STUDY OF LEFT VENTRICULAR THROMBI IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION BY 2-D-ECHO

THESIS

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BY

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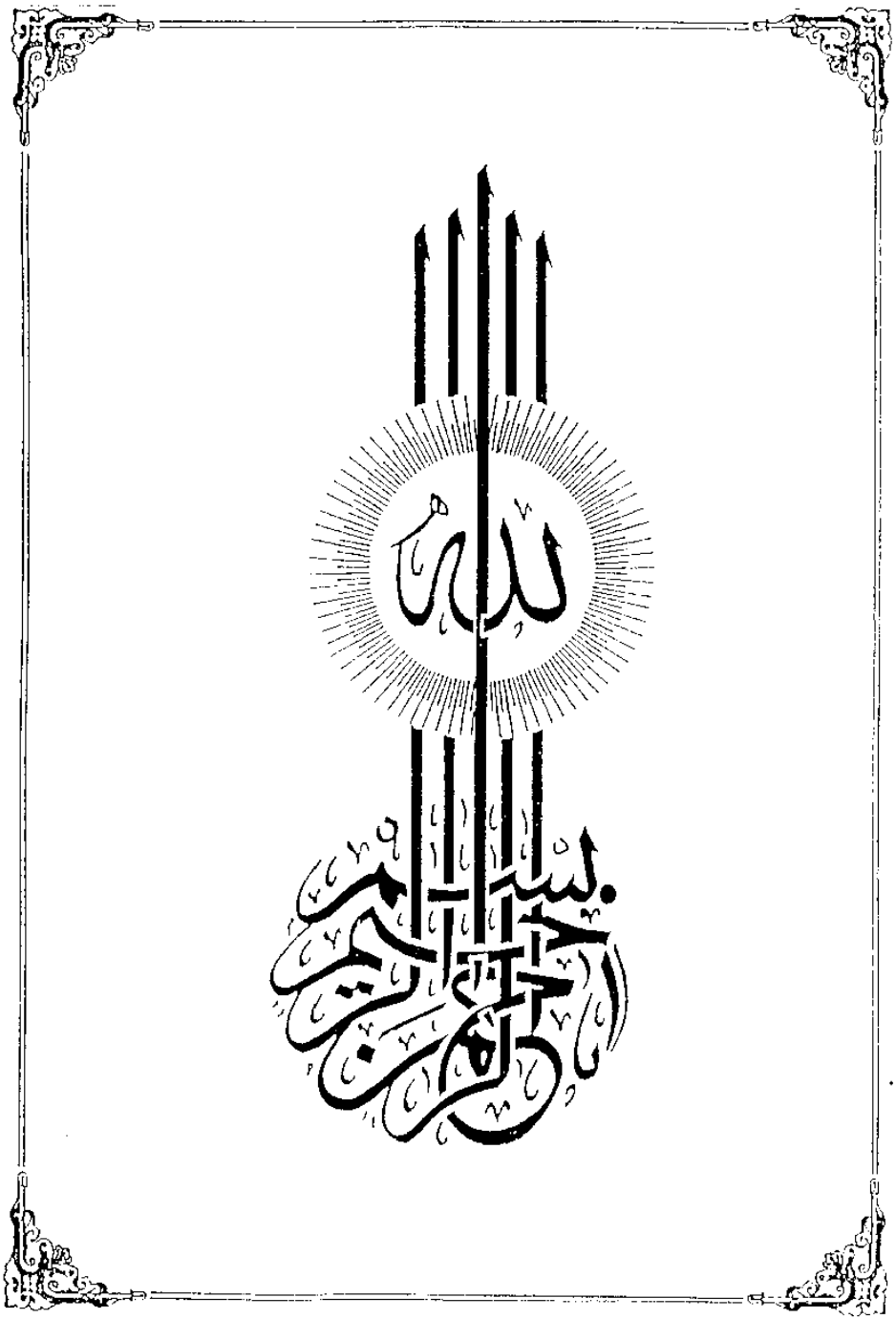

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INTRODUCTION AND AIM OF THE WORK

Left ventricular thrombi represent an important potentially catastrophic complication of acute myocardial infarction(1). Thrombus has been shown to occur early following an infarction and patients in this category are believed to be at a high risk for embolic complications(2,3).

