (Medtronic®) 30mm was placed and the valve was competent on the postoperative echocardiography.

The patient had an uncomplicated post-operative course and was discharged well after completing intravenous antibiotic therapy (see Supplementary text 1).

DISCUSSION

Erysipelothrix rhusiopathiae is a non-spore forming, facultative anaerobic, gram positive bacteria that is ubiquitous in the environment, present in soil and the gastrointestinal tract of certain animals such as swine, birds and fish.[5,6] It is a well-known pathogen in veterinary medicine and known to cause serious systemic illness in humans. It is an occupational disease and infected individuals usually have direct contact with animals or organic matter that contain the organism.[6] Infection can be acquired through mosquito bites, ingestion of undercooked pork or skin wounds.[5] Vaccines are commonly used for pigs, but less commonly used in poultry. For many decades it was considered to have minimal impact on livestock, but more recently due to changes in environmental conditions and an increase in outdoor organic farming, erysipelas appears to be re-emerging.[7]

Human infection can be classified into 3 categories:[5,6]

- Focal cellulitis, which is often mild and self-limiting and referred to as erysipeloid.
- Diffuse cellulitis.
- Septicaemia with or without skin involvement, which may be complicated with endocarditis, meningitis or arthritis.

Erysipelothrix rhusiopathiae infection is usually seen in adults between the ages of 40 - 60 years with a male preponderance, likely reflecting occupational exposure.[6] Alcohol abuse is a recognised risk factor and may predispose to severe disease and complications.[8] It is difficult to differentiate Erysipelothrix rhusiopathiae induced infective endocarditis from other causes of bacterial endocarditis,[5] although high index of suspicion is warranted in patients with classical presentation involving a history of occupational exposure, alcohol abuse and characteristic skin rash (erysipeloid), which is present in approximately a third of cases. Some patients may present with severe lower back pain[10] and abdominal pain,[11] which may be related to thrombosis of the kidney, spleen or mesenteric artery. The aortic valve is involved in up to 70% of cases.[6] Our patient did not give a clear history of erysipeloid or any other skin rash.

The reported mortality rate (40%) is almost twice as high[5] as other causes of bacterial endocarditis (18% - 23%).[12] This is due to increased occurrence of complications such as septic shock, heart failure, kidney failure, meningitis, mycotic aneurysm, valvular perforation, and myocardial abscess.[5,9]

Laboratory findings are similar to other causes of IE, with elevated infective markers, anaemia of chronic disease and a decrease in complement levels (C3/C4) the usual findings. Our

patient had elevated infective markers (CRP 38mg/l), but normal complement levels and only mild normocytic normochromic anaemia (Hb 11.2g/dL).

Erysipelothrix rhusiopathiae is mostly associated with native valve endocarditis and has a predilection for the aortic valve. It can also affect the mitral (as in our case) and tricuspid valves.[8] Valve involvement is often associated with extensive destruction and peri annular extension with abscess formation. Detailed transthoracic and transoesophageal echocardiography is essential to recognise peri annular extension and to plan surgical intervention.

Histological findings are similar to other bacterial endocarditis and characterised by valve degeneration, calcification or necrosis, and infiltration of numerous inflammatory cells.[5]

Blood culture is the gold standard for identifying Erysipelothrix rhusiopathiae as the causative organism of IE. Erysipelothrix rhusiopathiae can be difficult to identify with microscopy and maybe mistaken for other organisms, thus necessitating a thorough microbiological work up (see Supplementary text 2).

Anti-microbial therapy is the mainstay of treatment for Erysipelothrix rhusiopathiae sepsis and should be adjusted based on sensitivity patterns. They are mostly sensitive to penicillin[6] and often resistant to vancomycin, aminoglycosides and macrolides. Since vancomycin is often used in the treatment of endocarditis in penicillin allergic patients, it is important to identify the species correctly. In patients with penicillin allergy acceptable alternatives include erythromycin, clindamycin, cephalosporin and fluoroquinolones.[5,6,9]

Indications and optimal timing of valve replacement/repair should be based on accepted criteria.[14] Antibiotic treatment should be continued after surgery and for a duration of 4 - 6 weeks.[5] Our patient presented with severe mitral regurgitation with vegetation length less than 15mm, thus inpatient mitral valve repair was performed. The patient completed 4 weeks of intravenous penicillin G and made a complete recovery.

SUMMARY

A 37-year-old male who worked on a chicken farm was admitted with severe mitral regurgitation as a result of infective endocarditis caused by Erysipelothrix rhusiopathiae. Erysipelothrix rhusiopathiae is a rare cause of infective endocarditis (IE). A high index of suspicion is warranted in patients with IE and known occupational risk factors for this zoonotic disease. Penicillin is considered the most appropriate antimicrobial with up to a third of patients requiring valve surgery. This case report emphasises the need to consider this diagnosis if risk factors for a zoonosis are present, coupled with clinical features suggestive of Erysipelothrix rhusiopathiae.

TEACHING POINTS

- Erysipelothrix rhusiopathiae is a:
  - Zoonotic infection associated with occupational exposure.
  - Rare cause of infective endocarditis.
- The peripheral stigmata and imaging findings are similar to other causes of IE.
- Thorough microbiological work up in patients with risk factors are important, to enable early diagnosis and treatment, as the bacterium may mimic a variety of other pathogens.
- Erysipelothrix rhusiopathiae induced endocarditis have a higher mortality rate compared to other causes of bacterial endocarditis.

