Case Report

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Subacute Infective Endocarditis of Aortic Valve with Ascending Aorta Aneurysm Caused by Enterococcus Durans : A Case Report and Review of the Literature

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Abstract

Enterococcus durans endocarditis is very rare. A few reported cases of infective endocarditis caused by *Enterococcus durans* exist. Endocarditis with enterococcus durans is a potentially devastating infection and most often requires prolonged intravenous antibiotics with associated risks. We reported the case of aortic valve infective endocarditis caused by *Enterococcus durans.*

Keywords: Bacterial Endocarditis; Fusiform Aneurysm; *Enterococcus Durans.*

Introduction

Enterococcus durans is gram positive, ovoid, elongated cells grouped in pairs or short chains. The genus previously known as *Streptococcus durans*. Colonies on Nutrient Agar or Blood Agar 5% are circular, smooth and entire. By chemical reaction its catalase negative, PYR (Pyrrolidonyl-beta-naphthylamide) positive and growth in 40% bile[1]. Phenotypically very close to enterococcus faecium. Enterococcus are opportunistic pathogens in the urinary tract and blood stream.We are presenting the case of aortic valve infective endocarditis caused by *Enterococcus durans.*

Case Presentation

A 42 yrs old male patient presented with complain of high grade fever, loss of appetite, weakness, weight loss 4 kg since 25 days. Patient presented to us as a fever of unknown origin.The patient had no recent dental, gastrointestinal, genito- urinary procedure or foley's catheters prior to presentation. Patient taken IV ceftriaxone for 2 days for management of Fever. At the presentation time temperature 100.2 F, pulse 100/min. blood pressure 134/72mmHg, Respiratory rate 14/min and physical examination Levine III/IV systolic ejection murmur was heard in the aortic valve area. He had no Janeway lesions, Osler nodes or Roth spots. The Laboratory findings were as follows: Hemoglobin 12.1, white cells 10500/cmm (neutrophil 76%), platelets: 190000/cmm, RBS:100, ESR: 33/mm, SGPT: 45, BIL: T-1.2, D-0.8, I-0.4, Creatinine: 1.08mg/dl, Blood urea 30mg/ml, Na 138mEq/L, K 4.0mEq/L, Dengue NS1 by ELISA & profile (IGM & IGG) negative, S. WIDAL: negative, Urine analysis Pus cell 1-2/hpf, S.TSH: 1.65, LDH: 640, ANA by IF method: negative, R.A. factor: 57.1, Anti CCP level: <0.4, ASO titre: 100, X ray chest (P/A): normal, USG abdomen and Soft Tissue: normal study.

HRCT chest and abdomen shows mild fusiform aneurysmal dilatation of ascending aorta without

dissection or thrombosis (max. anterio-posterior diameter about 4.8 cm and transverse dimension about 4.4 cm), small calcification of aortic valve & non-enhancing wedge shaped hypodense areas involving spleen and left kidney possibly infarcts more likely (Figure 1,2). Transthoracic echocardiography was performed shows aortic valve thick, bicuspid with prolapse of anterior leaflet, small mobile structure is seen on anterior non-coronary leaflet on ventricular aspect p/o vegetation (Figure 3 and 4).

Blood culture Bactec aerobic plus with ½ hrs apart 3 sets were positive for gram positive cocci in angulated pairs in all six bottles in 26 hrs. Preliminary identification of the isolates over the next few days was based on gram stain, milky white colonies, negative catalase & pyrrolidonyl arylamidase reactive [2]. Definitive identification of isolate organism as Enterococcus durans by Vitek [2]. (Figure 5,6).

Antimicrobial susceptibility showed: Minimum inhibitory concentration : Benzylpenicillin >=64 was resistant, gentamicin with high level sensitive and synergy, linezolid 2, vancomycin <=0.5, teicoplanin <= 0.5, tigecycline <=0.12, according to the Clinical and Laboratory Standards Institute(CLSI) Guidelines.

These lead to diagnose of subacute infective endocarditis of aortic valve with ascending aorta aneurysm caused by Enterococcus durans. Treatment started on admission initially vancomycin 1gm/ 12hrly (30mg/kg/day) and gentamicin 80 mg/ 8hrly (3mg/kg/day) with explained drug nephrotoxicity to relative. After 3 days fever was subside and vancomycin trough level was 8.12 microgm/ml(1/2 hour prior to 4th dose). Nephrotoxicity develop after 14 days of therapy. Daptomycin(8mg/kg/day) used for rest of the therapy 4 weeks without side effect.

