

Evaluation of the Standard of Care Given to Patient with Acute Coronary  
Syndrome in General Hospital in Upper Egypt. A One Year Prospective  
Study

Thesis

*Submitted for Partial Fulfillment of Master Degree in  
Cardiology*

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**2011**

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مرضى متلازمة الشريان التاجي الحادة بالمستشفيات العامة  
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رسالة

توطئة للحصول على درجة الماجستير فى أمراض القلب والأوعية الدموية

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2011

### **abstract**

The diagnosis, management and treatment of the various forms of ACS which include STEMI, NSTEMI, and UA have been rapidly evolving in recent years.

Surveys and registries are an effective meaning of assessing the implementation of guidelines. Although the adherence to guidelines has been shown to be associated with improved outcomes, their implementation remains sub-optimal.

Examples for ACS registries include the GRACE study, the first and the second Euro Heart Survey for ACS.

In Egypt, it was observed the overwhelming increase in the number of ischemic heart disease patients and the change in the natural history of the disease affecting certain classes of patients particularly young age and women in addition to the aggressive nature in many patients. However, there are no documented registries to assess the criteria and parameters of ACS in Egyptian patients. Therefore, we aim for establishing a simple system for registering ACS in Egypt according to our resources. This could help to delineate for the first time the demographics of patients of ACS in Egypt.

This system could be a step for an Egyptian registry for ACS by applying it on different centers all over Egypt.

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## LIST OF ABBREVIATION

|                    |                                                                                             |
|--------------------|---------------------------------------------------------------------------------------------|
| <b>ACE</b>         | Angiotensin Converting Enzyme                                                               |
| <b>AHA</b>         | American Heart Association                                                                  |
| <b>ACC</b>         | American College of Cardiology                                                              |
| <b>ACTION</b>      | Acute Coronary Treatment & Intervention Outcomes Network                                    |
| <b>β-blockers</b>  | <b>Beta</b> -adrenergic <b>blockers</b>                                                     |
| <b>CABG</b>        | Coronary Artery Bypass Graft                                                                |
| <b>CAD</b>         | Coronary Artery Disease                                                                     |
| <b>CCU</b>         | Coronary Care Unit                                                                          |
| <b>CK</b>          | Creatine Kinase.                                                                            |
| <b>CK-MB</b>       | Creatine Kinase–MB                                                                          |
| <b>DM</b>          | Diabetes Mellitus                                                                           |
| <b>ECG</b>         | Electrocardiogram.                                                                          |
| <b>GP IIb/IIIa</b> | Glycoprotein IIb/IIIa Receptor Blocker                                                      |
| <b>HDL</b>         | High Density Lipoprotein.                                                                   |
| <b>HTN</b>         | Hypertension                                                                                |
| <b>HPS</b>         | Heart Protection Study                                                                      |
| <b>GRACE</b>       | Global Registry of Acute Coronary Events                                                    |
| <b>JNC VIII</b>    | The Joint National Committee on the detection, evaluation& treatment of high blood pressure |
| <b>LAD</b>         | Left Anterior Descending Artery                                                             |
| <b>LCx</b>         | Left Circumflex Artery                                                                      |
| <b>LDL</b>         | Low Density Lipoprotein                                                                     |
| <b>MACE</b>        | Major Adverse Cardiac Events                                                                |

### **LIST OF ABBREVIATION (CONT.)**

|               |                                                                      |
|---------------|----------------------------------------------------------------------|
| <b>MBF</b>    | Myocardial Blood Flow                                                |
| <b>MI</b>     | Myocardial Infarction.                                               |
| <b>NSTEMI</b> | Non- ST elevation myocardial infarction                              |
| <b>NO</b>     | Nitric oxide                                                         |
| <b>NRT</b>    | Nicotine replacement therapy                                         |
| <b>NTP</b>    | Nitroprusside                                                        |
| <b>OSIS</b>   | Organization for the Assessment of Strategies for Ischemic Syndromes |
| <b>PCI</b>    | Percutaneous Coronary Intervention.                                  |
| <b>PKC</b>    | Protein Kinase C                                                     |
| <b>RCTs</b>   | Randomized Controlled Trials                                         |
| <b>RCA</b>    | Right Coronary Artery.                                               |
| <b>STEMI</b>  | ST elevation myocardial infarction                                   |
| <b>TNF</b>    | Tumor Necrosis Factor.                                               |
| <b>UA</b>     | Unstable Angina                                                      |

## Introduction

Acute coronary syndrome encompasses a spectrum of coronary artery diseases, including unstable angina, ST-elevation myocardial infarction, and non-ST-elevation myocardial infarction. The term “acute coronary syndrome is useful because the initial presentation and early management of unstable angina, STEMI, and NSTEMI frequently are similar. They account for large numbers of emergency department visits and for more than 2 million annual admissions to intensive care and telemetry units across the U.S. When patients present with possible ACS, physicians must make quick assessments and treatment decisions because ACS progress rapidly (*Briege et al., 2004*).

