CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 28-2013: A 52-Year-Old Man with Cardiac Arrest after an Acute Myocardial Infarction

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PRESENTATION OF CASE

Dr. Lisa A. Arvold (Emergency Medicine): A 52-year-old man was brought to the emergency department at this hospital because of chest pain and ST-segment elevations on electrocardiography (ECG).

The patient had been in his usual health, with hypertension, dyslipidemia, and coronary artery disease, until approximately 8:30 on the morning of admission, when his girlfriend found him lying on the floor of his home, unresponsive; she called emergency medical services (EMS). Shortly thereafter, he regained consciousness, rose to sit in a chair, and reported chest pain and dizziness. He took two nitroglycerin tablets (sublingually). On examination by EMS personnel at 8:42 a.m., he was alert and oriented and appeared uncomfortable; he had pale and diaphoretic skin and was grasping at his sternum and moaning. He reported that he had taken his regular daily aspirin (325 mg, orally) earlier. Respirations and pulse were normal; a faint carotid pulse was palpated; jugular venous distention was not visible. In the ambulance, cardiac monitoring revealed a sinus rate of 56 beats per minute, without ectopy. Nausea and vomiting developed. Supplemental oxygen was administered through a nonrebreather face mask at a rate of 15 liters per minute, and intravenous catheters (18 gauge) were inserted into the antecubital vein in the right arm and a vein in the left forearm. At 8:50 a.m., the blood pressure was 110/90 mm Hg, the pulse 51 beats per minute, the respiratory rate 18 breaths per minute, and the oxygen saturation 98%, with the patient reporting chest pain rated at 7 on a scale of 0 to 10, with 10 indicating the most severe pain. A 12-lead ECG showed sinus rhythm at 60 beats per minute; ST-segment elevation of 2 to 3 mm in the inferior leads (II, III, and aVF) and in leads V_3 through V_6 and of 2 mm in right-sided leads V_4 and V_5; and ST-segment depression with T-wave inversion in leads aVL, V_1, and V_2. Transport to this hospital began at 8:52 a.m. Three minutes later, the patient vomited, became increasingly pale and diaphoretic, and was unresponsive to voice. Normal saline (a 500-ml bolus) was administered. An attempt at tracheal intubation was unsuccessful. However, since a gag reflex was present, a nasopharyngeal airway was inserted instead, without complication. Thereafter, the patient was noted to be breathing on his own. The ambulance arrived at the hospital at 9:04 a.m.

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The patient had a history of coronary artery disease with unstable angina; 12 years before admission, at another hospital, a bare-metal stent (3.0 mm by 24.0 mm) had been placed in the middle portion of the left anterior descending coronary artery at the level of bifurcation with the first diagonal artery. Seven years later, angina recurred, and a nuclear stress test was positive for anterior ischemia; repeat cardiac catheterization at the other hospital revealed obstructing lesions (95% and 60%) in the left circumflex artery, and two drug-eluting stents were placed.

Medications the patient was taking before admission were unknown; in the past he had reportedly taken atorvastatin, clopidogrel, metoprolol, and aspirin (325 mg) daily. He had reported no allergies. The patient was divorced and lived with his girlfriend, with visits by his children. He worked in the construction and entertainment industries. Family members reported that he drank alcohol in moderation and had stopped smoking 11 years earlier. There was a family history of coronary artery disease.

In the emergency department triage area, the patient was alert and oriented; the blood pressure was 110/90 mm Hg, and the pulse 70 beats per minute. Within seconds after he arrived, the pulse decreased to 36 beats per minute and the width of the QRS complexes on the cardiac monitor increased. External pacing was attempted, without mechanical capture. Seizure activity occurred briefly, and runs of ventricular tachycardia with wide ventricular complexes were seen; the blood pressure and carotid pulses could not be obtained, and the patient was unresponsive. The cardiac monitor revealed ventricular fibrillation, cardiopulmonary resuscitation (CPR) was begun, and the patient was moved to a resuscitation bay.

