Case Series

Prolonged Chest Compressions during Cardiopulmonary Resuscitation

for In-Hospital Cardiac Arrest due to Acute Pulmonary Embolism

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Patients with hemodynamic collapse due to acute pulmonary embolism have a dismal prognosis if not treated rapidly. Therapeutic options include systemic thrombolytic therapy, rheolytic thrombectomy, and surgical embolectomy. However, the efficacy of thrombolytic therapy is diminished because the low-output state hinders effective delivery of the lytic agent to the thrombus. In the absence of any form of mechanical circulatory support, such as extracorporeal membrane oxygenation or cardiac surgery on site, we think that prolonged vigorous manual compressions might be the only way to support the circulation during the initial critical state, when thrombolytic therapy has been administered.

We report the results of prolonged manual chest compressions (exceeding 30 minutes) on 6 patients who received tenecteplase in treatment of acute pulmonary embolism that induced in-hospital cardiopulmonary arrest. Four of 6 patients survived and were discharged from the hospital. In an era of increasing technologic complexity for patients with hemodynamic instability, we emphasize the importance of prolonged chest compressions, which can improve systemic perfusion, counteract the prothrombotic state associated with cardiopulmonary arrest, and give the lytic agent time to act. ***(Tex Heart Inst J 2015;42(2):136-8)***

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Despite considerable advances in the techniques of resuscitation, cardiopulmonary arrest still carries a dismal prognosis. Although thrombolytic therapy does not form part of any standard clinical algorithm, it can be useful in treating some clinical conditions that cause hemodynamic instability: especially acute myocardial infarction and acute pulmonary embolism (PE). However, the efficacy of thrombolytic therapy is decreased by cardiopulmonary arrest, which is an inherently prothrombotic state and restricts access of the thrombolytic agent to the pulmonary embolus.

For several years, guidelines have recommended thrombolytic therapy for acute PE patients who are experiencing cardiogenic shock or hemodynamic instability.[1] Recently, such therapy has also been suggested for patients with normal blood pressure and intermediate risk, provided that specified cardiac markers or imaging criteria are used in selecting those patients.[2]

In our unit, we have hypothesized that patients with acute PE and cardiopulmonary compromise can benefit from prolonged manual compressions, supplemented by transthoracic echocardiography (TTE), to improve the efficacy of thrombolytic therapy. Because we do not have, on site, any form of mechanical circulatory support, like extracorporeal membrane oxygenation (ECMO) or cardiac surgery, we think that prolonged vigorous manual compressions might be the only way to support the circulation during the initial critical state, when lytic therapy has been administered but has not yet taken effect. In an important study of in-hospital cardiac arrest,[3] the quality of chest compressions was deemed inadequate in more than one third of patients, when judged in terms of the depth of compression, and deemed inadequate in more than one quarter of patients, when judged in terms of rate. Manual compression devices have been developed[4] to overcome these difficulties, but their results have not been superior to those of conventional compression. Prompt surgi-

cal management has been advocated by some, but we think that simple steps—like effective prolonged chest compressions after thrombolytic therapy—should not be overlooked.[5] In acute PE, large central emboli can cause significant outflow obstruction. Because the right ventricle (RV) is principally a volume pump, it is unable to respond effectively to an acute challenge. Moreover, the thrombolytic agent needs prompt access to the large thrombus, to be efficacious.

Patients and Methods

We retrospectively studied the medical information of 6 patients who sustained in-hospital cardiopulmonary arrest due to acute PE, which had been confirmed by multidetector computed tomographic pulmonary angiography. Three patients had systemic hypotension (systolic blood pressure, <90 mmHg with or without hemodynamic support) preceding the cardiopulmonary arrest. During cardiopulmonary resuscitation (CPR) maneuvers, the thrombolytic therapy administered to all patients was tenecteplase, in accordance with a weight-based regimen. All patients received the thrombolytic agent within 10 min of the commencement of CPR maneuvers, administered by personnel trained in Basic and Advanced Cardiac Life Support. During CPR, echocardiography was done at the bedside to evaluate myocardial contractility. With the patient in the supine position, bedside TTE was performed in the standard 4-chamber view close to the apex of the heart and in the parasternal long-axis view close to the sternum, while chest compressions were stopped for 10 to 15 s. Even when contractility was noted, CPR was continued if the heart rate was low (usually <30 beats/min).

We define prolonged cardiopulmonary manual compressions as those continued for longer than 30 minutes after administration of the thrombolytic agent, because this was the longer time recommended for CPR maneuvers in one of the 2 seminal trials in which the use of thrombolytic agents in cardiopulmonary arrest was studied.[6] Return of spontaneous circulation (ROSC) was determined as the time when a pulse was palpable and the monitor registered concomitant electrical activity.

