

# Traumatic Liver Injury Complicating Cardio-pulmonary Resuscitation. The Value of a Major Intensive Care Facility: A Report of Two Cases

M. D. ZIEGENFUSS, D. V. MULLANY

*Intensive Care Facility, Prince Charles Hospital, Brisbane, QUEENSLAND*

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## ABSTRACT

*Complications associated with external cardiac compression include trauma to the heart, chest wall, lungs and gastrointestinal viscera, with gastrointestinal visceral injury including, ruptured stomach, liver, oesophagus, spleen and colon. However, the use of thrombolytics and anticoagulants in patients with an acute myocardial infarction increases the incidence of a visceral haemorrhage when these patients need cardiopulmonary resuscitation. We report two out-of-hospital cardiac arrest patients whose immediate post-resuscitation phase was complicated by hepatic injury and significant haemoperitoneum. Conservative management in a major intensive care unit of both the liver trauma and the induced coagulopathy was associated with a successful outcome in both cases. (Critical Care and Resuscitation 2004; 6: 102-104)*

**Key words:** Cardiopulmonary resuscitation, haemoperitoneum, liver trauma

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An out-of-hospital cardiac arrest has a poor prognosis if rapid access to cardiopulmonary resuscitation (CPR), early defibrillation and advanced medical care are not promptly implemented, with survival rates often varying between 3 - 10%. However, one study of patients who had a successful defibrillation following an out-of-hospital cardiac arrest from ventricular fibrillation reported a 40% survival to hospital discharge.<sup>1</sup>

Improved access to defibrillation and new therapies with intensive care management may potentially result in more survivors.<sup>2</sup> We report two out-of-hospital cardiac arrests whose post-cardiopulmonary resuscitation phase was complicated by traumatic liver injury and significant haemoperitoneum. Careful management in a major intensive care unit was associated with a successful outcome in both cases.

## CASE REPORTS

*Patient 1.* A 41-year old man had a witnessed cardiac arrest near an ambulance station. The first recorded rhythm was ventricular fibrillation. He

received ten minutes of CPR and was defibrillated twice before he reverted to sinus rhythm with restoration of his circulation. He was intubated at the scene and transferred to a tertiary referral center. Due to widespread anterior ST elevation, intravenous tenecteplase (100 mg) was administered, however with no resolution of the ST segment elevation he was transferred to our hospital for cardiac catheterisation.

Coronary angiography revealed a total occlusion of the mid left anterior descending coronary artery which was reopened using a stent. An intra-aortic balloon pump was also inserted. There was prompt resolution of the precordial ST elevation and the patient was transferred to the intensive care unit (ICU) after receiving 300 mg of aspirin, 300 mg of clopidogrel, and a continuous intravenous infusion of abximab and heparin. After an initial haemodynamically stable period, he became tachycardic, hypotensive with poor peripheral perfusion. His abdominal girth increased and his haemoglobin decreased from his admission value of 175 g/L to 53 g/L. An abdominal CT scan

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*Correspondence to:* Dr. M. Ziegenfuss, Intensive Care Facility, Prince Charles Hospital, Chermerside, Queensland 4032 (e-mail: marc\_ziegenfuss@health.qld.gov.au)

revealed a liver laceration, intra-hepatic haematoma and free intra-peritoneal fluid (Figure 1).



**Figure 1.** An abdominal CT scan showing the intrahepatic haematoma, free rupture of the liver and haemoperitoneum.

The liver laceration was managed conservatively. The heparin and abxycimab infusions were discontinued and he was transfused with 5 units of packed red cells, 4 units of fresh frozen plasma, 2 units of pooled platelets, 8 bags of cryoprecipitate and received 10 mg of vitamin K. However, due to the coronary artery stent, the clopidogrel (75 mg daily) and aspirin (300 mg daily) were continued. A percutaneous tracheostomy was performed on day 4 and remained in position until he was decannulated on day 15. He was discharged from the ICU on day 16 and from hospital on day 35.

He was readmitted with chest pain 1 year after his out-of-hospital cardiac arrest and, after informed consent, an hepatic MRA was performed which revealed a cystic hepatic lesion, which was thought to represent the residual defect from the liver laceration.

