Cardiac Complications of Cerebrovascular Stroke

Essay submitted for partial fulfillment of the Master Degree in Neuropsychiatry

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2010

المضاعفات القلبية الناتجة عن السكتة الدماغية

رسالة مقدمة توطئة للحصول على درجة
الماجستير في الأمراض العصبية و النفسية

مقدمة من

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جامعة عين شمس

2010

Acknowledgment

First I would like to thank ***ALLAH*** *for his care and blessing.*

I would like to express my deep feelings of gratitude to Prof. ***Dr. Hany Mohamed Ameen Aref*** *Professor of Neuropsychiatry, Ain Shams University,who was akind great supportive teacher, I will be forever grateful to him .*

Words cannot express the depth of my gratitude to my ***Prof. Dr. Nahed Salah Eldeen Ahmad*** *Professor of Neuropsychiatry, Ain Shams University for her valuable suggestions, generous assistance, kind support and continuous encorgagment during this work.*

I would like to express my deepest gratitude to doctor ***Dr. Ali Soliman Ali Shalash*** Lecturer *of Neuropsychiatry,* ***Ain Shams University for his willing help, patience, guidance, and support throughout this work.***

Last but not least, I want to extend my genuine thanks and gratefulness to my ***family*** *especially my* ***father****, and my* ***friends*** *for their assistance and supporting me.*

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List of abbreviation

ABS	**Apical ballooning syndrome**
AF	**Atrial fibrillation**
AV	**Atrioventricular**
CAMP	**Cyclic adenosine monophosphate**
CHF	**Congestive heart failure**
CK	**Creatine kinase**
CTnT	**Cardiac troponin T**
DMV	**Dorsal motor nucleus of the vagus**
ECG	**Electrocardiogram**
EF	**Ejection fraction**
ELR	**Event-loop recording**
HRV	**Heart rate variability**
ICH	**Intracerebral hemorrhage**
IHD	**Ischemic heart disease**
IML	**Intermediolateral column**
LV	**left ventricular**
LVFP	**left ventricular filling pressure**
MCA	**Middle cerebral artery**
MI	**Myocardial infarction**
MRI	**Magnetic resonance imaging**
NA	**Nucleus ambiguus**

NT-proBNP	**N-terminal pro-brain natriuretic peptid e**
NTS	**Nucleus tractus solitarii**
SAH	**Sub arachnoid hemorrhage**
SCAE	**Serious cardiac adverse events**
SG	**Stellate ganglion**
TIA	**Transient ischemic attack**
VF	**ventricular fibrillation**
VT	**ventricular tachycardia**

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Aim of the work

The aim of this work is to discuss, cardiac changes after acute stroke and pathophysiology of these complications and its role in management.

Introduction

Neurocardiology has many dimensions, but it may be conceptualized as divided into 3 major categories: the heart's effects on the brain (eg, cardiac source embolic stroke), the brain's effects on the heart (eg, neurogenic heart disease), and neurocardiac syndromes (eg, Friedreich disease) ***(Samuels, 2007).***

Serious cardiac events and cardiac death after acute stroke may be caused by acute myocardial infarction, heart failure, ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation, and cardiac arrest ***(Meyer et al., 2004).***

Cardiac complications, including focal myocytolysis, electrocardiographic changes, arrhythmias and left ventricular wall motion abnormalities, frequently occur following stroke and contribute to worsen the prognosis. Their clinical spectrum seems to be related to the type of cerebrovascular disease and its localization. Thus, the incidence of arrhythmias and pulmonary edema is

significantly higher in **SAH** than in **ischemic stroke**, and the lesions in the right insular cortex are a major risk for complex arrhythmias and sudden death. Elevated plasma norepinephrine levels are frequently associated with these events and strongly suggest an underlying sympathetically mediated mechanism ***(Zeppellini et al., 2001).***

Cerebrovascular events are known to produce changes in the electrocardiogram (ECG). Whether or not these changes are actually reflective of myocardial damage has been the topic of much research. There are indications that these ECG changes result from an imbalance in the autonomic nervous system, resulting in, a relative excess of sympathetic activity ***(Kocan, 1998)***

Cardiac injury occurs frequently after stroke; and the most widely investigated form of neurocardiogenic injury is aneurysmal **SAH**. Echocardiography and screening for elevated troponin and B-type natriuretic peptide levels may help prognosticate and guide treatment of stroke. The priority should be treatment of the

underlying neurologic condition, even in patients with left ventricular dysfunction (***Kopelnik and Zaroff, 2006).***

In contrast to **SAH** patients, patients with **ischemic stroke** are more likely to have concomitant significant heart disease. Stroke in the region of insula, (especially the right) leads to decreased heart rate variability and to increased incidence of sudden death ***(Sadberk et al., 1999).***

Local cardiac catecholamines are responsible for the cardiac damage. Norepinephrine, an adrenergic agonist with both alpha and beta activities, is the neurotransmitter of the sympathetic nerves in the heart. Intense sympathetic stimulation results in accumulation of norepinephrine that becomes cardiotoxic. With continuous beta-adrenergic activation, the calcium channels may fail to close. The cells remain contracted until all energy stores are exhausted and die from intracellular metabolic derangement ***(Cechetto and Hachinski., 1997).***

Abnormalities of the ECG, chiefly repolarization changes including T-wave inversion, QT prolongation, and STsegment elevation or depression, have been reported

after stroke. These changes are most commonly reported after **SAH**. It has been shown that such changes are a more frequent consequence of hemispheric than of brain stem lesions ***(Yutaka et al., 2001).***

Signs of myocardial involvement are common in patients with acute cerebrovascular events. ST segment deviations, abnormal left ventricular function, increased N-terminal pro-brain natriuretic peptide (NT-proBNP), prolonged QT interval, and/or raised troponins are observed in up to one third of the patients. The huge majority of these findings are fully reversible. The changes may mimic myocardial infarction, but are not necessarily identical to coronary thrombosis ***(Jespersen and Fischer, 2008).***

In patients with **ischemic stroke** and **intracerebral hemorrhage**, these ECG abnormalities (and QT prolongation) most often represent preexisting coronary artery disease. The specificity of ECG changes to diagnose acute myocardial infarction is low in the acute phase of stroke ***(Khechinashvili and Asplund, 2002).***

Atrial fibrillation is a common arrhythmia and a potent risk factor for **stroke**. The prevalence of AF is approximately 1% in the general population; in those older than 65 years, it is 5.9%. The relative risk of **stroke** in patients with AF ranges from 5.6 to 17.6 of that observed in individuals without AF, depending on whether associated valvular disease is present. AF is present in 15% to 21% of the patients with **stroke** ***(Henrik et al., 1996).***

Screening consecutive patients with **ischemic stroke** with routine Holter monitoring will identify new atrial fibrillation/flutter in approximately one in 20 patients. Although based on limited data, extended duration of monitoring may improve the detection rate. Further research is required before definitive recommendations can be made ***(Joy et al., 2007).***