Peer-Reviewed Case Series

Unresponsive Patient with LVAD: A Case Series

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Abstract

Mechanical circulatory support (MCS) for patients with advanced heart failure (HF) is becoming more commonplace as technology has progressed and reliability of these systems has improved. We report our experience with three patients with cardiopulmonary arrest in the hospital and propose a protocol for the prompt assessment and treatment of an unresponsive patient with a LVAD in place. We presented three cases of unresponsive patients on LVAD support with low flow or no flow through the LVAD. Although all three of them were alive after resuscitation, none survived to discharge. In all three cases, there were no flaws in LVAD per se, with power source and controller appropriately attached, and the pump functioning normally. The low flow state was secondary to non-cardiac conditions (respiratory acidosis, dehydration, and stroke). In early 2017 we developed our algorithm for unresponsive LVAD patients. Utilization of LVAD flow, which can be obtained quickly and non-invasively at the bedside of a patient is used to direct resuscitation efforts in this proposed protocol. Rapid and standardized protocol for resuscitation of LVAD patients with in-hospital cardiopulmonary arrest is necessary to decrease morbidity and mortality. Further investigation regarding the best practice in this clinical scenario is evolving and further studies are needed.

Keywords: CPR, ACLS, left ventricular assist device, cardiac arrest

Introduction

The use of mechanical circulatory support (MCS) for patients with advanced heart failure (HF) is becoming more commonplace as technology has progressed and reliability of these systems has improved. In-hospital cardiopulmonary arrest in patients with left ventricular assist devices (LVAD) is a deadly clinical scenario requiring rapid assessment and treatment. Often, healthcare providers and staff

are unfamiliar with the assessment and treatment of cardiopulmonary arrest in this unique patient population. We report our experience with three such patients who had cardiopulmonary arrest in the hospital and propose a protocol for the prompt assessment and treatment of an unresponsive patient with a LVAD in place.

Methods and Results

Three LVAD patients at our institution had a Code Blue called and each subsequently survived the initial event but later expired. The common etiologies of pump failure (power disconnect or driveline failure) or low flow state (de novo thrombus formation, new ischemic events, pulmonary embolism, or cardiac tamponade) were not implicated in these events. We report their initial resuscitative efforts based on review of code blue sheets and subsequent clinical course. We then describe our proposed MCS code algorithm.

Cases:

Patient A

Patient A was a 49-year-old obese male with chronic systolic heart failure, non-ischemic cardiomyopathy with a LVEF 10-15%. He also had chronic kidney disease, poorly controlled diabetes mellitus and obstructive sleep apnea. He underwent Heartware HVAD implantation in May of 2014 as bridge to transplantation. He was admitted in February 2017 for volume overload. Upon admission, he was hemodynamically stable and underwent appropriate diuresis. The Heartware device was found to be functioning well. The patient was placed on the transplantation list during the hospitalization. He required a controller exchange on hospital day 13 secondary to water damage which was completed without issues.

On hospital day 15, the patient removed his continuous positive airway pressure mask for comfort overnight, but had appropriate oxygen saturation on room air. The patient was soon noted to become bradycardic at 34 beats per minute on telemetry, and he was found to be unresponsive with the LVAD alarming low flow. Advanced cardiac life support (ACLS) was initiated, however, no compressions were given because of the fear of displacing the cannulas of the LVAD. Eight minutes into the code, a bolus of isotonic saline was administered, and at minute 11 the patient was intubated. At minute 12 , speed was decreased from 2840 revolutions per minute (rpms) to 2000 rpms. Power was 5Wt and pulsatility index was 3. The patient's heart rate increased to 50 beats/minute, and flow improved to 3.1L/minute. Arterial blood gas showed pH of 7.25, pCO2 of 55, and pO2 of 149. Mean arterial pressure (MAP) measured by Doppler was found to be 42 mmHg. A dopamine infusion was started at minute 18. Subsequently, heart rate improved to 76 bpm and arterial line was then placed showing a blood pressure 83/59 mmHg with a MAP of 69. The Heartware HVAD speed was increased to 2640 rpms, flow 7.1L/min, power 5Wt, and pulsatility index 3. The patient was hemodynamically stabilized, however had no subsequent improvement of his neurological status. A CT head showed cerebral edema likely secondary to anoxic injury. A repeat CT of

the head 24 hours later showed increasing cerebral edema and herniation. Comfort care measures were initiated, and the patient expired.

