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Avoidance of Increased Intrathoracic Pressure with High Velocity Oxygen Therapy: Managing a Patient with A LVAD in Shock – A Case Report

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Abstract

Patients with indwelling left ventricular assist devices represent a growing population presenting numerous novel diagnostic and treatment challenges. This case describes the complexities of managing a patient with a left ventricular assist device brought to the emergency department unresponsive following a motor vehicle collision. The arrival as a "trauma" patient complicated the diagnostic process, which ultimately revealed the patient had experienced ventricular tachycardia cardiac arrest but maintained minimal perfusion via the left ventricular assist device. The patient was evaluated for trauma and managed supportively with medications, fluid replacement, and high flow nasal oxygen therapy. The patient was successfully defibrillated. Balancing cardiac preload and afterload to maximize perfusion and stabilize the patient was particularly challenging and further complicated by the lack of availability of a left ventricular assist device specialty and the limited available patient history. This case highlights the cognitive challenges accompanying these patients and the need for further research and education to allow emergency department personnel to care for these patients optimally.

Keywords: Left ventricular assist device; Cardiogenic shock; High velocity therapy; High flow oxygen; Cardiac workload

Introduction

Emergency Medicine (EM) physicians care for patients with complicated medical conditions, such as those with a left ventricular assist device (LVAD). Limited interaction with LVAD patients can lead to framing bias, wherein providers fixate on the LVAD, thus neglecting the non-LVAD-related chief complaints [1]. Additionally, trauma evaluation in these patients remains undescribed [2]. Here, we present a case of a 40-year-old male with an LVAD who presented to the ED as a "trauma alert" following a single-driver, unrestrained motor vehicle crash. Vital signs were difficult to obtain due to electrical activity generated from the LVAD, which did not correlate with the physical exam.

Case Presentation

A 40-year-old male presented to the ED following a single-driver motor vehicle collision. The patient was unresponsive and unrestrained, with no evidence of airbag deployment or obvious bleeding at the scene. The patient was minimally responsive and declared a "trauma alert" with his mental status attributed to a head injury.

Initial examination revealed the patient was reactive to painful stimuli, could protect his airway, and the carotid pulse was faint but palpable. Repeated attempts to obtain a blood pressure (BP) measurement were unsuccessful, but capillary refill time remained 2-3 seconds. The providers questioned what an expected cardiac assessment should yield regarding peripheral pulses and BP due to the presence of the LVAD. Without the LVAD, lack of peripheral pulse would indicate no BP; thus,

cardiac compressions would have been indicated. However, compressions are contraindicated when an LVAD is present [3]. The patient had decreased perfusion and monomorphic ventricular tachycardia (VT), coinciding with a heart rate of 222 bpm. A bedside ECHO confirmed the arrhythmia while the LVAD coordinator was contacted. There were no remarkable electrolyte imbalances; initial arterial blood gas displayed a severe, partially compensated metabolic acidosis confirmed with an anion gap (21 mmol/L) (Table 1). An increased creatinine level of 2.6 mg/dL and BUN of 24 mg/dl suggested decreased renal perfusion. These results indicated dehydration-induced hypovolemic shock or conflicting congestive heart failure. A severely dehydrated patient could have had a high BUN due to insufficient fluid volume to excrete waste products. HCT was notably high at 50% PCV, leading the physician to treat the patient's dehydration issues. Coagulation studies were therapeutic with anticoagulation therapy. Radiologic images were unremarkable, and the LVAD showed no obvious misplacement or breakage.

Table 1: *Analysis of blood gas chemistry as a measurement of therapeutic effectiveness.*

Measurements	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	HCO_3 (mmol/L)	Anion Gap (mmol/L)	Lactate (mmol/L)	Creatinine (mg/dL)	BUN (mg/dL)
Upon Arrival	7.19	26.7	129	9.5	21	10.7	2.6	24
Post-Resuscitation*†	7.28	44.1	47	20.8	16	4.76	2.2	31

*Resuscitation included: Treatment for VT, fluid administration, and High Velocity Therapy (40 L/min, 55% FiO2)
†Value represents a venous blood gas measurement; thus, a significantly lower PaO_2 is expected.