Although many embolic events may be clinically silent particularly those in the spleen or kidneys(2,3), systemic emboli caused or contributed to death in 12-33 percent of autopsy patients with myocardial infarction(4). Embolic events, such as cerebrovascular accidents, mesenteric embolization, acute femoral and brachial artery occlusions, might complicate the care of patients convalescing from acute myocardial infarction(5).

In the past, studies of left ventricular thrombosis as a complication of acute myocardial infarction have largely been limited to autopsy series. They showed that a left ventricular thrombus is a common complication following acute myocardial infarction, its incidence being reported to be up to 68 percent(1, 6-9).

Since no particular non-invasive method to detect reliably a left ventricular thrombus has existed in the past, little information was available on the incidence of left ventricular thrombi in patients surviving acute myocardial infarction(10). The presence of a thrombus thus became clinically apparent only if an embolic event occurred or if detection was made by contrast ventriculography(11).

The advent of M-mode echocardiography was accompanied by the anticipation that ultrasound would provide a non-invasive diagnostic technique capable of detecting intracardiac thrombi. The ability of M-mode echocardiography to identify cardiac clots with an acceptable sensitivity and specificity, however was never established(12).

The failure of M-mode echocardiography to identify cardiac thrombi was primarily due to lack of spatial orientation inherent in this technique, which impaired the visualization of the cardiac apex, the site of most cardiac clots(13), and prevented the definition of a mass of echoes as a thrombus rather than a technical artifact.

Recently, cross-sectional or two-dimensional echocardiographic techniques have been developed that provide spatial orientation and enable the imaging of the entire left ventricle, including the cardiac apex(14-17). 2-D Echocardiography

also provides a safe non-invasive method for the diagnosis of ventricular thrombi with an estimated incidence of 17-40%(10,18,19).

The aim of the present work is to study the incidence, time and site of occurrence of left ventricular thrombi by two-dimensional echocardiography. Also to study their predisposing factors and possible complications in patients with acute myocardial infarction.

Review of Literature

PATHOGENESIS OF LEFT VENTRICULAR THROMBI

Left ventricular thrombosis is a common and early complication following acute myocardial infarction. The large majority of cases of left ventricular thrombosis develop in the first days after the onset of pain(20). Previous experimental studies have suggested that stasis of blood and endocardial changes play an important role in this context(21,22).

Since severe regional asynergy may be present on admission, especially in patients with an acute anterior myocardial infarction, stasis of blood might be present early following myocardial infarction(23-26). In addition, endocardial changes such as inflammation are seen early after the onset of symptoms(27).

Autopsy studies have shown that there are 3 patterns of left ventricular thrombi, small foci over the endocardial surface of full thickness infarction, large thrombi within infarcts showing aneurysmal dilatation and diffuse thrombi throughout the interstics of the whole ventricle. The first is related to endocardial damage and resultant inflammation, the second and third are related to local and general stasis (28).

Regarding the histopathology of the thrombus, the mural portion of the clot was found to be composed of lines of Zahn with platelet fibrin lamella and leukocyte margiration, while the tail portion, the part that embolize, consisted almost entirely of red blood cells held together by a delicate fibrin mesh(29).

Jordan et al.(1) studied the post mortem findings of 210 consecutive cases of acute fatal infarctions, in 110 of whom there were healed myocardial infarcts as well. In addition they studied 117 consecutive fatal cases from other causes, in which healed myocardial infarcts were found. In both groups the incidence of mural thrombi was found to be 33%(1).

In this study it was found that several factors are related to the development of mural thrombi in the left ventricle. They included the age of the infarction, its size, location, the presence or absence of congestive heart failure or myocardial hypertrophy(1).

The above factors were also found to be interdependant in their influence on the formation of mural thrombi in the left ventricle.