The plasma level of total carbon dioxide was 19.5 mmol per liter (reference range, 23.0 to 31.9), the anion gap 17 mmol per liter (reference range, 3 to 15), the level of phosphorus 2.3 mg per deciliter (0.7 mmol per liter; reference range, 2.6 to 4.5 mg per deciliter [0.8 to 1.5 mmol per liter]), and the level of glucose 145 mg per deciliter (8.0 mmol per liter; reference range, 70 to 110 mg per deciliter [3.9 to 6.1 mmol per liter]). Blood levels of other electrolytes, calcium, magnesium, total protein, albumin, and globulin were normal, as were the results of a complete blood count and tests of liver and renal function; screening for troponin I was negative.

During resuscitation, the trachea was intubated. Amiodarone and lidocaine were administered intravenously and nine direct-current countershocks were administered. There was no resolution of the ventricular fibrillation.

Management decisions were made.

DISCUSSION OF MANAGEMENT

Dr. David F.M. Brown: The case of this patient highlights two major issues that must be decisively addressed in the emergency department. One is the management of myocardial infarction with ST-segment elevation (STEMI), and the second is ventricular fibrillation that persists after a number of shocks from an external defibrillator (shock-refractory ventricular fibrillation).

MANAGEMENT OF STEMI IN THE EMERGENCY DEPARTMENT

The primary goal in the management of STEMI in the emergency department is to speed the time to coronary revascularization. First, the revascularization strategy should be chosen. This decision is generally made at an institutional level and then applied to all patients; in this hospital, as in most large U.S. hospitals, the preferred strategy is primary percutaneous coronary intervention (PCI). The 2013 STEMI guidelines indicate that primary PCI is the preferred strategy if it can be performed promptly (i.e., for most patients, within 90 minutes after first contact with the health care system).[1] Intravenous thrombolysis remains a viable option but is used much less often in this country. Because thrombolytic therapy is infrequently chosen as the revascularization strategy for STEMI in academic medical centers, current trainees do not have experience with this method of treatment.

This patient had indications for revascularization therapy, which include a history of chest pain typical of ischemia that is less than 12 hours in duration, and one of the following: ST-segment elevations greater than 1 mm in two contiguous leads, a left bundle-branch block not known to be old, or ECG evidence of an isolated posterior-wall infarction. If thrombolytic therapy is chosen, there must be no absolute contraindications; it is better to keep an accessible checklist of contraindications than to rely on the

physician's memory. In general, primary PCI is chosen because it not only provides better clinical outcomes in terms of reducing ischemic end points but also (and perhaps most important) reduces the risk of hemorrhagic stroke to less than 0.1% as compared with approximately 1% with thrombolysis.[2]

ACTIVATION OF THE CARDIAC-CATHETERIZATION TEAM

Since outcomes in STEMI depend on rapid treatment, it is critical in the emergency department to communicate smoothly and quickly with colleagues in interventional cardiology while proceeding in parallel with adjunctive medical treatments. A number of institutional strategies can reduce the "door-to-balloon time" (the time from the patient's arrival at the emergency department to restoration of coronary blood flow) for patients with STEMI. The most important of these is the empowerment of the emergency physician to activate the entire catheterization team on the basis of a prehospital ECG.[3] That is what occurred in this case, and it allowed the catheterization team to mobilize while the patient was still en route to the emergency department. This is routine procedure now at most hospitals but was, nonetheless, an important evolution in terms of trust and relationships between two clinical services (emergency medicine and cardiology) during the past 10 years.

Several adjunctive treatments can be performed in the emergency department while waiting for the catheterization team to be ready, but none should delay the transfer of the patient. All patients should receive aspirin, generally 325 mg by mouth (as this patient did), except for those with a true allergy. Further antiplatelet therapy, with ticagrelor, prasugrel, or clopidogrel, can also be administered in the emergency department. Anticoagulation therapy with either unfractionated heparin or low-molecular-weight heparin can be initiated. A glycoprotein IIb/IIIa inhibitor may be administered, but this decision is best deferred to the staff in the catheterization laboratory. Intravenous beta-blockers may be considered in the emergency department but should be withheld both from patients with bradycardia, heart block, or signs of heart failure and from patients with a heart rate above 100 beats per minute or who are older than 75 years of age. Nitrates may be administered to patients with STEMI, but since they are not associated with a decrease in mortality, their use should generally be deferred in patients with suspected concomitant right ventricular infarction, aortic stenosis, recent use of phosphodiesterase type 5 inhibitors, or hypotension.