The team concluded the VT was valid and the patient would benefit from cardioversion. Calcium gluconate (2 g), magnesium sulphate (2 g), lidocaine (100 mg), and a 500 ml bolus of normal saline were administered intravenously to optimize cardiac workload in preparation for defibrillation. Oxygen was applied via a non-rebreathing mask at 15 L/min, which produced an oxygen saturation of only 89%. Typically, intubation and mechanical ventilation would be utilized for managing the patient in a minimally responsive state; however, the physician was concerned that increased intrathoracic pressure (ITP) would impair cardiac preload and impede survival in this patient. Therefore, the decision was made to utilize high velocity oxygen therapy (HVT) to support and reduce breathing effort without increasing ITP to optimize preload and decrease the cardiac workload. Fourteen minutes after arrival, the patient was successfully defibrillated (200J x 1), leading to sinus tachycardia. Mean arterial BP was recorded at 70 mmHg, but the automated sphygmomanometer had difficulty registering separate systolic and diastolic pressures. HVT (40 L/min, 55% FiO2) continued as adjunctive therapy with an additional 500 ml lactated ringer and 500 ml of normal saline intravenously to reduce cardiac workload consumed by respiratory distress secondary to cardiogenic shock. Intravenous haloperidol (2.5 mg) was administered to reduce agitation as the patient became more responsive. Approximately 90 minutes after arrival, the patient was alert and stable. The patient's spouse arrived and revealed that the patient had been working in a hot environment during a heat wave. This history and elevated BUN and HCT led the physician to believe the patient had become dehydrated to the point of shock. LVAD patients are extremely fluid sensitive and therefore required to restrict fluids due to heart failure, and the patient had underlying renal insufficiency.

Repeated venous blood work revealed marked improvement in blood gas measurements and lactate levels (Table 1). The patient was transferred in stable condition to an LVAD center.

Discussion

This case report describes the diagnostic challenges for caring for an unresponsive patient with an LVAD. Patients presenting to the ED with activated "trauma alert" systems and those with LVADs pose a similar risk for anchoring bias, which can cause clinicians to focus on extraneous ideas, potentially leading to incorrect conclusions. In this case, a multidisciplinary and analytical approach ultimately led to this patient's proper diagnosis and treatment.

Patients with newer-generation LVAD systems providing continuous flow may have no palpable pulse, and ventricular arrhythmias have been reported to occur in 22-59% of all LVAD recipients [4]. Chest compressions are contraindicated, except when an LVAD team is present to replace any dislodged parts [3]. These patients are intravascular volume sensitive, balancing between heart failure from preload reduction or increased afterload and volume overload. Typically, it is reasonable to begin resuscitation with 250 – 500mL crystalloid when intravascular volume depletion is suspected [5]. LVAD patients may require intubation and mechanical ventilation when hypoxia, hypercarbia, or acidosis worsens or when the airway cannot be protected. However, high levels of positive ITP should be avoided to prevent worsening of right ventricular dysfunction in an already preload-dependent patient [6]. While acute heart failure (AHF) is typically managed with pressure-based respiratory

support there is a small amount of evidence that high flow nasal oxygen therapy (HFNO) may be an acceptable alternative for selected patients [7,8].

While HFNO generates a small amount of positive end-expiratory pressure, patients with sensitivity to cardiac preload may exhibit greater benefit from HFNO. Positive pressure within the nasopharyngeal space can be generated by overcoming the resistance against expiratory flow, secondary to rapid flush. This mild positive ITP has shown to be auto-titrated by the patient, dependent on inspiratory flow, minute ventilation and resistance, with peak effect occurring during expiration only [9]. This mild CPAP effect tends to promote recruitment of collapsed alveoli while enhancing lung aeration to further decrease the patient's breathing effort and perhaps impact alveolar ventilation [10,11]. HFNO can be an effective treatment for AHF patients, despite its relatively low and variable pressure generation compared to other more invasive and closed systems [10-12]. Overall, HVT reduces metabolic demand while decreasing myocardial workload, further fighting the sympathetic surge, as previously measured by inferior vena cava dynamics using ultrasound [13]. Therefore, usage of HVT warrants further study in this patient population. Another interesting observation was the rapid clearance of the lactate, which fell from 10.7 mmol/L to 4.76mmol/L in approximately 60 minutes. However, the dynamics of lactate clearance and the development of guidelines for respiratory failure prognosis warrant further research [14,15]. This case study demonstrated the collaborative effort of all medical and pharmaceutical interventions that led to a positive outcome for this patient. Decreased cardiac workload using HVT seemed evident in the rapid reduction in lactate and breathing effort, which suggests this concept may also warrant further exploration. Utilizing a comprehensive approach could prove beneficial in the acute management of respiratory failure in LVAD and other preload-dependent patients.

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Conflict of Interest

AB is Manager of Clinical Research for Vapotherm, Inc. MSP is Director of Professional Development and Education for Vapotherm, Inc. KDW has been employed within the past 12 months as a scientific consultant. JSW is VP of Clinical Research for Vapotherm, Inc – a manufacturer of high flow oxygen systems.

Author Contributions

AB, MSJ, and JW participated in this manuscript's conception, development, and writing. KDW assisted in the writing of this manuscript. All authors agree to be accountable for the content of the work.

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