Case Reports

A Case of Survival after Cardiac Arrest and 3½ Hours of Resuscitation

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Although survival rates after cardiac arrest remain low, new techniques are improving patients' outcomes. We present the case of a 40-year-old man who survived a cardiac arrest that lasted approximately 3½ hours. Resuscitation was performed with strict adherence to American Heart Association/American College of Cardiology Advanced Cardiac Life Support guidelines until bedside extracorporeal membrane oxygenation could be placed. A hypothermia protocol was initiated immediately afterwards. The patient had a full neurologic recovery and was bridged from dual ventricular assist devices to a total artificial heart. On hospital day 160, he underwent orthotopic heart and cadaveric kidney transplantation. On day 179, he was discharged from the hospital in ambulatory condition.

To our knowledge, this is the only reported case in which a patient survived with good neurologic outcomes after a resuscitation that lasted as long as 3½ hours. Documented cases of resuscitation with good recovery after prolonged arrest give hope for improved overall outcomes in the future. ***(Tex Heart Inst J 2014;41(2):222-6)***

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In general, outcomes after cardiac arrest remain poor, especially in patients with risk factors such as unwitnessed arrest, unfavorable initial rhythm, older age, and prolonged resuscitation without return of spontaneous circulation (ROSC).[1-4] Guidelines therefore exist for terminating resuscitative efforts in cases of cardiac arrest that are deemed futile.[5,6] Nonetheless, successful resuscitation and good recovery after prolonged arrest have been documented.[7-10] As the field of cardiopulmonary resuscitative medicine evolves, new techniques are being implemented to improve outcomes in patients who are in cardiac arrest. We present an example of how recent research findings in resuscitative medicine improved one patient's chances of survival.

Case Report

In June 2011, a 40-year-old white man with a history of hypertension, hyperlipidemia, obesity (body mass index, 34.9 kg/m^2), and chronic tobacco use presented with presyncopal symptoms. Severe pressure-like chest pain had started 24 hours previously and had completely resolved spontaneously 12 hours before the current presentation. An electrocardiogram (ECG) showed persistent ST-segment elevation in the anterior leads. He was hemodynamically well compensated. Initial laboratory reports showed cardiac troponin I elevation to a level above 50 ng/mL.

The patient was not a candidate for primary percutaneous coronary intervention (PCI) for STEMI because of his delayed presentation, the complete resolution of his chest pain, and the development of anterior Q waves on his ECG. He was admitted to the cardiovascular care unit (CCU) and was treated conservatively with antiplatelet, anticoagulant, and antiarrhythmic therapy.

The next morning, a coronary angiogram revealed an occluded proximal left anterior descending coronary artery, an occluded obtuse marginal branch, and a diffusely diseased right coronary artery. On hospital day 3, a positron emission tomographic viability scan showed only minimal viable myocardium and a large area of scar tissue; therefore, the patient was not a candidate for revascularization. He remained hemodynamically stable in the CCU and was subsequently transferred to the medicine floor.

At 5:37 AM on hospital day 5, the patient experienced sustained ventricular tachycardia and then lost consciousness. He had no spontaneous respirations, and neither the carotid nor femoral pulses could be palpated. His airway was secured, and positive-pressure ventilation was initiated. Simultaneously, cardiopulmonary resuscitation (CPR) was started by first responders. After the first 2 minutes of CPR, ventricular fibrillation (VF) was detected. The resuscitation continued in strict accordance with American Heart Association/American College of Cardiology Advanced Cardiac Life Support (ACLS) guidelines, including defibrillation. After the initial shock, the patient regained consciousness but for only a few moments, and VF was again detected. After CPR was resumed, the patient again became conscious (to the point of simple communication and responding when his name was spoken). It became evident that appropriate CPR was providing adequate cerebral perfusion, because the patient was conscious only during the chest compressions. Despite the patient's apparently extreme pain, the team decided to continue resuscitative efforts for as long as neurologic function remained intact. The patient had ROSC with stable hemodynamic status at 6:19 AM and was rapidly moved to the CCU.

The patient again became hypotensive despite normal infusions of saline solution and vasopressor agents. He was intubated and placed on mechanical ventilation. He became progressively bradycardic and again lost circulation at 6:50 AM. The patient was found to have pulseless electrical activity, and CPR was again initiated. All possible reversible causes of hemodynamic collapse were evaluated, and the patient was found to be in refractory cardiogenic shock. Spontaneous circulation eventually returned, and the patient's hemodynamic status was stabilized at 7:08 AM.

