Case Report

Infective Endocarditis by Pseudomonas Species Following Percutaneous Transluminal Mitral Commissurotomy

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Abstract:

Infective mitral valve endocarditis developed in a 35-year-old male patient after a percutaneous transvenous mitral commissurotomy (PTMC). The echocardiogram demonstrated vegetation in the anterior leaflet of the mitral valve and blood culture showed growth of Pseudomonas species which was sensitive to Ceftazidime, Ciprofloxacin, Cotrimoxazole and Imipenem and resistant to Amikacin, Ceftriaxone, Gentamycin and Nitilmycin. The patient underwent treatment with intravenous ceftazidime and ciprofloxacin for six weeks and patient improved significantly and got cure of the disease. Infective mitral valve endocarditis should be recognized as a potentially lethal complication after PTMC. The important measures to prevent bacteremia during PTMC and the appropriate role of antibiotics and operation are discussed.

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Introduction:

Percutaneous Transvenous Mitral Commissurotomy (PTMC) was introduced as a therapeutic option for acquired mitral stenosis in 1982.[1-4] Since then the therapeutic effectiveness of PTMC has been widely accepted despite the accompanying risk of mitral regurgitation and left to right atrial shunting.[1-6] Infective endocarditis after interventional catheter procedures is rare event.[7] Infective endocarditis is a disease that is associated with considerable morbidity and mortality in spite of advances in medical, surgical and critically care interventions.[8] Infective endocarditis caused by Pseudomonas species most commonly involves right sided heart valves in patients with intravenous drug abuse. Here we report a case of infective endocarditis after PTMC caused by Pseudomonas species.

Case Report:

A 35 years male, carpenter admitted into National Institute of Cardiovascular Diseases on 30.09.2009 with the complaints of high grade intermittent fever associated with chills and rigor and night sweating for 1½ months . There was history of significant weight loss and generalized weakness for last several days. He was diagnosed as a case of chronic rheumatic heart disease with mitral stenosis 4 years back and developed CVD with right sided hemi paresis 2 years back with no residual effect now. There is history of high grade intermittent fever that developed 15 days after PTMC that was done one year back. He was admitted into NICVD, Dhaka & Dhaka Medical College Hospital for several times and treated with antibiotics and other supportive drugs like aspirin, digoxin, diuretics, and penicillin. But his symptoms did not subside.

On examination patient was ill looking with poor nutrition, mildly anaemic, clubbing present, pulse-104/min, BP-110/70mmHg, JVP - not raised, apex beat in the left 5th intercostal space medial to mid clavicular line, tapping in nature. Left parasternal lift and palpable P2 present with diastolic thrill in mitral area. 1st and 2nd heart sounds are loud; there is low pitched, rough, rumbling, mid diastolic murmur in mitral area of grading 4/6. He is clinically diagnosed as a case of chronic rheumatic heart disease with residual mitral stenosis with pulmonary hypertension with infective endocarditis.

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Investigations showed , Hb-10.7gm/dl, total count 11.800/cu.mm with N-76%, L-20%, E-3% B-00%, ESR -90mm in 1st hr, CRP-18mg/dl, blood culture and sensitivity tests were done for several times. Previously four Blood cultures were negative blood culture done on 16.09.09 revealed Pseudomonas species and sensitive to Ceftazidime, Ciprofloxacin, Cotrimoxazole and Imipenem and resistant to Amikacin, Ceftriaxone, Gentamycin and nitilmycin. Echocardiography showed vegetation on tip of anterior mitral leaflet of mitral valve(Fig-1).

After six weeks of intravenous Ceftazidime and intravenous Ciprofloxacin patient improved significantly, feeling of wellbeing improved, fever subsided, appetite increased, ESR reduced. Follow up echocardiography after 8 weeks revealed no vegetations at tip of anterior mitral leaflet.

