Arrhythmias Following Acute Myocardial Infarction

Essay

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in General Intensive Care

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Abstract

Introduction: Clinical diagnosis of acute myocardial infarction (AMI) is defined as a rise in cardiac biomarker values [preferably cardiac troponin (cTn)] together with at least one of the following: symptoms of ischemia, electrocardiogram (ECG) changes indicative of new ischemia (new ST-T changes or new left bundle branch block), development of pathologic Q waves in the ECG, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Aims: The aim of the essay is to discuss arrhythmias following acute myocardial infarction, their pathophysiology, diagnosis and management.

Summary: The indications for treatment of cardiac arrhythmia may be considered in the following situations; (1) when the arrhythmia produces immediate deterioration in cardiovascular function (as in VF and VT and sometimes in AF) or (2) when the arrhythmia gives no immediate deterioration in cardiac function, but when its persistent presence may be expected to give rise to such deterioration or (3) when the arrhythmia carries a risk of giving rise to a subsequent more serious arrhythmia.

Conclusion: Patients who present to the emergency department with an AMI will usually be diagnosed and treated before admission to the ICU. Treatment includes; oxygen supplementation if oxygen saturation is less than 90%, morphine for analgesia, beta blockers, nitrates, aspirin, inhibitors of the platelet p2y12 receptor, anticoagulation therapy and PCI if accessible or fibrinolysis.

Keywords: Arrhythmias, Acute Myocardial Infarction, Pathophysiology

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List of Abbreviations

ACC	American College of Cardiology
ACCF	American College of Cardiology Foundation
ACE	Angiotensin-converting enzyme
ACS	Acute coronary syndrome
AF	Atrial fibrillation
AHA	American heart association
AMI	Acute myocardial infarction
apoB	Apolipoproteins B
ARB	Angiotensin receptor blocker
ARP	Absolute refractory period
ATP	Adenosine triphosphate
AV Node	Atrioventricular node
BMI	Body mass index
BNP	B-type natriuretic peptides
Ca2+	Calcium ion
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
CCUs	Cardiac care units
CHF	Congestive heart failure
CK-MB	Creatine kinase MB isoenzyme
CRP	C-reactive protein
CRTD	Cardiac resynchronization therapy device
CRTP	Cardiac resynchronization therapy Pacemaker
CTA	Computed tomography angiography

cTn	Cardiac troponin
cx	Circumflex arteries
ECF	Extracellular fluid
ECG	Electrocardiograph
EF	Ejection fraction
EPS	Electrophysiological studies
ERP	Effective refractory period
ESC	European Society of Cardiology
ESR	Erythrocyte sedimentation rate
hFABP	Heart-type fatty acid binding protein
ICD	Implantable cardioverter-defibrillator
ICF	Intracellular fluid
ICU	Intensive care unit
LAD	Left anterior descending artery
LBBB	Left bundle branch block
LDH	Lactate dehydrogenase
LMWH	Low-molecular-weight heparin
LV	Left ventricule
LVEDP	Left ventricular end-diastolic pressure.
MR	Mitral regurgitation
MRA	Magnetic resonance angiography
Na+	Sodium ion
NPO	Nil per os
NSTEMI	Non ST elevation myocardial infarction
NSVT	Non-sustained ventricular tachycardia
PaO2	Arterial oxygen tension

PCI	Percutaneous coronary intervention
PCWP	Pulmonary capillary wedge pressure
PSVT	Paroxysmal supraventricular tachycardia
PVCs	Premature ventriculr complexes
PVO2	Venous oxygen tension
RAAS	Renin-angiotensin-aldosterone system
RBBB	Right bundle branch block
RCA	Right coronary artery
RMP	Resting membrane potential
RRP	Relative refractory period
RV	Right ventricule
RWMAs	Regional wall motion abnormalities
SA Node	Sinoatrial node
SBP	Systolic blood pressure
STEMI	ST elevation myocardial infarction
SVT	Supraventricular tachycardia
TMP	Transmembrane potential
TNK	Tenecteplase
UFH	Unfractionated heparin
URL	Upper reference limit
VF	Ventricular fibrillation
VT	Ventricular tachycardia
WHF	World heart federation

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Introduction

Clinical diagnosis of acute myocardial infarction (AMI) is defined as a rise in cardiac biomarker values [preferably cardiac troponin (cTn)] together with at least one of the following: symptoms of ischemia, electrocardiogram (ECG) changes indicative of new ischemia (new ST-T changes or new left bundle branch block), development of pathologic Q waves in the ECG, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality ***(Thygesen et al., 2012)***.

In-hospital mortality after acute myocardial infarction is caused primarily by a number of complications that can be broadly classified into: *mechanical*, *ischemic*, *arrhythmic*, *embolic* or *inflammatory* complications ***(Adusumalli and Yeh, 2014)***.

About 90% of patients who have AMI develop some form of cardiac arrhythmia during or immediately after the event. In 25% of patients, such rhythm abnormalities manifest within the first 24 hours. In this group of patients, the risk of serious arrhythmias, such as ventricular fibrillation, is greatest in the first hour and declines thereafter. Coronary artery reperfusion due to thrombolytic therapy may also cause some arrhythmias ***(Shah et al., 2014)***.

Arrhythmias in the setting of AMI may be due to reentry, abnormal automaticity, or conduction block. These mechanisms are modulated by ischemia, LV failure, and variations in autonomic tone. They can be classified into: (a) *Ventricular tachycardias (VT)* which encompass: Ventricular fibrillation, ventricular tachycardia, and accelerated idioventricular rhythm. (b) *Supraventricular tachycardias (SVT*) which encompass: Sinus tachycardia, atrial fibrillation or flutter, paroxysmal SVT, and non-paroxysmal junctional tachycardia. (c) *Bradyarrhythmias* which encompass: Sinus bradyarrhythmia, junctional escape, and high degree AV block. (d) *Reperfusion arrhythmia* ***(Adusumalli and Yeh, 2014)***.

Arrhythmias are a major dilemma confronting the intensivist. They represent a major source of morbidity, and they lengthen hospital stay. They often lead to significant hemodynamic compromise because of the presence of multisystem disease with potentially life threatening consequences. The aim of arrhythmia treatment is to control the ventricular rate, restore sinus rhythm and to minimize complications if sinus rhythm cannot be restored ***(Mega and Morrow, 2015).***

Aim of the Essay

The aim of the essay is to discuss arrhythmias following acute myocardial infarction, their pathophysiology, diagnosis and management.

Cardiac Physiology and Electricity

Coronary blood flow:

The coronary circulation is unique in that the heart is responsible for generating the arterial pressure that is required to perfuse the systemic circulation and yet, at the same time, has its own perfusion impeded during the systolic portion of the cardiac cycle. The normal coronary blood flow in the resting human being is about 225 ml/min, which is about 4 to 5 percent of the total cardiac output ***(Canty Jr. and Duncker, 2014).***

Because myocardial contraction is closely connected to coronary flow and oxygen delivery, the balance between oxygen supply and demand is a critical determinant of the normal beat-to-beat function of the heart. When this relation is acutely disrupted by diseases affecting coronary blood flow the resulting imbalance can immediately precipitate a vicious cycle, whereby ischemia-induced contractile dysfunction precipitates hypotension and further myocardial ischemia. Thus knowledge of the regulation of coronary blood flow, determinants of myocardial oxygen consumption, and the relation between ischemia and contraction are essential for understanding the pathophysiologic basis and management of many cardiovascular disorders ***(Canty Jr. and Duncker, 2014).***