Primary Percutaneous Coronary Intervention for Refractory Cardiac Arrest

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The identification and treatment of reversible causes is paramount to the success of resuscitation in cardiac arrest, particularly when standard therapy has failed. Acute coronary occlusion is one such cause, and the introduction of primary percutaneous coronary intervention services may provide an opportunity for emergency revascularization in this setting. This article describes 2 patients with cardiac arrest as a result of coronary occlusion, in which standard therapeutic measures proved futile. The first patient had refractory ventricular fibrillation, and the second had an episode of ventricular fibrillation followed by true pulseless electrical activity: total cessation of ventricular activity. In both examples, external mechanical compression and primary percutaneous coronary intervention facilitated coronary revascularization and achieved return of spontaneous circulation, leading to survival to hospital discharge. [Ann Emerg Med. 2014;64:192-194.]

INTRODUCTION

Resuscitation guidelines stipulate that reversible causes "must be sought during any cardiac arrest."1 Thrombotic coronary occlusion is well recognized as such a cause, but revascularization is not commonly attempted during resuscitation. Primary percutaneous coronary intervention is increasingly available and may be useful in this setting.

CASE REPORT

Case 1

A 59-year-old male smoker, receiving celecoxib for rheumatoid arthritis, presented to the emergency department (ED) with a 24-hour history of central chest pain. An ECG demonstrated an extensive anterolateral ST-elevation myocardial infarction. Before transfer to the cardiac catheter laboratory, the patient developed ventricular fibrillation refractory to defibrillation, despite aggressive cardiopulmonary resuscitation, epinephrine, and amiodarone. The patient received intubation and ventilation, and an AutoPulse mechanical chest compression device (Zoll, Chelmsford, MA) was deployed.

During continuous mechanical compression, which maintained catheter-transduced blood pressure at 175/115 mm Hg (Figure 1), coronary angiography demonstrated proximal occlusion of the left anterior descending artery (Figure 2). After thrombus aspiration, balloon dilatation, and stenting, normal flow was restored to the distal vessel. Subsequent defibrillation was then successful, resulting in sinus bradycardia and return of spontaneous circulation and allowing cessation of chest compressions 66 minutes after initial arrest.

An intra-aortic balloon pump was inserted and the patient was transferred to the ICU where therapeutic hypothermia was induced. He was rewarmed, woken, and extubated the following day and discharged on day 11, without neurologic deficit. He currently lives independently at home.

Case 2

A 59-year-old female smoker arrived at the hospital by ambulance in ventricular fibrillation, having collapsed at home after an episode of chest pain. Defibrillation in the ED briefly restored spontaneous circulation, and an ECG demonstrated sinus rhythm with left bundle branch block. Shortly after this, the patient experienced a further arrest with pulseless electrical activity. Focused echocardiography was performed, which demonstrated complete absence of ventricular activity. An AutoPulse compression device was again deployed, and she was transferred to the catheter laboratory.

Angiography demonstrated total occlusion of the proximal left anterior descending artery. Thrombus aspiration, predilatation, and stenting were performed as before, restoring distal coronary flow. Return of spontaneous circulation was achieved, and the patient was transferred to the ICU for therapeutic hypothermia, although she was extubated later that day. She was discharged home on day 9 without neurologic deficit and is living independently.

DISCUSSION

It is now widely recognized that, after resuscitation from cardiac arrest, patients with ST-elevation myocardial infarction should undergo early coronary angiography and percutaneous coronary intervention.2 In addition, some authorities recommend emergency angiography for all successfully resuscitated patients when there is no obvious alternative cause3 because traditional ECG changes may be a poor predictor of coronary occlusion in the postarrest period.1,2,3
The use of external mechanical compression devices is somewhat controversial, and evidence has been mixed when their use is examined as a single, novel intervention. However, their ability to facilitate diagnostic imaging or therapy for patients in cardiac arrest has been repeatedly described. The use of such bridging techniques to assist the delivery of definitive care is of increasing interest not only with mechanical compression but also with deep therapeutic hypothermia, or even emergency cardiopulmonary bypass, in trauma, in refractory cardiac arrest, or to facilitate percutaneous coronary intervention.

Undertaking resuscitative percutaneous coronary intervention for the treatment of refractory cardiac arrest is uncommon, and in our primary percutaneous coronary intervention facility, we have undertaken angiography during resuscitation in only 4 other cases. One patient arrived in asystole after a prolonged out-of-hospital resuscitation attempt and developed hemorrhagic pulmonary edema during angiography. No attempt at percutaneous coronary intervention was therefore undertaken. Two others arrived in ventricular fibrillation, having experienced out-of-hospital cardiac arrest with a delay to basic life support (one of 7 minutes and the other of 14 minutes). Both underwent angioplasty to the left anterior descending artery and survived to hospital admission, but not to discharge. The fourth patient was morbidly obese and arrived in cardiogenic shock, with an extensive anterior ST-elevation myocardial infarction. He experienced a ventricular fibrillation arrest before percutaneous coronary intervention, which was completed with technical success during external mechanical compression. Unfortunately, the patient failed to wake in the ICU and therapy was withdrawn.

It seems likely that patient selection is key to optimizing the response to this procedure. The patients in the successful cases above were relatively young, with little previous comorbidity, and both had a history of confirmed or highly likely cardiac event,
with ECG changes supportive of the diagnosis, meaning that angiography was undertaken with therapeutic rather than solely diagnostic intent. Both individuals benefited from prompt resuscitation, known to improve survival, the first arresting in the resuscitation room of our ED and the second found collapsed by paramedics 4 minutes after she had called for help, complaining of chest pain. The first-contact-to-balloon times were 110 minutes and 122 minutes, respectively, with a door-to-balloon time of 69 minutes for the second case.

Even with these “favorable” characteristics, in situations in which ventricular fibrillation is unresponsive to defibrillation the prognosis is poor. Only amiodarone has been shown to improve survival, and then only to hospital admission rather than discharge. Although reversible myocardial dysfunction after cardiac arrest is well recognized, including transient “myocardial stunning” caused by the ischemic insult or subsequent defibrillation, the complete absence of mechanical ventricular activity on targeted echocardiography has been described as another poor prognostic sign. This “true pulseless electrical activity” is generally regarded as a marker of futility and in many cases may contribute to decisions about the cessation of resuscitation efforts.

In cases such as these, which would be otherwise futile in the absence of an alternative efficacious therapy, correction of the underlying cause of arrest becomes imperative. Ventricular arrhythmia and myocardial dysfunction are common complications of ischemia and so may potentially be reversed with treatment of the substrate. Trials of thrombolysis in cardiac arrest of unknown or suspected cardiac cause have been disappointing, particularly compared with its use in pulmonary embolism, but percutaneous coronary intervention offers an alternative revascularization strategy for patients with refractory cardiac arrest caused by coronary obstruction. Research into the potential benefits of resuscitative percutaneous coronary intervention is required.

CONCLUSIONS

Myocardial ischemia is an important cause of cardiac arrest, with an effective treatment. Early coronary angiography is recommended by some authorities after cardiac arrest in patients without an obvious noncardiac cause, and ECG changes may be unreliable in this setting. In appropriately selected patients, resuscitative invasive coronary revascularization could be considered to reverse the cause of refractory cardiac arrest.

REFERENCES