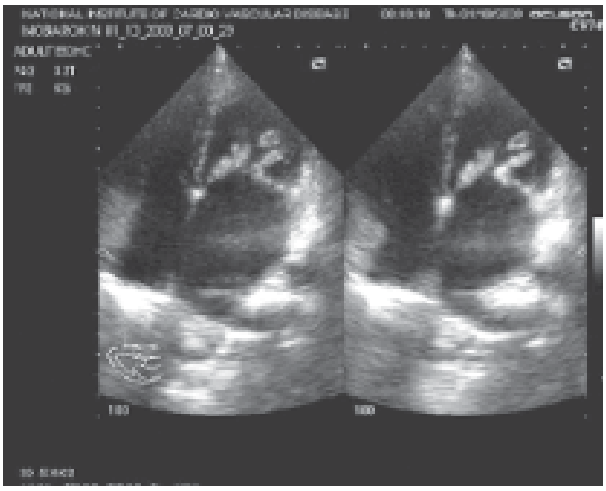

Fig-1: *Vegetation in AML of Mitral Valve.*

Discussion:

Most (95%) patients with *P. aeruginosa* endocarditis have abused intravenous drugs, and nearly all Intravenous Drug Abusers(IDUs) have abused Tripelennamine and Pentazocine ("T's and blues").[9,10,12–16] The male:female ratio is 2.5:1, and the mean age is 30 years. The organism affects normal valves in most cases. Major embolic phenomena, inability to sterilize valves, neurological complications (53%), ring and annular abscesses, splenic abscesses, bacteremic relapses, and rapidly progressive CHF are common. Ecthyma gangrenosum, the necrotizing cutaneous lesion characteristic of *Pseudomonas* bacteremia, has occasionally been noted, especially in cases of IE caused by *P (Burkholderia) cepacia*.[17] The disease carries the highest mortality rate in patients >30 years of age (73% versus 33% in younger patients) when the duration of illness is<5 days (which increases mortality from 41% to 76%) and when there is left-sided cardiac involvement.[12,15] Because of the gloomy outlook and frequent complications,[13] many authorities recommend early surgery for left-sided *Pseudomonas* endocarditis.[10,16] In contrast, high-dose regimens of Antipseudomonal Penicillins combined with Aminoglycosides have had a salutary effect in a majority of patents with isolated right-sided Pseudomonal IE.

Medical therapy may be successful in *P. aeruginosa* IE involving the right side of the heart in 50% to 75% of cases. If the disease is refractory to antibiotics, then partial tricuspid valvulectomy or "vegetectomy"[18] without valve replacement is indicated.[19] Although valve replacement often is necessary for curing left-sided IE caused by *P aeruginosa*,[20] results in a series of 10 patients (7 with left-sided involvement alone or in combination with tricuspid disease) suggest that medical therapy alone is occasionally curative.[11] Studies in animals with experimental *Pseudomonas* endocarditis[21] offer a potential explanation for these disparate results: The penetration into vegetations and the time during which antibiotic concentrations exceeded the MBC were both significantly greater with tricuspid than with aortic vegetations for both Ceftazidime and Tobramycin. Problems have emerged with all potential regimens in animal models of *P. aeruginosa* IE, including failure of β-lactam (eg, Ceftazadime) therapy as a result of constitutive hyperproduction of type β-lactamase by isolates within valve vegetations in animal models[21] and clinically;[22] isolates demonstrating Aminoglycoside resistance caused by permeability defects that emerge during therapy; absence of a postantibiotic effect of B-lactams against *P. aeruginosa* in vivo,[23] thus necessitating frequent (or continuous) drug administration; and reduced host mediated clearance of mucoid strains from the valvular vegetation resulting from alginate exopolysaccharide.[24] On the basis of clinical experience,[12,14,15] however, the preferred regimen for IE caused by *P. aeruginosa* is high-dose tobramycin (8 mg/kg per day IV or intramuscularly in once-daily doses) with maintenance of peak and

trough concentrations of 15 to 20 μgm/mL and ≤2 μgm/mL, respectively, in combination with either an extended-spectrum penicillin (eg, Ticarcillin, Piperacillin, Azlocillin) or Ceftazidime or Cefepime in full doses (Class IIa, Level of Evidence: B). The toxicity associated with this regimen is surprisingly low; combination treatment should be given for a minimum of 6 weeks. The use of Quinolones (in combination with an Aminoglycoside) for the treatment of *Pseudomonas* endocarditis appears promising, based on favorable results in animal models[21] and humans,[24] but the development of stepwise resistance during therapy may limit the efficacy of this class of drugs in the future. On the basis of limited experimental data,[25] Ceftazidime-Tobramycin is preferred over Aztreonam - Tobramycin for this disease. Approximately 7 cases of *P aeruginosa* endocarditis have been successfully treated with imipenem plus an aminoglycoside,[26] but the potential for the development of resistance exists with any of these regimens.