From 7:08 to 7:33 AM, the patient had palpable pulses; however, he remained hypotensive despite continuous saline infusion and increased vasopressor administration. For the 3rd time, VF recurred and CPR was initiated. In accordance with ACLS protocol, the patient was defibrillated multiple times and was given appropriate pharmacologic support. At 8:15 AM, an intra-aortic balloon pump (IABP) was placed to provide immediate cardiac support and potentially bridge the patient to a ventricular assist device (VAD), should sinus rhythm return; however, it became evident that the patient was in refractory VF and would not survive without more definitive therapy. A 21F-to-17F venous-to-arterial extracorporeal membrane oxygenator (ECMO) was placed at the bedside at 9 AM and enabled adequate perfusion. Throughout IABP and ECMO placement, CPR was performed by a regularly rotating team of healthcare personnel. As a result of sedation during ECMO placement, the patient's neurologic function could not be reliably determined; therefore, therapeutic hypothermia was initiated, with a temperature goal of 32 to 34 °C. The entire cardiac arrest lasted approximately 3½ hours.

The patient was transferred to a tertiary-care facility, where he remained in stable condition on ECMO and IABP support for several days. After being rewarmed and weaned from sedation, he had a full neurologic recovery. However, because of extreme rhabdomyolysis, he developed persistent renal failure that necessitated hemodialysis. On hospital day 10, he was bridged from the IABP and ECMO to left and right CentriMag® VADs (Thoratec Corporation; Pleasanton, Calif). On day 49, he underwent implantation of a SynCardia® temporary Total Artificial Heart (SynCardia Systems, Inc.; Tucson, Ariz). On day 160, he underwent orthotopic heart and cadaveric kidney transplantations. He was discharged from the hospital on day 179, at which time he was ambulatory.

Discussion

To our knowledge, ours is the only reported case in which a patient survived with good neurologic outcomes after a resuscitation that lasted as long as 3½ hours. We found 4 published reports of survival after prolonged resuscitation for cardiac arrest. The first patient was a 56-year-old man with no history of cardiac illness who collapsed and was found to be in VF. He was resuscitated by means of CPR, defibrillation, and the administration of epinephrine, atropine, and magnesium for 60 minutes; spontaneous circulation returned for 9 minutes but did not persist. The patient's initial ECG suggested an anterior myocardial infarction. Stenting of his occluded proximal anterior descending artery enabled his recovery and discharge from the hospital 3 months after admission.[8] The 2nd patient was an 82-year-old man in whom asystole developed after electroconvulsive therapy. He was treated with CPR and epinephrine for 54 minutes before placement of a pulmonary artery catheter and a pacemaker. Initial improvements in his vital signs and mental status were temporary, and his deteriorating condition necessitated increasing ventilatory and circulatory support. Care was withdrawn, and he died.[9] In the 3rd case, a 72-year-old man collapsed while mountain-climbing, and CPR was performed for 25 minutes while rescuers waited for an emergency team. The patient was defibrillated from VF, transported to a hospital, diagnosed with myocardial infarction, and treated by means of PCI. He recovered and was discharged from the hospital 24 days later.[7] The 4th patient was a 69-year-old woman who presented with non-STEMI and cardiogenic shock and underwent 60 minutes of CPR for VF and asystole. She underwent stenting and was discharged from the hospital 16 days after admission.[10]

Current guidelines suggest that asystole persisting for longer than 20 minutes without a known reversible cause is grounds for terminating resuscitation in a patient with atraumatic cardiac arrest.[5,6] However, it is generally understood that resuscitative efforts should continue for as long as VF persists. The guidelines take into account that every situation is different and that further research might yield better ways of predicting good and poor outcomes. As the field of cardiopulmonary resuscitative medicine develops and new techniques are implemented to improve survival outcomes in cardiac-arrest patients, some cases—such as our patient's—will defy the general rules. Following are some of the techniques that we think contributed to our patient's successful outcome.

The 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations describes good-quality CPR as the cornerstone of resuscitative efforts.[11] All rescuers, trained or not, should administer chest compressions to victims of cardiac arrest. High-quality compression involves pushing hard to a depth of at least 2 in (5 cm) at a rate of at least 100 compressions/min, allowing full chest recoil after each compression, and minimizing interruption of the process. In addition, trained rescuers should provide ventilations at a compression–ventilation ratio of 30:2.[12] Compression should resume immediately after defibrillation, without pauses to analyze a rhythm.[13] Evidence suggests that minimizing pauses in CPR improves the chance of recovery from shock. While our patient awaited more definitive treatment, chest compressions were executed sufficiently to maintain his mean arterial pressure above 60 mmHg.

