Veiled Bacterial Endocarditis: Case Study

Sinha A*, Shetkar UB**, S.C. Tewari***, M.B. Chandurkar*, Sinha NK**

Abstract

We report two case of infective endocarditis (IE) who were admitted and treated for other medical and surgical complications in the hospital. The first case was admitted for abdominal infection following seven month amenorrhea and the second case was admitted for bleeding esophageal varices. The risk factor in the first case was vaginal hysterectomy and mitral valve prolapse while in the second it was variceal sclerotherapy. Both patients developed infective endocarditis (IE) while being managed for their respective diseases. IE prophylaxis was not undertaken as the cardiac status at the time of admission was not available. Echocardiography played an important role in the diagnosis and management of both cases. We conclude that greater emphasis has to be placed in managing patients with cardiac condition undergoing any high risk upper gastrointestinal procedure / genitourinary procedure / abdominal surgeries.

Key Words: *Infective endocarditis, vaginal hysterectomy, variceal sclerotherapy, echocardiography.*

Introduction

Infective endocarditis (IE) caries a mortality rate of 20 to 40 % and perhaps as high as 70%[1]. Prevention of this disease is very important and identification of high risk group patients is a clinical priority. We report two cases who were admitted for other reasons to the hospital.

Case 1

A 20 year old female, primigravida, with history of seven month amenorrhea; was admitted to hospital with chief complains of abdominal pain of one day duration. Her initial work up revealed the following positive findings: Blood pressure (BP): 160 / 100 mm of Hg, sinus tachycardia, bilateral pedal edema and hemoglobin: 4.8 g/ dl. An emergency ultrasonography for fetal viability was carried out, which revealed an intrauterine dead fetus, oligohydramnios and retro placental clot. Other examination and history were unremarkable. She did not have past history of hypertension, other cardiac illness or diabetes. She was nonalcoholic, nonsmoker and non drug abuser. Her other systemic examination findings were within normal limits. A provisional diagnosis of primigravida with severe pregnancy induced hypertension (PIH) in latent phase of labour was made. An artificial rupture of membrane was carried out and blood filled liquor drained. Within hours of admission her mental status deteriorated and her diagnosis was modified to include concealed accidental hemorrhage with severe anemia with cerebral hypoxic encephalopathy with PIH. An immediate vaginal delivery was planned and within five hours of admission a preterm dead baby was delivered. Post delivery, uterus was floppy and non-contractile. Her retroplacental clot weighed about 600g. Even after transfusion of five units of fresh frozen plasma, her systolic BP did not rise above 90mm of Hg. She gradually kept deteriorating with onset of drowsiness (Glasgow coma scale 10 /15), restless and developed profuse vaginal bleeding. Considering her critical clinical condition, therapeutic hysterectomy was done. This was followed by increase in pulse rate (150 / min) and BP (134/94 mm of Hg). Due to the waxing and waning consciousness levels and aberrant arterial blood gas level she was put on assisted ventillatory support (SIMV mode).

On second post operative day (POD) she developed bilateral crepitations over chest which improved with diuretic drug administration. On fifth POD a chest X-ray showed right lower zone consolidation of the lung while the patient was still on ventilator. Her drain culture was positive for coagulase negative staphylococcus but blood cultures were repeatedly negative on 3 consecutive days.

* Lecturer, ** Professor, *** HOD
Dept. of Medicine Rural Medical College, Loni

Corresponding Author :
Dr. Sinha A.
PG Student, Dept. of Medicine
Rural Medical College, Loni, Tal. Rahata, Dist. Ahmednagar (MS)

She continued to be treated with ampicillin and metronidazole injections. She developed continuous high fever (103°F) and tachycardia. She was transfused with packed red cells following which her Hb level rose to 6.7 g/ dl. She extubated herself on POD: 8, during which she was still febrile (101°F) with BP of 134/100 mm Hg. She was then put on fluroquinolones. There was derangement of renal function test during this period. A provisional diagnosis of septicemia with acute renal failure based on her present situation was made. On POD 20, a two dimensional trans-thoracic echo-cardiogram (2DTTE) was carried out, which revealed severe mitral stenosis (valve area = 1.34cm sq), mitral valve prolapse (MVP) of both anterior and posterior leaflets, moderate mitral regurgitation and enlarged left atrial diameter (4.5cm). Considering her recently revealed cardiac status she was managed more aggressively with restriction of intravenous fluids. She continued to have high fever (105°F) despite being subject to all available antibiotics in the armamentarium. These included antimalarials and third generation cephalosporins, quinolones, antimalarials and azole groups. By POD: 23, she also developed hemi-paresis of left upper and lower limbs as well as congestive cardiac failure.