Y. Moriyama et al [27] reported first case of Infective mitral valve endocarditis after percutaneous transvenous mitral commissurotomy in 1995. Bacteremia occurs very infrequently during routine diagnostic cardiac catheterization. According to the literature [1-4] infective endocarditis occurred in only 3 of 12,367 cases evaluated in the cooperative study on cardiac catheterization. The therapeutic effectiveness of PTMC is, however, based on mechanical fracturing of the fused commissure. The site of damage induced by balloon dilatation on an already compromised valve would be ideal for bacterial implantation. Animal experiments using rabbits have demonstrated that bacteremia in association with catheter induced endothelial damage may result in endocarditis [5,28].

It must therefore be underscored that a sterile technique for the PTMC procedure and prompt removal of the intra- venous catheters should decrease the incidence of bacteremia. All patients undergoing PTMC should be treated with prophylactic antibiotics, achieving therapeutic serum bacteriocidal levels in the periprocedure period. This may be especially important in immunocompromised patients. Infective endocarditis after interventional catheter procedures is a rare event; however, whenever this complication is suspected, echocardiography should be performed to detect any new lesions on the infected valve such as vegetation. Prompt surgical treatment with valvuloplasty or replacement of the infected valve is indicated in patients who failed to respond to the appropriate antibiotic treatment.

References:

1. Rahman MT, Haque SA , Islam KQ, Chowdhury AW, Chowdhury AU, Khair MA; Immediate Outcome and Safety of Percutaneous Transvenous Mitral Commissurotomy In Patients with Restenosis After Closed Mitral Commissurotomy. Bangladesh Heart journal.2008; 23(1):44-47.
2. Islam KQ , Rahman MT, Haque SA . Percutaneous Transvenous Mitral commissurotomy In Juvenile Rheumatic mitral stenosis, Immediate and In – Hospital Results. Chest and heart Journal. 2007;31(2):94-99.
3. Md. Toufiqur Rahman, Syed Azizul Haque, Khandakar Qamrul Islam, Abul Khair, Ashraf Uddin Chowdhury;" Immediate Outcome of Percutaneous Transvenous Mitral Commissurotomy(PTMC)"16th ASEAN Congress of Cardiology , Bali, Indonesia, Poster Session B, Friday, April 20, 2007, no.8 . Abstract Supplement.
4. Braunwald E, Swan HJC (eds) (1968) Cooperative study on cardiac catheterization. Infectious, inflammatory and allergic complications. Circulation 37 (suppl III): 49-51
5. Garrison PK, Freedman LR (1970) Experimental endocarditis I. Staphylococcal endocarditis in rabbits resulting from placement of a polyethylene catheter in the right side of the heart. Yale J Biol Med 42:394-410.
6. Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N (1984) Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 87:394- 4O2.
7. Nobuyoshi M, Hamasaki N, Kimura T, Nosaka H, Yokoi H, Yasumoto H, Horiuchi H, Nakashima H, Shindo T, Mori T, Miyamo- to AT, and Inoue K (1989) Indications, complications, and shortterm clinical outcome of percutaneous transvenous mitral commissurotomy. Circulation 80:782-792.
8. Tuzcu EM, Block PC, Palacios IF (1991) Comparison of early versus late experience with percutaneous mitral balloon valvuloplasty. J Am Coll Cardiol 17:1112-1124.
9. Snyder N, Atterbury CE, Pinto Correia J, Conn HO. Increased concurrence of cirrhosis and bacterial endocarditis. A clinical and postmortem study. *Gastroenterology*. 1977;73:1107–1113.
10. Komshian SV, Tablan OC, Palutke W, Reyes MP. Characteristics of left-sided endocarditis due to *Pseudomonas aeruginosa* in the Detroit Medical Center. *Rev Infect Dis*. 1990;12:693–702.

