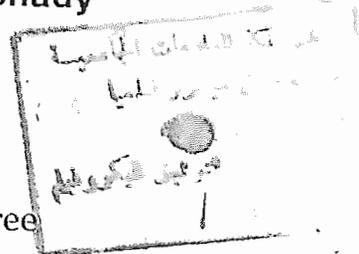


**"IN-HOSPITAL COURSE, MORBIDITY & MORTALITY,
OF ACUTE INFERIOR WALL MYOCARDIAL INFARCTION
IN DIABETIC AND NON-DIABETIC PATIENTS"**

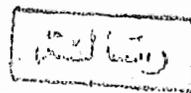
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**FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY**

1993

بسم الله الرحمن الرحيم

"وما أوتيتم من العلم الا قليلاً"

صدق الله العظيم

سورة الإسراء (٨٥)



**Thanks to the Almighty God,
who supported me throughout this work**

**Then thanks to my parents,
who I love and care for very much**

ACKNOWLEDGEMENT

I would like to express my deep gratitude to *Prof. Mamdouh El Ashry*, Professor of Cardiology, Ain Shams University for his continuous encouragement and kind guidance.

I am greatly honoured to express my deep gratitude to *Dr. Said Khalid*, Assistant Professor of Cardiology, Ain Shams University. He gave me much of his time and experience, keenly supervised my work and guided every step till it was completed.

I also wish to thank *Dr. Mohamed Gamal Abdel-Ber*, Lecturer of Cardiology, Ain Shams University, for his kind help and guidance.

Finally, I wish to thank all the members of the Cardiology Department, Ain Shams University, for all the help they offered.

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***INTRODUCTION
&
AIM OF THE WORK***

INTRODUCTION & AIM OF WORK

Acute inferior wall myocardial infarction is relatively benign infarction, with good prognosis and low mortality, when compared with acute anterior wall myocardial infarction, and it is usually associated with normal left ventricular hemodynamics (Hamby et al., 1974, Pfisteror et al., 1990).

Diabetes mellitus is known to accelerate coronary atherosclerosis, but evidences had accumulated for the existance of myocardial dysfunction in diabetic patients beyond that attributable to coronary artery obstruction (Kannel et al., 1974).

When the insultation of left ventricle, by acute inferior myocardial infarction, is summated to the subclinical insultation caused by diabetes mellitus the prognosis becomes worse than expected (Stone et al., 1989 and Jaffe et al., 1984).

The aim of the work is to evaluate the in-hospital course, morbidity and mortality, of acute inferior wall myocardial infarction in diabetic patients, and to unmask the bad effect of associated diabetes mellitus by comparing them to non-diabetic patients with acute inferior wall infarction.

*REVIEW
OF
LITERATURE*

DIABETES MELLITUS AS A RISK FACTOR FOR CORONARY HEART DISEASE :

Diabetes mellitus (D.M.) is a strong, independent risk factor for the development of coronary artery disease (C.A.D.). Numerous clinical and autopsy studies have demonstrated that the incidence of C.A.D. is higher in diabetics more than non diabetics (Kannel, 1985).

Glucose intolerance doubles the occurrence of coronary disease in men, and triples to quadruples the incidence in women, particularly prior to age 50 (Barrett - Connor, 1983).

A study for the status of the coronary arteries at necropsy in diabetes mellitus with onset after age 30 years was made in 1980 by Waller et al. The clinical and morphological observations were made in diabetic patients with and without clinical evidence of C.A.D. The average number of three major (right, left anterior descending, left circumflex) coronary arteries per patient narrowed >75 percent in cross sectional area by atherosclerotic plaques was identical in all the diabetic patients with and without C.A.D.

Yet the diabetic patients with C.A.D. had more severe narrowing of the three major coronary arteries than did the diabetic patients without C.A.D.

The amount of severe (>75 percent in cross sectional area) narrowing of the left main coronary artery was greater in the patients with D.M. than the non diabetic controls.

The similarity in the amount of coronary arterial narrowing was present irrespective of the age of onset, duration or the type of treatment recieved by the diabetic patients.

The precise reasons for the diabetic's increased susceptibility of coronary atherosclerosis are under continuing investigations, a variety of reasons may be blamed.