This patient took sublingual nitroglycerin in the prehospital setting; thereafter, on arrival at the emergency department, he collapsed. I believe that, in this case, the nitrates may have tipped the balance in terms of his arrest. Nitrates dilate both the arterial and the venous beds. Dilatation of the venous beds leads to a reduction in venous return, which can be very destabilizing in a patient with a right ventricular infarct.

MANAGEMENT OF SHOCK-REFRACTORY VENTRICULAR FIBRILLATION

High-quality chest compressions performed in the emergency department are critical to the survival of patients with STEMI complicated by shock-refractory ventricular fibrillation. Advanced airway management, antiarrhythmic agents, and epinephrine or vasopressin can be given while rapidly moving the patient to revascularization in the catheterization laboratory. This patient was moved to the catheterization laboratory while chest compressions were ongoing.

CORONARY REVASCULARIZATION IN STEMI

Dr. Farouc A. Jaffer: We were alerted to the arrival of this 52-year-old man who had an acute inferior STEMI with probable right ventricular infarction and shock-refractory ventricular fibrillation and on whom CPR was currently being performed. Our initial plan was to continue the advanced cardiovascular life-support protocol in the emergency department until the ventricular fibrillation was terminated, and then to transfer the patient to the cardiac catheterization laboratory for emergency primary PCI. However, in this case, ventricular fibrillation persisted. Since the shock-refractory ventricular fibrillation was most likely driven by ongoing ischemia and infarction, we next recommended immediate transfer of the patient to the cardiac catheterization laboratory, with ongoing CPR en route. We postulated that reperfusion with PCI might facilitate termination of the ventricular fibrillation. The patient spontaneously moved during CPR, almost sitting up at one point, and required sedation with midazolam and propofol. We thought this indicated reasonable neurologic function and proceeded with salvage PCI during ongoing CPR.

Videos showing coronary angiography are available at NEJM.org

Immediate coronary angiography was performed (Fig. 1A; and Video 1, available with the full text of this article at NEJM.org). Angiography revealed a thrombotic occlusion in the middle-through-distal portions of the right coronary artery due to coronary artery disease. First, we advanced a coronary guidewire into the distal right coronary artery. We then performed thrombectomy and retrieved a large, red thrombus.

