Dear Sir,

Enterococci are normal inhabitants of the human gastrointestinal tract, increasingly becoming important human pathogen posing therapeutic challenges for long. The unique natural resistance of enterococci to several types of antibiotics and their inherent capacity to survive in different environments allows them to survive, proliferate, and fill the void when antibiotic-susceptible organisms are eliminated, contributing to the frequency of enterococci in nosocomial infections and unusual clinical consequence.¹

A three and a half year old girl child from low socioeconomic status weighed 12 kg (<-2 zscore), height 92 cm (<-2 score) moderately malnourished as per WHO criteria presented with complaints of fever moderate to high grade, cough for 8 days, and breathing difficulty 1 day. She was receiving some oral antibiotic 1 day prior to admission; next pleurocentesis was done for respiratory distress, 200 mL pus was removed and referred immediately. She had no significant past, family history, child was sick looking, febrile, had respiratory distress, features of parapneumonic effusion. Cardiac and abdominal examination was unexceptional except mild nontender hepatomegaly. She was put on supportive treatment, broad spectrum antibiotics after sending cultures and other investigations. Immediate ICD was placed and 400 mL pus was drained out. Laboratory analysis of pus revealed TLC 220,000/mm³ with 98% neutrophils, sugar 20 mg/dL, protein 5 gm/dL, ADA 452 IU/L and AFB +ve, so ATT was started. Hematological analysis showed HB 10 gm%, TLC 20200/mm³ with 86% polymorphs. Blood culture was sterile at 72 hours; LFT, RFT normal. Her serology was negative for HIV. Her USG abdomen was not significant other than mild hepatomegaly. She continued to have fever spikes although her toxicity was decreased. On day 4, pus culture grew vancomycin-resistant Enteroccus faecalis (VRE) and antibiotics were changed to linezolid and ciprofloxacin as per sensitivity report. She became afebrile by day 6, her ICD was removed on day 10 and was discharged after completion of 2 weeks of antibiotics on ATT, hematinics. X-ray chest showed lung expansion; CT chest not done due to financial constraint but USG abdomen and chest did not show any other abnormality. Family screen for TB was negative.

Discussion

Enterococci is an important cause of nosocomial infection, infective endocarditis, wound infections, and urinary tract infections but very rarely causes spontaneous pleural empyema. The infection sometimes occurs in the presence of peritonitis, possibly because of a dysfunctional reticuloendothelial system, but cases of E. faecalis empyema in the absence of peritonitis have been reported as well.² They are uncommon but emerging agents of upper and lower airway diseases, in particular, pneumonia and thoracic empyema and may jeopardize the clinical outcome of compromised, hospitalized hosts, as well as affect outpatients.³,⁴ In Indian scenario pertaining to children etiological profile of empyema mainly remains staphylococci and streptococci, Gram-negative and anaerobes are next in order and enterococcus has been reported

¹Asst. Professor, ²Professor and Head, ³Professor, Dept. of Pediatrics, Shri Guru Ram Rai Institute of Medical and Health Sciences, Patel Nagar, Dehradun-248001, Uttarakhand, India.

Correspondence: Dr. Sudhir Rana, Dept. of Pediatrics, Shri Guru Ram Rai Institute of Medical and Health Sciences, Patel Nagar, Dehradun-248001, Uttarakhand, India.

E-mail Id: drsudhirrana@gmail.com

Orcid Id: http://orcid.org/0000-0001-7018-7067

How to cite this article: Agrawal A, Rana S, Aggarwal B. A case of Enterococcus Fecalis Spontaneous Empyema with Coexistent Tuberculosis. J Commun Dis 2017; 49(4): 1-2.

Digital Object Identifier (DOI): https://doi.org/10.24321/0019.5138.201726

ISSN: 0019-5138
rarely till recently.\textsuperscript{5,6} Our patient presented with frank spontaneous empyema with coexistent tuberculosis. She underwent pleurocentesis in a peripheral setting contacted \textit{E. faecalis} infection either due to poor immunological status or from nosocomial settings. Patient was referred here immediately post tapping and there was no significant past history hence chances of nosocomial infection were minimal. The continuing progress of modern medical care toward more intensive and invasive medical therapies has undoubtedly contributed to the increased prevalence of these remarkable opportunistic pathogens. This trend has been attributed to the increasing antibiotic resistance among clinical isolates of \textit{enterococci}. The rapid spread of \textit{enterococci} with resistance to vancomycin (VRE) has been of particular concern and many strains that are resistant to vancomycin also show resistance to penicillin, as well as high-level resistance (HLR) to aminoglycosides. Also, over the past two decades, \textit{E. faecium} has emerged as a leading cause of multidrug-resistant enterococcal infection.\textsuperscript{7} Finally, as has historically been the case with enterococci, resistance is emerging to newer agents used to treat VRE infections, such as linezolid, quinupristin/dalfopristin, and daptomycin.\textsuperscript{8} The solution lies in prevention of emergence and dissemination of newer antimicrobial resistance with rational use of antibiotics and infection control policies.

**Key Message**

Enterococcus poses a threat as an emerging community acquired, rational use of antibiotics is warranted.

**References**