CASE REPORT

Ruptured subcapsular liver haematoma following mechanically-assisted cardiopulmonary resuscitation

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SUMMARY

A 64-year-old man with a history of ascending aortic surgery and pulmonary embolus presented with shortness of breath. He rapidly decompensated, prompting intubation, after which he lost pulses. Manual resuscitation was initiated immediately, with subsequent use of a LUCAS-2 mechanical compression device. The patient was given bolus thrombolytic therapy and regained pulses after 7 min of CPR. Compressions were reinitiated with the LUCAS-2 twice more during resuscitation over the subsequent hour for brief episodes of PEA. After confirmation of massive pulmonary embolism on CT, the patient underwent interventional radiology-guided ultrasonic catheter placement with local thrombolytic therapy and experienced immediate improvement in oxygenation. He later developed abdominal compartment syndrome, despite cessation of thrombolytic and anticoagulation therapy. Bedside exploratory abdominal laparotomy revealed a ruptured subcapsular haematoma of the liver. The patient's haemodynamics improved following surgery and he was extubated 11 days postarrest with intact neurological function.

BACKGROUND

In their 2015 update, the American Heart Association reaffirmed the priority of chest compressions in CPR, and set new upper limits for compression depth and frequency, narrowing the range for compression depth to 2.0–2.4 inches and for frequency to 100–120/min.¹ Efforts to implement these guidelines consistently could implicitly strengthen the argument for use of mechanical compression devices. Such devices deliver consistent timed compressions without fatiguing, providing a theoretical advantage shown to improve outcomes in CPR during transport or interventional procedures.^{2–3} The theoretical advantage in consistency, however, has not been shown to improve outcomes in the emergency department (ED) setting. The risks posed by these devices have been reported in small studies and several case reports but are difficult to weigh against possible advantages.^{4–5} This case report describes a rare complication of mechanically-assisted CPR, which led to significant patient morbidity, and highlights the importance of individualising CPR efforts.

CASE PRESENTATION

A 64-year-old man presented to the ED via ambulance for acutely worsening shortness of breath after 2 weeks of productive cough and symptoms consistent with an upper respiratory tract infection.

His medical history included hypertension, thoracic aortic aneurysm repair with bioprosthetic aortic valve replacement and coronary artery bypass graft 2 years prior, and remote history of pulmonary embolism of unknown aetiology, not on anticoagulation.

On presentation, the patient was in significant respiratory distress and reporting chest and upper back pain. He was tachypnoeic to 50 bpm, with a systolic blood pressure of approximately 90 mm Hg in both arms. Physical examination revealed cool, clammy skin, engorged neck veins and use of accessory respiratory muscles.

INVESTIGATIONS

A focused bedside transthoracic echocardiograph showed evidence of right heart failure (figure 1), with signs of significant pressure overload in the right ventricle. Before central or arterial access could be secured, the patient decompensated and was urgently intubated with a rapid sequence technique. Immediately following intubation, the patient experienced a pulseless electrical activity (PEA) arrest and manual CPR was initiated. After approximately 2 min of manual CPR, trained ED staff placed a Lund University Cardiac Arrest System–V.2 (LUCAS-2; Redmond, Washington, USA) mechanical compression device. Initial placement was deemed too caudal and efforts were made to move cephalad prior to initiation of compressions. After placement was agreed on (total placement time <1 min), mechanical compressions were initiated and 7 min completed before return of spontaneous circulation.

Based on the history obtained, initial echocardiographic findings and PEA arrest, the decision was made to empirically bolus 50 mg of tissue

Figure 1 Transthoracic echocardiographic ultrasound image showing septal bowing and right atrial dilation, consistent with right heart strain.

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plasminogen activator (tPA) for presumptive pulmonary embolism during CPR. Arterial and central venous cannulae were emergently placed and high-dose vasopressor therapy was initiated (epinephrine at 0.8 µg/kg/min). The patient experienced two subsequent arrests within the hour, with prompt return of spontaneous circulation with use of the LUCAS-2, which was placed by trained staff. A second bolus dose of 50 mg tPA was given during this time. A CT was subsequently obtained, which confirmed the diagnosis of massive bilateral pulmonary emboli; interventional radiology was contacted to evaluate suitability for intervention. Given persistent hypoxia with oxygen saturations in the range of 40–50%, the extracorporeal membrane oxygenation team was also consulted and recommended prioritising placement of targeted catheter therapy, given the significant clot burden.

DIFFERENTIAL DIAGNOSIS

Acute respiratory distress with ultrasonic evidence of right heart strain is pathognomonic for pulmonary embolism, though ARDS is a consideration. End-stage cor pulmonale could also present similarly, however, with no evidence of myocardial hypertrophy on ultrasound and no history of symptomatology, this is much less likely.

TREATMENT

Per recommendations, the patient was switched to vasopressin for runs of ventricular tachycardia and nitric oxide was initiated at 10 ppm. Placement of bilateral EkoSonic Endovascular System catheters (EKOS, Bothell, Wash) was successful and targeted tPA therapy was initiated at 0.5 mg/h, with an immediate improvement in oxygen saturation and reduction in vasopressor requirement. An inferior vena cava filter was placed and systemic anticoagulation initiated with heparin at 500 units/h. The patient was transferred to the surgical intensive care unit following the procedure.