The etiology for his unresponsiveness was likely respiratory acidosis resulting in vasodilation, bradycardia, hypotension, and hemodynamic compromise. Log files for this patient's HVAD device were unfortunately unavailable, however review of such files in the future would be very valuable to assess adequate resuscitation. Review of this case suggests he should have been intubated sooner, and timely chest compressions could have been helpful to improve brain perfusion and avoid anoxic injury.

Patient B

Patient B was a 74-year-old male with a history of chronic HF secondary to ischemic cardiomyopathy, mitral valve repair, hemochromatosis, and stage III chronic kidney disease who was referred to our institution for evaluation of LVAD, which was implanted. He was successfully managed as an outpatient until he complained of some shortness of breath on exertion, and his home diuretics were increased. Several days later, he became dizzy, but did not call the coordinator. He fell while walking outside, sustained a head injury, and was eventually found unconscious by a family member. During the first call to the LVAD coordinator from the family member a low flow alarm was identified. He was brought to the hospital unresponsive.

Upon arrival, a CT of the head showed subdural and subarachnoid hemorrhages. His INR was found to be within therapeutic limits on arrival to the emergency department. The patient again had a low flow alarm shortly after arrival but initially had carotid pulse detected by Doppler. Then the pulse by Doppler was lost, thus ACLS was initiated, including chest compressions. Epinephrine was administered (1 mg) and repeated twice for a total of 3mg during resuscitation. Chest compressions were stopped when a carotid pulse became palpable, partial pressure of exhaled carbon dioxide was 27 mmHg, and LVAD flow was 3.3L/min. The patient remained hypotensive with MAP of 50mmHg. Boluses of normal saline were given, and epinephrine, vasopressin, and dopamine infusions were initiated. Following the resuscitation, the patient remained in cardiogenic shock with worsening renal failure and hypoxemia. The patient's neurological function also declined, and following discussion with the patient's next-of-kin, the decision was made to withdraw care several days later. The etiology of the code was likely dehydration with low flows, precipitated by traumatic brain injury.

Patient C

Patient C was a 43-year-old male with history of large acute anterior ST-elevation myocardial infarction. His clinical course was complicated by cardiogenic shock requiring intra-aortic balloon pump placement. He subsequently underwent Heartmate II LVAD placement. A year later, the LVAD was exchanged due to pump thrombosis. The patient had been successfully managed as an outpatient until he developed pump thrombosis and had an LVAD exchange in June of 2016. On post-operative day 12 he had acute respiratory decline and was intubated. Blood pressure was 82/22 mmHg and heart rate began to decrease after the

intubation. Several minutes later, the patient was found to be pulseless with LVAD showing no flow. ACLS was initiated with one mg of epinephrine and chest compressions. Chest compressions were ceased after two minutes when blood pressure improved to 110/60mmHg. The patient remained hemodynamically stable but blood pressure began decreasing and he was started on a norepinephrine infusion. Sanguineous output from the left subcostal incision was noted after chest compressions ceased. The patient was taken to the operating room for exploration of the wound. There was no evidence of pump damage or left ventricular injury, and the bleeding was felt to be secondary to local tissue trauma secondary to chest compressions in the setting of severe coagulopathy. The patient was stabilized, however, he was found to have fixed and dilated pupils. A CT scan showed extensive bilateral middle cerebral artery, posterior cerebral artery, and basilar artery territory infarcts with diffuse severe cerebral edema and tonsillar herniation. After multi-disciplinary discussion with family, care was withdrawn and the patient subsequently expired. The respiratory arrest and unresponsiveness in this patient was likely secondary to acute stroke.

