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Review

Post cardiac arrest syndrome☆

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ABSTRACT

Background: Resuscitation from cardiac arrest with global ischemia restores spontaneous circulation in some patients; however, survival depends on many factors associated with post cardiac arrest syndrome. During the last ten years, the understanding and control of these factors have improved the prognosis in a subgroup of patients.
Objective: To describe the pathophysiology and current management of the post cardiac arrest syndrome (PCAS).
Methodology: Narrative review of the literature using Medline via PubMed and Clinical Trials, using the terms MeSH cardiac arrest – Cardiopulmonary Resuscitation and (no term MeSH) Post cardiac arrest syndrome.
Results: Clinical trials have established a set of management protocols and guidelines based on therapeutic objectives with survival rates exceeding 50% of the cardiac arrest victims.
Conclusions: The management of this syndrome has actually strengthened the last link in the survival chain by standardizing the evaluation and selection of cardiac arrest victims via a therapeutic hypothermia protocol and early percutaneous coronary intervention.

Síndrome posparo cardiaco

Palabras clave:
Paro cardiaco
Resucitación cardiopulmonar
Intervención coronaria percutánea
Hipotermia inducida
Circulación sanguínea

RESUMEN

Antecedentes: La reanimación en el paro cardiaco con isquemia global logra restablecer la circulación espontánea en algunos pacientes; sin embargo, la sobrevida depende de muchos factores que explican el síndrome posparo cardiaco. El entendimiento y el control de estos factores durante la última década han logrado mejorar el pronóstico en un subgrupo de pacientes.
Objetivo: Describir la fisiopatología y el manejo actual del síndrome posparo cardiaco.
Metodología: Revisión narrativa de la literatura a través de las bases electrónicas de Medline vía PubMed y Ensayos Clínicos usando los términos MeSH *Cardiac arrest – Cardiopulmonary Resuscitation* y (el término no MeSH) *Post cardiac arrest syndrome*.

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Resultados: Los estudios clínicos han establecido una serie de protocolos y guías de manejo basadas en objetivos terapéuticos con tasas de sobrevida que superan el 50% de las víctimas de paro cardiaco.

Conclusiones: Actualmente el manejo de este síndrome ha fortalecido el último eslabón dela cadena de supervivencia al estandarizar la evaluación y la selección de víctimas de paro cardiaco con un protocolo de hipotermia terapéutica e intervención coronaria percutánea precoz.

Introduction

In 1972 the Russian pathophysiologist Vladimir Negovsky described the syndrome as "a post-resuscitation disease".[1] However, since it involves a series of uncontrolled events, the International Liaison Committee on Resuscitation (ILCOR) adopted the term post cardiac arrest syndrome.[2]

The incidence rate due to all cardiac causes is 460,000 deaths/year.[3,4] Prospective trials refer to 350,000 coronary disease-related deaths/year; this is 1–2/1000 people for the American population.[5] There are survival reports of patients with extra-hospital cardiac arrest of 23.8% at the time of admission and of 7.6% at the time of discharge.[6]

Biological death depends on the cardiac arrest mechanism, on the underlying disease and on the delay in starting the resuscitation maneuvers (CPR). A poor neurological prognosis after 4–6 min of an unattended arrest is irreversible,[7] and hence resuscitation must be a constant mission.[8]

When the arrest mechanism is an asystole or a pulseless electrical activity (PEA), the progression to neurological injury is faster and leads to a worse prognosis.[7]

Mild therapeutic hypothermia (32–34 °C) is the gold standard in post-arrest care.[9]

A narrative review of the literature using Medline via PubMed and Clinical Trials using the terms MeSH cardiac arrest – Cardiopulmonary Resuscitation and no term MeSH Post cardiac arrest syndrome was carried out.

Clinical evolution

Once the patient recovers spontaneous cardiac circulation, a cascade of events develops, mainly characterized by anoxic brain injury, post cardiac arrest myocardial dysfunction, "ischemic/reperfusion" systemic response, and the typical pathology of the triggering cause of the cardiac arrest. The clinical evolution shall be dependent on clinical conditions such as the patient's co-morbidities, the duration of the ischemic lesion and the cause that triggered the cardiac arrest.[10]

Pathophysiology

Oxygen deficiency and generalized acidosis develop during cardiac arrest. If the victim is resuscitated using CPR/defibrillation maneuvers, with resumption of spontaneous circulation, PCSS develops, which is characterized by a systemic inflammatory response of the immune system and of coagulation.[11] Cell damage seems to affect the enzyme calpain and peroxidation caused by oxygen free radicals that begin to develop during the phase of global ischemia and perpetuates during reperfusion.[12]

The main cause of extra-hospital cardiac arrest in the adult is acute myocardial infarction.[13] There are many other pathologies leading to multisystem failure and subsequent cardiac arrest in the hospitalized patient.[13]

Treatment

According to the ILCOR[2,10] document, the PCAS classification follows physiological criteria in five phases:

1. Immediate care: the initial 20 min following the patient's spontaneous recovery of circulation.
2. Early phase: from 20 min to 6–12 h, when critical protective and therapeutic measures are required for a successful outcome.
3. Intermediate phase: from 6–12 h to 72 h a close surveillance and ICU treatment are required consistent with the therapeutic objectives.
4. Recovery phase: comprises the patient's condition after the initial 72 h when there is a clearer diagnosis and a more predictable result.
5. Rehabilitation phase: focuses on the patient's complete recovery. Any electrolytic abnormalities shall be corrected during phases 1 and 2, in addition to providing inotropic support and optimized oxygenation.[14]

Goal-targeted therapy

Ventilation support

1. Normocapnia ($PaCO_2$ between 40 and 45 mmHg). However, arterial gasometry should be properly interpreted in patients undergoing therapeutic hypothermia. When a patient reaches a central body temperature of close to 33 °C, the actual $PaCO_2$ may be up to 7 mmHg below the value in the arterial gases machine.[15] Hyperventilation has been associated with a drop in coronary perfusion and venous return, in addition to cerebral vasoconstriction.[16]
2. Normoxia. Both, hypoxia and hyperoxia ($PaO_2 > 300$ mmHg) may result in secondary neurological injury. Using an inspired oxygen fraction to maintain the arterial saturation between 95% and 99% or a $PaO_2 > 100$ mmHg is