Critical illness induced acute left ventricular myocardial dysfunction

(Stress Related Cardiomyopathy)

Essay

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List of Abbreviations

ACS: ACUTE CRONARY SYNDROME

AHA: AMIRICAN HEART ASSOCIATION

ALVD: ACUTE LEFT VENTRICULAR DYSFUNCTION

AMI: ANTERIOR MYOCARDIAL INFARCTION

AP: ACTION POTENTIAL

AR: ADRENERGIC RECEPTOR

ARVC: ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

ATP: ADINOSINETRIPHOSPHATE

AV: ATRIOVENTRICULAR

BNP: BRAIN NATRIURETIC PEPTIDE

cAMP: CYCLIC ADENOSINE MONOPHOSPHATE

CMR: Cardiac Magnetic Resonance

CNS: CENTRAL NERVOUS SYSTEM

CPVT: CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA

CRP: C REACTIVE PROTIEN

CVA: CARDIO VASCULAR SYSTEM

DCM: DILATED CARDIOMYOPATHY

ECG: ELECTROCARDIOGRAM

eNOS: ENDOTHELIAL NITRIC OXIDE SYNTHASE

EPI: EPINEPHRINE

ESCL EUROPIAN SOCIATY OF CARDIOLOGY

FDG-PET: FLUORODEOXYGLUCOSE POSITRON EMISSION TOMOGRAPHY

GIT: GASTROINTESTINAL TRACT

HCM: HYPERTROPHIC CARDIOMYOPATHY

IABP: INTRAAORTIC BALLOON COUNTERPULSATION

ICH: INTRACRANIAL HAEMORRHAGE

ICU: INTENSIVE CARE UNIT

IDDM: INSULIN DEPENDENT DIABETIS

IL: INTERLEUKIN

ISFC: THE INTERNATIONAL SOCIETY AND FEDERATION OF CARDIOLOGY

LAD: LEFT ANTERIOR DESCENDING ARTERY

LBB: LEFT BUNDLE BRANCH

LEG: LATE GADOLINIUM ENHANCEMENT

LQTS: LONG QT SYNDROME

LV: LEFT VENTRICLE

LVEF: LEFT VENTRICLE EJECTION FRACTION

LVNC: LEFT VENTRICULAR NON-COMPACTION

LVOT: LEFT VENTICULAR OUT FLOW

MI: MYOCARDIAL INFARCTION

NE: NOREPINEPHRINE

NO: NITRIC OXIDE

NOS: NITRIC OXIDE SYNTHASE

NSC: NEUROGENIC STRESS CARDIOMYOPATHY

ONOO-: PEROXYNITRITE

PCI: PERCUTANEOUS CORONARY INTERVENTION

RBB: RIGHT BUNDLE BRANCH

RCM: RESTRICTIVE CARDIOMYOPATHY

SA: SINOATRIAL

SAH: SUBARACHINOID HAEMORRHAGE

SAM: SYSTOLIC ANTERIOR MOTION

SPET: SINGLE-PHOTON EMISSION TOMOGRAPHY

SQTS: SHORT QT SYNDROME

SRC: STRESS RELATED CARDIOMYOPATHY

STEMI: ST ELEVATION MYOCARDIAL INFARCTION

SUNDS: SUDDEN UNEXPLAINED NOCTURNAL DEATH SYNDROME

TC: TAKOTSUBO CARDIOMYOPATHY

TCM: TAKOTSUBO CARDIOMYOPATHY

TIMI: THROMBOLYSIS IN MYOCARDIAL INFARCTION

TTC: TACOTSUBO CARDIOMYOPATHY

TTP: THROMBOTIC THROMBOCYTOPENIC PURPERA

TUNEL: TERMINAL DEOXYNUCLEOTIDYL TRANSFERASE-MEDIATED NICK END-LABELING

UCP: UNCOUPLING PROTEINS

WHO: WORLD HEALTH ORGANIZATION

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INTRODUCTION

Acute left ventricular (LV) dysfunction is common in the critical care setting and more frequently affects the elderly and patients with comorbidities. Because of increased mortality and the potential for significant improvement with early revascularization, the practitioner must first consider acute coronary syndrome. However, variants of stress cardiomyopathy may be more prevalent in ICU settings than previously recognized.

Early diagnosis is important to direct treatment of complications of stress cardiomyopathy, such as dynamic LV outflow tract obstruction, heart failure, and arrhythmias. Global LV dysfunction occurs in the critically ill because of the cardio-depressant effect of inflammatory mediators and endotoxins in septic shock as well as direct catecholamine toxicity. Tachycardia, hypertension, and severe metabolic abnormalities can independently cause global LV dysfunction, which typically improves with addressing the precipitating factor. **(Chockalingam, 2010)**

Routine troponin testing may help early detection of cardiac injury and biomarkers could have prognostic value independent of prior cardiac disease. Echocardiography is ideally suited to quantify LV dysfunction and determine its most likely cause. LV dysfunction suggests a worse prognosis, but with appropriate therapy outcomes can be optimized. **(Samuels, et al. 2007)**

Neurocardiology has many dimensions, namely divided in three categories: the heart's effects on the brain (i.e., embolic stroke); the brain's effects on the heart (i.e., neurogenic heart disease); and neurocardiac syndromes, such as Friedreich disease. The present review will focus on the nervous system's capacity to injure the heart. The relationship between the brain and the heart, i.e., the brain-heart connection, is central to maintain normal cardiovascular function. This relationship concerns the central and autonomic nervous systems, and their impairment can adversely affect cardiovascular system and induce stress-related cardiomyopathy (SRC). Even if it is unclear whether myocardial adrenergic stimulation is the only pathophysiological

mechanism associated with SRC, enhanced sympathetic tone inducing endogenous catecholamine's stimulation of the myocardium was always reported. **(Samuels, et al. 2007)**

The first description of suspected SRC was reported by W.B. Cannon in 1942 cited by Engel et al. who published a paper entitled "Voodoo death," which reported anecdotal experiences of death from fright. This author postulated that death can be caused by an intense action of the sympathico-adrenal system. In 1971, Engel et al. collected more than 100 accounts from the lay press of sudden death attributed to stress associated with disruptive life events and provided a window into the world of neurovisceral disease (i.e., psychosomatic illness). It is now widely admitted that this autonomic storm, which results from a life-threatening stressor, can be observed in the four following situations that induce left ventricle (LV) dysfunction:

- Takotsubo cardiomyopathy or apical ballooning syndrome.
- Acute LV dysfunction associated with subarachnoid haemorrhage.
- Acute LV dysfunction associated with pheochromocytoma and exogenous catecholamine.
- Acute LV dysfunction in the critically ill patients (severe sepsis, post cardiac resuscitation, post tachycardia...). **(Bybee, 2004, Prasad, 2008)**

Cardiac toxicity was mediated more by catecholamines released directly into the heart via neural connection than by those reaching the heart via the bloodstream. The mechanisms underlying the association between this generalized autonomic storm secondary to a life-threatening stress and myocardial toxicity are widely discussed. Takotsubo cardiomyopathy has been reported all over the world and has been acknowledged by the American Heart Association as a form of reversible cardiomyopathy. **(Kim, 2010)**

Chapter 1

Anatomical and physiological review of cardiovascular system

- Anatomical review of cardiovascular system:
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  - Structure of the cardiac myocyte.
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