

Changing Pattern of Infective Endocarditis

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SUMMARY:

Fortyfive indoor patients admitted during 1985-87 with mean age of 19 years are being reported. 37 patients (82%) had pre-existing rheumatic valvular lesions and 8 patients (81%) had underlying congenital heart disease. Two patients had prosthetic valve endocarditis while right-sided endocarditis was found in 8 cases (80%). Fever, anaemia, clubbing, splenomegaly and microscopic haematuria were the common features. Portal of entry could not be established in any case. Blood cultures were positive in 10 cases (22%). On Echocardiography vegetations were seen in 25 cases (55%). Mortality, irrespective of causative organism was 29%. Best therapeutic response was obtained with combination therapy of Penicillin or Cephalosporins, with Gentamycin in 47% and 37% cases respectively. Two cases were sent for urgent surgery.

Introduction:

Bacterial endocarditis or better called Infective endocarditis which was first described by Osler (1) and Horder (2) remains one of the most serious complications of heart disease. It was a fatal disease till the introduction of Penicillin when the mortality fell from almost 100% to around 30% where it has generally stayed(3), though lower figures have been quoted from some major centres(4) in recent years.

In spite of new diagnostic techniques, potent antimicrobial agents and advances in cardiac surgery, the morbidity and mortality still remains significantly high.

The clinical pattern of Infective Endocarditis is changing because of alteration in the

population at risk, wide spread use of antibiotics, invasive monitoring and cardiac surgery(5). These factors make the diagnosis of the disease much more difficult.

Patients and Methods:

It was a prospective study covering all patients suspected of infective endocarditis admitted to cardiology unit, P.G.M.I./R.H. from January 1985 to November, 1987.

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A proforma was used giving details of under-

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lying cardiac disease, duration of fever, detailed clinical history and physical examination. All patients had routine investigation like full blood count, ESR blood Urea and electrolytes, Urine exam. Chest X-Ray and E.C.G. Other investigations for P.U.O. like blood film for Malaria parasites, Widal, liver function tests, ASO titre etc. were also done. Some patients had Rheumatoid and Antinuclear factor checked.

Arrangements were made with PMRC, Peshawar who kindly agreed to provide blood culture specimen bottles as well as culturing facilities for all patients.

Three blood cultures were taken from all patients at random intervals with all necessary precautions and were subcultured aerobic and anaerobically. Patients with known history of recent antibiotic use, the drug was stopped for at least three days prior to blood cultures.

M-Mode and Cross-Sectional Echocardiography was done on all patients on admission and repeated in some for follow up.

Those patients whose fever settled down within a week after admission or where the diagnosis other than Infective Endocarditis was established, were excluded from the study.

Antibiotic therapy was given for six weeks,

irrespective of causative organism. It was changed only if there was no clinical improvement and fever response within two weeks.

Patients were declared cured after completion of therapy with signs of clinical improvement, apyrexia and falling ESR.

Results:

A total of 65 proformas were collected. Out of which only 45 were accepted with most probable Infective Endocarditis. 20 cases were discarded, as they proved to be non-endocarditic.

**TABLE II
HISTORY OF 45 CASES OF
INFECTIVE ENDOCARDITIS**

Palpitation + Dyspnoea	36	80%
Fever	40	88%
Anorexia + Wt. loss	30	70%
Joint pains	09	20%
Known Previous Antibiotic Therapy	12	27%
Recent Dental Tx, Minor Surgical		
OR		
Diagnostic Procedures		NIL

The age range was 7-15 years (Mean 19 years) with greater proportion of males (M : F Ratio 3 : 2). Table-I gives the details of underlying cardiac lesions. One case of mixed rheumatic heart disease had vegetations on the Tricuspid valve. All cases of C.H.D. except one, had right heart endocarditis. Table-II & III give details of common presenting symptoms and signs. Table-IV deals with investigations while V & VI give details of therapy, course and mortality.

**TABLE I
UNDERLYING CARDIAC LESION IN 45
CASES OF I.E.**

(A) R H D	37	82%
Pure mitral valve disease	10	27%
Pure aortic valve disease	08	22%
Mixed valvular diseases	17	46%
(B) C H D		
Rt. heart :		
PDA	03	
TOF	02	
VSD + PS	01	
ASD = PS	01	
Lt. heart :		
Bicuspid Aortic valve	01	

**TABLE III
COMMON SIGNS IN 45 CASES OF
INFECTIVE ENDOCARDITIS**

Anaemia	30	66%
Clubbing	20	45%
Splenomegaly	18	40%
Hepatomegaly	36	80%
Heart murmurs	45	100%
Congestive cardiac failure	29	64%
Embolisation	05	11%
Cerebral	02	
Peripheral	03	

TABLE IV
INVESTIGATIONS OF
45 CASES OF I.E.

