ELSEVIER

Resuscitation 66 (2005) 175–181

RESUSCITATION

www.elsevier.com/locate/resuscitation

Reversible myocardial dysfunction after cardiopulmonary resuscitation☆

Manuel Ruiz-Bailén[a,*], Eduardo Aguayo de Hoyos[b], Silvia Ruiz-Navarro[a], Miguel Ángel Díaz-Castellanos[c], Luis Rucabado-Aguilar[a], Francisco Javier Gómez-Jiménez[d], Sergio Martínez-Escobar[e], Rafael Melgares Moreno[f], Javier Fierro-Rosón[c]

[a] *Intensive Care Unit, Critical Care and Emergency Department, Hospital Universitario Médico-Quirúrgico, Complejo Hospitalario de Jaén, Spain*
[b] *Intensive Care Unit, Critical Care and Emergency Department, Virgen de las Nieves University Hospital, Granada, Spain*
[c] *Intensive Care Unit, Critical Care and Emergency Department, "Poniente" Hospital, El Ejido, Almería, Spain*
[d] *Department of Medicine, Granada University, Granada, Spain*
[e] *Intensive Care Unit, Critical Care and Emergency Department, Hospital Torrecárdenas, Spain*
[f] *Interventional Cardiology Unit, Virgen de las Nieves University Hospital, Granada, Spain*

Received 5 July 2004; received in revised form 27 January 2005; accepted 28 January 2005

Abstract

Objective: Myocardial stunning frequently has been described in patients with an acute coronary syndrome. Recently, it has also been described in critically ill patients without ischaemic heart disease. It is possible that the most severe form of any syndrome, leading to cardio-respiratory arrest, may cause myocardial stunning. Myocardial stunning appears to have been demonstrated in experimental studies, though this phenomenon has not been sufficiently studied in human models. The aim of the present work has been to study and describe the possible development of myocardial dysfunction in patients resuscitated after cardio-respiratory arrest, in the absence of acute or previous coronary artery disease.
Design: Descriptive study of a case series.
Setting: The intensive care unit (ICU) of a provincial hospital.
Patients and participants: The study period was from April 1999 to June 2001. All patients admitted to the ICU with critical, non-coronary artery pathology, with no past history of cardiac disease, and those who were resuscitated after cardio-respiratory arrest, were included in the study.
Measurements and results: Transthoracic and transoesophageal echocardiography was used to assess left ventricular ejection fraction (LVEF) and disturbances of segmental contractility. This study was carried out within the first 24 h after admission, during the first week, during the second or third week, after 1 month, and between 3 and 6 months. Twenty-nine patients with a median age of 65 years (range 24–76) were included in the study. Twelve patients died. Twenty patients developed myocardial dysfunction; the initial LVEF in these patients was 0.28 (0.12–0.51), showing improvement over time in the patients who survived. All of these patients presented disturbances of segmental contractility which also became normal over time.
Conclusions: After successful CPR, reversible myocardial dysfunction, consisting of systolic myocardial dysfunction and disturbances of segmental contractility, may occur.

Keywords: Myocardial dysfunction; Echocardiography; Myocardial stunning; Critical pathology; Cardiac arrest; Apnoea

1. Introduction

In 1975, Heyndrickx and colleagues were the first to describe the phenomenon of reversible post-ischaemic left ventricular dysfunction [1]. In 1982, Braunwald and Kloner [2] called this syndrome "myocardial stunning", and since that

☆ A Spanish translated version of the Abstract and Keywords of this article appears as Appendix at 10.1016/j.resuscitation.2005.01.012.

* Corresponding author. Present address: C/Las Torres 57, Torredonjimeno, Jaén, Spain. Tel.: +34 679178994.
E-mail address: mrb1604@terra.es (M. Ruiz-Bailén).

0300-9572/$ – see front matter © 2005 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.resuscitation.2005.01.012

time it has been extensively documented [3]. Myocardial stunning is defined as the reversible myocardial dysfunction (RMD), which persists after myocardial reperfusion, in the absence of an irreversible lesion, after the restoration of normal or near-normal coronary artery flow. Since its initial description, it has been widely reported and is recognised in patients with various forms of ischaemic heart disease. Phenomena of reversible myocardial dysfunction, consisting of systolic dysfunction, altered segmental contractility and electrocardiographic changes have been described in patients with critical, non-coronary pathology such as sepsis, trauma, stress, acute neurological pathology, asthma and pancreatitis [4].

