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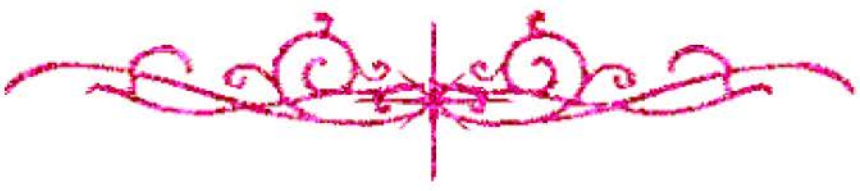

EARLY ONSET PROSTHETIC VALVE ENDOCARDITIS

THESIS

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INTRODUCTION

INTRODUCTION

Although early prosthetic valve endocarditis is a rare complication occurring during the early post operative period, but it can be considered the most serious one as early infection is usually severe and invasive and most probably leads to further complications and sometimes ends in patient's death.

Diagnosis of early prosthetic valve endocarditis is rather a difficult mission as its clinical presentations may be deceiving and its signs and symptoms always simulated by many other medical problems, so definite diagnosis always not established at the optimum time.

Treatment of early prosthetic valve endocarditis is another major problem as the severity of infection in these cases may always lead to both intracardiac complications such as prosthetic valve dehisence and dysfunction and extension of intracardiac infection beyond the valve, and also extracardiac complications such as systemic emboli that may affect any of the body systems or organs.

For all of the above, it is mandatory when dealing with early prosthetic valve endocarditis to apply the golden rule:

" Protection is better than treatment "

We have to study carefully the possible risk factors that may predispose to occurrence of early prosthetic valve endocarditis and take all precautions in order to avoid these risk factors aiming to reduce the incidence of occurrence of early prosthetic valve endocarditis as much as possible.

All of the above were the great stimulus for us to make this study to clarify and explain some points about early prosthetic valve endocarditis in our cardiac surgery center in order to improve our work and decrease morbidity and mortality of open heart surgery patients as much as possible.

REVIEW OF LITERATURE

HISTORICAL NOTES

Not long after the initial reports of valve replacement by Starr and Harken, the First reports of prosthetic valve endocarditis (PVE) appeared in the literature.

Before the routine use of prophylactic antibiotics, Geraci and associates (1963)[1] and Stein and co-workers (1966)[2] reported incidences of early prosthetic valve endocarditis of 10 and 12 % respectively. Routine prophylactic antibiotics markedly reduced the incidence of this devastating complication.

From the outset, the surgical management of prosthetic valve endocarditis has been a formidable challenge and remains so today. Early reports described debridement and drainage of the infection, which was associated with an extremely high mortality rate. Discouraged by such early surgical experience, cardiac surgeons made strong efforts to avoid operation for prosthetic valve endocarditis.

In 1972, Ross successfully performed aortic root replacement for prosthetic valve endocarditis using an aortic homograft (Ross, 1990)[3]. His reports stressed the importance of complete surgical debridement of all infected tissues, and the use of homograft for reconstruction offered greater surgical flexibility and the advantage of minimizing the placement of foreign material area.

Plinger and Maloney[4] reporetd replacement of the infected aortic prosthesis and external felt buttressing for correction of aortic ventricular discontinuity in 1977.

The following year, Frantz, Murray, and Wilcox[5] reported repair of ventricular-aortic discontinuity from endocarditis and abscess formation by aortic root replacement using a synthetic valve conduit.

Danielson and associates (1974)[6] described a technique for treating extensive periannular abscess formation in the native aortic valve endocarditis by translocation of a prosthetic aortic valve into the ascending aorta and saphenous vein coronary artery bypass grafting.

In 1981, Reitz and co-workers[7] successfully applied this technique to treatment of prosthetic aortic valve endocarditis.

In 1982, Symbas and colleagues[8] combined aortic valve replacement with patch repair of periannular abscess cavity.