Regarding the age of the infarct, it was found that left ventricular mural thrombi occur more frequently with acute myocardial infarctions than with healed ones(1).

The size of the infarction is the 2nd determinant of the outcome of acute myocardial infarction. In order to determine the relationship of the size of myocardial infarction to the frequency of left ventricular mural thrombosis, they multiplied the length of each infarct by its width. The product was assumed to be the approximate area involved by the infarct. Using this method it appeared that as the total area involved by the infarct increases, so does the incidence of mural thrombi in the left ventricle(1).

Out of 240 cases in which the area of myocardial infarction was less than 40 sq cm,52 had mural thrombi (21.7%). On the other hand in the 87 cases with infarcts of 40 sq cm or more, 56 cases had left ventricular mural thrombi (64.4%)(1).

The location of the infarct in the left ventricle appeared to have a rather marked effect on the incidence of mural thrombi being more frequent in association with anterior wall infarcts.

This fact was also confirmed by more recent studies. In 1981 Asinger et al.(10) performed serial two-demensional echocardiographic studies in 70 consecutive patients, 35 had recent inferior infarcts none had a left ventricular thrombus. In the other 35 cases with recent anterior wall infarctions 12 had left ventricular thrombi.

There may be several reasons for the greater incidence of left ventricular mural thrombi in the cases with anterior wall myocardial infarctions. Patients with anterior infarcts are at a great risk for expansion, acute regional dilatation, and thinning of the infarct zone(30). Infarct expansion is a deterimental alteration, it leads to overall cardiac dilatation and may be a factor in cardiac rupture and late aneurysmal formation(31).

It is conceivable that anterior wall expansion is partly responsible for the increased tendency towards thrombosis because of stagnation of blood in an expanded region. In addition the local release of factors from the stretched and disrupted myocardium may promote thrombosis(32).

Since infarct size alone cannot fully explain the particular hazards of anterior infarction, it is believed that qualitative differences in the anterior myocardial

wall that are both intrinsic and extrinsic to the heart should be considered(32).

The anteroseptal myocardium, as defined by its blood supply, includes the apex of the heart. As the thinnest part of the left ventricular wall, the apex may be the most "stressed" by overall cardiac dilatation and therefore particularly susceptible to expansion, aneurysm and thrombus formation. The posterior wall of the heart is entirely covered by pericardium and to a certain extent buttressed by the spinal column and lungs(31).

Also a major part of the anteroseptal myocardium can expand freely into the right ventricular cavity and is less restricted by the pericardial sac or extrincic thoracic structures. Accordingly, if the anteroseptal wall of the left ventricle is softened and necrotic, it may be more susceptible to the changes in shape that can adversely affect global left ventricular function and predispose to regional low blood flow and thrombus formation(32).

Intrinsic tissue factors may also contribute to differences in the characters of infarcts. The conduction system is located primarily in the anteroseptal myocardium. In

contrast, myocardial chemoreceptors which are responsible for the parasympathetic vasodilatation causing hypotension and bradycardia after acute infarction (the Bezold-Jarisch reflex), appear to be distributed preferentially in the posterior wall of the left ventricle in dogs and probably also in human beings. This explains why enhanced vagal tone is more prominent in acute inferior infarction(33). Conversely in anterior wall infarction, the higher heart rates, blood pressure, and contractile stimulation may lead to preferential anterior wall expansion(32).

Local differences in coronary anatomy and physiology could also affect the quality of an infarct, by determining factors such as the magnitude of reperfusion after coronary occlusion or the extent of transmural necrosis. Indeed, the extent to which an infarct involves the full thickness of the left ventricular wall is another qualitative feature of acute infarction that independantly of size alone, influences the outcome. Rupture, expansion, aneurysm formation and thrombus deposition are almost exlusively limited to infarcts that are transmural(32).

Hochman et al. in 1984(27) found that there are two morphologically distinct types of left ventricular aneurysms that may behave differently in the clinical setting, particularly with regard to systemic emboli and ventricular arrhythmias.