A repeat 2 DTTE, on POD: 35 was carried out to rule out left atrial thrombus, which incidentally revealed mobile vegetation in anterior mitral leaflet (7mm in diameter). Her left atrium was enlarged (6 cm in diameter) but no clots were seen in the appendages (Fig. 1).

A working diagnosis of infective endocarditis was made. Distinction between acute and sub-acute endocarditis was blurred as the organism responsible for acute endocarditis could not be isolated. A repeat blood culture was negative on three consecutive samples. She was started on infective endocarditis regime with heparin to which there was favorable response. Her fever subsided, general condition improved, power in limbs returned to near normal and by the 45th day of admission there were no signs of heart failure.

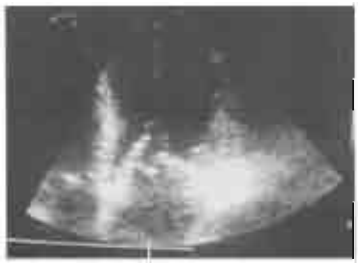

Fig. 1: (Case 1): Two dimensional trans-thoracic echocardiography image: Vegetation on anterior mitral leaflet

Case 2

A 55 year old male underwent variceal sclerotherapy on two occasions for grade IE esophageal varices. He was not given bacterial endocarditis prophylaxis before the procedures. He reported back to hospital on post procedure day 20, with history of continuous fever with, chills, and mild splenomegaly. Blood culture was positive for S aureus. Two dimensional TTE revealed vegetation on both anterior and posterior mitral leaflets (Fig. 2. 3). He was treated with vancomycin and gentamycin for 6 weeks after which a repeat echo showed that vegetation on the mitral valve had disappeared. Presently both patients are doing well.

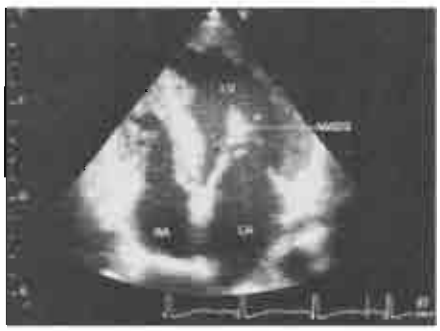

Fig 2: Case 2: Two dimensional trans-thoracic echocardiography image: Vegetation on anterior mitral leaflet

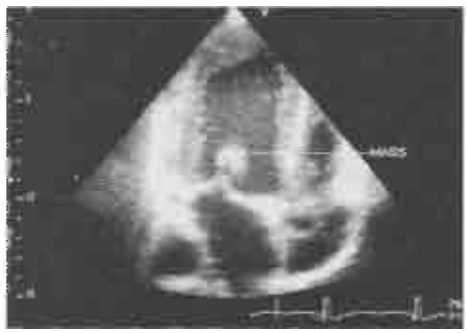

Fig.3: Case 2: Two dimensional trans-thoracic echocardiography image: Vegetation on anterior mitral leaflet

Discussion

Infective endocarditis, at present, represents the fourth leading cause of life-threatening infectious disease syndromes, after urosepsis, pneumonia, and intra-abdominal sepsis. The intracardiac effects of infective endocarditis include severe valvular insufficiency, which may lead to intractable congestive cardiac failure and myocardial abscesses. Systemic embolization occurs in 22% to 50% of cases. It produces a wide variety of systemic signs and symptoms through several mechanisms,

including both sterile and infected emboli and various immunological phenomena[8]. Mitral valve prolapse, vaginal hysterectomy and variceal sclerotherapy are the three high risk factors for infective endocarditis. The use of new clinical criteria, emphasizing echocardiography, can guide the practitioner to a correct diagnosis of this disease[9]. Prompt recognition and management of the major complications of infective endocarditis, such as heart failure, periannular extension of infection, splenic abscess, and mycotic aneurysm, are also essential for successful patient outcome. Chronic heart failure may develop acutely from perforation of a native or bio-prosthetic valve leaflet, rupture of infected mitral chordae, valve obstruction from bulky vegetations, or sudden intracardiac shunts from fistulous tracts or prosthetic dehiscence. Chronic heart failure may also develop more insidiously, despite appropriate antibiotics, due to progressive worsening of valvular insufficiency and ventricular dysfunction. Patients who have normal ventricular function or only mild chronic heart failure at initial diagnosis of infective endocarditis may progress to severe chronic heart failure during treatment, and two thirds of those patients will do so within the first month of therapy.[3] Chronic heart failure in infective endocarditis, irrespective of the cause or mechanism, portends grave prognosis with medical therapy alone. It is also the most powerful predictor of poor outcome with surgical therapy.[4] The first patient had mitral valve prolapse with moderate mitral regurgitation and thickened valves which according to American Heart Association, is a moderate risk condition. In the largest retrospective study of population risk factor for endocarditis to date, MVP was as significant a risk factor as the presence of a prosthetic valve. It is not MVP that increases the risk, but the jet of mitral regurgitation, causing spearing forces which damages the atrial structure of the valve.'

