International Journal of Research in Medical Sciences
Rafiq K et al. Int J Res Med Sci. 2023 Sep;11(9):3414-3417
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20232488

Case Report

A rare case of bilateral septic arthritis of hip: in a patient of idiopathic angiodema

Kanwal Rafiq[1], Faisal Saeed Bhutta[2]*

[1]Primary Care Physician Greater Manchester, U. K.
[2]Primary Care Physician Haughton Thornly Medical centres Manchester, U. K.

Received: 13 July 2023
Revised: 02 August 2023
Accepted: 03 August 2023

***Correspondence:**
Dr. Faisal Saeed Bhutta,
E-mail: drfaisalbhutta@yahoo.com

ABSTRACT

Enterococcal infection can lead to gastroenteritis and bacteraemia. However, osteoarticular infections caused by Enterococci are rarely encountered. We report the case of a 27-year-old female patient with bilateral hip infection caused by enterococci. This young female patient initially presented with flare up of angioedema symptoms followed by septic arthritis of one hip and then 2nd hip while patient was admitted in hospital. Thankfully this patient was successfully treated and discharge back home. Early detection and proper treatment are essential for the eradication of the infection. The use of prolonged antimicrobial therapy can be considered in the management of bilateral hip joint destruction due to delayed diagnosis of enterococci infection. The patient was informed that data from the case would be submitted for publication, and she provided his consent.

Keywords: Bilateral hip infection, Enterococci, Antibiotics, Hip washout

INTRODUCTION

This is interesting but rare case of a young female patient with existing history of idiopathic angioedema. She was admitted with acute flare up. Her hospital stay was complicated by enterococcal septic arthritis of one hip followed by other hip.[1] This case report signifies importance of thorough history examination and prompt treatment which saved patients hip and life.[2] Bilateral septic arthritis of hip is very rare condition, particularly enterococcal septic arthritis of hip antibiotic resistance is emerging threat and clinicians need to be mindful of antibiotic use.[3]

CASE REPORT

A 27 old female patient presented with h/o of upper abdominal pain of moderate to severe intensity and colicky in nature, vomiting 5-6 episodes diarrhoea loose watery 5-6 times and red blotchy erythematous rash with flushing and sweating.

She used to have idiopathic urticaria and angioedema and had appendicectomy, cholecystectomy in the past. She was on steroids, montelukast, ranitidine, fosamax, calcichew for existing conditions. She was allergic to penicillin, NSAIDS, Morphine and its derivatives, on first examination pulse 155/min BP155 /105 temp 35.5º. Flushing of face, sweating erythematous rash, tenderness in upper abdomen.

She was diagnosed with acute episode of generalized idiopathic angioedema which was treated with high doses of IV steroids and antihistamines.

Patient started getting better urticaria, vomiting, diarrhoea stopped. Patient was happy to go home in a day or so. After one week patient became very unwell with rigors,

chills and cramps. High temperature of 40 degree centigrade. Tachycardic and hypotensive

Table 1: Laboratory findings related to the patient.

Index	Episode		
	First episode	Second episode	Normal range (unit)
Hemoglobin	15.1	11.6	12-16 g/dL
WBC[a]	0.9	13.1	4-10 (10^3)
RBC[b]	2.19	3.46	4.2-5.5 (10^3)
Platelet	135	467	150-450 (10^3)
ESR[c]	58	95	<20 mm/h
Serum creatinine	0.6	0.6	0.5-1 mg/dL
CRP[d]	85	180	<20 mg/L

So, blood cultures and tip of femoral line sent and patient started on iv antibiotics. Blood culture and femoral line tip came out to be positive for MRSA after two weeks Patient diagnosed with MRSA septicaemia and treated with antibiotics (gentamicin, vancomycin, rifampicin, teicoplanin)

At this stage echocardiogram, transoesophageal echo and CT scan of abdomen was done which was normal.

Patient started getting better WCC, CRP started coming down. While she was getting better, she started having pain in left hip.

Examination of the hip joint revealed tenderness and marked limitation of range of movements with SLR of 10 degrees in left hip.

She was diagnosed as septic arthritis of hip for which she had a Arthrotomy and washout of left hip joint on same day which revealed frank pus coming out from hip joint.

Pt. Started feeling better, pain in his settled inflammatory markers came down.

After three weeks patient started having same pain on right side hip joint with limited range of movements and on clinical suspicion of septic arthritis arthrotomy and washout of right hip done which revealed turbid fluid Pt. Started feeling better hip pain settled.

