EFFECT OF STREPTOKINASE ON EARLY COMPLICATIONS FOLLOWING ACUTE MYOCARDIAL INFARCTION

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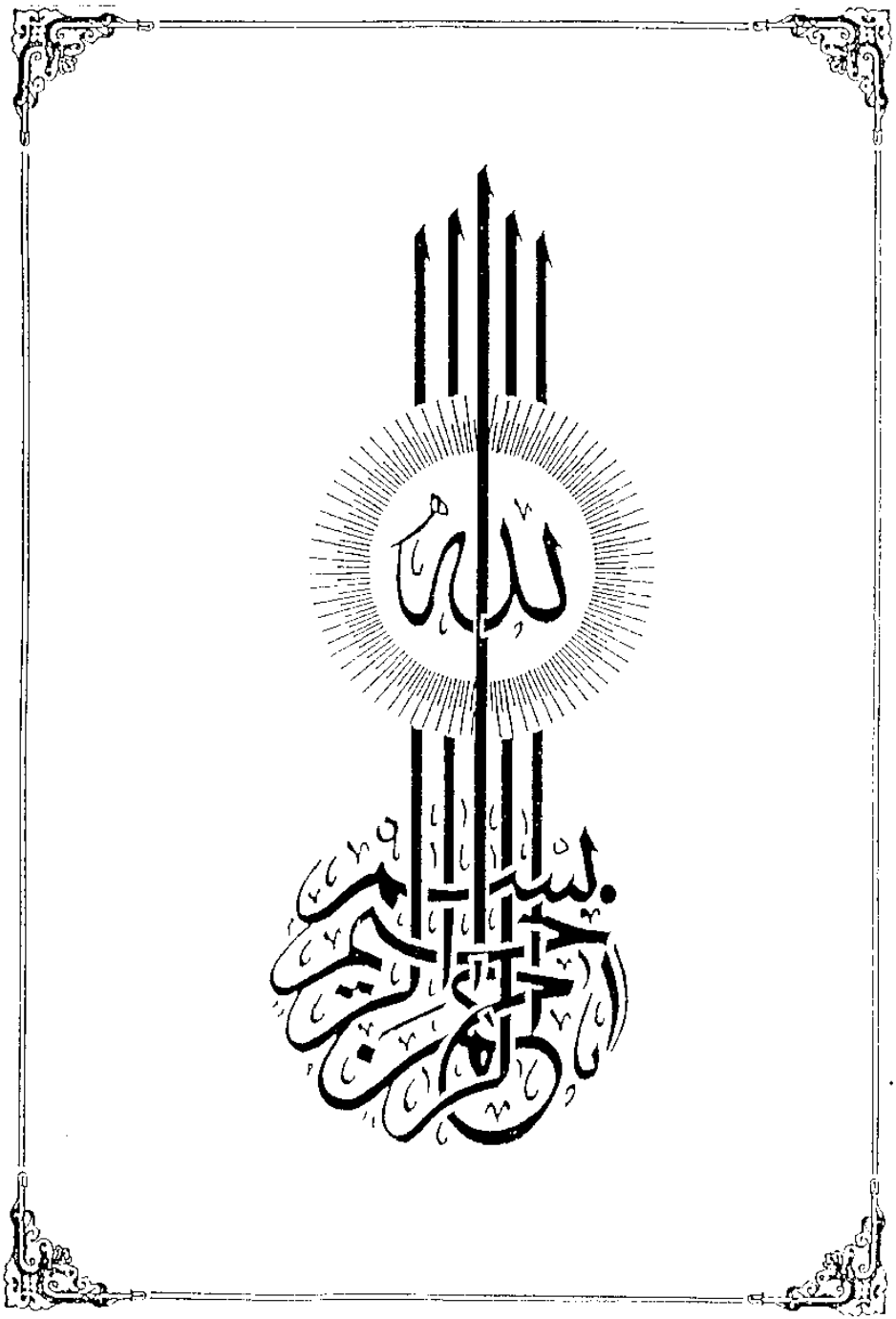

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INTRODUCTION

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- Thrombolysis in myocardial infarction was studied by (TIMI) Trial, Phase I. The study proved that, intravenous streptokinase produced reperfusion of totally occluded artery by 31%, when studied by angiography 90 minutes after the starting of the thrombolytic therapy.
- Another trial was done, called the Western Washington intravenous streptokinase in acute myocardial infarction (Kennedy, 1988), (Martin 1988), concerning the effect of thrombolytic therapy on ventricular function and size of infarction.
- It was found that overall ejection fraction was improved from 54% to 57% in treated patients with thrombolytic therapy (Simoons, 1985).
- Also, this study proved the long term benefit of intravenous streptokinase therapy on reducing the size of infarction (Serruys, 1986).
- GISSI trial found a significant 20% reduction of 21 day mortality in treated patients with thrombolytic therapy (GISSI, 1987).
- Previous studies did not show a clear-cut difference in short term complications following acute myocardial infarction between patients receiving thrombolytic therpay and those who did not.

AIM OF THE WORK

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To study the effect of streptokinase on early complications following acute myocardial infarction during hospital stay.

ACUTE MYOCARDIAL INFARCTION

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Definition:

An evolving acute myocardial infarction is said to be present when a patient has chest discomfort or other symptoms, usually but not always lasting longer than 20 minuites, believed to be due to myocardial ischemia, and when less than 4 to 6 hours has elapsed since the onset of symptoms.

- When the chest discomfort or other symptoms due to myocardial ischemia last longer than 4 to 6 hours, the infarction should be classified as completed myocardial infarction.