11. Rodriguez C, Olcoz MT, Izquierdo G, Moreno S. Endocarditis due to ampicillin-resistant nontyphoid *Salmonella:* cure with a third-generation cephalosporin. *Rev Infect Dis*. 1990;12:817– 819.

12. Bassetti S, Battegay M. *Staphylococcus aureus* infections in injection drug users: risk factors and prevention strategies. *Infection*. 2004;32: 163–169.

13. Levine DP, Crane LR, Zervos MJ. Bacteremia in narcotic addicts at the Detroit Medical Center, II: infectious endocarditis. A prospective comparative study. *Rev Infect Dis*. 1986;8:374 –396.

14. Reyes MP, Brown WJ, Lerner AM. Treatment of patients with *Pseudomonas* endocarditis with high dose Aminoglycoside and Carbenicillin therapy. *Medicine*. 1978;57:57– 67.

15. Reyes MP, Lerner AM. Current problems in the treatment of infective endocarditis due to *Pseudomonas aeruginosa. Rev Infect Dis*. 1983;5: 314–321.

16. Wieland M, Lederman MM, Kline-King C, Keys TF, Lerner PI, Bass SN, Chmielewski R, Banks VD, Ellner JJ. Left-sided endocarditis due to *Pseudomonas aeruginosa*: a report of 10 cases and review of the literature. *Medicine*. 1986; 65:180 –189.

17. Noriega ER, Rubinstein E, Simberkoff MS, Rahal JJ. Subacute and acute endocarditis due to *Pseudomonas cepacia* in heroin addicts. *Am J Med*. 1975;59:29 –36.

18. Hughes CF, Noble N. Vegetectomy: an alternative surgical treatment for infective endocarditis of the atrioventricular valves in drug addicts. *J Thorac Cardiovasc Surg*. 1988;95:857– 861.

19. Arbulu A, Thoms NW, Chiscano A, Wilson RF. Total tricuspid valvulectomy without replacement in the treatment of *Pseudomonas* endocarditis. *Surg Forum*. 1971;22:162–164.

20. Mammana RB, Levitsky S, Sernaque D, Beckman CB, Silverman NA. Valve replacement for left-sided endocarditis in drug addicts. *Ann Thorac Surg*. 1983;35:436–441.

21. Bayer AS, Hirano L, Yih J. Development of beta-lactam resistance and increased quinolone MICs during therapy of experimental *Pseudomonas aeruginosa* endocarditis. *Antimicrob Agents Chemother*. 1988;32: 231–235.

22. Jimenez-Lucho VE, Saravolatz LD, Medeiros AA, Pohlod D. Failure of therapy in *Pseudomonas* endocarditis: selection of resistant mutants. *J Infect Dis*. 1986;154:64–68.

23. Parr TR Jr, Bayer AS. Mechanisms of aminoglycoside resistance in variants of *Pseudomonas aeruginosa* isolated during treatment of experimental endocarditis in rabbits. *J Infect Dis*. 1988;158:1003–1010.

24. Bayer AS, Park S, Ramos MC, Nast CC, Eftekhar F, Schiller NL. Effects of alginase on the natural history and antibiotic therapy of experimental endocarditis caused by mucoid *Pseudomonas aeruginosa*. *Infect Immun*. 1992;60:3979 –3985.

25. Pefanis A, Giamarellou H, Karayiannakos P, Donta I. Efficacy of ceftazidime and aztreonam alone or in combination with amikacin in experimental left-sided *Pseudomonas aeruginosa* endocarditis. *Antimicrob Agents Chemother*. 1993;37:308 –313.

26. Fichtenbaum CJ, Smith MJ. Treatment of endocarditis due to *Pseudomonas aeruginosa* with imipenem. *Clin Infect Dis*. 1992;14: 353–354.

27. Moriyama, H. Toyohira, H. Saigenji, S. Shimokawa, A. Taira Infective mitral valve endocarditis after percutaneous transvenous mitral commissurotomy. *Eur J Cardio-thorac Surg* 1995; 9: 111-112.

28. Rahman MT, Zaman MK, Haque SA , ALI M, Chowdhury AU, Sayeed MZ. A case of Congenital Mitral Stenosis. *Bangladesh Heart Journal*.2006; 21(1):60-62.