Hepatic Rupture after Cardiac Arrest

– A Case Report –

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A closed-chest cardiac massage, although a lifesaving procedure, invariably is fraught with potential complications[1–4]. We describe liver tauma brought about by closed-chest cardiac massage for cardiac arrest due to pulmonary embolism after laparotomy.

Case Report

A 49-year-old woman, 154 cm in height and 58 kg in weight, underwent a radical hystrectomy for uterine cancer. Five days later, she suddenly complained of a severe chest pain, and subsequentry fell into cardiac arrest. Cardiopulmonary resuscitation (CPR), including endotracheal intubation, closed-chest cardiac massage, and an intravenous injection of epinephrine was immediately instituted.

She fortunately was resuscitated, and dispatched by ambulance to our hospital. On the way, however, she received closed-chest cardiac massage three times. On arrival in our emergency room, she was in deep coma, and had a heart rate (HR) of 108 beats per minute (bpm) and an arterial blood pressure (BP) of 60/40 mmHg despite norepinephrine infusion at a rate of 0.2–0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Further, a chest x-ray revealed cardiomegary. A blood gas analysis revealed a Pa_{CO_2} of 44 mmHg, a Pa_{O_2} of 175 mmHg and a BE of -15 $mEq \cdot l^{-1}$ with an F_{IO_2} of I.O.

Echocardiography and pulmonary angiography confirmed a pulmonary embolism to suspiciously be the cause of cardiac arrest. At this time pulmonary arterial pressure (PAP) was 54/23 mmHg, and we start anticoagulation therapy with intravenous heparin 5,000 units, followed by an infusion of heparin of 500 units per hour. Additionally, we administered urokinase, a thrombolytic agent, at a rate of 24,000 units per hour. Four hours after pulmonary angiography, PAP decreased to 23/10 mmHg, and the administration of urokinase was discontinued.

Her laboratory data on admission were as follows: hematocrit, 23%; platelets, 185,000 ul^{-1}; SGOT, 1611 $IU \cdot l^{-1}$; SGPT, 1088 $IU \cdot l^{-1}$; LDH, 13257 $IU \cdot l^{-1}$; total bilirubin, 0.5 $mg \cdot dl^{-1}$. After transfusion of 8 units (1600 ml) of packed red blood cells over twenty-four hours, hematocrit was 31% and hemodynamics improved; BP was 104/68 mmHg, HR was 115 bpm, cardiac output was 5.0 $l \cdot min^{-1}$, pulmonary capillary wedge pressure (PCWP) was 5 mmHg and PAP was

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23/10 mmHg with a continuous infusion of dopamine at a rate of 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

Her neurological condition also improved to wince in response to pain stimuli and she began to rub her epigastric area making grimace. Then her abdomen slightly distended. An ultrasound study revealed echo-free space in the abdominal cavity, which proved to be blood on aspiration. We first thought that this abdominal bleeding originated from the surgically manupulated area and might be provoked by urokinase and heparin. Fourty-two hours after admission to ICU, BP suddenly fell to 54/37 mmHg, HR was 184 bpm and PCWP was 2 mmHg. At the same time, her abdomen further distended. Hematcrit dropped to 21% despite transfusion of 4 units (800 ml) of packed red blood cells.

Emergency laparotomy revealed this bleeding did not originate from the area of her hysterectomy but from the liver. A large subcapusular hematoma and two lacerations near the broad ligament were identified. As the lacerations and hematoma were localized in the left lobe, the left-lateral lobe was resected. Only then we deduced that the damages were induced by the closed-chest cardiac massage. Her postoperative course was uneventful and she left ICU eight days later without any neurological defecits.

Discussion

Although closed-chest cardiac massage is the essential procedure of CPR, this may cause some complications[1-4]. Pneumothorax following rib or sternal fractures is common complications. As rare and more serious complication, gastric rupture[6], laceration of the lier[5], or rupture of the heart itself[7] have been reported. If the lower part of the sternum or the xiphoid is compressed, liver trauma may occur. Additionally, should cardiac massage be performed at an unstable place such as in an ambulance and/or be done frequently, the risk of liver trauma will increase.

Almost all liver traumas due to CPR are discovered on autopsy, and a composite review of 557 autopsies of patients who received CPR demonstrates a 2.9% incidence of liver traumas[5], and the range in each study extending from 0% to 11%[1,2]. To our knowledge, our patient is the second case that survived a liver trauma after CPR. The first case was reported by Adler et al. in 1983[5], wherein two patients with a liver trauma resulting from CPR for ventricular fibrillation due to an acute myocardial infarction were described. One patient was saved by conservative therapy for liver trauma but the other died of multiple organ failure in spite of liver surgery.

In contrast to Adler's patients, who were neurologically intact soon after defibrillation and could complain of severe epigastralgia, our patient was comatose and could not verbally explain her condition. We first suspected that postsurgical bleeding caused hypovolemia and speculated that SGOT, SGPT and LDH increased as a result of postresuscitation hepatic ischemia. However, based on our subsequent findings, we found that liver laceration induced by CPR was a possible cause of bleeding.

Heparin was used as the anticoagulation therapy in both our case and Adler's case for pulmonary embolism and for acute myocardial infarction[5]. In addition, our case received thrombolytic therapy, which might enhance bleeding, then led to hypovolemic shock. Thrombolytic and anticoagulation therapy after CPR may increase the risk of bleeding from the liver trauma.

If abdominal bleeding with hypovolemic shock occurs in a patient after CPR who had received a laparotomy, postsurgical bleeding may

first be suspected as a cause of hypovolemia. However, more attention should be paid to liver trauma. This is even more imperative in the patient who received CPR at an unstable place and/or needed frequent closed-chest cardiac massage.

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