Diabetic patients have been found to have abnormal blood lipids including elevated very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL) and triglycerides with reduced levels of high density lipoproteins (HDL) (Kannel W, 1985). Diabetic individuals in general have higher serum cholestrol values. However, this observation is only partially confirmed by data from the Framingham study, which showed female but not male diabetic subject to have higher serum cholestrol levels than their nondiabetic peers. Diabetic men - both those who did and did not develop artherosclerotic cardiovascular disease had lower total serum cholestrol than non diabetic males. Diabetic women - both those who did and did not develop cardiovascular disease had

higher total serum cholesterol values than their non diabetic counterparts (Kannel W.B., 1979).

Several studies called attention to the inverse relation between HDL and C.A.D., and proposed that HDL might promote the clearance of cholesterol from the arterial wall (Miller et al., 1977, Miller and Miller, 1975). Since their report, numerous clinical and epidemiological studies have shown an inverse relation between CAD and HDL, where decreased levels of HDL have been associated with premature C.A.D. (Avogaro et al., 1980). The protective effect of HDL has been described as being due to the role of this lipoprotein in "reversed cholesterol transport" whereby excess cholesterol is taken up by the HDL and removed by the liver. Alternatively, HDL may reduce delivery of cellular cholesterol by impairing LDL binding to the cell surface (Carew et al., 1976).

Another mechanism relates to the role of HDL as a scavenger during other lipoproteins (Chylomicron and VLDL) hydrolysis, where by HDL availability would determine the amount and kind of remnants taken up by the macrophages of the arterial wall.

So, low levels of HDL in diabetics could result in impaired cholesterol removal or give risk to the accumulation of remnants after hydrolysis of triglycerides rich particles such as chylomicrons

and VLDL. This accumulation of remnants might promote atherosclerosis (Zilversmit et al., 1979).

Elevation of intermediate density lipoprotein (IDL) cholesterol concentration in diabetic patients might be responsible for the higher incidence of coronary and peripheral vascular disease.

Studies on the atherogenic effect of IDL on CAD demonstrated that IDL was closely related to the degree and in particular, the frequency of CAD, independent of LDL and HDL cholesterol. IDL concentration was significantly correlated with glucose intolerance in the total patients studied by (Tatani et al., 1981).

Serum of diabetic patients can stimulate vascular smooth muscle hyperplasia independent of the other factors including hyperglycemia, insulin and hyperlipidemia (Zilversmit et al., 1979).

Insulin even in small concentrations has been reported to promote proliferation of arterial smooth muscle and incorporation of glucose into lipids.

Also there are some ways by which diabetes may be implicated in producing endothelial injury. Diabetes may have impaired erythrocyte oxygen transport and hence may develop arterial injury on a hypoxic basis.

Another explanation is that hypoxic arterial damage could result from intracellular accumulation of sorbitol and the consequent increase in the cellular water (Steiner et al., 1981). The endothelial injury permits platelets to interact with collagen and start the following cycle:- The platelets discharge their stored granules and aggregate. The stored granules release their ADP and platelet derived growth factor which stimulates smooth muscle cell proliferation. ADP activate phospholipases that lead to the release of arachidonic acid which in turn is converted to prostaglandins and then to thromboxane. The thromboxane promotes smooth muscle contraction. Furthermore, thromboxane and ADP together with locally generated thrombin cause further platelets aggregation. Thrombin also causes polymerization of fibrinogen and the generation of a thrombus.

All of this is counter balanced by prostacyclin, as the most active site of prostacyclin production is the endothelial cell. One can understand why platelets don't aggregate on a healthy endothelium. However, when the endothelium is injured and platelets can interact with collagen, this balance tips towards thromboxane effect. This leads both to further endothelial damage and to the release of more of growth factor that stimulates smooth muscle proliferation.

Platelets from diabetics have been found to have an increased sensitivity to aggregating agents. They were also shown to produce more thromboxane, the amount produced being positively correlated to the patients fasting blood sugar (Steiner et al., 1981).

Numerous studies have demonstrated platelet function abnormality in D.M. and have demonstrated platelet hyperactivity may be involved in the pathogenesis of the vascular complications of D.M. ADP threshold for producing irreversible platelet aggregation was lower in patients with vascular complications and normal in those without vascular complications (Leone et al., 1974).

Plasma β -thromboglobulin levels have been evaluated as a measure of platelet release activity in the peripheral circulation, and found to be elevated in patients with D.M. compared to normal individuals.

There was no correlation between plasma levels of β -thromboglobulin and complications of D.M. suggesting that platelet function abnormalities may be related directly to D.M. rather than to complications (Burrows et al., 1978).

Platelets from diabetic patients are very active in synthesizing a prostaglandin E-like material when stimulated with ADP, epinephrine or collagen. Platelets of these patients also