Haemoglobin 10 Gm or below	31	68%
Leucocytosis 10,000/cmm or more	15	33%
ESR more than 20 mm 1st hr	30	33%
RBC in urine more than 5/HPF	27	60%
Proteinuria + 1 or more	20	45%
Blood urea more than 40 mg%	09	20%
Positive blood culture	10	22%
Strep. viridans	03	
Staph. aureus	03	
Staph. epidermidis	02	
Citobacter	01	
Micrococcus	01	
Electrocardiography		
LVH	30	67%
RVH	09	20%
Normal	07	13%
Chest X-Ray		
Cardiomegaly (CTR. more than 50%)	36	80%
Echocardiography (M-MODE + SECTOR)		
Vegetations:		
Absent	20	45%
Present	25	55%
On Mitral valve	12	48%
Aortic valve	05	20%
Mitral + Aortic	02	08%
Tricuspid valve	02	08%
Pulmonic	03	12%
Pul. artery (Ductal)	01	04%

Discussion:

The mean age of 19 years for our patients was extremely low which was even lower than 31 year of pre-antibiotic era (6).

In last 2-3 decades the mean age has steadily increased to 57 years (6,7) because of increase longevity and falling incidence of rheumatic heart disease.

Aortic Valve is now much more commonly affected with decline in mitral valve involvement. Therefore the sex ratio has also changed, men now account for 2-3 times as many cases as women (8,9).

TABLE V
THERAPY OF 45 CASES OF I.E.

Pencillin + Streptomycin	05	11%
Pencillin + Gentamycin	21	47%
Cephalosporins + Gentamycin	17	37%
Fucidin + Gentamycin	02	04%

TABLE VI
COURSE AND MORTALITY IN 45
CASES OF I.E.

Cured	27	60%
Died	13	29%
LAMA	03	07%
sent for emergency surgery	02	04%

This is in contrast to our study where all cases had pre-existing heart disease with rheumatic heart-disease making the main bulk (82%). Mitral Valve was still commonly affected. Though male to female ratio was 3:2 but females predominated the younger age group and in isolated mitral valve involvement.

Fever, anaemia, clubbing, hepato-splenomegaly and microscopic haematuria were still the common features (10). Since majority of our patients had advanced shenmatic heart disease, 64% presented with congestive cardiac failure which explains the high percentage of cases (80%) with hepatomegaly. All our patients had murmurs. 88% presented with fever on admission while remaining 12% developed fever within a week after admission.

It was interesting to note that none of our patients gave the history of recent dental extraction, minor surgical or diagnostic procedures. However, they were not referred to the dentist for proper check up since they were all young with good oro-dental hygiene.

Ocular, Skin manifestations, and Splinter haemorrhages were rarely seen in our patients as reported by various studies of antibiotic era(11).

Blood cultures were positive in only 10 cases (22%). Out of which two cases of citobacter and Micrococcus were most probably contaminants while the rest 8 cases grew the same bacteria in at least two sets. This was in sharp contrast to other studies where the causative organisms were isolated from 90% to 98% of cases (9, 12).

The possible reasons for such a high incidence of blood culture negative endocarditis in our study are follows:—

1. Indiscriminate use of antibiotics prior to hospitalisation.
2. Faulty techniques of sampling and storage.
3. Inadequate laboratory facilities for diagnosis and identification of bacteria.
4. High incidence of rare infecting organisms.
5. Lack of financial resources.

On M-Mode and 2-D Echocardiography definite vegetations were seen in 55% of cases. Our figures were little low probably due to strict diagnostic criteria. Other studies have shown the sensitivity ranging from 50% to 80% (13). It can be difficult to diagnose vegetations on badly deformed rheumatic valves. The active vegetations have to be differentiated from a lump of calcium, Non-infective vegetations of rheumatic and non-rheumatic origin and from healed infective vegetations(14) which may take almost three years to disappear. It must also be mentioned that negative vegetation will not rule out possibility of infective endocarditis.