*Patient 2.* A 51-year old man had a cardiac arrest in a hospital car park after 4 hours of central chest pain. Medical and nursing staff who witnessed the collapse commenced CPR. The first rhythm recorded was ventricular fibrillation. Return of spontaneous circulation occurred after 30 minutes following 20 defibrillations, 7 mg of adrenaline and 300 mg of amiodarone. An adrenaline infusion was commenced to manage his hypotension and aspirin 300 mg and tenecteplase 50 mg were given during his 90 minute transfer to our hospital.

Therapeutic cooling was also initiated. At our hospital the patient underwent coronary angiography, which revealed severe coronary artery disease but no arterial occlusion. He received 5000 units of heparin followed by an intravenous heparin infusion. He was then admitted to the ICU where he became hypotensive with a low central venous pressure. A transoesophageal echocardiogram demonstrated a small and underfilled left ventricle with inferolateral hypokinesis.

As his abdomen became progressively distended an ultrasound was performed which revealed a laceration and intra-parenchymal hematoma of the left lobe of the liver and a large amount of free peritoneal fluid. Intravenous protamine was administered and the heparin infusion was discontinued. He was also rewarmed to 36°C to reduce the effect of a hypothermic coagulopathy. The liver laceration was managed conservatively with the patient receiving a total of 12 units of packed red cells, 4 units of fresh frozen plasma and 16 units of platelets within 48 hours. Anticoagulant and antiplatelet agents were not administered for the next 7 days. An abdominal CT scan on day 2 confirmed the hepatic contusion with a large amount of fluid surrounding the liver and spleen (Figure 2).

The patient made a satisfactory neurological recovery and was able to obey complex commands by day 2. A percutaneous tracheostomy was performed on day 4 and remained in position until he was decannulated on day 12. An implantable defibrillator was inserted on day 24. He was discharged from hospital on day 28 in good health.



**Figure 2.** An abdominal CT scan demonstrating the subxiphisternal hepatic contusion and fluid surrounding the liver and spleen.

## DISCUSSION

Generally, only a small percentage of patients survive an out-of-hospital cardiac arrest with less than 50% surviving for more than 3 days and less than 10% surviving to hospital discharge. Recent trials have shown improvements in survival to hospital discharge due to improved access to defibrillators, modified CPR techniques, hypothermia and use of thrombolytics, anticoagulants and coronary artery intervention. While many of these interventions may result in improved survival, there is an increased incidence of bleeding complications.<sup>3</sup>

Effective CPR in an adult requires the sternum to be depressed 4 - 5 cm with each compression. Rib fractures are the most common complication associated with CPR and may occur in up to 50 percent of patients. Other thoracic complications include haemothorax, pneumothorax, tension pneumothorax, haemopericardium, and bone marrow embolus. Intra-abdominal injuries include hepatic, splenic, or bowel trauma and retroperitoneal haematoma, all of which have been reported before thrombolytic and anticoagulant therapies were used routinely for acute myocardial infarction.<sup>4,5,6</sup> In one autopsy study, liver trauma was recorded in 2.9% of patients who died after CPR,<sup>7</sup> and is probably caused by caudal sternal compression of a liver which is distended by high venous pressures. In both our patients the abdominal CT scan demonstrated a laceration beneath the xiphoid process.

Hemodynamic instability after cardiac arrest is not uncommon. If persistent shock with low central venous pressures and abdominal distension are noted, an abdominal ultrasound may demonstrate the liver injury and intra-peritoneal fluid. The indications for surgery are ongoing bleeding or the need to exclude other injuries (e.g. splenic injury).<sup>8</sup> Surgery in our two patients would have been hazardous due to the recent myocardial infarction, impaired hemostasis and limited access to the dome of the liver. Both patients were successfully

managed using conservative treatment, although antiplatelet agents were continued in the first patient to facilitate patency of the intra-coronary stent. Late complications of a liver laceration include a bile leak, an area of infarction or a rebleed, which may occur up to 6 weeks after the liver injury.

Liver injury after cardiopulmonary resuscitation should be considered in the haemodynamically unstable patient and may be managed successfully by conservative means using a major intensive care facility.

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