During next coming three weeks she kept on having multiple spikes of temperature and pain in right hip for which she had five washouts of hip

Nothing grown on hip aspirates, washouts and blood cultures as she was already on antibiotics.

The last washout fluid and blood culture grown *enterococcus* She was started on antibiotics according to sensitivities. Vancomycin, gentamicin, rifampicin, linezolid, fluconazole. Patient did very well and discharged home.

DISCUSSION

Bilateral arthritis of hip (Enterococcal)

It is a rare condition usually involving one joint.[5] Incidence between 2-10 cases per 100000 of population. More prevalent in immunocompromised where it is 30-70 cases per 100000. Most common organisms are staphylococcus and streptococcus.[6] The 25-50% of patients develop irreversible joint damage despite advances in diagnostic studies, strong antibiotics and early drainage.[7]

Aetiology and pathology

The 80% of cases are caused by gram positive (60% *S. aureus*; 15% beta-haemolytic *Streptococci*; 5% *Streptococcus pneumoniae*).[8] 20% of cases are caused by gram negative organisms. Bacteria may enter joint by direct trauma, nearby osteomyelitis or soft tissue infections and hematogenous.[9] Clinically patient presents with fever with hot, painful joint with restricted range of movements both active and passive

Investigations

Bloods demonstrated leucocytosis and raised CRP and ESR which were also helpful for monitoring treatment. Blood cultures positive for *enterococcus.* Imaging studies AP and lateral views plain radiographs demonstrated soft tissue swelling around joint, displacement of tissue planes, bony erosion joint space narrowing.

Figure 1: X-ray Hip.

Ultrasonography demonstrated joint effusion. Other modalities of investigations can be used to define the extent of septic arthritis and help guide treatment such as nuclear scanning, needle aspiration, synovial fluid analysis.[10]

Treatment

Patient was treated with broad spectrum IV antibiotics after aspiration/ drainage, once culture results are back than according to sensitivities. Surgical drainage is cornerstone to success otherwise rapid destruction of joint follows quickly. If improvement is not achieved than open drainage arthroscopically or via arthrotomy is recommended. Postoperative early weight bearing with frequent passive range exercises improves outcome.

Outcome and prognosis

Despite quick and proper treatment prognosis is poor in one study 21% resulted in death and severe functional deterioration and 33% resulted in poor joint function

Enterococcal pathology causing septic arthritis

Enterococcal pathogenicity was initially addressed at the end of the 19th century by MacCallum and Hastings, who isolated an organism from a case of acute endocarditis and designated it *Micrococcus zymogenes* based on its fermentative properties.[11] The organism was shown to be resistant to desiccation, heating to 60°C, and several antiseptics, including carbolic acid and chloroform.[12] It was also found to be lethal when injected intraperitoneally in white mice, and capable of producing endocarditis in a canine model. A century later, enterococci are prominent among nosocomial pathogens, ranking second only to *E. coli* in total nosocomial infections, accounting for more than 12% of all cases.[13]

Infections caused by the genus *Enterococcus* (most notably *Enterococcus faecalis*, which accounts for ~80% of all infections) include urinary tract infections, bacteraemia, intra-abdominal infections, and endocarditis. The problem of nosocomial enterococcal infection is compounded by multiple antibiotic resistance. A comparison of outcomes for patients with bacteraemia due to vancomycin-resistant *Enterococcus faecium* or vancomycin-susceptible *E. faecium* found a median length of stay of 46 days after the first episode of bacteraemia in the group of patients with vancomycin-resistant *E. faecium*, as compared to 19 days for patients infected by a susceptible strain.[19] The presence of VRE (vancomycin-resistant enterococci) in the bloodstream has also been associated with increased mortality. Patients with enterococcal bacteraemia were observed to be twice as likely to die (37% vs. 16%) when the infecting isolate was resistant to vancomycin.[15]

However, more recent studies indicate that VRE-status is not a major predictor for clinical outcome. Whether more recent findings describe cases that benefit from further evolution in the treatment of VRE requires further analysis.[16] As vancomycin frequently represents the last available therapeutic for multiple antibiotic resistant enterococci, the rapid increase in vancomycin resistance indicates that enterococcal infection will pose an increasing therapeutic challenge.

Antibiotic resistance

The intrinsic ruggedness of enterococci also confers an unusual level of tolerance to several classes of antibiotics including aminoglycosides, beta-lactams and quinolones. For example, the resistance of enterococci to aminoglycosides results from the ability of enterococci to block the uptake of the drug at the cell wall Consequently, aminoglycosides are only effective against enterococci when used in combination with cell wall active antibiotics.[17] This combination treatment modality has been compromised, however, by the rapid spread of high-level aminoglycoside resistance among enterococci (>2000 µg/ml). Although the mechanism of high-level resistance was determined to be the result of a bifunctional enzyme, the molecular basis for the intrinsic resistance of enterococci to low levels of aminoglycosides remains to be determined.