Acute coronary syndromes represent the acute life threatening phase of coronary artery disease (*Hamm, 2001*).

Decisions regarding medical and interventional treatments are based on the presence or absence of ST segment elevation on the presenting electrocardiogram and abnormal elevations of myocardial enzymes (*Thom et al., 2006*).

Registries and surveys have the potential to define the gaps between evidence and practice as well as implementation of guidelines (*Bassand et al., 2005*).

The observed increase in the numbers of patients admitted with acute coronary syndromes (ACS), all over the world, as well as in the middle - east has raised many questions about the precipitating factors and etiological background.

Epidemiological data have shown the importance of traditional and other risk factors in the pathogenesis of atherosclerosis.

Recent studies, however suggest that the pattern of risk factor profile may differ according to age, gender as well as racial and geographical factors.

The study of such data in our region may demonstrate the proper risk profile of our patients and whether it will be different from the European standards whose guidelines are currently applied in Egypt as well as most of the middle - east countries. The availability of such data could be an appropriate database allowing the initiation of national guide lines.

### **Aim of the Work:**

Epidemiological study of patients admitted to Luxor International Hospital with the diagnosis of acute coronary syndrome through one year, and evaluation of care given to them within the hospital, and to compare the results with those of other international registries.

## **Pathophysiology of Acute Coronary Syndrome**

Theories of atherosclerotic plaque formation have changed such that cholesterol is no longer the lone culprit in CAD. Cellular signaling associated with inflammation is increasingly implicated in the initiation and progression of atherosclerotic plaques. Plaques have been shown to develop early in life and remain subclinical for years or decades, depending on the accelerating effects that risk factors and genetic predisposition to CAD will have on disease progression (*Libby et al., 2002*).

An etiologic approach has been proposed (*Libby et al., 2005*). Five pathophysiological processes may contribute to the development of ACS. These include:

1. Plaque rupture or erosion with superimposed thrombus (by far the most common cause of ACS).
2. Dynamic obstruction (i.e., coronary spasm of an epicardial artery, as in Prinzmetal angina or constriction of the small muscular coronary arteries).
3. Progressive mechanical obstruction.
4. Inflammation.
5. Secondary unstable angina related to increased myocardial oxygen demand or decreased supply (e.g., anemia).

Individual patients may have several of these processes coexisting as the cause of their episode of ACS. The major pathophysiologic mechanism is rupture or fissuring of an atheromatous plaque with superimposed thrombus (*Braunwald, 1998*).

- ***Plaque Formation***

Inflammatory processes participate quite heavily all the way from the initial stages of vascular endothelial cell perturbations to the later eventual rupture of the "vulnerable plaque" that leads to an acute coronary syndrome (ACS). This is final degenerative phase that is responsible for the great majority of devastating clinical events. Inflammation is increasingly implicated in plaque formation. At the cellular level, plaque accumulates in response to many signals that cause blood cells, such as monocytes, to adhere to the endothelium of the arterial lumen. Inflammatory responses to insults such as bacterial toxins, in addition to traditional risk factors, such as dyslipidemia, hypertension, hyperglycemia, and obesity, can initiate monocyte adherence (*Libby et al., 2005*).

***Endothelial Dysfunction***

The cause and progression of atherosclerosis are intimately related to the health of the inner arterial wall. The inner lining consists of a thin layer of endothelial cells (the endothelium) that

provides a smooth, protective surface. Endothelial cells prevent toxic, blood-borne substances from penetrating the smooth muscle of the artery. They also respond to changes in blood pressure and release substances into the cells of the smooth muscle that help change the muscle tone of the artery. Furthermore, endothelial cells secrete chemicals that provoke a protective response in the artery after an injury. This protective response includes signaling smooth muscle cells and white blood cells to congregate at the site of an injury (*Cominacini et al., 2001*).

Vascular endothelial cell dysfunction begins well before any morphologic manifestations of atherosclerosis are visible, and continues throughout the entire course, probably waxing and waning along the way (*Behrendt et al., 2002*).

We now know that in fact this dysfunction is characterized by several features typical of inflammation, including expression of numerous cellular adhesion molecules, like vascular cell adhesion molecule (VCAM-1), intercellular adhesion molecule (ICAM-1) and E-selectin. The abnormally functioning endothelial cells also release proinflammatory cytokines such as interleukin (IL-1 $\beta$ ), tumor necrosis factor (TNF- $\alpha$ ) and C - reactive protein (CRP). Furthermore, the dysfunctional endothelium produces less nitric oxide (NO), a leukocyte and platelet inhibitor and a vasodilator that also helps maintain vascular smooth muscle cells in a nonproliferative state. On the other hand, there are more