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Case report

Therapeutic hypothermia after prolonged cardiopulmonary resuscitation due to pulmonary thromboembolism. Case report☆

Julian Alvarado Ramírez[a], *Hector Dario Arrovave Paramo*[b], *Fabian David Casas Arroyave*[c,*]

[a] *Physician, Third Year Resident of Anesthesiology and Resuscitation, University of Antioquia, Medellín, Colombia*
[b] *Anesthesiologist, Specialist in Medicine and Critical Care. Intensivist. University Hospital San Vicente Fundación, Medellin. Professor of Anesthesiology, University of Antioquia, Medellín, Colombia*
[c] *Anesthesiologist, University Hospital San Vicente Fundación, Medellin. Professor of anesthesiology, University of Antioquia, Medellín, Colombia*

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ABSTRACT

This is a report of a 41-year-old patient undergoing femur osteosynthesis (OS) who develops intraoperative cardiac arrest (CA) with pulseless electrical activity (PEA). Massive pulmonary thromboembolism (PTE) was diagnosed as the cause for the CA and a thrombolysis performed 30 min later reestablished spontaneous circulation with no new CA events. Therapeutic hypothermia (TH) was then established with local measures for 18 h for brain protection. The patient was extubated 24 h later with no neurological deficit. There is an increasing evidence of TH and its protective mechanisms in patients with non-shockable arrest rhythms leading to a widespread use of the technique in various institutions around the world, with particular emphasis on neurological outcomes. This article discusses a review of the current literature on TH, in addition to describing each of the stages in TH and how to approach these stages.

Hipotermia terapéutica post-reanimación cardiopulmonar prolongada en paro cardiaco debido a tromboembolismo pulmonar. Reporte de caso

Palabras clave:
Hipotermia
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RESUMEN

Se reporta un caso de un paciente de 41 años quien es llevado a osteosíntesis (OS) de fémur y que presenta paro cardiaco (PC) intraoperatorio con ritmo de actividad eléctrica sin pulso. Se diagnostica tromboembolismo pulmonar masivo como causa del PC y 30 min después se

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* *Corresponding author at*: Hospital Universitario San Vicente Fundación, Calle 64N 51 D-154, Medellín, Colombia.
E-mail address: fabiandavid68@yahoo.com (F.D.C. Arroyave).

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hace trombólisis, obteniéndose circulación espontánea sin nuevos episodios de PC. Posteriormente se instaura hipotermia terapéutica (HT) con medidas locales durante 18 h para protección cerebral. El paciente es extubado 24 h después sin ningún déficit neurológico. Es importante entender que la evidencia actual de la HT en pacientes con ritmos de paro no desfibrilables y sus mecanismos de protección es creciente, y que cada vez más se está imple-mentando esta técnica en los diferentes centros del mundo, sobre todo haciendo énfasis en desenlaces neurológicos. En este artículo se hace una revisión de la literatura actual sobre HT, además de describir cada una de las etapas de la HT y la forma en que se deben abordar.

Introduction

Good neurological results following a CA are difficult to achieve. Any intervention during cardiopulmonary resuscitation (CPR) and in the next few hours after recovering spontaneous circulation (SC) is critical to reach this goal. Experimental trials both in humans and in animals show that therapeutic hypothermia (TH) improves the neurological and cardiovascular outcomes in these patients.[1,2] There is enough evidence to use moderate TH (32–34 °C) in ventricular tachycardia (VT) or ventricular fibrillation (VF), since it improves both, the neurological prognosis and mortality.[1,3]

Clinical case

41-year-old patient with distal femoral shaft fracture referred two days later to the University Hospital San Vicente Fundación (HUSVF) in Medellin for osteosynthesis. The patient has a history of untreated diabetes mellitus type II. Paraclinical tests: HbA1c, blood count, glycaemia, ionogram, renal function, PT and PTT normal. Functional class and cardiovascular examination are normal.

The procedure is performed under spinal anesthesia, hyperbaric bupivacaine plus morphine, achieving a T10 level of aesthesia. 30 min into surgery, suddenly and unexpectedly, the patient develops cardiorespiratory arrest with PEA. CPR is administered, with capnography monitoring above 15 mmHg. The patient had not received thromboprophylactic treatment and the transthoracic ECG showed dilatation of the right ventricle and left-IV septal deviation, leading to a diagnosis of massive pulmonary thromboembolism (PTE). The patient received thrombolysis with tissue plasminogen activator as follows: initial 25 mg bolus followed by 25 mg in 30 min; then 50 mg in the next 30 min and 100 mg during the next hour, for a total of 200 mg.

After initiating the resuscitation maneuvers, the patient recovers spontaneous circulation; a femoral arterial catheter is inserted and esophageal temperature monitoring is established after CPR. When the patient was admitted to the ICU, the temperature reported was 33 °C (Philips Mx 600 monitor) just with exposure to the OR temperature. The patient is left uncovered, keeping the esophageal temperatures under control between 32.5 and 33.5 °C for 18 h. No ionotropic support was required and the norepinephrine vasopressors initiated during resuscitation were gradually tapered and well tolerated in the first 24 h. The patient received target CVP and diuresis-guided water therapy (approximately 7000 ml of crystalloids and transfusion of 2 leukocyte depleted red blood cells). During this time the patient did not develop any new episodes of CA or severe arrhythmia. Volume controlled mechanical ventilation was used – 6 ml/kg – and no additional neuromuscular relaxation was needed besides the rapid intubation sequence with 1.2 mg/kg rocuronium, sedation analgesia with 100 mg/h fentanyl plus midazolam 2 mg/h. At the end of 18 h, the patient was warmed up to 37 °C with blankets at room temperature. 6 h later the patient was extubated free of complications. The PTE was confirmed through ventilation/perfusion ultrasound, visualizing multiple apparently residual subsegmental thrombi. The patient experienced no significant bleeding and no neurological deficit.

Discussion

Survival and neurological recovery following a CA are the most significant outcomes that vary depending on the underlying pathology, the time elapsed prior to receiving care after the arrest, the initial rhythm and the resuscitation modality.[4,5] The survival rate at discharge of the cases that achieve spontaneous circulation has been below 10%.[6]

The stages of the post-cardiac arrest syndrome are: phase one includes the period immediately after returning to spontaneous circulation and up to 20 min later. It is characterized by cardiovascular dysfunction, with a corresponding 63% mortality. During the intermediate phase – between 6 and 12 h – neurological damage develops accounting for 17% mortality and, to a larger extent, for morbidity at discharge. Finally, the recovery period accounts for 7% mortality from infectious complications and multiple organ failure (MOF). At the end of these three stages, the survival rate is 13%, of which 4% are free from any neurological damage.[2,7]

Neurological damage is mediated by several mechanisms.[8] Early on, the lack of cerebral blood flow depletes the ATP reserves. In the intermediate stage, the release of excitatory amino acids activates the cytotoxic pathways. Then at the late stage, the rupture of the blood–brain barrier worsens the cerebral edema and cell death.