It is easy to imagine that in a pathology that leads to cardio-respiratory arrest (CA), the phenomena of post-resuscitation myocardial dysfunction could occur. Furthermore, in the post-resuscitation syndrome described after cardiopulmonary resuscitation, with multi-organ failure, including renal insufficiency, acute pulmonary or cerebral lesions, myocardial dysfunction could easily exist within this context [5]. Experimentally, many studies have investigated this hypothesis, all demonstrating variable degrees of myocardial dysfunction after cardiopulmonary resuscitation (CPR) [6–9]. However, there are very few studies using human models, most of these being case series in which no convincing results on the existence or absence of post-resuscitation myocardial dysfunction following CPR have been found [10–13]. In view of these findings, the objective of the present study has been to investigate the existence of myocardial dysfunction in patients following successful resuscitation after cardio-respiratory arrest.

2. Material and methods

A descriptive study carried out on a series of cases gathered prospectively from the patients admitted to a nine-bed medico-surgical ICU in a provincial hospital during the study period running from April 1999 to June 2001.

The inclusion criteria for the study were respiratory or cardio-respiratory arrest with survival for at least 72 h. Any patient with a past history of cardiovascular pathology was excluded, except for those with, or having treatment for, arterial hypertension. Patients were excluded who: (1) had a known history of cardiovascular pathology or previously abnormal ECG or elevated cardiac enzymes; (2) had been admitted for coronary artery pathology or other acute cardiovascular disturbance such as valve pathology, cardiomyopathy, or other anatomical disturbances which could be associated with myocardial dysfunction; (3) presented with critical pathology known to be associated with myocardial dysfunction, such as myocarditis, sepsis, septic shock, pulmonary embolism or peri-partum cardiomyopathy; (4) had suffered multiple trauma with thoracic trauma; (5) survived for less than 72 h after successful CPR.

2.1. Echocardiographic study

The first echocardiographic examination was performed within the first 24 h after achieving successful CPR. Thereafter, transthoracic or transoesophageal echocardiography were performed depending on the acoustic window in the patient. These studies were carried out within the first 48 h, and repeated during the first week, during the second or third week, and after 1, 3 and 6 months. During the echocardiography, the left ventricular ejection fraction (LVEF) was calculated. The value accepted for the LVEF was the arithmetic mean of a minimum of three measurements. The methods used for its measurement were M mode, the area–length method, the modified Simpson method, or visual estimation in cases in which it was impossible to make a measurement. An assessment was also made of alterations in segmental contractility using the recommendations of the American Society of Echocardiography [14], establishing the following score: 0 = hyperkinesia, 1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia [12]. Ventricular aneurysm has been considered as an area of ventricular dilatation or expansion, showing systolic dyskinesia without a return to normality during diastole. Diastolic function was not studied. With respect to the right ventricle, only dilatation and severe dysfunction were taken into account. The diameter of the left ventricle was considered normal up to 57 mm (parasternal projection, long axis); above this diameter, left ventricular dilatation was considered to be present. The maximum diameter of the right ventricle in the same projection was taken as 27 mm. Other echocardiographic abnormalities, such as the development of valve pathology, pericardial effusion and the development of ventricular aneurysms, were also studied and followed up. Myocardial dysfunction was defined as the presence of an LVEF of <0.55.

2.2. Electrocardiography

A 12-lead ECG was carried out on admission in all the patients and repeated on a daily basis. The ECG was recorded using conventional parameters (paper speed 25 mm/s, calibration 10 mm = 1.0 mV). Alterations of the ST segment, Q-wave, alterations of the T-wave, and the QTc segment were studied. Patients with previous ECG abnormalities were excluded.

Peak creatine kinase (CK) values were included, including the MB fraction. Data relating to the presence of the classical cardiovascular risk factors were recorded, and the degree of severity was quantified using the APACHE III scale. All complications that developed were also recorded.

A Swan-Ganz catheter was inserted according to diagnostic and therapeutic needs as assessed by the intensivist responsible for the patient.

Coronary angiography and/or exercise testing was requested if there was a suspicion of acute ischaemic cardiomyopathy.