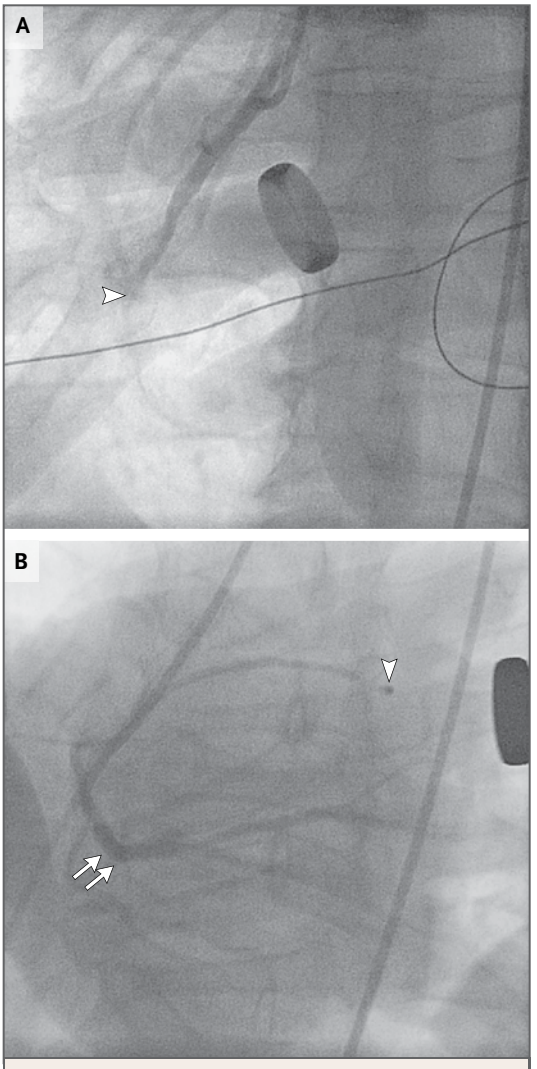

Figure 1. Coronary Angiogram.
A still frame from a cineangiogram shows that the middle portion of the patient's right coronary artery is occluded (Panel A, arrowhead). Flow in the right coronary artery is restored after placement of a bare-metal stent (Panel B, arrows); a temporary pacing wire is visible (arrowhead). In both panels, the elliptical radiopacity is a ring on the finger of a staff member in the cardiac catheterization laboratory who was performing CPR on the patient.

Next, during a 5-second pause in CPR, we implanted a bare-metal stent (3.5 mm by 23.0 mm) to restore flow in the right coronary artery (Fig. 1B and Video 2). CPR was resumed, and advanced cardiovascular life-support protocols were continued, with antiarrhythmic therapy and correction of metabolic derangements. An intraaortic balloon pump was then placed for hemodynamic support in case the patient's heart returned to an organized rhythm. Defibrillation was again attempted, and a few seconds of bradycardia were noted, but ventricular fibrillation returned. Electrophysiology consultation was requested, and a temporary pacing wire was placed in case bradycardia recurred.

Because of the persistence of ventricular fibrillation, we next considered the possibility that there was an additional coronary occlusion, especially since the patient had previously had stents implanted in the left anterior descending (LAD) coronary artery and the left circumflex coronary artery. Left coronary angiography was performed during CPR and revealed an acute occlusion of the middle portion of the LAD artery in the area of the previous bare-metal stent, a finding consistent with very late stent thrombosis (stenosis that occurs >1 year after implantation of the stent) (Video 3). The previous drug-eluting stents in the circumflex coronary artery were widely patent. Angioplasty of the LAD artery was performed, resulting in a 40% residual stenosis and restoring coronary flow (Video 4).

Defibrillation was attempted again but remained unsuccessful. CPR continued. We suspected that the patient's heart was too distended to permit termination of ventricular fibrillation, because we had performed CPR for more than an hour. A decision was made to consult cardiac surgery about the initiation of extracorporeal membrane oxygenation (ECMO) for full hemodynamic support and to decompress the heart and facilitate termination of ventricular fibrillation.

VENOARTERIAL ECMO FOR HEART SALVAGE

Dr. Joshua N. Baker: On the basis of evidence that early venoarterial ECMO improves the rate of survival among patients with cardiogenic shock after STEMI,[4] we were called for emergency venoarterial ECMO support after 91 minutes of very aggressive CPR and persistent ventricular fibrillation. Our team took the ECMO circuit to the catheterization laboratory. Our system consisted of a magnetically levitated, continuous-flow cen-

trifugal pump (CentriMag, Thoratec); a hollow-fiber oxygenator (Quadrox D, Maquet); and tubing with a bioactive coating. We achieved an activated clotting time of more than 250 seconds, and then performed percutaneous cannulation with a 25-French venous-return cannula placed through the right femoral vein into the right atrium. We then placed an 18-French arterial cannula percutaneously through the left common femoral artery for retrograde arterial perfusion. Careful, sterile connections to the circuit were made so as to not entrain any air into the system. After initiation of flows to approximately 4 liters per minute and decompression of the left ventricle, a 10th countershock (high-output defibrillation attempt) administered by the cardiology service resulted in conversion to sinus bradycardia, and the temporary pacing wire was adjusted to provide a ventricular pacing rate of 90 beats per minute. In addition, we placed an antegrade perfusion line down the superficial femoral artery on the left side for additional flow to the leg beyond the cannulation site. The acidosis and the requirement for vasopressors rapidly diminished.