- Pathology of Acute Myocardial Infarction:

- ***The acute Coronary Event:***

Acute myocardial infarction is the end result of prolonged, unrelieved ischemia. It is caused by total interruption in blood supply to a segment of myocardium and is largely unrelated to myocardial demand. The cause of the abrupt interruption in blood supply is usually an acute disruptive event within an atherosclerotic coronary segment, that event is most often thrombosis over a complicated atheroma which had previously narrowed the lumen by 75 to 80 percent (Buja; 1987) (DeWood, 1980).

Plaque rupture, hemorrhage into the plaque, or erosion of the intima over the fibrous cap is the event which incites the thrombus formation, converting a critical narrowing into a total occlusion.

- ***Coronary Thrombosis:***

  Myocardial infarction may be divided into two major types: transmural infarcts, in which myocardial necrosis involves the full thickness of the ventricular wall, and subendocardial (non transmural) infarcts in which the necrosis involves the subendocardium, the intramural myocardium or both without extending all the way through the ventricular wall to the epicardium.

- In fatal acute transmural myocardial infarction more than 90 percent have associated coronary thrombosis. (Buja, 1987). Significantly fewer thrombi (less than 33 percent) are found in cases of either subendocardial infarction or sudden death.
- De wood et al evaluated patients in the first 4 hours and found 87 percent had thrombotic occlusion, whereas this percentage decreased to 65 percent when patients were examined 12 to 24 hours after onset. These data suggest that thrombi may undergo spontaneous thrombolysis, fragmentation, and distal embolization with infarct evolution, which would account for their deminished frequency in older infarcts (DeWood, 1980).

- ***Coronary Artery Spasm:***
- In the setting of acute myocardial infarction, there is evidence of increased production of vasodilating and vasoconstricting prostaglandins (Fox, 1984), (Friedrich, 1985). But it appears that the vasoconstricting activity of thromboxane A_2 predominates (Willerson, 1984) (Hirsh, 1987). Thus the presence of thromboxane A_2 or other vasoconstricting substances released by the aggregating platelets at the site of a coronary artery stenosis has the potential to initiate or maintain coronary artery constriction. It may be responsible for some observed cases of coronary artery spasm occuring with and perhaps contributing to the pathogenesis of acute myocardial infarction.
- In addition to causing acute myocardial infarction in rare patients with normal coronary arteries, coronary artery spasm may also play a broader role in patients with atherosclerotic coronary artery disease (Feld man, 1987), (Conti, 1985).
- It has been postulated that spasm may cause intimal damage that can initiate formation of an atherosclerotic plaque (Gertz, 1978), (Marzilli, 1980) Epicardial caronary artery spasm has been identified in patients with fixed atherosclerotic coronary artery stenosis before, during and after acute myocardial infarction (Bertrand, 1983). An

association between coronary artery spasm and coronary artery thrombosis has also been documented clinically (Vincent, 1983).

- ***Pathology of the myocardium:***

After several hours of acute interruption of blood flow, the myocardium undergoes irreversible injury. There are three type of irreversible cell injury that can be identified pathologically.

1. ***Coagulation Necrosis:***

It is the pattern of necrosis observed when blood flow is parmenantly interrupted, as occurs typically in the core of an infarct after a thrombotic occlusion. The newly formed granulation tissue replaces the necrotic cells over the course of the next several weeks, and finally by 4 to 6 weeks a largely healed infarct is present. The completness of healing at any given point in time is in part a function of the size of the infarct and its type: a large transmural infarct takes at least 6 weeks to heal, a small non transmural infarct may be healed in 3 to 4 weeks. (Mallory, 1939).

2. ***Contraction Band Necrosis:***

Is the type of cell death which occurs when a period of

lethal myocardial ischemia is followed by reperfusion. It may develop in a variety of circumstances in which myocardial blood flow is temporarily interrupted. This type of reperfusion necrosis may occur in patients with coronary spasm and may be seen on the margins of acute myocardial infarct which is predominantly coagulation necrosis, probably due to retrograde collateral blood flow. This pattern of necrosis can be produced experimentally by interruption flow for 40 minutes and then reperfusing the vessel (Reichenbach, 1968).

3. ***Myocytolysis:***

It occurs in focal nests of cells mainly on the border of infarcts or in the subendocardium. The ultimate fate of these damaged cells is not clear, but at least some of them die and are replaced by scar tissue.

- ***Site of Necrosis:***

Although infarct size is a major determinant of left ventricular function and mortality after acute myocardial infarction, site or location of the myocardial injury is also an important factor in determining outcome (Bulkley, 1981).

- Anterior wall infarcts which result from occlusion of the left anterior descending coronary artery tend to be large,

associated with greater left ventricular dysfunction, are more likely to cause permenant complete heart block, develop mural thrombosis and late aneurysms and carry a higher mortality.

Occlusionof left circumflex coronary artery causes infarction of the lateral wall of the left ventricle, infarction in this location are the smallest, least to produce haemodynamic complications except with a less common left dominant circulation, a left circumflex occlusion can lead to a more extensive posterior wall infarct.

- Occlusion of the right coronary artery typically causes an infarct of the posterior wall of the left ventricle and may lead to infarction of the right ventricle, associated with ischemia or infarction of the posteromedial papillary muscle.
- The relation of location of necrosis to outcome is also illustrated in the difference between transmural and non transmural infarcts (Freifeld, 1983).
- Transmural infarcts are most likely to be associated with infarct expansion, cardiac dilatation, cardiac rupture, mural thrombosis and late aneurysm formation (Hochman, 1983).

A preserved epicardial rim of myocardium which is present in the non transmural infarct appears to protect against the shape change of expansion and aneurysms and protects against rupture (Schuster, 1979).