Induced mild therapeutic hypothermia following cardiac arrest in the setting of acute myocardial infarction and successful primary percutaneous coronary intervention: a case report

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ABSTRACT

It was presented the case of a 50-year-old male, with no previous history of cardiovascular disease, who was successfully treated with induced mild hypothermia and primary percutaneous coronary intervention after cardiac arrest and prolonged resuscitation (twelve electrical cardioversions), following acute ST-segment elevation myocardial infarction. The patient had full recovery and uneventful short and long-term neurological course. Mild hypothermia has been considered a safe option for cerebral preservation after cardiopulmonary resuscitation. Several mechanisms mediate brain protection during hypothermia, including a possible prevention of apoptosis (programmed cell death). The role of apoptosis following ischemic and/or hypoxic brain injury is also reviewed.

Keywords: Myocardial infarction/therapy; Hypothermia; Apoptosis; Angioplasty, transluminal, percutaneous coronary; Death, sudden; Case reports

INTRODUCTION

Complete neurological recovery after cardiac arrest with temporary cerebral ischemia is uncommon. Mild hypothermia has been described as an effective strategy in the treatment of traumatic brain and neurological emergencies, including comatose state post ventricular fibrillation (V-fib) cardiac arrest.

Use of mild hypothermia in acute myocardial infarction (AMI) has profound multifactorial benefits to the brain and ischemic myocardial tissues. It is reported the case of a patient who suffered sudden cardiac death due to primary V-fib in the setting of AMI, and was successfully resuscitated. The patient underwent percutaneous revascularization and interventional hypothermia, progressing without any neurological damage. Hypothermic protective mechanisms, including a possible prevention of apoptosis and during brain damage, are reviewed and discussed.

CASE REPORT

A 50-year-old man was brought by the emergency rescue service due to recent onset of acute exertional chest pain. He had no significant past medical history and lost consciousness few minutes prior to arrival to the Emergency Department in V-fib cardiac arrest.
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Immediate cardiopulmonary resuscitation (ACLS protocol) was instituted. The patient underwent immediate orotracheal intubation and 12 (twelve) direct cardioversion (DC) shocks, alternated with intravenous administration of epinephrine, lidocaine and amiodarone. Total resuscitation time was approximately of 20 to 25 minutes. His postcardiac arrest electrocardiogram revealed sinus bradycardia with heart rate of 60 bpm, mild prolongation of QT-interval (0.456 sec) and acute anterior wall ST-segment elevation MI. There were no malignant post-resuscitation arrhythmias. An emergency coronary angiography showed total thrombotic occlusion (100%) at the mid-left anterior descending (LAD) artery (Figure 1). A primary percutaneous coronary intervention (PPCI) was performed using a 2.5 x 20 mm conventional balloon catheter followed by deployment of a 3.5 x 16 mm CoStar drug-eluting stent with post-stent TIMI grade III flow (Figure 2). Complementary “kissing balloon” technique of LAD and third diagonal branch was also successfully performed. Infusion of abciximab (Reopro – IIb IIIa GP inhibitor) was started. His total door-to-balloon time (including resuscitation) was approximately 40 to 45 minutes. Doppler echocardiogram revealed apical and mid-septal akinesia, and anterior wall hypokinesia with left ventricular ejection fraction of 43%.

He was taken to the Intensive Care Unit (ICU) under full cardiac monitoring and invasive mechanical ventilation. He was mildly hypotensive, fully sedated and under mechanical ventilation. His blood pressure was 90/60 mmHg, heart rate of 55 to 65 under continuous amiodarone IV drip, and not receiving vasopressors or inotropes.

In the ICU, the patient was wrapped in a water mat and covered by cooling blankets, and the rectal temperature was maintained between 33 and 34 °C. He was kept at this temperature for 24 hours, then he was slowly rewarmed at a rate of 0.2 - 0.3 °C per hour, to a final temperature of 36.5 °C. A head computerized tomography was performed and did not show abnormalities. During therapeutic hypothermia, the cardiac output and mixed venous blood saturation (SVO\textsubscript{2}) were monitored continuously, and acidosis did not occur. He had no recurrent arrhythmias.

Over the following two days, sedation was discontinued and he was successfully weaned from mechanical ventilation. He regained gradual consciousness without any focal neurological deficit. After his fifth day at the hospital, he was transferred to the Stepdown Coronary Care Unit (CCU). He started a rehabilitation program with complete recovery. The patient was also able to tolerate up titration of beta blockers and ACE-inhibitors. A follow-up echocardiogram showed minimal apical akinesia and a new ejection fraction of approximately 56%. A second head computed tomography was normal. The patient did well and was discharged 17 days after hospital admission.

In consecutive re-evaluations at 3, 6 and 12 months after myocardial infarction, the patient is in New York Heart Association class I, without any ventricular arrhythmias on 24-hour Holter recordings, and remains with fully preserved mental status.
The degree of hypoxia is critical for determination of metabolism and degradation of these proteins are. In spite of current knowledge of several related proteins into such pathways, it is still unclear what the true mechanisms role through the knowledge of mediated process and pathways of cell-cycle and cell-death. In spite of current knowledge of several related proteins into such pathways, it is still unclear what the true mechanisms of metabolism and degradation of these proteins are. The degree of hypoxia is critical for determination of brain cell death or survival in cardiac arrest patients and acute ischemic cerebrovascular accidents. Basic science studies have demonstrated overproduction of inflammatory cytokines, release of mitochondrial c-cytochrome, among other phenomena. There is microthrombotic platelet formation, and increase in intravascular fibrin also contributing for brain infarct development.

In summary, it is presented a case of a middle aged male who suffered V-fib cardiac arrest in the setting of AMI, and was successfully resuscitated and subsequently treated by PPCI. He underwent inteventionlal mild hypothermia with no neurological damage during the follow-up. Current evidence supports prompt institution of mild hypothermia after successful cardiopulmonary resuscitation and clinical stabilization. Prevention of apoptosis by hypothermia may be one of the several brain protective mechanisms.

REFERENCES