Hypothermia has a neuroprotective effect after cardiac arrest. This effect might occur through reduction of cerebral oxygen demand and destructive enzymatic reactions, suppression of free-radical reactions, protection of the blood–brain barrier, reduction of intracellular acidosis, and inhibition of several neurotransmitters. The favorable effect of mild hypothermia on the mental status of patients who survive an out-of-hospital cardiac arrest is well documented.[14,15] No randomized trials have been conducted to test the effect of hypothermia in survivors of malignant arrhythmias after myocardial infarction specifically. However, results of one retrospective, nonrandomized study showed that patients with STEMI who were treated with primary PCI and externally induced hypothermia had a better calculated cerebral performance upon hospital discharge than did patients who underwent PCI under normothermic conditions.[16] Accordingly, therapeutic hypothermia is internationally recommended as early treatment for comatose survivors after cardiac arrest caused by VF.[17] We initiated a hypothermia protocol for our patient immediately after ECMO was begun.

As an adjunctive therapy to traditional ACLS management, ECMO has improved outcomes in terms of short-term resuscitation, survival to hospital discharge, and outcomes at 1 year.[18-27] Current techniques enable 44% of patients to survive to hospital discharge, and approximately 30% can be weaned from support directly. For those who cannot be weaned, ECMO can be used as a bridge to VAD placement or to heart transplantation. Because results in adults are modest when compared with those in children, risk and cost might make ECMO a poor option as adjunctive therapy for adult patients in cardiac arrest. However, many risks can be minimized by selecting appropriate patients, using ECMO as a bridge, and avoiding its prolonged use. When used as a bridge to VAD placement in patients who are too ill to receive a VAD directly, ECMO can improve outcomes to levels comparable to those of patients who are stable enough to receive direct VAD placement or heart transplantation.[28]

One technique not used in our patient but worth mention (because it is an area of ongoing interest) is the use of empiric thrombolytic agents for prolonged CPR. Multiple case reports have described successful resuscitation when thrombolytic agents were used after other interventions had failed. The rationale behind this therapy is the high prevalence of cardiopulmonary arrest triggered by pulmonary embolism or acute coronary syndrome. Early prospective controlled trials associated this technique with a low rate of complications.[29-33] However, subsequent larger trials have revealed no such benefit.[33,34] As a result, the current American Heart Association guidelines report no convincing evidence that the routine use of thrombolytic agents during resuscitation improves survival rates.[35]

The overall survival rate after cardiac arrest remains low. A meta-analysis[36] associated out-of-hospital cardiac arrest with survival rates of 24% to the time of hospital admission and 8% to hospital discharge. Survival was somewhat more likely when the cardiac arrest was witnessed, when the witness initiated immediate CPR, when the patient had a comparatively favorable presenting rhythm such as ventricular tachycardia or VF, and when the patient had intermittent ROSC. Survival rates did not vary significantly in distance from the hospital, the age of the patient, or emergency medical services' mean response time.[36] In regard to in-hospital cardiac arrest, observational investigators determined success rates of 39% to 72% for any ROSC, 25% to 42% for survival at 24 hours, 1% to 21% for survival to hospital discharge, and 11% to 14% for survival at 6 to 12 months.[1-4] Higher survival rates were associated with daytime cardiac arrest, younger age, shorter CPR duration, primary cardiac disease as the cause, cardiac monitoring, no intubation requirement, favorable initial rhythm, and witnessed arrest. Sex differences were not significantly associated with mortality rates.[1]

Survival rates after cardiac arrest remain low. However, despite poor overall outcomes, case reports of successful resuscitation after prolonged cardiac arrest give hope for improved overall outcomes in the future. Several factors contributed to our patient's recovery: he was relatively young, his arrest resulted from a ventricular arrhythmia caused by cardiac ischemia, and the arrest was witnessed on telemetry. The obvious preservation of his neurologic function throughout the arrest was pivotal in our decision to continue resuscitative efforts and pursue advanced cardiopulmonary support. We realize that our case is different from previously described cases of prolonged cardiac arrest, and the hope for patient survivability without substantial morbidity should be tempered with realistic expectations on an individual-patient basis. The substantial costs of aggressive cardiac therapies are another consideration before invasive intervention is undertaken. We submit our case as an example of how advances in resuscitative medicine can be used to promote survival in the appropriate setting.

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