CONCLUSION

This case report illustrates the unusual presentation of enterococcal septic arthritis in a patient with underlying disease. Antibiotic treatment and surgical intervention should be initiated by the time the clinical diagnosis is made. Correct diagnosis and successful treatment in this case in addition to hip arthroplasty and prolonged antimicrobial therapy can be considered in the management of bilateral hip joint destruction and have saved the patient life.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Raymond NJ, Henry J, Workowski KA. Enterococcal arthritis: case report and review**.** Clin Infect Dis. 1995;21(3):516-22.
2. Arduino RC, Jacques-Palaz K, Murray BE, Rakita RM. Resistance of *Enterococcus faecium* to neutrophil-mediated phagocytosis. Infect Immun. 1994;62:5587-94.
3. Smith JW, Chalupa P, Shabaz Hasan M. Infectious arthritis: clinical features, laboratory findings and treatment. Clin Microbiol Infect. 2006;12:309-14.
4. Arduino RC, Murray BE, Rakita RM. Roles of antibiotics and complement in phagocytic killing of *Enterococci*. Infect Immun. 1978;62:987-93.
5. Rutherford AI, Subesinghe S, Bharucha T, Ibrahim F, Kleymann A, Galloway JB. A population study of the reported incidence of native joint septic arthritis in the United Kingdom between 1998 and 2013. Rheumatology. 2016;55:2176-80.

6. Osiri M, Akkasilpa S, Reinprayoon S, Deesomchok U. Streptococcal arthritis in Thai adults: case series and review. J Med Assoc Thai. 1996;79:755-61.
7. Stutz G, Kuster MS, Kleinstuck F, Gachter A. Arthroscopic management of septic arthritis: stages of infection and results. Knee Surg Sports Traumatol Arthrosc. 2000;8:270-4.
8. Shanthi M, Sekar U, Sridharan K. Septic arthritis of Hip caused by *Salmonella typhi*: a case report. Case Rep. Infect Dis. 2012;2012:464527.
9. Ross JJ. Septic arthritis. Infect Dis Clin N Am. 2005;19:799-817.
10. Fowler ML, Zhu C, Byrne K, Lieber SB, Moore A, Shmerling RH et al. Pathogen or contaminant? Distinguishing true infection from synovial fluid culture contamination in patients with suspected septic arthritis. Infection. 2017;45:825-30.
11. Booth MC, Bogie CP, Sahl HG, Siezen RJ, Hatter KJ, Gilmore MS. Structural analysis and proteolytic activation of *Enterococcus faecalis* cytolysin, a novel lantibiotic. Mol Microbiol. 1996;21:1175-84.
12. Bhakdi ST, Klonisch PN, Fischer W. Stimulation of monokine production by lipoteichoic acids. Infect Immun. 1991;59:4693-7.
13. Bottone EJ, Patel L, Patel P, Robin T. Mucoid encapsulated *Enterococus faecalis*: an emerging morphotype isolated from patients with urinary tract infections. Diagn Microbiol Infect Dis. 1998;31:429-30.
14. Caballero-Granado FJ, Cisneros JM, Luque R, Torres-Tortosa M, Gamboa F, Diez F et al. Comparative study of bacteremias caused by *Enterococcus* spp. with and without high-level resistance to gentamicin. J Clin Microbiol. 1998;36:520-25.
15. Chow JW, Thal LA, Perri MB, Vazquez JA, Donabedian SM, Clewell DB, Zervos MJ. Plasmid-associated hemolysin and aggregation substance production contribute to virulence in experimental enterococcal endocarditis. Antimicrob. Agents Chemother. 1998;37:2474-7.
16. Chenoweth C, Schaberg D. 1990. The epidemiology of *Enterococci*. Eur J Clin Microbiol Infect Dis. 1990;9:80-89.
17. Coburn PS, Sahl HG, Siezen RJ, Gilmore MS. The *Enterococcus faecalis* cytolysin: a novel toxin with roots in the lantibiotic family. In Abstracts of the Eighth European Workshop on Bacterial Protein Toxins, Gustav Fischer, Jena, Germany. 1997;409-10.

Cite this article as: Rafiq K, Bhutta FS. A rare case of bilateral septic arthritis of hip: in a patient of idiopathic angioedema. Int J Res Med Sci 2023;11:3414-7.