For neurologic protection in this patient, we used a modified cooling protocol and cooled to 32°C. He was supported with ECMO for 72 hours. Initially, his left ventricle was essentially nonpulsatile, but within 24 hours after the initiation of ECMO, it started improving. The arterial pressure tracing during the first 24 hours, which had initially been flat, became increasingly pulsatile. There was rapid improvement in the ejection fraction on the third day, to 45%. To wean the patient from the ECMO device, we progressively reduced flows with additional anticoagulation under transesophageal echocardiographic guidance. A brief clamping trial proved that his ventricular recovery was sufficient for decannulation and the repair of arterial access, which were performed on the third hospital day.

THERAPEUTIC HYPOTHERMIA

Dr. M. Edip Gurol: The neurology service was consulted regarding the role of therapeutic hypothermia for this patient who had undergone prolonged CPR after having had a cardiac arrest due to ventricular fibrillation. Before arriving at the hospital, the patient had lost consciousness twice, at home and in the ambulance. For a brief period in the emergency department triage area, the patient was awake with normal neurologic function, but then he quickly had cardiac arrest due to ventricular fibrillation. CPR was immediately started. The success rate for out-of-hospital CPR after cardiac arrest is less than 10%, and of every five patients resuscitated in the hospital, an average of less than one survives to discharge.[5,6] Even among patients who have a return of spontaneous circulation, the mortality rates are exceedingly high (>70% for out-of-hospital cardiac arrest, and 67% for in-hospital cardiac arrest).[3,7,8] Several studies suggest that a delay between collapse and the initiation of CPR, a longer duration of CPR, and nonshockable cardiac arrhythmias can predict poorer outcomes.[9,10] At this hospital, decisions on whether to treat patients with hypothermia are not based on these indicators, since the indicators may exclude some patients who may benefit from the treatment (http://www2.massgeneral.org/stopstroke/protocolHypothermia.aspx). This patient had one of these three risk factors (prolonged CPR).

Hypothermia has been shown to improve neurologic outcomes in patients who are still in a coma after cardiac arrest and the subsequent return of spontaneous circulation. In two large, randomized trials, patients who were cooled to 32 to 34°C shortly after the return of spontaneous circulation and maintained at that temperature for 12 to 24 hours were more likely to have good recovery or mild-to-moderate disability than were controls (relative risk, 1.4 to 1.9).[11,12] A smaller study showed some benefit from a shorter course of hypothermia (4 hours) among patients who had cardiac arrest due to asystole or pulseless electrical activity.[13,14] A Cochrane review confirmed that, among patients who are in a coma after cardiac arrest, those who undergo therapeutic hypothermia are more likely to have a good neurologic outcome (relative risk, 1.55; 95% confidence interval, 1.22 to 1.96) than are those who receive standard care.[14] At this hospital, we use therapeutic hypothermia regardless of the type of cardiac arrhythmia (ventricular fibrillation, ventricular tachycardia, asystole, or pulseless electrical activity) for both out-of-hospital and in-hospital cardiac arrests, as long as other criteria are fulfilled.[15]

In most cases, the initial postarrest neurologic examination is performed after the return of spontaneous circulation. However, in this case it was performed while the patient was receiving ECMO. After the patient had been unsedated for 10 minutes, he was unresponsive to verbal and

painful stimuli. He had no eye opening, no response to commands, no brain-stem reflexes, and no meaningful motor response in the arms or legs. Cooling was started immediately after the examination was performed, and the administration of propofol was restarted. One critical issue for this patient was that his arrest was witnessed while he was in the hospital. Patients who have cardiac arrest in the hospital have a better chance of recovery if the event was witnessed or monitored.[16] Earlier induction of mild hypothermia might also be a determinant of good neurologic outcome, although contradictory results have been reported.[17,18]

