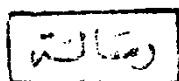


*Coronary Neovascularization as a
Specific Sign for Left Atrial Thrombus
in Mitral Stenosis*

THESIS
SUBMITTED IN PARTIAL FULFILLMENT OF
MASTER DEGREE IN CARDIOLOGY



BY

Yasser G. El Kashlan

Yasser G. El Kashlan

616-125
y.g

SUPERVISORS

u8104

Prof. HASSAN EZZELDIN ATTIA

M.D., F.A.C.C.

CARDIOLOGY DEPARTMENT, CHAIRMAN

AIN SHAMS UNIVERSITY

DR. OSSAMA HASSAN, M.D.

DR. TAREK ZAKI, M.D.

LECTURER OF CARDIOLOGY

LECTURER OF CARDIOLOGY

AIN SHAMS UNIVERSITY

AIN SHAMS UNIVERSITY



CONTENTS

INTRODUCTION AND AIM OF THE WORK	1
REVIEW OF LITERATURE	
ANATOMY OF THE LEFT ATRIUM AND LEFT ATRIAL APPENDAGE	3
ANATOMY OF THE CORONARY ARTERIES	4
PATHOPHYSIOLOGY OF MITRAL STENOSIS	6
THROMBOEMBOLISM IN MITRAL STENOSIS	8
SPONTANEOUS ECHO CONTRAST(SEC)	16
LEFT ATRIAL THROMBI IN MITRAL STENOSIS	23
SCOPE AND MAGNITUDE OF THE ISSUE	23
THE MECHANISM OF LEFT ATRIAL THROMBUS FORMATION.....	26
THE STRUCTURE OF THROMBI	31
DIAGNOSIS OF LEFT ATRIAL THROMBUS.....	35
CORONARY NEOVASCULARITY AND FISTULA FORMATION.....	51
CONGENITAL CORONARY ARTERIOVENOUS FISTULA.....	51
CORONARY NEOVASCULARITY WITH CARDIAC NEOPLASMS.....	52
CORONARY NEOVASCULARITY AND FISTULA FORMATION- A	
SIGN OF MURAL THROMBUS	53
CORONARY ARTERIOGRAPHY AS A DIAGONISTIC TECHNIQUE	
FOR LEFT ATRIAL THROMBOSIS IN MITRAL STENOSIS.....	63
PATIENTS AND METHODS.....	67
RESULTS.....	76
DISCUSSION	103
CONCLUSION.....	111
SUMMARY.....	112
REFERENCES.....	115
ARABIC SUMMARY	129



INTRODUCTION

INTRODUCTION

In 1975 Standen, using selective coronary angiography, described tumour vascularity with abnormal vessels arising from the left circumflex artery to the left atrium in patients with severe mitral stenosis. A left atrial thrombus was found at surgery. Since then sporadic case reports have also described abnormal vessels arising from the circumflex arteries or coronary neovascularization with fistula formation to the left atrium in patients with severe mitral stenosis who had left atrial appendage thrombi.

Colman and associates (1981), on the other hand, in a retrospective study of a large number of patients with mitral valve disease, have shown coronary neovascularization with fistula formation to be a specific sign for the presence of left atrial thrombi.

AIM OF THE WORK

The aim of the work is to study the presence of abnormal vessels arising from the circumflex artery or coronary neovascularization with fistula formation to the left atrium in patients with severe mitral stenosis who had left atrial thrombi.

PATIENTS AND METHODS

Two groups of patients with rheumatic mitral stenosis will be studied. *Group one* will include fifteen (15) patients in whom left atrial thrombi are detected by transesophageal echocardiography. *Group two* will also include fifteen (15) patients in whom no left atrial thrombi are detected by transesophageal echocardiography.

For both groups the following will be done:

1. Thorough clinical assessment.
2. Twelve (12) lead surface electrocardiogram.
3. Plain X-ray chest, posteroanterior and left lateral views.
4. Transthoracic echo-Doppler study, to detect the presence of mitral stenosis and to assess its severity, and to detect the presence of left atrial thrombi.
5. Coronary arteriography, to detect the presence of abnormal vessels arising from the circumflex arteries or coronary neovascularization with fistula formation to the left atrium in patients with mitral stenosis who had left atrial thrombi.
6. Results will be tabulated, analyzed and statistically studied by *T-Student Test*.
7. Results will be discussed.

REVIEW OF LITERATURE

ANATOMY OF THE LEFT ATRIUM AND LEFT ATRIAL APPENDAGE

The left atrium is located superiorly, in the midline and posterior to the other cardiac chambers. It forms the base of the heart, the esophagus abuts directly upon its posterior surface, while the aortic root impinges upon its anterior wall. The right atrium is located to the right and anterior. The left ventricle is to the left, anterior and inferior. Anteriorly, the long, narrow left atrial appendage projects forwards and partly overlaps the beginning of the pulmonary trunk.

