

Cardiac Complications of Cerebrovascular Stroke

Essay submitted for partial fulfillment of the Master Degree
in Neuropsychiatry

Presented by
Hany Mohamed Badr Eldeen
(M.B.B.Ch)

Supervisors

Prof. Dr. Hany Mohamed Amin Aref

Professor of Neuropsychiatry,
Faculty of medicine,
Ain Shams University

Prof. Dr. Nahed Salah Eldeen Ahmad

Professor of Neuropsychiatry,
Faculty of medicine,
Ain Shams University.

Dr. Ali Soliman Ali Shalash

Lecturer OF Neuropsychiatry
Faculty of Medicine
Ain Shams University

2010

المضاعفات القلبية الناتجة عن السكتة الدماغية

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

مقدمة من

الطبيب: هانى محمد بدر الدين

بكالوريوس الطب و الجراحة

تحت إشراف

ا.د/ هانى محمد امين عارف

أستاذ الأمراض العصبية و النفسية

جامعة عين شمس

ا.د/ ناهد صلاح الدين احمد

أستاذ الأمراض العصبية و النفسية

جامعة عين شمس

د/ علي سليمان علي شلش

مدرس الأمراض العصبية و النفسية

جامعة عين شمس

كلية الطب

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 a kind 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 encouragement 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.*

List of Contents

Title	Page
• List of Figures	i
• List of Tables	ii
• List of Abbreviations	iii
• Introduction	1
• Aim of the Work	6
• Review of Literature:	
○ Brain heart connections	7
○ Cardiac electrophysiological disturbances after stroke	
♦ ECG abnormalities.....	20
♦ Arrhythmias.....	37
♦ Mechanisms of arrhythmias and ECG abnormalities...	47
○ Cardiac structural abnormalities after stroke	50
○ Cardiac mortality and prognosis after stroke	66
○ Management of cardiac abnormalities after stroke	73
• Summary	87
• Discussion	93
• Recommendations	104
• References	106
• Arabic Summary	--

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 peptide**

NTS **Nucleus tractus solitarius**

SAH **Sub arachnoid hemorrhage**

SCAE **Serious cardiac adverse events**

SG **Stellate ganglion**

TIA **Transient ischemic attack**

VF **ventricular fibrillation**

VT **ventricular tachycardia**

List of figures

No	Title	Page
1	Basic circuitry of the medullary cardio respiratory reflexes	12
2	Neural control of circulatory system.....	14
3	Sympathetic heart–brain connections	19
4	CT (A) and ECG (B) on admission of a patient with SAH	24
5	Torsade de pointes.....	27
6	Broad, deeply inverted “cerebral” T waves	28
7	Electrocardiogram from a patient with ICH.....	30
8	Inverted, symmetric T waves are seen on the ECG of a patient with SAH	31
9	A; The CT scan of the head shows acute hemorrhage...	33
	B; The ECG reveals sinus tachycardia with ST-elevation	34
	C; Electrocardiography in a patient with SAH.....	35
10	A prominent U wave	36
11	Ventricular tachycardia.....	39
12	Atrial fibrillation	41
13	Brain MRI map of cerebral infarction sites associated with cardiac troponin elevation	55

14	Daily median B –type natriuretic peptide (BNP) levels in 31 SAH patients	58
15	Section of myocardium of left ventricle showing focal area of myocytolysis	60
16	The neurocardiac lesion: Gross specimen of 30 years old male patient with an acute fresh endocardial hemorrhage	62
17	A; Cardiac MRI of a patient with takotsubo syndrome B; Left ventriculography in a patient with apical ballooning syndrome	63
18	Atrial flutter	70
19	Possible therapeutic approaches aimed to prevent neurocardiac damage.....	84
20	Follow up electrocardiogram in patient with ICH.....	85
21	The ECG shows resolution of ST-elevation	86

List of tables

No	Title	Page
1	Rates of ECG abnormalities and arrhythmias following stroke	46
2	Risk factors that may need continuous heart monitoring after a stroke	73
3	Results of cardiac investigations of stroke patients of different types.....	101

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*).