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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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Dobutamine Induced ST- Segment Changes in patients with Acute Myocardial Infarction and its role in detection of Myocardial Ischemia, Viability and Ventricular Dysynergy

Thesis
Submitted to the Faculty of Medicine
Alexandria University

In partial fulfillment for the requirement of the degree of Master of cardiology and angiology

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Acknowledgment

I would like to express my deepest thanks and appreciation to Professor Doctor EL-SAYED SALAMA EL-SAKKA, Professor of Cardiology, Faculty Of Medicine, Alexandria University who has devoted much of his time in surveying the whole work.

I wish to express my profound gratitude and appreciation to Professor Doctor EBTHAG AHMED HAMDY, Professor Of Cardiology, Faculty Of Medicine, Alexandria University for her close endless supervision, valuable advice and great support.

My deepest thanks and appreciation to Dr. SAHER HAMDY AZAB, Lecture of Cardiology, Faculty Of Medicine, Alexandria University, for her continuous support and encouragement to facilitate the completion of this study.

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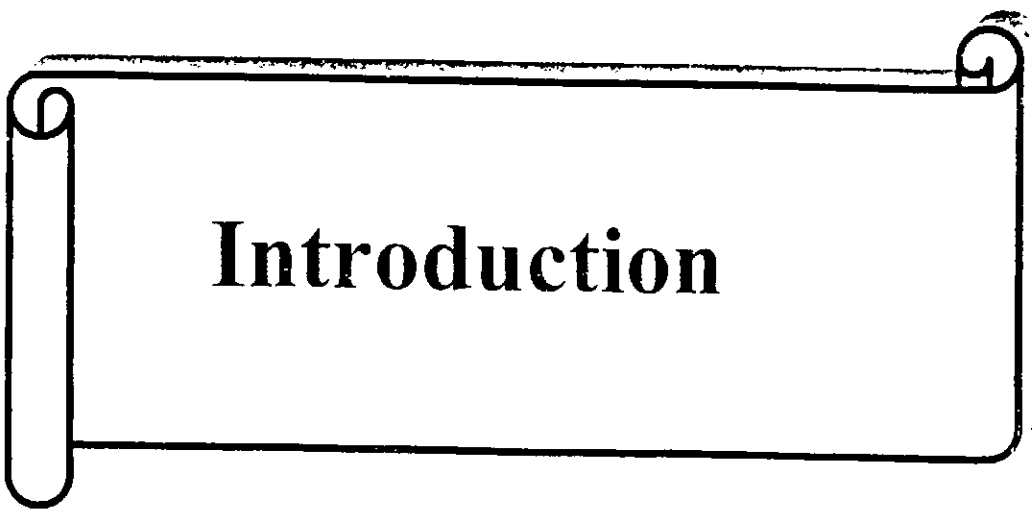

Introduction

Acute Myocardial Infarction

Despite impressive strides in diagnosis and management over the last three decades, acute myocardial infarction (AMI) continues to be a major public health problem in the industrial world. [1]

More than one million patients with suspected AMI are admitted yearly to coronary care units in the united states; in only 30 to 50 percent of patients the diagnosis is confirmed [2].

Although the death rate from AMI had declined by about 30 percent over the last decade, its development is still a fatal event in approximately one third of patients. [3]

About 50 percent of the deaths associated with AMI occur within 1 hour of the event and are attributed to arrhythmias, most often ventricular fibrillation. [4]

Limitations of current therapy

Despite the gratifying success of medical therapy for AMI, several observations indicate that considerable room for improvement exists.[5]

The short term mortality of patients with AMI, who receive aggressive reperfusion therapy as part of a randomized trial, is in the range of 6.5 percent,[6] whereas observational data bases, such as the national registry of MI suggest that the mortality rate in AMI patients not receiving reperfusion therapy is about 13 percent [7], in addition, the mortality rate from AMI in patients enrolled in randomized trials is considerably lower than that observed in patients who are excluded from such trials.