The wall of the left atrium is 3 mm in thickness. The endocardium of the left atrium is smooth and slightly opaque. Pectinate muscles are present only in the left atrial appendage (*Hurst, 1990*).

The trabecular left atrial appendage is a remnant of the original embryonic left atrium that develops during the third week of gestation. The left atrial cavity develops later and is formed from an outgrowth of the pulmonary veins.

The function of the left atrial appendage is unknown. *Bercher et al (1963)* speculated that the atrial appendage fills the space that is created within the pericardial sac during ventricular systole as the ventricles eject blood and decrease in size. The appendage passively fills during ventricular systole and then passively empties during ventricular diastole.

ANATOMY OF THE CORONARY ARTERIES

The left main coronary artery travels anteriorly, slightly inferiorly, and leftward from the left coronary sinus to emerge from behind the pulmonary trunk. Within a short distance, usually 2 to 10 mm, the left main stem divides into 2 or more major branches of nearly equal diameter: *the left anterior descending, the left circumflex and sometimes a diagonal (or intermediate) branch.*

The left anterior descending coronary artery is a direct continuation from the left main coronary artery and courses anteriorly and caudally over the ventricular septum within the anterior interventricular sulcus. It usually circles around the apex and terminates in the inferior aspect of the cardiac apex.

The branches of this artery, in their usual order of origin, are the first diagonal, the first septal perforator, other septal perforators, and other diagonal branches.

The left circumflex coronary artery arises from the left main coronary artery at an obtuse angle and turns posteriorly as it courses around the left side of the heart within the left atrioventricular sulcus. The branches of the left circumflex are variable but may include the sinus node artery (40 - 50%), the left atrial circumflex branch, the anterolateral marginal, and the posterior descending artery (10-15%).

The right coronary artery leaves the right coronary sinus and descends in the right atrioventricular groove curving posteriorly at the acute margin of the right ventricle. In 85 to 90% of hearts; it makes a 90° turn at the crux of the heart and continues downward in the posterior atrioventricular groove, finally terminating as a left ventricular branch.

The branches of the right coronary artery include the conus artery, the artery to the sinus node, several anterior right ventricular branches, right atrial branches, the acute marginal branch, the artery to the atrio-ventricular node and proximal bundle branches, the posterior descending artery, and terminal branches to the left ventricle and left atrium (*Hurst, 1990*).

PATHOPHYSIOLOGY OF MITRAL STENOSIS

In normal adults the cross-sectional area of the mitral valve orifice is 4 to 6 cm². When the orifice is reduced to approximately 3cm², which is considered to represent mild mitral stenosis, blood can flow from the left atrium to the left ventricle only if propelled by an abnormal, though small, pressure gradient. When the mitral valve opening is reduced to 1 cm², which is considered to represent critical mitral stenosis a left atrioventricular pressure gradient of approximately 20 mm Hg (and therefore, in the presence of a normal left ventricular diastolic pressure, a mean left atrial pressure of approximately 25 mm Hg) is required to maintain normal cardiac output at rest. The elevated left atrial pressure in turn raises pulmonary venous and capillary pressures, resulting in exertional dyspnea. The first bouts of dyspnea in patients with mitral stenosis are usually precipitated by exercise, emotional stress, sexual intercourse, infection, or atrial fibrillation, all of which increase the rate of blood flow across the mitral orifice and result in further elevation of the atrial pressure.

In order to assess the severity of obstruction of the mitral valve, it is essential to measure both the transvalvular pressure gradient and the flow rate. The latter depends not only on cardiac output but on heart rate as well. An increase in heart rate shortens diastole proportionately more than systole and diminishes the time available for flow across the mitral valve. Therefore at any given level of cardiac output, tachycardia augments the transmitral valvular pressure gradient and elevates left atrial pressure further. This explains the sudden development of dyspnea and pulmonary edema in previously asymptomatic patients with mitral stenosis who experience atrial fibrillation with a rapid ventricular rate and also accounts for the equally rapid improvement in these patients when the ventricular rate is slowed by means of cardiac glycosides

and/or beta-adrenoreceptor blocking agents even when the cardiac output per minute remains constant.

Hydraulic considerations dictate that at any given orifice size the transvalvular gradient is a function of the square of the transvalvular flow rate. Thus, a doubling of flow rate will quadruple the pressure gradient, so that stress such as exercise in patients with moderate or severe mitral stenosis will cause marked elevation of left atrial pressure.