Right-sided endocarditis was found in 18% of cases which was exclusively due to congenital heart disease. Other studies have given the figures from 8.2% to 14% depending on the number of drug addicts. (7,15).

Though drug addiction is quite common in our province, intravenous drug abuse is hardly even seen and no case of endocarditis was reported during the study.

Right sided endocarditis runs relatively benign course with very good response to therapy and low mortality.

There were two cases of late onset prosthetic-valve endocarditis who did not respond to therapy and died. The overall incidence of PVE is estimated to be between 1-10% with most recent reported average of 4.4% (16). The mortality of PVE ranges from 23-86% (17). Poor prognostic

factors include non-streptococcal origin, early incidence of aortic valve involvement, presence of congestive heart failure and prosthetic dysfunction.

Management was found to be rather difficult because infective organisms were not known in majority of cases. However, combination therapy of Penicillin or Cephalosporins with Gentamycin gave best results. Our mortality was around 30%.

The exact cause of death could not be established because of lack of autopsy but uncontrolled infection, advanced cardio-hepato-renal failure and cerebro-vascular episodes were the most probable causes.

The bad prognostic signs were late diagnosis, in starting therapy and late referral to surgery.

Prophylaxis is extremely important especially for those at risk, since bacteraemia which is becoming more common, must be recognised more often. Early diagnosis can be greatly helped if when a patient with known heart disease develops fever of unknown cause, to take blood cultures before antibiotics are given.

The combination of enhanced awareness on the part of patient and physician, together with meticulous prophylaxis, early consultation with cardiac surgeon and effective treatment with antibiotics should considerably reduce the mortality.

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REFERENCES:

1. Osler, W. Chronic Infective Endocarditis. Q.J. Med. 1908-9-2:219-30.
2. Horder, T. Infective Endocarditis with an analysis of 150 cases and with special reference to the chronic form of disease Q.J. Med. 1908-9; 2:89-324.
3. Smith, R.H. Radford, D.J., Clark, R.A., Julian, D.G. Infective Endocarditis: A Survey of cases in the South-East Region of Scotland, 1969-72. Thorax 1976; 31:373-9.

4. Von Reyn, Infective Endocarditis, An analysis based on strict case C.F. et al. Definitions. Ann. Intern. Med. 94:505,1981.
5. Come, P. Infective Endocarditis,; Current Prespectives Compr. Ther. 8:57, 1982.
6. Cherubin, C.E. & New, H.C., Infective Endocarditis and the Presbyterian Hospital in New York city From 1938-1970 Am. J. Med. 5: 83, 1971.
7. Pelletier, L:L. & Petersdorf R.G., Infective Endocarditis: A Reveiw of 125 cases from the University of Washington Hospital, 1962-72 Medicine, (Baltimore) 56:287,1977.
8. Lerher, P.I. & Weinstein, L. Infective Endocarditis in Antibiotic era. N. Engl. J. Med. 274:199,259,323, 388,1966.
9. Bayliss, R. Clarke, C. Qakley C.M., Somerville, W. Whitfeild, AG.W., Young, S.E.J. The Microbiology and Pathogenesis of Infective Endocarditis, Br. Heart. J. 50:513-9.1983.
10. Cates, J.E. Christie, R.V. Subacute Bacterial Endocarditis: A Reveiw of 442 patients in 14 centres appointed by the Penicillin Trials Committee of Medical Research Council; Q.J; Med. 1951; 20:93-130.
11. Prondfit, W., Skin sign of Infective Endocarditis Am. Heart. J. 106: 1451, 1983.
12. Werner, A.S. et. al; Studies on the bacteraemia of Bacterial Endocarditis J.A.M.A. 202:199. 1967.
13. O'Brieu, J. & Geiser, E. Infective Endocarditis and Echocardiography. Am Heart J. 108 : 386, 1984.
14. Stafferd, A, et al. Echocardiographic appearance of healing bacterial vegetations. Am. J. Cardiol., 44: 754,1979.
15. Michael. J. Robbins, Right sided Valvular Endocarditis: Etiology, Diagnosis et al. and an approach to therapy. Am Heart J. 128:111. 1968.
16. Moore-Gillon, J., Eykyn, S.J. & Philips, 1. Prosthetic valve endocarditis: Br. Med. J. 287 : 739, 1983.
17. Calderwood, J.B., et al. Risk factors for the development of Prosthetic valve endocarditis: Circulation 72:31:1985.