OUTCOME AND FOLLOW-UP

After initial improvement, the patient experienced progressively worsening haemodynamics overnight, with an increased vasopressor requirement to maintain mean arterial pressure greater than 55 mm Hg. During this time, he was noted to have increased abdominal distension, and a bedside abdominal ultrasound examination demonstrated significant free peritoneal fluid concerning for active bleeding. Despite cessation of heparin and tPA, the patient experienced increasing bladder pressures and worsening haemodynamics, consistent with abdominal compartment syndrome. A bedside exploratory laparotomy was performed on the second day of admission. Seven litres of bloody fluid was evacuated from the abdomen, with prompt improvement in haemodynamics. Active haemorrhage was noted in the area of a subcapsular liver haematoma, with apparent rupture just medial to the falciform ligament ([figure 2](#)). These findings were deemed most consistent with a compression injury, likely sustained during CPR. The abdomen was packed and reopened 3 days later, at which time definitive repair was completed with fibrin sealant, argon coagulation and tissue patching. The patient was successfully extubated on postarrest day 11, with grossly intact neurological function.

DISCUSSION

Mechanical assist devices for chest compressions during CPR were first introduced in the 1970s but were not widely available. An expanded focus on resuscitation efforts and improvements in resuscitation technology in recent years have led to increased

Figure 2 View of liver with ruptured subcapsular haematoma before repair. Image courtesy of Lena M Napolitano, MD.

use of such devices among first responders and EDs, although specific data are lacking on the prevalence of adoption. This development has prompted several studies comparing manual and mechanical CPR. Analysis of the effectiveness of compressions in animal and human studies appears to show an advantage for mechanical compression, which did not, however, impact more clinically relevant outcomes, such as survival to discharge and neurological outcome.^{2 6} In fact, the LUCAS-2's manufacturer, Physio-Control, only claims to improve the quality and consistency of compressions during transport and in the cardiac catheterisation laboratory.⁷ Furthermore, the list of contraindications for use of the LUCAS-2 device is short and includes the following: patient too small, patient too large and, 'if it is not possible to position LUCAS safely or correctly on the patient's chest'.⁸

Complications arising from chest compressions are generally accepted as sequelae of the life-saving therapy. A postmortem analysis by Smekal *et al*⁵ compared liver capsule injury rates in patients who had received manual versus LUCAS compressions, and found a higher rate among patients who had received LUCAS therapy (4.3% vs 2.4%). Though this increase was not deemed significant, there are other studies that show a trend towards increased incidence.^{9 10} Only one patient experienced what was considered a 'life-threatening' liver haemorrhage of 500 mL—significantly less than the blood loss we have described. It is likely that the combination of systemic tPA therapy and acute right heart failure, with engorged hepatic vasculature, predisposed the patient we treated to significant bleeding. We would question the appropriateness of use of the LUCAS-2 device in patients with a similar presentation.

The most commonly found injuries following CPR are rib/sternal fracture, retrosternal bleeding and epicardial bleeding.^{5 9} Abdominal organ injury, such as liver trauma, gastric perforation

and splenic rupture are a rare, but not insignificant, occurrence. Mechanical compression devices have been shown to produce higher numbers of rib fractures, but investigation into the relative incidence of internal organ injury has been limited by small sample size.^{5 9 11} Several case reports have described liver injuries related to CPR with both manual^{12 13} and mechanical^{14–16} compressions. Three of the cited cases resulted in haemodynamic compromise requiring intervention, with one subsequent death attributed to haemorrhagic shock.¹⁵ As manual compressions should always precede mechanical devices, it is impossible to definitively attribute any complication to mechanical compressions. Furthermore, it will be difficult to ascertain the contribution of device malposition or migration to such complications. More studies are needed to evaluate complications associated with mechanical CPR, especially when compared to manual CPR, in order to inform categories of patients who may be predisposed to complications with the use of mechanical CPR devices.

Patient's perspective

Patient

"I'm sorry, I get a bit emotional talking about this. I don't remember anything about the CPR or the LUCAS device, but I'm really grateful for the care I received. I know that I could've had serious brain damage but, to be honest, I'm as sharp as I ever was. I'm really glad you guys used the LUCAS device."

Spouse

"Because of that LUCAS machine, my husband had minimal time down. I know he arrested three separate times, things could have ended up much worse. I know we've had a lot of complications related to the machine but I think it was worth it—I really don't think he'd be sitting here right now if you hadn't used it. I'm not sure if the machine should be used on everyone, but I'm glad it was used on my husband. I think everyone should at least know about the machine and how to use it. They should also know about the complications. Honestly, though, I think it should be in every ER in the country—a machine is better than a human, in my mind."

Learning points

- There is no evidence for improved outcomes with the use of mechanical compression devices in the emergency room setting.
- Conditions leading to hepatic congestion and systemic anticoagulation may increase risk for liver injury during CPR.
- It is important to consider visceral injury as a cause of haemodynamic instability after CPR.

In summary, we have described a case of significant patient morbidity following use of the LUCAS-2 mechanical compression assist device during CPR. We believe that this case highlights the importance of individualising therapy during CPR. Providers should consider the risks and benefits of use of the LUCAS-2 device in patients undergoing systemic anticoagulation and in patients with acute right heart failure.

Contributors All the authors assisted in care of the patient while in the emergency department. JRJ drafted the report with the assistance of REF. MA and REF assisted with manuscript edits. All the authors reviewed and approved the final draft for submission.

Competing interests None declared.

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