Vaginal hysterectomy is a risk factor for bacteraemia in Case no 1. Insertion of Foley's catheter is another added risk. The recommended prophylaxis for genitourinary procedures and abdominal surgeries is oral amoxicillin or intravenous ampicillin or vancomycin or vancomycin together with gentamycin[2,5].

In Case no 1, ampicillin but not vancomycin was given on the first day of admission as her mitral valve status was unknown. It is noteworthy that this patient developed infective endocarditis of the mitral valve within 15 days which was evident from the two 2D TTE done on the 20^{th} and 35^{th} post operative days with the first one showing no vegetation while the second one showing a mobile pedunculated mass. This can be explained by the presence of two of the most important risk factors.

In Case no 2, gastrointestinal manipulation namely variceal sclerotherapy proved to be a high risk factor for bacteraemia.

Conclusion

Echocardiography plays an important role in the diagnosis and management of infective endocarditis. Characteristic vegetations, abscesses, dehiscence of new prosthetic valves, and new regurgitation, in combination with other clinical parameters, are four powerful identifiers of infective endo-carditis[6,7]. Echocardiography is not an appropriate screening test in the evaluation of patients with fever or a positive blood culture that is unlikely to reflect infective endocarditis, nevertheless, some form of echocardiography should be performed in all patients suspected of having this lesion. Infective endocarditis prophylaxis has to be strictly adhered to, in any patient with cardiac condition undergoing any highrisk upper gastrointestinal procedure, genitourinary procedure or abdominal surgeries, if this complication has to be avoided. It is a cause for alarm because it is known that this disease caries a mortality rate of 20-40% and perhaps as high as 70% in some cases.

Acknowledgement

Authors want to thank Dr. A.K.Sinha of K.K.Nursing Home, Bhagalpur, Bihar for the second case report

References

1. Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis: Recommendations by the American heart association. Clin lnfect Dis; 25:1448-1458.
2. Durack DT. Prevention of infective endocarditis. N Engl J Med; 332:38-44, 1995.
3. Mills J, Utley J, Abbott J. Heart failure in infective endocarditis: predisposing factors, course and treatment. Chest. 1974; 66:151-157.
4. Stinson EB. Surgical treatment of infective endocarditis. Prog Cardiovasc Dis. 1979; 22: 145-168.
5. Wilson WR, Karchmer AW, Dajani AS, Taubert KA, Bayer A, Kaye D, Bisno AL, Ferried P, Shulman ST, Durack DT. Antibiotic treatment of adults with infective endocarditis due to streptococci, enterococci, staphylococci, and HACEK

microorganisms; American Heart Association. JAMA. 1995; 274:1706-1713.

6. Durack DT, Lukes AS, Bright DK. New criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings: Duke Endocarditis Service. Am J Med. 1994;96:200- 209.
7. Shapiro SM, Young E, De Guzman S, Ward J, Chiu CY, Ginzton LE, Bayer AS. Transesophageal echocardiography in diagnosis of infective endocarditis. Chest. 1994;105:377- 382.
8. Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Ferried P, Gewitz MH, Shulman ST, Noun S, Newburger JW, Hutto C, Pallasch TJ, Gage TW, Levison ME, Peter G, Zuccaro G Jr. Prevention of bacterial endocarditis: recommendations by the American Heart Association. JAMA. 1997;277:1794-1801.
9. Bayer AS. Infective endocarditis. Clin lnfect Dis. 1993; 17:313-320.
10. Heiro M, Nikoskelainen J, Hartiala JJ, Saraste MK, Kotilainen P. Diagnosis of infective endocarditis: Sensitivity of the Duke vs von Reyn criteria. Arch Intern Med. 1998; 158:18-24.