Discussion

We presented three cases of unresponsive patients on LVAD support with low flow or no flow through the LVAD. Although all three survived initial resuscitation, none survived to discharge. In all three cases, the LVAD was not suspected to have initially contributed to the arrest, all power sources and controllers were appropriately attached, and the pumps were functioning normally. The low flow state in these cases was thought to be secondary to non-cardiac conditions (respiratory acidosis, dehydration, and stroke). In two of three cases, chest compressions were performed and there were no signs of cannulae dislodgement.

Existing literature regarding management of the unresponsive patient with MCS is limited (1-5). In the recent past, chest compressions were discouraged for fear of dislodgement of the cannulae (6), but a number of recent case reports suggests chest compressions are safe and should be performed when necessary (3). With increasing LVAD prevalence in the community, providers with little to no training in the management of LVAD face the need to provide urgent or emergent care for patients on assistive pumps. Several algorithms, written in order to assist providers in taking care of critically ill patients on mechanical circulatory support, have been published (5,7). While very detailed, these algorithms may be challenging to follow when decisions have to be made very quickly.

Our center has developed its own adapted ACLS algorithm for LVAD patients. Predating our algorithm, we launched the MCS Code team comprised of providers and staff who are familiar with care for MCS patients. At our institution, when a patient with MCS has a cardiopulmonary arrest event, a specific phone number is called that notifies specialized personnel. Rather than internal medicine residents responding to the code, a cardiology fellow is called. The critical care anesthesia team is responsible for airway management instead of emergency medicine, which is the standard at general codes. The respiratory therapist called to the code is directly from the cardiovascular ICU where LVAD patients are cohorted and is expected to have advanced knowledge and experience with this population. Lastly,

the attending cardiologist and CT surgeon are notified of the code as well. The creation of this team prevents MCS patients from receiving chest compressions inappropriately and helps coordinate care of patients more effectively. Even with a specialized code team, we have continued to reevaluate its performance and its standards. In early 2017 we developed our algorithm for LVAD patients (Fig. 1). The initial provider must first assess LVAD connections and power sources prior to moving through the algorithm. If LVAD hum is not present, pulseless electrical activity leads immediately into ACLS protocol, including chest compressions. If VAD hum is present, flow is assessed. If flow is greater than 2.5 L/min and MAP obtained by Doppler or arterial line is greater than 50 mmHg, cardiovascular collapse is likely not the etiology of the patient's unresponsiveness and other etiologies should be sought. If the MAP is found to be less than 50 mmHg by Doppler or arterial line and flow is greater than2 .5L/min, the provider should assess the patient's rhythm and deliver defibrillation as needed, provide volume support, inotropes, vasopressors, and seek reversible causes. If flow is found to be less than 2.5 L/min and the patient is pulseless by palpation, ACLS protocol including chest compressions should be initiated. Given the unreliable nature of presence of pulse by palpation in LVAD patients, in practice pulse checks should be assessed by Doppler after initial palpable pulse is not felt. If efforts are unsuccessful, emergent ECMO cannulation at the bedside is recommended. Utilization of LVAD flow, which is obtained quickly and non-invasively at the bedside, is used to direct resuscitation efforts in this proposed protocol.

Further changes our algorithm are necessary as more data emerges. Urgent, bedside echocardiography in an experienced operator's hands could significantly assist in resuscitative efforts. Future revisions may include parameters to allow for adjustments of VAD settings based on echocardiographic data obtained at the bedside. This, however, requires more evaluation before inclusion in our algorithm. It is also unclear if a hypothermia post-cardiac arrest has its role in LVAD patients. Given the unclear etiology of cardiopulmonary arrest in LVAD patients, autopsies were considered. However, the families of all three patients declined. Tactful encouragement of families to allow for autopsy and coordination with pathologists would be helpful in the future to better understand the effect of CPR on LVAD cannula dislodgement and function.