Cooling was maintained for 24 hours, followed by rewarming. When sedation was subsequently lightened, the patient was immediately able to follow simple commands. He opened his eyes spontaneously and responded to verbal and tactile stimuli. He had no focal motor deficits. The good neurologic response seen early in this patient is consistent with data from 2011 that suggest that therapeutic hypothermia in itself does not delay the time to awakening in patients with cardiac arrest.[19] This patient had no seizures after the initial presentation, and the head CT after cardiopulmonary stabilization revealed no hemorrhage or other acute pathologic features. His cognitive status improved quickly after extubation, and he has not had any subsequent neurologic problems. Starting CPR immediately and early institution of hypothermia might have contributed to the good neurologic recovery that the patient has made, despite the prolonged CPR that he needed, further suggesting that this factor need not exclude a patient from receiving therapeutic hypothermia.

Dr. Jaffer: Dr. Baker and I have followed the patient for the past 30 months. His hospital course was fairly complicated. He required a tracheostomy, a feeding tube, and renal-replacement therapy. He was eventually discharged to a rehabilitation facility, 2.5 months after admission.

Before the patient's first hospital discharge to rehabilitation, a cardiac ultrasound examination revealed nearly normal ventricular function. Soon after discharge, he was readmitted to this hospital with pancreatitis. During this admission, congestive heart failure developed, and the ejection fraction declined to 34%. Cardiac ultrasonography revealed a left ventricular thrombus, and the administration of warfarin was begun. He did not have evidence of angina or ischemia; therefore, coronary angiography was deferred. As the pancreatitis and associated inflammatory response improved, the symptoms of congestive heart failure resolved. The ejection fraction improved to 43%, and the left ventricular thrombus resolved at 1 month after the initial diagnosis.

Three months later, mild congestive heart failure developed. The administration of diuretics was initiated. A chemical stress test revealed evidence of a moderate-to-large incomplete anterior, anteroseptal, and anterolateral infarction associated with moderate ischemia and hypokinesis. Coronary angiography revealed severe two-vessel (right coronary artery and LAD coronary artery) disease. The stent that was placed in the right coronary artery during the STEMI was patent, but there were severe lesions flanking the stent. In addition, the previous stent in the LAD artery, on which angioplasty was performed during the initial hospitalization, had severe restenosis. The patient underwent PCI of the right coronary artery and the LAD artery, and three drug-eluting stents were placed. Congestive heart failure subsequently resolved. He completed cardiac rehabilitation. For the past 2 years, he has had no angina, heart failure, or cardiovascular limitation. He continues to receive aggressive medical treatment for secondary prevention of coronary artery disease.

The patient has normal neurologic function. He has returned to his former profession as a musician and to his role as father to two children.

Dr. Baker: Ischemia developed in the distal left foot and toes from vasopressor usage during the patient's initial hospitalization (Fig. 2A). After consultation with vascular surgery, a suggestion was made for a midfoot amputation to speed his recovery, but the patient is a professional drummer and wanted to keep the foot. Troops injured in Iraq used extracellular matrix in situations such as this one; on this basis, we applied extracellular matrix (Wound Matrix, Oasis) to the patient's wounds, initially every 3 days and then weekly; after 30 outpatient visits, the foot healed (Fig. 2B) to the extent that he is able to drum with it, although he has had episodes of osteomyelitis requiring débridement and antibiotics.

Dr. Brown: Are there any questions?

A Physician: What would you have done if the patient had had ventricular fibrillation without