SUPPLEMENTARY MATERIAL

Supplementary text 1

A verbal consent was provided by the patient to write this case report. He is presently lost to follow up and unreachable telephonically, therefore a written consent was not possible.

Supplementary text 2

Erysipelothrix rhusiopathiae can be difficult to identify with microscopy and maybe mistaken for other organisms such as Lactobacillus, Enterococcus and Streptococcus viridans.[5,10] Biochemical testing and gram stain may assist in differentiating it from other non-spore forming gram positive bacilli. Two distinct morphological forms grow on solid media - smooth and rough colonies.[6] Smooth colonies are pinpoint to 1.5mm in size, maybe transparent or bluish in colour and convex.[6] Rough colonies are larger with irregular border, flat and often more virulent.[6] Colonies are alpha haemolytic on blood agar after prolonged incubation.[5,6] They are positive for pyrrolidonyl aminopeptidase (PYR) activity similar to Enterococcus, and alpha haemolytic and catalase negative similar to Streptococcus viridans and can be differentiated by their ability to produce hydrogen sulfide (H2S) on triple sugar iron (TSI) agar.[14] This ability can also assist in differentiating it from Lactobacillus, a gram positive bacilli that is also catalase negative and can also be resistant to vancomycin and be associated with endocarditis. Negative catalase reaction can differentiate it from Corynebacterium spp. and Listeria spp.[14]

Conflict of interest: none declared.

REFERENCES

1. Pecoraro AJ, Doubell AF. Infective endocarditis in South Africa. Cardiovasc Diagn Ther. 2020;10:252-61. http://dx.doi.org/10.21037/cdt.2019.06.03.
2. Pecoraro AJK, Colette P, Herbst PG, et al. Causes of infective endocarditis in the Western Cape, South Africa: A prospective cohort study using a set protocol or organism detection and central decision making by an endocarditis team. BMJ Open 2021; 0:e053169. doi:10.1136/bmjopen-2021-053169.
3. Pecoraro AA, Herbst PP, Pienaar CC, et al. Bartonella species as a cause of culture-negative endocarditis in South Africa. Eur J Clin Microbiol Infect Dis 2021;40:1873-9.
4. Yamamoto Y, Shioshita K, Takazono T, Seki M, Izumikawa K. An autopsy case of Erysipelothrix rhusiopathiae endocarditis. Intern Med. 2008;47:1437-1440. https://doi.org/10.2169/internalmedicine.47.1150.
5. Hua P, Liu Jialiang, Tao J, Liu Jianyang, Yang Y, Yang S. Erysipelothrix rhusiopathiae-induced aortic valve endocarditis: Case report and literature review. Int J Clin Exp Med. 2015;8:730-736.
6. Gorby GL, Peacock JJ. Erysipelothrix rhusiopathiae endocarditis: Microbiologic, epidemiologic, and clinical features of an occupational disease. Rev Infect Dis. 1988;10:317-325. https://doi.org/10.1093/clinids/10.2.317.
7. Opriessnig T, Forde T, Shimoji Y. Erysipelothrix Spp.: Past, present, and future directions in vaccine research. Front Vet Sci. 2020;7:174. https://doi.org/10.3389/fvets.2020.00174.
8. Tomaszuk-Kazberuk A, Kaminska M, Sobkowicz B, Hirnle T, Prokop J. Infective endocarditis caused by Erysipelothrix rhusiopathiae involving 3 native valves. Kardiol Pol. 2011;69:827-829.
9. Nassar IM, De la Llana R, Garrido P, Martinez-Sanz R. Mitro-aortic infective endocarditis produced by Erysipelothrix rhusiopathiae: Case report and review of the literature. J Heart Valve Dis. 2005;14:320-324.
10. Kodera S, Nakamura A, Ooe K, Furukawa K, Shibata N, Arakawa Y. One case with Erysipelothrix rhusiopathiae endocarditis. Kansenshogaku Zasshi. 2006;80:413-417. https://doi.org/10.11150/kansenshogakuzasshi1970.80.413.
11. Heidrich JP, Stahl M, Dittmann R, Maass M, Solbach W. Mitral valve endocarditis caused by Erysipelothrix rhusiopathiae. Dtsch Med Wochenschr. 2001;126:431-433. https://doi.org/10.1055/s-2001-12726.
12. Wallace SM, Walton BI, Kharbanda RK, et al. Mortality from infective endocarditis: Clinical predictors of outcome. Heart. 2002;88:53-60. https://doi.org/10.1136/heart.88.1.53.
13. Jean S, Lainhart W, Yarbrough M. The Brief Case: Erysipelothrix bacteremia and endocarditis in a 59 year immunocompromised male on chronic high dose steroids. J Clin Microbiol. 2019;57:2031-18. https://doi.org/10.1128/JCM.02031-18.
14. Baumgartner H, Falk V, Bax JJ, De Bonis M, Hamm C. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. European Heart Journal. 2017;38:36:2739-2791. https://doi.org/10.1093/eurheartj/ehx391.