Aortic and Mitral Valve Affection In Patients With Chronic Renal Failure

Thesis

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M.S. Degree In Cardiology

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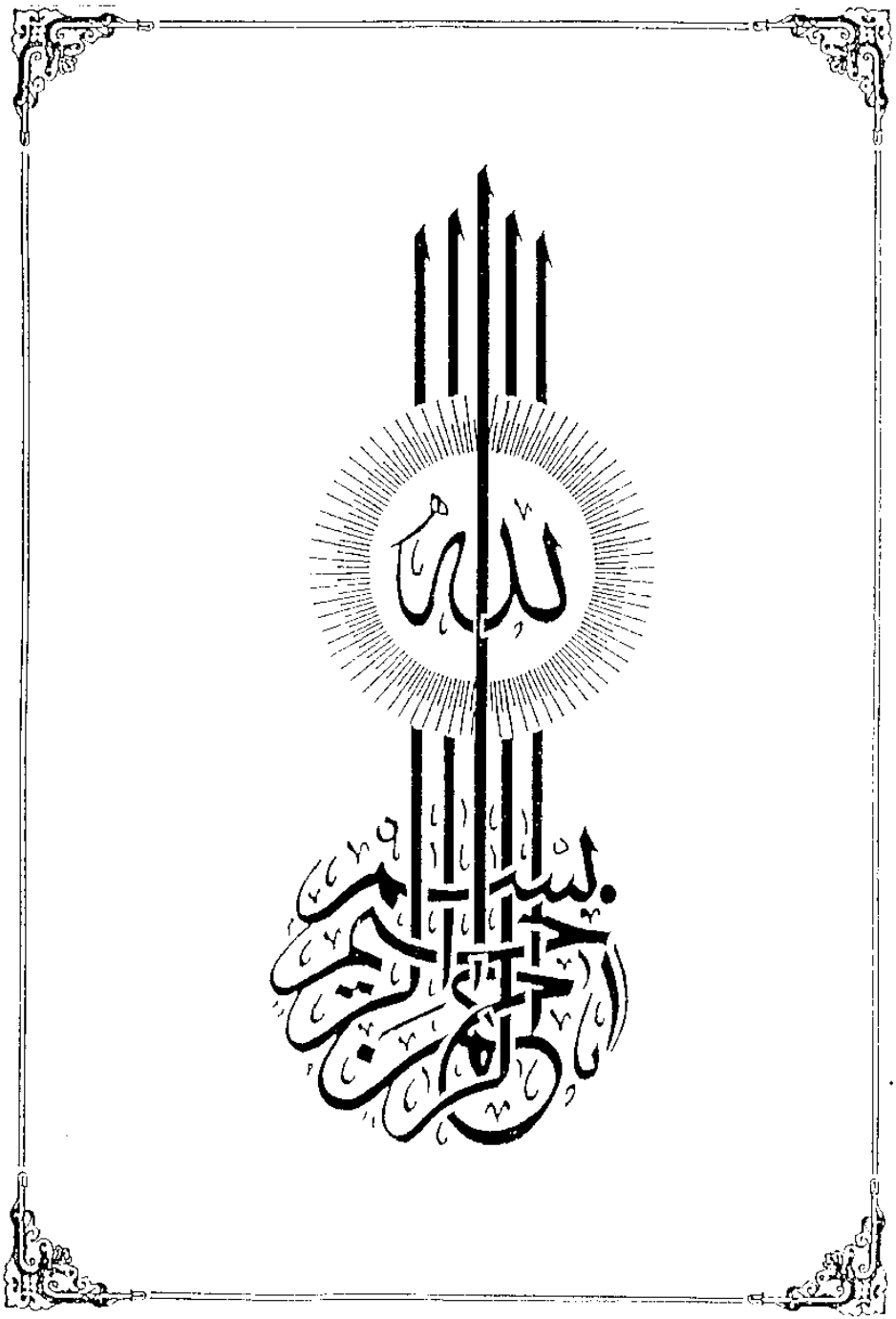

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To

My Beloved Parents

&

My Sincere Wife

Contents

Page

Introduction & Aim Of The Work

Introduction

Cardiac valvular affection is an under-recognized complication of chronic uremia ***(Maher et al., 1987)***.

Calcification of the mitral annulus was common in patients with chronic renal failure than in age matched controls ***(Fulkerson et al., 1979)***. Calcification of the mitral annulus can cause mitral regurgitation or stenosis or both and is also often associated with aortic valve calcification. Severe aortic valve calcification can cause aortic stenosis and aortic regurgitation ***(Lombard et al., 1987)***. Prospective echocardiographic study identified aortic valve calcification in 28% and mitral valve calcification in 36% of uremic patients ***(Maher et al., 1987)***. Furthermore, clinically significant valvular stenosis of tricuspid aortic valve and less frequently of the mitral valve may occur. The cause of premature calcification in uremic patients of the mitral and aortic valves is uncertain, however, secondary hyperparathyroidism, hypertension and hypercholesterolaemia are thought to be essential risk factors.

Case control studies have shown annular calcification in uremic patients with higher ionized calcium, phosphorus and Ca X P product levels.

These findings suggest that vigorous attempts to normalize calcium-phosphorous metabolism may decrease the incidence of this complication in patients with renal failure ***(Nestico et al., 1983)***.

Aim of The Work

The aim of this study is to identify the valvular heart affection in patients with chronic uremia by conventional and color coded Doppler echocardiography.