Over the past decade, the incidence of prosthetic valve endocarditis has remained fairly stable: most series have reported an incidence of 2-4% (Cowgill et al., 1986)[9] and prosthetic valve endocarditis continues to be one of the most serious and surgically challenging complications of valve replacement.

DEFINITION AND EPIDEMIOLOGY

Karchmer (2001)[10] Defined infective endocarditis in general as a microbial infection of the endothelial surface of the heart.
The Characteristic lesion, the vegetation, is a variably sized amorphous mass of platelets and fibrin in which abundant microorganisms and scant inflammatory cells are enmeshed.
Heart valves are most commonly involved; however, infection may occur at the site of a septal defect or on chordae tendineae or mural endocardium.

Many species of bacteria and fungi, mycobacteria, rickettsiae, chlamydiae,and mycoplasmas cause infective endocarditis; nevertheless, streptococci, staphylococci, enterococci, and fastidious gram-negative coccobacilli cause the majority of cases of infective endocarditis. (Karchmer, 2001)[10]

The terms acute and subacute are often used to describe infective endocarditis. Acute infective endocarditis presents with marked toxicity and progresses over days to several weeks to valvular destruction and metastatic infection. In contrast, subacute infective endocarditis evolves over weeks to months with only modest toxicity and rarely causes metastatic infection. Acute infective endocarditis is caused typically, although not exclusively, by staphylococcus aureus, whereas the subacute syndrome is more likely caused by viridans streptococci, enterococci, coagulase-negative staphylococci, or gram-negative coccobacilli. (Karchmer, 2001)[10]

The incidence of infective endocarditis remained relatively stable from 1950 through 1987at about 4.2 per 100,000 patients-years.
During the early1980`s, the yearly incidence of infective endocarditis per 100,000 population was 2.0 in the United Kingdom and Wales

and 1.9 in the Netherlands. (Van der Meer, et al. 1992)[11]

A higher incidence was noted from 1984 through 1990; 5.9 and 11.6 per 100,000 population were reported from Sweden and Metropolitan Philadelphia, respectively. Endocarditis usually occurred more frequently in men; gender-derived ratios range from 1.6 to 2.5. The age-specific incidence of endocarditis increased progressively after 30 years of age and exceeded 15 to 30 cases per 100,000 person-years in the sixth through eighth decades of life. (Hogevik, et al. 1995) [12]

From 55 to 75 percent of patients with native valve endocarditis have predisposing conditions: rheumatic heart disease, congenital heart disease, mitral valve prolapse, degenerative heart disease, assymetrical septal hypertrophy, or intravenous drug abuse. Predisposing conditions cannot be identified in 25 to 45 percent of patients. (Kazanjian, 1993)[13]

As regards prosthetic valve endocarditis:

Epidemiological studies suggest that prosthetic valve endocarditis comprises 10 to 30 percent of all cases of infective endocarditis in developed countries. (Pulvirenti, et al. 1996)[14]

In Metropolitan Philadelphia, 0.94 cases of infective endocarditis per 100,000 population involved prosthetic valves. (Berlin, et al. 1995) [15]

The cumulative incidence of prosthetic valve endocarditis estimated actuarially has ranged from1.4 to 3.1 percent at 12 months and 3.2 to 5.7 percent at 5 years. (Agnihotri, et al. 1995) [16]

The risk of prosthetic valve endocarditis overtime, however, is not uniform. The risk is greater during the initial 6 months after valve surgery (particularly during the first 5 to 6 weeks) and thereafter declines to a lower but persistent risk (0.2 to 0.35 percent per year). (Horskotte, et al. 1995)[17]

Studies to identify risk factors for prosthetic valve endocarditis have not resulted in a coherent picture. Data suggest that, during the initial months after valve implantation, mechanical prostheses are at greater risk of infection than bioprosthetic valves but after 12 months the risk of infection of bioprostheses exceeds that of mechanical valves (Ivert, et al. 1984)[18]