Atrial contraction augments the presystolic transmitral valvular gradient by approximately 30% in patients with mitral stenosis. Withdrawal of atrial transport when atrial fibrillation develops decreases cardiac output by about 20%. The more rapid ventricular rate that occurs in atrial fibrillation until it is pharmacologically controlled, raises the transvalvular pressure gradient. Thus, haemodynamic considerations indicate the desirability of maintaining sinus rhythm in patients with mitral stenosis (*Stott et al, 1970*).

THROMBOEMBOLISM IN MITRAL STENOSIS

Systemic arterial embolization imparts a significant risk of serious complications throughout the lives of patients with rheumatic heart disease. Left atrial thrombi have been thought to be the major source of emboli (Hwang *et al*, 1992).

Thromboembolism is a major cause of morbidity and mortality in patients with mitral stenosis (Braunwald, 1992). Weiss and Davis (1933) found that 15% of mortalities among patients with rheumatic heart disease were caused by systemic emboli. Graham *et al* (1951) evaluated the causes of death in 101 patients with mitral stenosis. In 33 patients, systemic emboli were the direct cause of death. Wallach *et al* (1953) found that embolization is responsible for death in more than 20% of patients who die of rheumatic heart disease.

Incidence:

Weiss and Davis (1933) found evidence of embolism at autopsy in 45% of 164 patients with rheumatic heart disease. Several studies have since confirmed that rheumatic heart disease is frequently associated with systemic embolization. Graham *et al* (1951) found in a clinicopathologic study evidence of systemic emboli in 49 of 101 patients with mitral stenosis. Wood (1954) reported a fourteen percent incidence of systemic embolism in patients with mitral stenosis. Casella *et al* (1964) found that about 9.6 to 20.1% of patients with mitral stenosis being evaluated for operation have clinical evidence of systemic emboli. Hwang *et al* (1992) found evidence of systemic embolization in 15% of patients with mitral valve disease. Systemic emboli involving the brain were diagnosed by clinical assessment beside head computed tomography scan. Angiography was used to confirm the diagnosis of peripheral emboli involving the kidneys,

mesenteric arteries or extremities. *Vigna et al (1993)* found that arterial embolization had occurred in 12 (20.3%) of 59 patients with rheumatic mitral stenosis.

Sites:

- (1) The central nervous system is the commonest site of lodgment of systemic emboli in patients with mitral stenosis.

Wood (1954) found that 75% of the emboli were cerebral, 33% were peripheral and 6% were visceral. *Askey (1957)* found that about one half of systemic emboli involve the brain. *Erbel et al (1986)* found that 54% of systemic emboli were in the cerebral arteries. *Daniel et al (1988)* reported that 64% of patients with systemic emboli had cerebral emboli. Finally, *Hwang et al (1992)* found that 71% of systemic emboli were located in the central nervous system.

- (2) Other sites include: the kidneys, mesenteric arteries and extremities

Prognosis:

The prognosis of systemic embolization is sometimes grave. Approximately one sixth to one third of patients do not survive the first cerebral embolism, and one third of survivors die within a year, mostly because of recurrent embolism. Delay in the management of emboli to the extremities may lead to amputation of the affected limb. Clearly, systemic embolization is a constant and undesirable threat in patients with rheumatic heart disease.

Risk Factors:

There are several studies that have correlated various clinical, haemodynamic and echocardiographic variables with the incidence of systemic embolization in patients with mitral stenosis.

The most significant risk factors that correlate closely with a high frequency of systemic embolization are:

1. The patient's age.
2. Atrial fibrillation.
3. Mean cardiac index.
4. Left atrial thrombosis.
5. Left atrial spontaneous echo contrast

Some of the studies that evaluated one or more of the above factors are enlisted below:

Graham et al (1951) found that systemic embolization occurs at one time or another in about one half of the patients with mitral valve disease who have thrombus material in the left atrium.

Askey (1957) found that 75 to 85 percent of patients with systemic emboli in the setting of mitral stenosis were found to have atrial fibrillation.

Casella et al (1964) compared the clinical and haemodynamic data of 21 patients with mitral stenosis and systemic embolization with those of another 36 patients with mitral stenosis but without evidence of systemic embolization.

They found that:

Patients with mitral stenosis and emboli differed from those without emboli by being 8.6 years older and by having a higher incidence of atrial fibrillation (85.7% versus 33.3%). The mean cardiac index in the embolic group was significantly lower than that of the group without emboli. This relationship was independent of atrial fibrillation. The incidence of systemic embolization was less than 5% under the age of 30 years and increased progressively thereafter at a rate of 1.5% per year to reach an incidence of approximately 40% by the seventh decade.