Spinal Cord Ischemia Caused by Cardiac Arrest Secondary to Pericardial Effusion

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The reported rates of pericardial effusion after cardiac surgery are as high as 64%.1 However, it is rare for this to lead to life-threatening conditions such as cardiac arrest. It is very difficult to maintain cardiac output during cardiopulmonary resuscitation (CPR) in the setting of pericardial effusion. In such cases, declining perfusion pressure primarily results in neurologic complications, specifically anoxic brain damage, cerebrovascular accident, and/or seizures. It is uncommon to see paraplegia alone (ie, without accompanying cerebral complications) after CPR.2 However, other predisposing factors such as diabetes, hypertension, and atherosclerotic disease of the lower extremities may contribute to an increased incidence of spinal cord ischemic injury in this setting. The authors present a rare case in which acute paraplegia developed after prolonged cardiac arrest secondary to pericardial effusion.

CASE REPORT

A 68-year-old woman was admitted to the hospital with the complaint of difficulty of breathing. She had undergone coronary artery bypass grafting 1 month previously and had diabetes mellitus, hypertension, and atherosclerotic occlusive disease of the lower extremities. The patient was also seropositive for hepatitis-B surface antigen. Her blood pressure, heart rate, and respiratory rate on admission were 99/45 mmHg, 110 beats/min, and 20 breaths/min, respectively. Physical examination revealed decreased respiratory sounds in the lower left hemithorax. Bleeding time, prothrombin time, and a complete blood count were normal. A chest x-ray showed moderate cardiomegaly. Hematocrit and white blood cell count were normal. A chest x-ray showed moderate cardiomegaly. Echocardiography revealed moderate pericardial effusion but no cardiac tamponade.

On arrival at the intensive care unit, a peripheral intravenous catheter was placed. After electrocardiographic and pulse oximetry monitoring, a left radial artery catheter was inserted with local anesthesia. Meanwhile, the patient became bradycardic and rapidly progressed to electromechanical dissociation culminating in ventricular fibrillation. CPR was started immediately, and she was rapidly intubated. The algorithm for advanced cardiac life support was followed for a total of 50 minutes.1 There was no initial response to epinephrine boluses and 100% oxygen. Circulation was restored after fluid administration (200 mL of 5% human albumin and 1,000 mL of other colloid solution). During the course of CPR, the patient received a total of 15 mg of epinephrine.

Two attempts at pericardiocentesis were made during CPR, but they were both unsuccessful. Immediately after the patient responded to CPR, a tube was placed in the pericardiac sac via a subxiphoid approach, and 200 mL of serosanguineous fluid were drained. The patient’s heart rate, cardiac rhythm, and blood pressure gradually improved. She regained full consciousness 6 hours after the arrest and complained of weakness and sensory abnormalities in her lower limbs at that time. Neurologic examination revealed flaccid paralysis of the lower extremities and loss of anal tone. Intravenous dexamethasone (8 mg 4 times a day) was initiated. This regimen was maintained for 1 week, and the dose was then gradually tapered off over 2 weeks. On detection of the paralysis, a cerebrospinal fluid catheter was immediately inserted by the anesthesiologist. Fifty-five milliliters of fluid were drained over the first 48 hours. The drainage kept the cerebrospinal fluid pressure at 10 mmHg, but there was no change in the patient’s neurologic deficit. Magnetic resonance imaging showed an abnormal hyperintense signal within the spinal cord extending from T7 to L1 (Figs 1 and 2).

The patient was extubated on day 6 after cardiac arrest, but respiratory distress and hypotension recurred on day 15. Echocardiography at that time revealed recurrence of the pericardial effusion: it was decided to operate and drain the fluid. After 17 days of mechanical ventilation, a tracheostomy was performed, and the patient was gradually weaned to spontaneous ventilation. At 3 weeks after the arrest, spasticity of both lower limbs and improved anal tone were noted. However, the muscle power in the lower extremities did not improve further. Gastrointestinal bleeding was detected on day 28, and endoscopy was performed 3 times. A duodenal ulcer was found and electrocautery with sclerotherapy were used. Because of the recurrence of bleeding, the patient underwent a laparotomy. Pyloroplasty together with bilateral truncal vagotomy were performed on day 31. No active bleeding site was found during the operation. The patient’s general status continued to deteriorate gradually, and she died on day 38 because of multiple-organ failure.

DISCUSSION

Spinal cord infarction can be caused by a variety of circulatory abnormalities, including embolic disease, vasculitis, cardiac arrest, aortic dissection, and complications after aortic surgery or spinal angiography.4 The exact incidence of paraplegia after cardiac arrest is not known. In a large series of 44 cases of ischemia and infarction of the spinal cord, 9% of the cases occurred after cardiac arrest.5 The literature contains only 1 case report of paraplegia after cardiac tamponade.2 In that case, a 16-year-old patient went into shock and then cardiac arrest 2 weeks after surgical repair of an atrial septal defect. He responded to CPR after pericardiocentesis was performed but was paraplegic when he regained consciousness 4 hours after the cardiovascular event.

Hypertension, diabetes, and atherosclerotic occlusive disease of the lower extremities were present in addition to pericardial effusion in the present case. This patient also developed infarction of the lower thoracic and lumbar cord with no accompanying cerebral deficit. In 1 autopsy study of 83 cardiac arrest patients who had anoxic-ischemic central nervous system injury, the investigators found a lumbar sacral spinal cord injury in 49.4% of the cases.6

The anatomic distribution of arteries supplying the spinal cord includes a region called the “ischemic watershed zone” between T4 and T8. This zone is so named because it has comparatively fewer radicular arteries than the supply to other portions of the cord. Considering this anatomy, the thoracic

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1053-0770/07/2101-0018$32.00/0


Key words: pericardial effusion, cardiac arrest, spinal cord ischemia
region of the spinal cord would be expected to have a higher frequency of ischemia than other segments, but in fact it is the lumbar portion that is most prone. There are 2 main reasons for this susceptibility: (1) lumbosacral neuronal tissue has higher metabolic demands than other cord regions because of the extensive motor neuron activity that occurs in this zone and (2) atherosclerosis tends to be more pronounced in vessels of the lower body. The latter include branches that supply the spinal cord, and this means that the distal spinal cord has lower perfusion pressure than more proximal portions.

The incidence rates of spinal cord ischemia in studies in which patients’ neural status has been assessed solely by clinical examination is much lower than the rates noted in autopsy evaluations. The reason for this is that spinal cord ischemia is underdiagnosed because most patients have coincidental cerebral ischemia, which is the main determinant of the clinical presentation. The present case involved a prolonged course of CPR. Because of pericardial effusion during this period of CPR, the patient’s arterial pressure might have been sufficient to maintain cerebral perfusion but not spinal cord perfusion. Also, hypoperfusion might have been aggravated by associated aortic atherosclerosis involving the proximal thoracic artery or the origins of radicular arteries. Another potential underlying factor in this case might have been rupture of an atherosclerotic plaque from compressions during prolonged CPR. The patient had known atherosclerotic disease in her aorta and could have had a plaque at the origin of the radicular arteries, which provide critical supply to the spinal cord.

This patient had a pericardial effusion that was not relieved during CPR. As a result of the effusion, during CPR her mean arterial pressure remained in the 40- to 55-mmHg range. The authors suspect that this pressure was adequate for cerebral perfusion but not for spinal cord perfusion. This case shows that a patient with pericardial effusion who has undergone prolonged resuscitation can develop spinal cord infarction without cerebral infarction.

REFERENCES