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DELAYED VISCERAL BLEEDING FROM LIVER INJURY AFTER CARDIOPULMONARY RESUSCITATION

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□ **Abstract—Background: Visceral injury is a life-threatening complication of cardiopulmonary resuscitation (CPR); however, the clinical significance has been masked by the lethal outcome of out-of-hospital cardiac arrest (OHCA). Objective: The objective is to share our experience of successful treatment of OHCA patients with serious, CPR-related visceral complications. Case Reports: We report two cases of cardiac-origin OHCA with liver injury exacerbated by heparinization during mechanical circulatory support. Although both patients presented with delayed massive liver bleeding (intrahepatic or peritoneal) that compromised hemodynamic status, one patient was successfully treated by selective transcatheter arterial embolization and the other by a surgical procedure. Conclusion: Preventive measures such as careful CPR, as well as interventional or surgical repair after the early diagnosis of visceral injury, are required to improve the outcome in some cases of OHCA.** © 2012 Elsevier Inc.

□ **Keywords—liver injury; cardiopulmonary resuscitation; cardiac arrest; heparinization; mechanical circulatory support**

INTRODUCTION

Visceral injuries from cardiopulmonary resuscitation (CPR) can affect the morbidity and mortality of cardiac arrest patients despite early successful resuscitation. We report two cases of successful management of serious liver bleeding associated with CPR, and subsequent anticoagulation therapy.

CASE REPORT

Case 1

A 53-year-old man with a history of coronary artery bypass grafting 7 years prior presented with a witnessed, out-of-hospital cardiac arrest. Bystander CPR was not performed. Because the initial cardiac rhythm was ventricular fibrillation (VF) and there was no return of spontaneous circulation (ROSC) despite automatic external defibrillation, basic life support was performed during transportation by emergency medical technicians. These individuals had passed a state examination for qualification and were permitted to insert an intravenous line and an alternative airway management device and to use a semi-automated external defibrillator provided that they had online confirmation by a medical doctor. Soon after arrival at the emergency department (ED), ROSC was achieved by a monophasic shock of 150 J. The duration of CPR was 10 min. The emergent coronary angiography showed the patency of the bypass grafts and good collateral perfusion; therefore, VF was presumed to be a consequence of the old

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myocardial infarction. An intra-aortic balloon pump (IABP) was inserted with concomitant administration of heparin (625 IU per hour) and mild hypothermia was induced to prevent post-cardiac arrest brain injury. The target body temperature of 34 °C was achieved 7 h after admission. The patient's hemodynamics were stable and the ejection fraction measured by echocardiography was approximately 40%. Twenty hours after admission, the hemodynamics gradually deteriorated to blood pressure 70/40 mm Hg and heart rate 70 beats/min. Electrocardiogram and left ventricular function by echocardiography showed no significant change, and the hemoglobin concentration was 15.9 g/dL. Sedation dose reduction and low doses of noradrenaline (norepinephrine) were required to stabilize the hemodynamics. Four hours after the first episode of hypotension, the hemodynamics remained unstable and the hemoglobin concentration dropped to 11.4 g/dL, and the activated partial thromboplastin time (aPTT) was within the appropriate range, suggesting the existence of bleeding. Because echocardiography showed a massive heterogeneous echoic mass in the right lobe of the liver, contrast-enhanced computed tomography (CT) scan was performed after stabilizing the hemodynamics by plasma and red blood cell transfusion. There was a massive liver parenchymal hematoma found with active arterial bleeding (Figure 1A), and emergent angiography and selective transcatheter arterial embolization were performed (Figure 1B). After embolization, the hemodynamics became stable without transfusion, and the patient was weaned off mechanical ventilation with good neurological recovery on the 12th hospital day.

