Mechanical Chest Compressions in Prolonged Cardiac Arrest due to ST Elevation Myocardial Infarction Can Cause Myocardial Contusion

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Abstract

Keywords

- cardiac arrest
- mechanical chest compressions
- ST-elevation myocardial infarction
- percutaneous coronary intervention
- myocardial contusion

Acute coronary syndrome is a common cause of sudden cardiac death. We present a case report of a 60-year-old man without a history of coronary artery disease who presented with ST-elevation myocardial infarction. During transportation to the hospital, he developed ventricular fibrillation (VF) and later pulseless electrical activity. Chest compressions with LUCAS 2 (Medtronic, Minneapolis, MN) automated mechanical compression–decompression device were initiated. Coronary angiography showed total occlusion of the left main coronary artery and primary percutaneous coronary intervention (PCI) was performed. After the PCI, his heart started to generate effective contractions and LUCAS could be discontinued. Return of spontaneous circulation was achieved after 90 minutes of cardiac arrest. The patient died of cardiogenic shock 11 hours later. An autopsy revealed a transmural anterolateral myocardial infarction but also massive subepicardial hemorrhage and interstitial edema and hemorrhages on histologic samples from regions of the myocardium outside the infarction itself and also from the right ventricle. These lesions were concluded to be a myocardial contusion. The true incidence of myocardial contusion as a consequence of mechanical chest compressions is not known. We speculate that severe myocardial contusion might have influenced outcome of our patient.

Outcomes of patients resuscitated for out-of-hospital cardiac arrest (OHCA) complicating ST-elevation myocardial infarction (STEMI) who achieve return of spontaneous circulation (ROSC) before urgent coronary angiography have already been investigated in several retrospective and prospective series. In-hospital mortality or poor neurological outcome has been reported in 23% of these patients.^{1,2} Thirty-day mortality was 33%³ and complete neurological recovery or moderate neurological disability was found in 58% of patients with STEMI successfully resuscitated for OHCA.⁴ This is in contrast with the population of unselected patients treated for OHCA of supposedly cardiac origin where survival rates remain still under 20%. Predictors of deleterious outcome were found to be delayed in

basic life support and OHCA before arrival of the emergency medical services. On the contrary, predictors of favorable outcome used an automated external defibrillator and ventricular fibrillation (VF) as first observed rhythm.¹ However, we do not know the extent of damage that prolonged cardiopulmonary resuscitation with mechanical chest compression device causes to the heart and whether it has an impact on outcomes of resuscitated patients.

Case Report

A 60-year-old man without a history of coronary artery disease called an ambulance for typical chest pain at rest.

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Fig. 1 An electrocardiogram of acute anteroseptal ST-elevation myocardial infarction.

An electrocardiogram recorded by the emergency medical staff upon arrival showed ST-elevations in multiple leads (► **Fig. 1**). It was decided to transport the patient directly to a percutaneous coronary intervention (PCI) center. During transportation, he developed VF and after first defibrillation he developed bradycardia with severe hypotension (pulseless electrical activity). Closed-chest compressions with LUCAS had to be initiated together with orotracheal intubation and mechanical ventilation before arrival at the hospital. A trans-thoracic echocardiography (TTE) performed in the emergency department during a short cessation of chest compressions showed an akinetic left ventricle without pericardial effusion. The patient was transported from the emergency department directly to the catheterization laboratory still in cardiac arrest. Coronary angiography, which showed a totally occluded left main coronary artery (► **Fig. 2**), was followed by PCI with implantation of bare-metal stent with good antegrade flow. An intra-aortic balloon pump (IABP) was advanced via

the femoral artery. Needle to first balloon inflation time was 10 minutes and the whole procedure took 20 minutes. Mean arterial blood pressure during mechanical chest compressions with LUCAS varied between 20 and 50 mmHg. After the reperfusion, his heart started to generate effective contractions and LUCAS could be discontinued. Arterial blood pressure of 120/90 mmHg could be achieved with the use of an IABP and catecholamines. ROSC was achieved after 90 minutes of cardiac arrest. Another TTE performed after the PCI showed akinetic anterolateral segments and an apical septum with left ventricular ejection fraction 20%. The patient was in cardiogenic shock with repeated episodes of ventricular tachycardia/fibrillation requiring defibrillations. Increasing doses of intravenous catecholamines including epinephrine were needed to maintain circulation. The patient subsequently died of cardiogenic shock 11 hours after admission to the hospital. An autopsy was performed that revealed transmural myocardial infarction of the anterior septum and anterior and lateral walls of the left ventricle but also massive

Fig. 2 Coronary angiography during mechanical chest compressions with LUCAS shows totally occluded left main coronary artery (arrow).

Fig. 3 Histologic sample of the myocardium (hematoxylin and eosin staining) from location outside the infarction itself shows marked interstitial edema and hemorrhages with preserved viability of myocytes—signs of myocardial contusion.

subepicardial hemorrhage under the inferior part of the sternum over which the suction cap of the LUCAS had been placed. Histologic samples of the myocardium from areas outside the infarction itself showed marked interstitial edema and hemorrhages with preserved viability of myocytes (–Fig. 3). These findings were concluded as being myocardial contusion due to 90 minutes of mechanical chest compressions.

Discussion

The use of automated mechanical compression–decompression device such as LUCAS enables uninterrupted continuous chest compressions while an operator is performing PCI, which would hardly be possible without LUCAS. In a retrospective analysis of STEMI patients from a single center, technical success of PCI in patients treated with LUCAS was similar to patients without cardiac arrest.⁵ The incidence of myocardial contusion in patients resuscitated with mechanical chest compression devices (MCCDs) such as LUCAS is unknown. According to the review of Gates et al, injuries caused by MCCD LUCAS were reported by five human studies and two animal studies.⁶ Human studies demonstrated a trend toward more injuries with MCCD, in one study statistically significant. However, the studies neither investigated cardiac contusion nor mentioned it in results. The effect of prolonged resuscitation with mechanical chest compressions on ischemic heart injury in patients with acute myocardial infarction is also unknown. In infarction, hypoxia/anoxia causes cell necrosis and there is generalized coagulation necrosis and gradual transition from the infarcted to the normal tissue. In contusion, edema and hemorrhage are more pronounced, while there are regions of viable cells with distinct boundary to necrotic ones (patchy necrosis).⁷ Diagnosis of a myocardial contusion in a critically ill patient due to acute myocardial infarction is difficult to make. Plasma markers of myocardial injury and clinical signs and symptoms are of no use in this setting. Echocardiographic findings early after the ROSC were not suggestive of myocardial contusion. Whether myocardial contusion in our patient might have contributed to the severe systolic dysfunction and cardiogenic shock cannot be answered with the present knowledge on this topic. We can only hypothesize that part of the stunned myocardium due to ischemia caused either by coronary artery occlusion or cardiac arrest itself would recover. Patients resuscitated from cardiac arrest due to acute coronary syndrome may need some advanced circulatory support including extracorporeal mem-

brane oxygenation (ECMO) to bridge the time to recover systolic function of the left ventricle. Recent work of Kagawa et al showed favorable 30-day survival and neurological outcome (29 and 24%, respectively) in 86 patients with acute coronary syndrome and cardiac arrest unresponsive to conventional cardiopulmonary resuscitation who were treated with ECMO.⁸

Conclusion

This case report demonstrates that ROSC could be achieved after 90 minutes of cardiac arrest and only after intra-arrest PCI facilitated with mechanical chest compression device LUCAS. Prolonged mechanical chest compressions with LUCAS can cause myocardial contusion.

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