Four of 6 patients survived to the time of their discharge from the hospital. One of the 2 patients who did not survive eventually had anoxic encephalopathy and died a few days later. The durations of manual compressions shown on the clinical notes were 40, 48, 55, 61, 80, and 91 minutes for the 6 patients. Table I shows the clinical characteristics of the patients.

Discussion

Some studies have been performed on the use of thrombolytic therapy for patients who present in cardiac arrest. In one study of 333 patients with pulseless electrical activity (who were randomized),[6] only one patient survived to hospital discharge. In that study, it was stipulated that at least 15 more minutes of manual compression be performed after the administration of the lytic agent. There was an interval of approximately 35 minutes between collapse and the administration of thrombolytic therapy. Moreover, only 1 of 42 patients who underwent autopsy was confirmed to have had an acute PE.

In another study,[7] performed a few years later, outcome improved and more than 14% survived to hospital discharge, yet thrombolytic therapy was deemed to be ineffective in cases of out-of-hospital cardiac arrest. However, the study might have included very few patients with acute PE, because, if PE was suspected to be the cause of cardiac arrest, open-label use of thrombolytic agent was permitted, rather than randomization. The study also mandated the continuation of CPR for at least 30 min-

TABLE I. Characteristics and Outcomes of the 6 Patients Who Underwent Prolonged Chest Compressions

Patient No.	Age (yr)	ECG Rhythm	Time to ROSC (min)	Total Chest Compression Time (min)	PE Location	Outcome
1	49	Severe bradycardia	40	61	Central and saddle	Lived
2	61	Asystole	None	91	Central	Died
3	93	Paced rhythm	40	48	Central	Lived
4	70	Asystole	30	55	Central	Lived
5	32	Electromechanical dissociation	30	40	Central	Lived
6	68	Asystole	40	80	Central	Died*

ECG = electrocardiographic; PE = pulmonary embolus; ROSC = return of spontaneous circulation

*Anoxic encephalopathy

The no-flow time in each patient was 0.

utes after the administration of the study drug. Despite the increased CPR time in comparison with that of the previous study, tenecteplase was not superior to placebo. However, the interval between the patient's collapse and the administration of the study drug was shorter than in previous reports (18 min). Moreover, only 37 patients out of a total of 1,050 had confirmed PE.

In yet another retrospective review and analysis of patients with central PE who sustained cardiac arrest due to PE, only 2 patients of 21 who received thrombolysis survived to hospital discharge.[8] The only difference between patients who did and did not receive thrombolysis was that ROSC occurred in 81% of the thrombolysis patients and in only 43% of the nonthrombolysis patients. In that study, ROSC occurred between 3 and 105 minutes after initiation of CPR.

Our approach has been slightly different. The absence of any form of mechanical circulatory support or cardiac surgery in our district hospital limits what we can offer patients who present in cardiogenic shock with acute PE. However, we have strengthened our approach with a few crucial steps that we think can improve survival rates.

If we suspect acute PE in patients with out-of-hospital cardiac arrest, we emphasize vigorous CPR followed by TTE as soon as possible. If our suspicion of acute PE is reinforced by severe dilation of the RV or by the presence of thrombus in the right atrium or ventricle, we administer thrombolytic therapy as soon as possible. We prefer tenecteplase because of its simple regimen: the administration of a single weight-adjusted bolus. We strongly recommend that vigorous manual chest compressions be performed after the provision of thrombolytic therapy, because the dilated RV is proximal to the sternum and therefore amenable to improved output by this means. Although intermittent echocardiography during CPR maneuvers is not mentioned in any guidelines, every 5 minutes we scan the thorax rapidly for a few seconds (coupled with palpation of the pulses) to determine whether contractility has returned. Even if vigorous contractility is seen upon TTE, we continue with manual compressions if the heart rate is low, in order to ensure adequate cardiac output.

In our cohort of patients with acute PE and cardiopulmonary arrest, 4 of 6 patients survived to hospital discharge. Prolonged compression augments RV output, because the RV is proximal to the sternum and is even more proximal when dilated. Moreover, prolonged compression probably improves right-sided heart flow and circulation of the thrombolytic agent; and this counteracts the systemic prothrombotic state induced by cardiopulmonary arrest. We certainly acknowledge the possible need of embolectomy and the superiority, under ideal circumstances, of mechanical circulatory support in the form of ECMO. Yet we wish to emphasize the worth of the simple bedside maneuvers described here, in treating patients who do not have rapid access to ECMO or cardiac surgery.

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