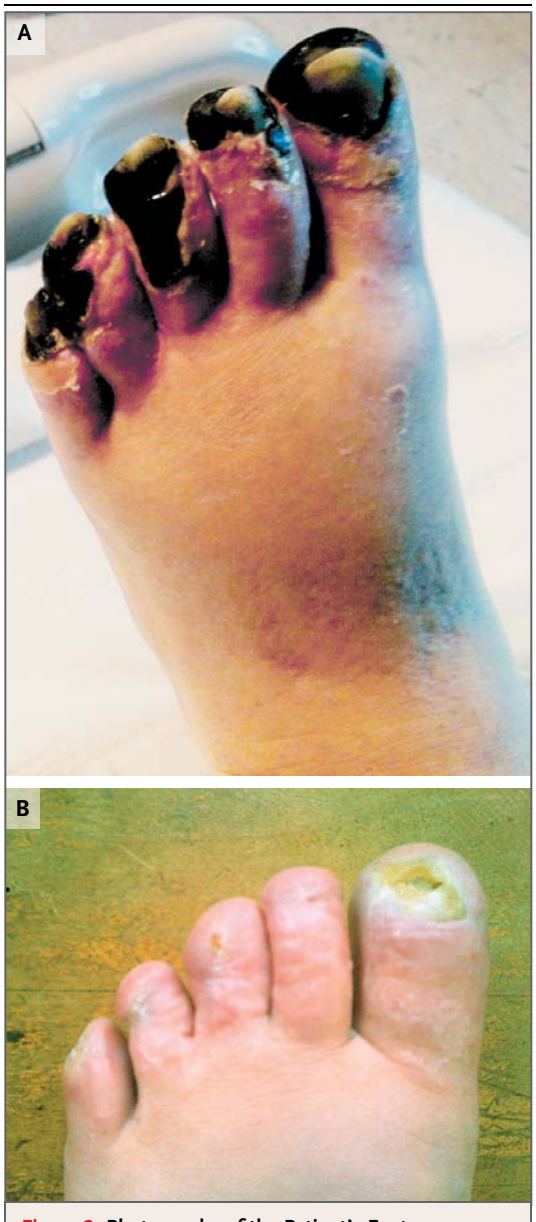

Figure 2. Photographs of the Patient's Foot.
While the patient was in the intensive care unit, there was gangrene of all toes on the left foot (Panel A). One year later, after multiple weekly applications of extracellular matrix, substantial healing is evident (Panel B).

ST-segment elevations on his ECG? Should we activate the catheterization laboratory for people who have had an arrest caused by ventricular fibrillation but who have do not have ST-segment elevation on their ECG?

Dr. Jaffer: Ventricular fibrillation may have causes other than acute myocardial ischemia or infarction; therefore, an episode of ventricular fibrillation without ST-segment elevation or a pattern of new left bundle-branch block on the previous or subsequent ECG is not a routine indication for emergency activation of the cardiac catheterization laboratory. Cardiology consultation is indicated to guide further management.

A Physician: Is there a role for initiating ECMO before catheterization?

Dr. Jaffer: Yes. Dr. Baker and I have had two similar cases since this initial one, and we have initiated ECMO in the cardiac catheterization laboratory before PCI for the following three reasons: full cardiac support is attained immediately, allowing perfusion of the vital organs and limiting the risk of multisystem organ failure; cerebral cooling through the ECMO circuit is initiated earlier; and it is technically easier to perform PCI with ECMO support rather than during ongoing CPR. Both of these patients also survived with intact neurologic function.

A Physician: I can easily see that at any stage during this CPR, any of you could have decided not to continue with it. How did you decide to continue?

Dr. Jaffer: Patient selection is important in determining who will benefit from ECMO support during fulminant cardiogenic shock in the context of an STEMI. Chances of survival are higher in younger patients, those who receive immediate CPR, those without chronic illnesses, and those who undergo successful primary PCI.

Dr. Brown: Most compelling for me, as the physician who saw him essentially at the front door of the hospital, was that the patient was awake, alert, and neurologically normal on arrival and that he had what appeared to be a reversible condition — ventricular fibrillation. If he had had an out-of-hospital arrest or if the prehospital ECG had not shown clear ST-segment elevation, then the likelihood of a good clinical outcome would have been extremely low, and I don't think we would have activated the interventional team.

ANATOMICAL DIAGNOSIS

Cardiac arrest due to ventricular fibrillation caused by acute myocardial infarction with ST-segment elevation (STEMI).

This case was discussed at Emergency Medicine Grand Rounds.

Dr. Jaffer reports receiving consulting fees from Boston Scientific. No other potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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