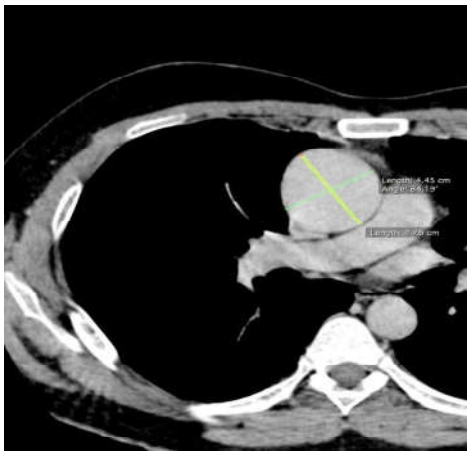

Fig. 1: Ascending aorta Aneurysm

Follow up after 6 weeks – 3sets of blood culture: negative, 2D Echo – freely mobile but fibrosed vegetation seen on AV(size:19-6mm). CT Abdomen – contraction of infarct - Sequelae of old infarct in spleen and kidney. Patient advise to underwent Bentall surgery(AVR + AO-Root replacement). Aortiv valve replacement done. Pre operative CAG & blood culture for bactec aerobic plus (3set) normal. CPK Total: 299.Intraoperative findings: aortic dilated and thinned out(reconstruct the demaged ascending aorta), NCC vegetation +, NCC tear +, below which

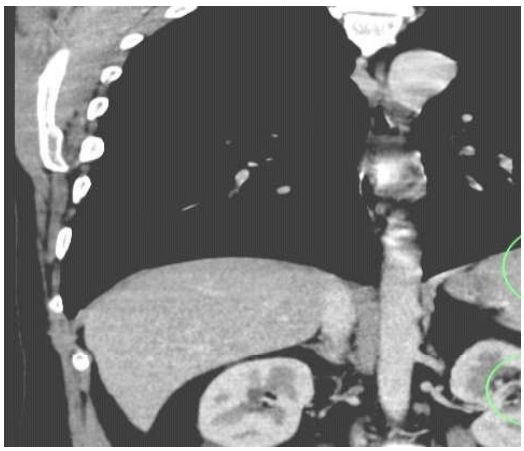

Fig. 2: Infarct in spleen and Kidney

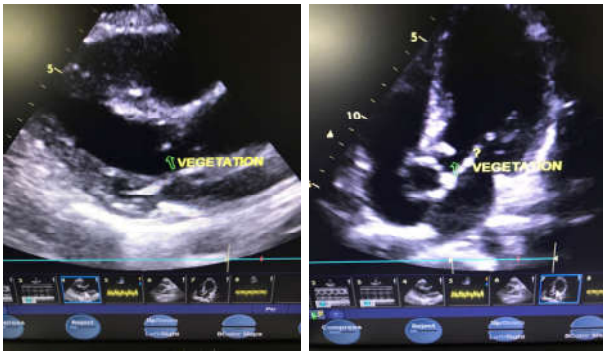

Fig. 3 and 4: Aortic valve Vegetation

Fig. 5: Colonies of Enterococcus durans on Nutrient agar

Fig. 6: Colonies of Enterococcus durans on Blood agar

there was an healed abscess cavity. Investigation of post surgery fungal culture and anaerobic culture was no organism. Tissue for HPE fibrosed valve leaflet with myxoid changes. 2D echo after 1 week of surgery - concentric LVH, mechanical AV in situ, with no complication. Post surgery patient treated with Daptomycin for 4 weeks.

Discussion

The incidence of infective endocarditis is approximately 1.7- 6.2 cases per 100000 patients years. Enterococci are the third most common organism (approximately 8% of cases) causing endocarditis, after streptococci and staphylococci [3], and are emerging as an important cause of infection, especially in elderly. Among all enterococcal infections, 63- 81% are *E. faecalis*, 13- 23% are *E. faecium* & 1% are other remainings (*E. avium, E. durans, E. casseliflavus, E. gallinarum, E. hirae, E. raffinosus*).

E.durans is a very rare species of non- faecalis, non- faecium enterococci. E. durans has been isolated from clinical specimens (usually from intestine of animals and less frequently in human) but rarely causes human infection as it is known to have low virulence.It has been estimated that E. durans is responsible for <1% of all enterococcal IE episodes (Olaison & Schadewitz,2002). E.durans was a rare cause of enterococcal bacteraemia (0.1%) in a large series [4]. Aneurysms of ascending aorta caused by infection are referred to as mycotic aneurysms. These aneurysms are rare and most commonly seen after an episode of valvular endocarditis.

Optimal therapy for IE caused by enterococci requires a synergistic bactericidal combination of a cell- wall- active antimicrobial agent to which the organism is susceptible and an aminoglycosides. In this case vancomycin and gentamicin should be administered because isolate is resistant to penicillin. Enterococci are relatively impermeable to aminoglycosides. High concentrations of aminoglycosides in the extracellular environment are required to achieve sufficient concentrations of the drug at the site of the ribosomal target within the bacterial cell for bactericidal activity [5].

Combinations of penicillin or ampicillin with gentamicin are preferable to combined vancomycin-gentamicin because of the potential increased risk of ototoxicity and nephrotoxicity with the vancomycin-gentamicin combination. Morever, combinations of penicillin or ampicillin and gentamicin are more active than combinations of vancomycin and gentamicin in vitro and in animal models of experimental IE. It is reasonable that patients with NVE receive 6 weeks of vancomycin-gentamicin therapy and that patients with PVE receive at least 6 weeks of therapy.

Rarely, strains of E.faecalis produce an inducible Beta- lactamase. These Beta- lactamase producing strains are susceptible to ampicillin-sulbactum and to vancomycin. Intrinsic penicillin resistance is uncommon in E.faecalis but is common in E. faecium. It is reasonable to treat patients with E. faecalis IE caused by strains that are intrinsically resistance to penicillin with a combination of vancomycin plus gentamicin.

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