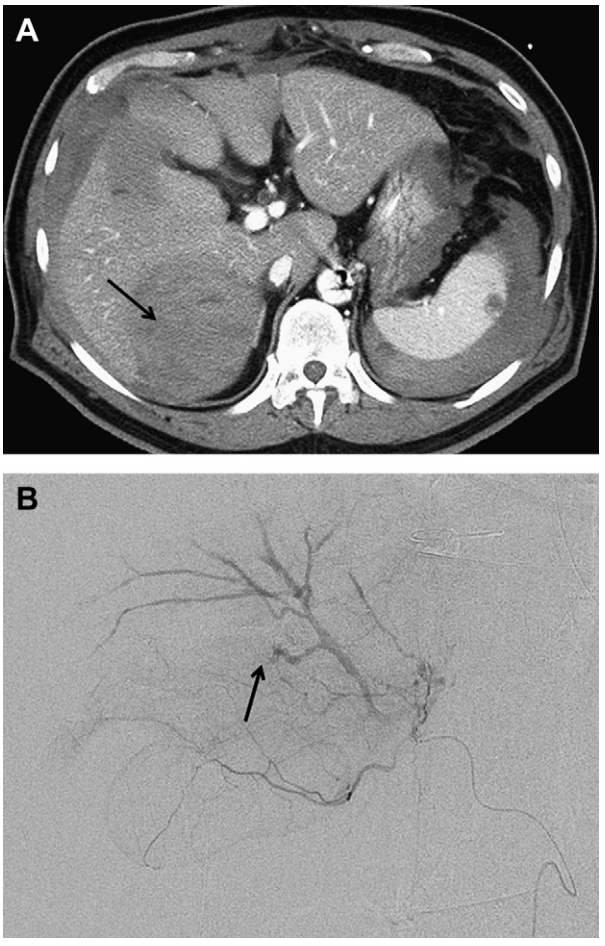

Figure 1. (A) Contrast-enhanced computed tomography scan in the portal phase showing a massive liver parenchymal hematoma in the right robe (arrow). (B) Selective angiography reveals successful embolization of the posterior branch of the right hepatic artery (arrow).

Case 2

A 50-year-old man with diabetes mellitus and dyslipidemia developed a witnessed out-of-hospital cardiac arrest due to acute myocardial infarction. Bystander CPR was performed for 19 min. However, the initial cardiac rhythm was VF and the automatic external defibrillation failed to achieve ROSC. Mechanical chest compressions were delivered with the AutoPulse™ (ZOLL Medical Corporation, Chelmsford, MA) during transportation by helicopter. Because refractory VF continued, a cardiopulmonary bypass (CPB) was performed immediately upon arrival at the ED. Emergent coronary angiography revealed occlusion of the left anterior descending artery, and successful revascularization was performed using a coronary stent. An IABP was inserted and mild hypothermia was induced. Loading doses of aspirin and ticlopidine (160 mg and 300 mg per day, respectively) and heparin (625 IU per hour) were administered. After the revascularization, the hemodynamics became stable. On the second hospital day, the usual doses of aspirin and ticlopidine were administered (100 mg and 75 mg per day, respectively), but heparin was titrated down to 100 IU per hour due to an aPTT of more than 150 s and anemia refractory to transfusion. Transfusion of massive loading doses of fluids was required to maintain the extracorporeal circulation. Abdominal distension appeared on the third hospital day. Echocardiography showed massive intraperitoneal fluid, and subsequent contrast-enhanced CT scan revealed liver injury with active bleeding in the right lobe (Figure 2A). The severely reduced left ventricular function at that time made it impossible to wean the patient from CPB, which principally would require anticoagulation therapy. Emergent surgery was performed, showing massive hemoperitoneum (approximately 5.5 L of blood) due to the liver laceration. Hemostasis was performed surgically and was successful without significant cardiac events (Figure 2B). Platelet inhibitors and heparin were discontinued, and the patient was weaned from CPB and IABP successfully, without thromboembolic events, on the 4th and 6th hospital day, respectively. Unfortunately, the patient ended up with