was working the night shift in the small emergency room of a satellite clinic during my internal medicine residency in India. I was still a jittery intern with little hands-on experience, and I had not handled a code before. As interns, my coworkers and I were the only medical staff taking care of urgencies and we had little backup, a familiar scenario to most of my colleagues in India and other developing countries.

Around 12 AM, a male patient in his mid-40s presented with severe crushing substernal chest pain of 45 minutes’ duration. He had no other comorbidity except mild obesity. He had accelerated hypertension and a blood pressure of 230/130 mm Hg with no congestive failure. Electrocardiography revealed ST segment elevation in precordial leads V1 to V4.

As I was thinking about thrombolysis in this patient, I was also faced with the problem of lowering his blood pressure. Sodium nitroprusside was not available; only intravenous nitroglycerin was on hand. Only one nurse was available to help me with the patient, and no telephone contact with other medical staff or resources was possible. We rapidly built up the nitroglycerin dose and were able to decrease the patient’s blood pressure in 15 minutes, but, as we began thrombolysis with intravenous streptokinase, the patient went into cardiac arrest.

We had a portable cardiac monitor-defibrillator and just as the patient was beginning to blue, the monitor showed ventricular fibrillation. I had little time to think about the patient’s airway, and I was still shaky about what to do first as I defibrillated him. Fortunately, a normal sinus rhythm returned and the patient’s color was promptly restored. We then administered a lidocaine infusion.

A few heartbeats later, the patient went into accelerated hypertension again. I immediately discontinued the streptokinase infusion and again built up the dose of intravenous nitroglycerin. After his blood pressure decreased, I resumed intravenous thrombolysis.

As I prayed that nothing else bad would happen, a calm 15 minutes passed—but then the patient went into ventricular fibrillation again. We reenacted the whole scenario: defibrillate, decrease blood pressure, resume thrombolysis. I could do nothing other than increase the maintenance infusion of intravenous lidocaine. Amiodarone was not available at that time, and I could not think of any other treatment strategies.

Throughout the night, the patient experienced cardiac arrest six times and was successfully resuscitated each time. My shift ended around 7 AM, and, at that time, the patient had kept a normal sinus rhythm for more than 1 hour. He was pain free, his blood pressure was well under control, and repeat electrocardiography showed a non-Q infarct.

The patient underwent cardiac catheterization later that day. The procedure revealed a proximal, tightly occluded, left anterior descending coronary artery, which was opened and stented. The patient had normal left ventricular function, and no echo regional wall motion abnormality was detected. He was successfully discharged 72 hours after the procedure.

When I think about this patient, I always wonder at the rapid sequence of events, what the final outcome was, and what the outcome could have been. Most gratifying is that I did everything possible under the circumstances and that the patient responded so well.

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