Conclusion

Rapid and standardized protocol for resuscitation of LVAD patients with in-hospital cardiopulmonary arrest is necessary to decrease morbidity and mortality. Further investigation regarding the best practice in this clinical scenario is evolving and further studies are needed.
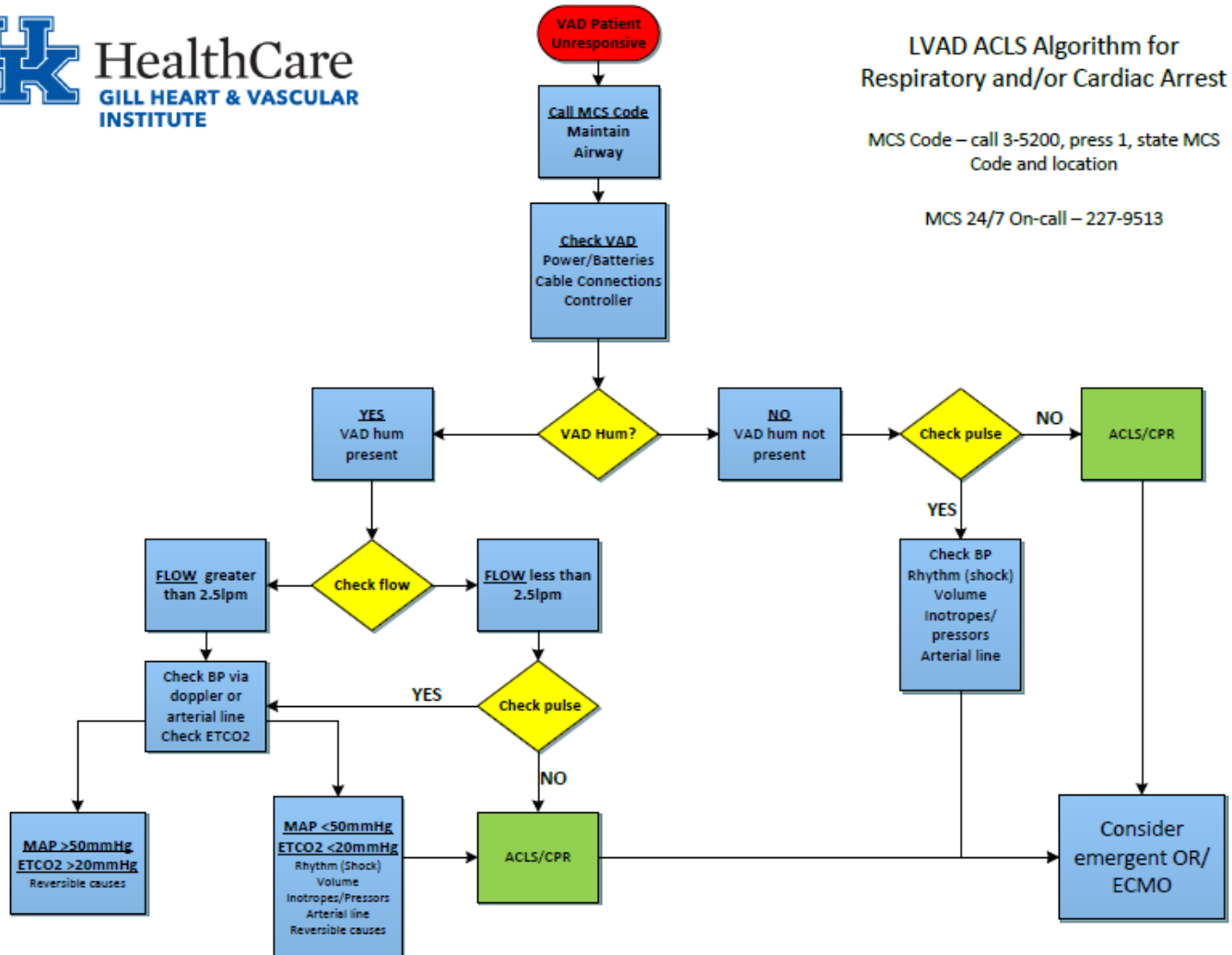
Figure 1. MCS code algorithm, University of Kentucky

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