Review Of Literature

Effects of Renal Disease on The Cardiovascular System

Pericarditis:

Pericarditis is a common complication of both acute and chronic renal failure. In the past, when dialysis was not available, the occurrence of pericarditis meant impending death ***(Wacker and Merill, 1954)***. In the dialysis era, pericarditis no longer denotes such grim prognosis. The early use of dialysis in the treatment of uremia not only assures the improvement and correction of uremic pericarditis but virtually guarantees its prevention if it has not yet occurred. With the introduction of chronic or repetitive dialysis in the treatment of chronic renal failure, with which one would expect the total eradication of pericarditis, a new clinical syndrome has evolved: an effusive pericarditis that occurs with varying incidence and has been difficult to control. It is quite evident that dialysis has dramatically altered the clinical features and course of renal failure - associated pericarditis ***(Comty CM, et al., 1971)***.

Prevalence:

Pericarditis associated with chronic renal failure was first noted only 150 years ago. In 1954, the prevalence was 51 %. In more recent years, with the combination of effective conservative management and the early use of dialysis in the treatment of renal failure patients, the

prevalence of uremic pericarditis has been reduced to 16 % in 1970 and 6.0 % in 1980. However, among patients with end-stage renal disease (ESRD) undergoing chronic, intermittent dialysis, the prevalence of pericarditis has declined but still occurs at a predictable rate of 12 to 20 %. Echocardiography has revealed pericardial effusions in 32 to 56 per cent of patients at the initiation of dialysis, most of which are asymptomatic ***(Frommer, et al., 1985)***.

Clinical features:

Pericarditis associated with chronic renal failure occurs in the following clinical settings: ***(Ventura and Garella, 1990)***

1. Before initiation of dialysis.
2. Within the first 8 weeks after starting chronic, intermittent dialysis treatments (early dialysis-related pericarditis).
3. Any time after the 2nd month of chronic dialysis treatments (late dialysis - related pericarditis).

They assumed that the pericarditis seen in (1) and possibly (2) was truly uremic in nature. It is a clinical manifestation of the serositis seen in uremia which also can involve the pleura ***(Hoops, 1955)***. Fibrinous pleuritis, pleural friction rubs ***(Luft, et al., 1980)***, hemorrhagic pleural effusion, and pneumonitis also have been reported to occur in uremia.

The pericarditis seen in (3) is due to a multitude of factors such as bacterial infection, cytomegalovirus infection and dialysis itself may be one of them ***(Luft, et al., 1980)***.

The clinical presentations of pericarditis occurring in any of the clinical settings described above are virtually identical. The variations are in the frequency or intensity of certain symptoms occurring in one group compared with the others, e.g., chest pain is more frequent and severe in (1), whereas pericardial effusion is almost always the presentation in (2) and (3). Chest pain is the most common symptom. Its intensity varies from substernal pressure to severe excruciating pain when breathing: coughing or changing the body position. The pain is usually worst in the recumbent position, and partial relief is obtained by sitting and bending forward with the elbows resting on the knees. The remaining symptoms are non-specific and may represent mainly uremic symptoms.

The most impressive physical examination finding is pericardial friction rub. The sound has been described variously as scratchy and leathery. It has a high frequency, invariably has two components and occurs following the heart sounds. When present with its typical features, the friction rub presents no difficulty being identified, but when the rub is heard only in systole, it could be easily mistaken as a harsh systolic murmur and its significance may be overlooked.

Although the pericardial friction rub may be audible throughout the anterior chest wall, it is usually best heard at the area of the precordium where the heart is closet to the chest wall. The intensity of the rub depends on the extent of the inflammation ***(Rutsky and Rostand, 1989)***.

Uremic pericarditis is almost always associated with effusion. The clinical features of pericardial effusion are related to the volume of pericardial fluid accumulation and the compliance of the pericardial sac. With small effusions, no subjective complaints or objective findings are evident. When the effusion is large enough to be hemodynamically significant or if the fluid accumulation is quite rapid, the patient usually complain of dyspnea on exertion, PND, orthopnea and non-productive cough together with the chest pain. Right upper abdominal quadrant pain from congestive hepatomegaly and increasing abdominal girth from ascites are frequent features of worsening pericardial effusion, as are the clinical features of diminished cerebral blood flow. The physical examination findings include enlargement of the area of pericardial dullness, positive Ewart's sign and with impending or overt cardiac tamponade, hypotension, distended jugular veins and paradoxical pulse ***(Kleiman et al., 1978)***

Two other clinical features of interest have been observed in ***Comty, et al. (1971)*** patients with hemodynamically significant pericardial effusions.

1. Clotting of the arteriovenous fistula or shunt and,
2. In patients who still had measurable urine volume (≥ 1000 ml / day) at the time pericardial effusion evolved, a marked decrease in urine volume and sodium concentration with a concomitant increase in urine urea nitrogen, creatinine and potassium concentrations.

Clotting of the cannula or fistula preceded or occurred simultaneously with the development of pericardial effusion. Clinically evident pericardial effusion was observed in 15 to 55 % of patients with uremic pericarditis of those patients with pericardial effusion ***(Comty, et al., 1971).***

Cardiac tamponade occurred in 10 to 35 %. An impending or fully developed cardiac tamponade is characterized clinically by the fast, weak pulse, the rapidly declining systolic blood pressure, and the acute rise in venous pressure ***(Specter, et al., 1983).***

Cardiac catheterization findings in such clinical settings would show:

1) Marked reduction in cardiac output / index.
2) Diminution in mean systemic arterial pressure, and
3) Elevation in mean right atrial and right ventricular end-diastolic pressures. If not corrected, cardiac tamponade is an acute medical