For example, the 18 months mortality in the 2180 patients excluded from the Danish Verapamil Infarction Trial II in 1983 (DAVIT II) was 25.6 percent, whereas it was only 13.9 percent in the placebo group enrolled in the trial.[8]

Variation has also been observed in the treatment patterns of certain population subgroups with AMI notably women and blacks. [9]

Although the unadjusted rate of thrombolytic use and referral for cardiac catheterization and angioplasty are lower and mortality rates are higher in women in AMI [10-11] ,gender differences are less apparent once adjustment is made for baseline variables such as comorbidities and age. [12]

Although black patients with AMI undergo fewer cardiac procedures such as cardiac catheterization than their white counter parts, [13] (even after adjustment for patients and hospital characteristic), they experience equivalent survival rates.[14]

Clinical picture of acute myocardial infarction

1) History:

A typical history includes retrosternal pain, which may either develop abruptly, or over a period of minutes to hours, the pain is described as constricting, crushing, oppressing or compressing.

The patient complains of something sitting on or squeezing the chest. It may also be described as burning discomfort. [15]

This pain is usually spreading to both sides of chest with predilection to the left side radiating to ulnar side of left arm with tingling to the left wrist, hand and fingers, in some patients the pain of AMI radiate to shoulders, neck, jaw and inter scapular region. [16]

The pain of Acute Myocardial Infarction may begin in other patients in the epigastrium and simulate a variety of abdominal disorders, nausea and vomiting occur in more than 50 percent of patients with transmural Myocardial Infarction with sever chest pain, this occurs commonly in patients with inferior MI than those with anterior MI. [17] In many cases the diagnosis is not made presumably because of atypical presentation. [18]

Predisposing factors :-

In one half of patients with AMI, a precipitating factor or prodromal symptoms can be identified. [19]

Although adequate controlled studies have not been carried out, evidence suggests that usually heavy exercise (particularly in fatigued or emotionally stressed patients) may play a role in precipitating AMI, such infarction could be the result of marked increases in myocardial oxygen consumption in the presence of significant coronary arterial narrowing. [20]

It has been suggested that unusually heavy exertion or mental stress such as that caused by anger [21] may trigger plaque disruption, leading to AMI. [22]

Patients with coronary disease who have been hospitalized for treatment of an acute coronary syndrome related event and who subsequently report a high level of stress in their life have an increased risk of rehospitalzation for cardio vascular events and also for "hard" events such as re- infarction. [23]

Accelerating angina and rest angina, two patterns of unstable angina, may culminate as AMI, surgical procedures associated with acute blood loss have been also been noted as precursors of AMI. [24]

Reduced myocardial perfusion secondary to hemorrhagic or septic shock and increased myocardial oxygen demands secondary to aortic stenosis [25], fever, tachycardia, and agitation can also be responsible for myocardial necrosis [26]

Other factors reported as predisposing to AMI include respiratory infection, hypoxemia, pulmonary embolism, hypoglycemia, administration of ergot preparations, use of cocaine, sympathomimetices, serum sickness, and allergy. [27]

Trauma may precipitate an AMI in one of two ways :- [28]

Myocardial contusion and hemorrhage into the myocardium may actually cause cell necrosis, or the injury may involve a coronary artery, causing occlusion of that vessel with resultant AMI. Neurological disturbance (transient ischemic attacks or strokes) may also precipitate AMI. [29]

2) Clinical findings:

The patient is typically anxious and restless, with coolness of extremities, temperature elevation in the range of 37 to 38 °c (a main specific response to tissue necrosis, within 24 to 48 hours of the onset of infarction and returns to normal by the seventh or eighth day following infarction). [30]

Alteration of pulse rate is non specific, the heart rate may vary from marked bradycardia to rapid regular or irregular tachy- cardia, depending on the underlying rhythm and the degree of left ventricular failure. [31]

Most commonly, the pulse is rapid and regular initially slowing, as the patient's pain and anxiety are relieved. [32]

Blood pressure elevation is common especially in the first hours of AMI, as a consequence of increased adrenergic discharge secondary to pain and agitation. [33]

A considerable fall in blood pressure occurs 6 hours after the onset of the attack in patients with massive infarction . [34]

Cardiac examination may reveal a presystolic pulsation synchronous with an audible fourth heart sound, reflecting a vigorous left atrial contraction filling a