

Post Cardiac Arrest Syndrome

Essay

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Intensive Care Medicine*

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْحَكِيمُ
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Abbreviation

ACS: acute coronary syndrome

APACHE :Acute Physiology and Chronic Health
Evaluation

BLS: Basic Life Support.

CBF :cerebral blood flow

CMRO₂:cerebral metabolic rate of oxygen consumption

CPP : cerebral perfusion pressure

DO₂:oxygen delivery

EEG: electroencephalography

FPRs :high false-positive rates

HACA: hypothermia after cardiac arrest study

ICP :intracranial pressure

MAP : mean arterial blood pressure

NOS : inducible nitricoxide synthase

NRCPR : **National Registry of Cardiopulmonary
Resuscitation**

OHCA: Out Of Hospital Cardiac Arrest

PCI: primary percutaneous coronary intervention

ROSC: rapid occurrence of spontaneous circulation

ScvO₂: systemic mixed venous oxygen .

SSEPs: somatosensory-evoked potentials

VF: ventricular fibrillation

Introduction

Introduction

There has been increasing recognition that the care of patients after return of spontaneous circulation (ROSC) has been achieved plays an important role in determining patient outcome. This is reflected by the addition of post cardiac arrest care as a link to the ‘chain of survival’ (*Nolan et al.,2006*).

The International Liaison Committee for Resuscitation (*Nolan et al.,2008*) and the Intensive Care Society have produced guidelines and clinical standards for the management of patients during the post cardiac arrest phase of care (*Nolan, 2008*).

The concept of a post cardiac arrest syndrome was first described by Negovsky, a Russian pathophysiologist in 1972. High mortality seen in cardiac arrest is a result of the pathology that caused the cardiac arrest compounded by global ischaemia and subsequent reperfusion injury. The theories describe the release of pro-inflammatory cytokines (IL-1 β , IL-8, TNF α) leading to a systemic inflammatory response and ‘sepsis-like syndrome’ following resuscitation (*Adrie and Laurent 2002*).

In 2005, the chain was updated to include post cardiac arrest care in response to growing evidence that treatments in this phase have a significant influence on overall outcomes . The concept of care bundles, popularised by the ‘Surviving Sepsis’ campaign has been applied to the management of postresuscitation care. A combination of protocol-guided therapeutic hypothermia, early revascularisation and glycaemic control have been associated with improved process and clinical outcomes in this population of patients (*Sunde et al.,2006*).

The guidelines recommend that hypothermia is induced as soon as possible after cardiac arrest and that a core temperature of 32-34°C is maintained for at least 12-24 hours. Rewarming should be controlled to ensure the rewarming rate does not exceed 0.25-0.5°C per hour. Hyperthermia during rewarming should be aggressively treated (*Arrich., 2007*).

The ability to predict the likely neurological outcome and prognosis of a cardiac arrest survivor following admission to critical care is important. Up to two thirds of Out Of Hospital Cardiac Arrest(OHCA) patients die in intensive care as a consequence of poor neurological recovery and a quarter of those following

in-hospital arrest. A variety of clinical, electrophysiological and laboratory tests have been evaluated as predictors of outcome from 24 hours after cardiac arrest, but few centres have the facilities to routinely perform many of the electrophysiological or laboratory tests. Reliable tests predicting outcome would help target aggressive treatment to patients likely to have a good outcome, while allowing early palliation and withdrawal of life-sustaining treatments in cases where continuing care is futile(*Yeung et al.,2010*).

The development of post-cardiac arrest syndrome is common in initial survivors of cardiac arrest. Postresuscitation care has rightly become part of the chain of survival given the growing evidence demonstrating that the care received during this phase of resuscitation has a significant influence on final patient outcomes. The development of postresuscitation care bundles which include protocols for therapeutic hypothermia, primary percutaneous coronary intervention, control of blood sugar and a systematic approach to prognostication, is likely to play a significant part in improving process and patient (*Yeung et al.,2010*).

Aim Of The Work

AIM OF THE ESSAY

Raising attention to accurate post cardiac arrest care, systematic approach to prognostication and assessment of outcome is likely to play a significant part in improving an important part of the chain of survival process to lower the morbidity and mortality in the post cardiac arrest interval.

Review Of Littrature

Pathophysiology

Clinically, the post cardiac arrest syndrome presents with neurological dysfunction, cardiovascular instability and multi-organ dysfunction.

The brain is uniquely susceptible to ischaemia and reperfusion injury. Brain injury is the result of complex interactions of excitotoxicity, disrupted calcium homeostasis, formation of free radicals, protease cascades and combination of apoptosis and neuronal necrosis(*Lemiale et al., 2008*). There is some evidence to suggest that hyperoxia during the reperfusion stages can lead to increased free radical formation and mitochondrial oxidative injury, thereby exacerbating neuronal injury (*Balan et al., 2006*).

Heart rate and blood pressure can be labile immediately post ROSC and a normal or elevated reading can be a result of catecholamine release and can mask underlying myocardial dysfunction. Post-cardiac arrest myocardial dysfunction is a result of myocardial stunning and can be seen without evidence of reduced coronary blood flow or infarction. Increased levels of myocardial inflammatory cytokines (IL-8) and inducible nitric oxide synthase (iNOS) have been implicated in