Case Report

Traumatic Refractory Ventricular Fibrillation Successfully Treated with Intra-Arrest Esmolol

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Keywords

Esmolol · Refractory ventricular fibrillation · Resuscitation · Emergency medicine · Cardiology

Abstract

Here, we describe a case of refractory ventricular fibrillation (RVF) in an undifferentiated trauma patient that responded to intra-arrest esmolol. To our knowledge, this is the first case of RVF in an undifferentiated trauma patient that responded to intra-arrest esmolol. This case continues to support the growing evidence for use of esmolol as a treatment of refractory fibrillation and should be considered even in undifferentiated traumatic arrest patients prior to the cessation of resuscitation efforts.

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Introduction

The current advanced cardiac life support (ACLS) algorithms for the management of ventricular fibrillation (VF) and pulseless ventricular tachycardia consist of high-quality cardiopulmonary resuscitation (CPR) and defibrillation as well as the administration of epinephrine and antiarrhythmic agents such as amiodarone [1]. Refractory ventricular fibrillation (RVF) is thought to be defined as failure to obtain return of spontaneous circulation (ROSC) within 10 min despite 3 defibrillation attempts, 3 mg of epinephrine, and 300 mg of amiodarone [2]. To this date, there is no clear consensus on the definition of RVF, nor the establishment of best practices, when it comes to treating patients that present with RVF [3, 4]. When RVF presents, the mortality rates are thought to be between 85% and 97% [5, 6].

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Esmolol, a potent beta-1 antagonist, has been shown to improve ROSC, increase successful defibrillation attempts, and improve the duration of post-resuscitation survival in RVF. The proposed mechanism of how esmolol works in the treatment of RVF is that it blocks the high endogenous and exogenous catecholamine state of cardiac arrest (CA) [7, 8]. Various experimental models involving esmolol in the treatment of RVF show promise, but data are overall limited supporting the use of esmolol in RVF [9–14]. Even further, to our knowledge, there have been no published case reports discussing the use of esmolol in an undifferentiated trauma patient that presented in RVF. Here, we present a case of RVF in a traumatic drowning patient that responded to intra-arrest esmolol.

Case Presentation

A 21-year-old Caucasian male, with no significant past medical history, was involved in a motor vehicle accident, in which he was a restrained passenger. The vehicle was traveling approximately 30 mph when it drove off the road into the Gulf of Mexico. The accident occurred when the average temperature of the Gulf of Mexico in that area was approximately 20°C. The patient was submerged underwater for approximately 10 min. The patient was extricated from the vehicle by first responders, and resuscitation was immediately begun. The patient's initial presenting rhythm was VF. The standard ACLS algorithm was followed, which consisted of high-quality CPR, serial epinephrine 1:10,000 1 mg interosseous (IO) boluses as well as 300 mg of amiodarone IO bolus, biphasic defibrillation at appropriate intervals, and the placement of a definitive airway. The patient was defibrillated a total of 3 times on scene. ROSC was achieved briefly, and the patient was transported to our facility. During transport, the patient did return to a rhythm of VF.

The patient arrived at our facility in VF with CPR in progress. Primary survey showed diminished breath sounds bilaterally, cold skin, and faint seat belt sign across the chest, with an even greater defined seat belt sign across the lower abdomen. Extended Focused Assessment Sonography for Trauma (eFAST) performed during resuscitation showed no evidence of cardiac tamponade, intra-abdominal free fluid, nor pneumo/hemothorax but did show decreased ventricular function consistent with VF CA. Three rounds of ACLS protocol were followed in the trauma bay, which again consisted of high-quality CPR, 2 additional defibrillations, and epinephrine 1 mg 1:10,000 every 3–5 min. Due to the patient being in RVF, esmolol bolus was given during resuscitation at a dose of 50 mg intravenously. The patient subsequently experienced ROSC not long after the administration of esmolol. The total resuscitation time was approximately 30 min, not including the 10 min of possibly being submerged underwater.

Following ROSC, post-resuscitation care was initiated immediately. Care included vasopressor therapy with epinephrine drip and bicarbonate drip and boluses due to extreme metabolic acidosis, most likely from prolonged CA. The patient's temperature was then assessed at 35°C due to cold-water submersion and cool ambient temperatures. The patient was actively warmed after some discussion with the trauma team due to concerns of possible traumatic internal injuries and development of possible coagulopathy. CT of the thorax was remarkable for diffuse pulmonary edema consistent with the patient's history of drowning. Other imaging was overall unremarkable. A full neurologic exam was unable to be performed prior to initiation of sedation; however, in the immediate post-resuscitation setting, the patient was making no spontaneous or purposeful movements.

The patient was admitted to the Trauma ICU for a total of a 3-day hospital stay. He was subsequently discharged with no focal neurological deficits and only retrograde amnesia of the accident.

Discussion

Ventricular arrhythmia and CA in the trauma patient have many etiologies. Traumatic brain injury and hypovolemic shock account for 62–90% of CA in trauma [15, 16]. Blunt cardiac injury most often presents with cardiac arrhythmias [17, 18], and commotio cordis is a well-described phenomenon of VF induction from blunt cardiac impact during the repolarization phase of the cardiac cycle [19, 20]. Additionally, in a drowning patient, prolonged hypoxia can induce CA. In our patient, TBI and hypovolemia were ruled out in the primary evaluation. Blunt cardiac injury is primarily a clinical diagnosis that would be difficult to confirm in the present case since he received prolonged CPR which would cause elevated serum troponin and myocardial dysfunction on echocardiogram – the 2 primary means of diagnosis of blunt cardiac injury [21]. He did have a seatbelt sign on the chest wall without sternal or rib fractures, so either blunt cardiac injury or commotio cordis from rapid deceleration and chest wall impact is a possible source of his ventricular arrhythmia. Finally, he was submerged underwater for approximately 10 min which could have led to a primary hypoxic CA.

Prolonged RVF is a critical state that is associated with very poor outcomes, especially if the resuscitation persists beyond 10 min. The reported 1-month survival rate of out-of-hospital RVF is 20.4%, with only 5.6% of patients regaining good neurological outcomes [22, 23]. Many case reports discussing the treatment of esmolol in RVF do so in the clinical scenario of cardiac ischemia, as this is the most common cause of RVF [11, 12]. In the present case however, cardiac ischemia is a very unlikely source given the patient's age and lack of coronary risk factors. There are many different theories on how RVF may occur in a healthy heart; however, one of the most convincing theories is due to the excessively high sympathetic tone in the peri-arrest state. CA has high levels of endogenous and exogenous catecholamines which are proarrhythmic and may exacerbate ventricular arrhythmias [8, 9, 24, 25]. A traumatic injury could induce the original arrhythmia, and the elevated sympathetic state and catecholamine excess inhibit successful conversion of the arrhythmia leading to RVF.

Epinephrine is a mainstay in the current ACLS algorithm and has been shown to increase the likelihood of the ROSC [26]. Epinephrine increases peripheral vasoconstriction thereby increasing coronary perfusion pressure and peripheral blood pressure via its alpha receptor mechanism. However, most of the potentially harmful effects of epinephrine are a by-product of its potent beta-agonism and include dysrhythmias, increased myocardial oxygen demand, greater risk of recurrent arrest, thrombosis through platelet activation, and cerebral ischemia [2, 8, 9, 27].

In light of this knowledge we gained from physiology, there has been a recent push to introduce beta-adrenergic blockade during a CA situation in order to mitigate the harmful effects of high sympathetic tone while maintaining the beneficial alpha effects. Two pharmacological β -blockers, esmolol and propranolol, have been studied in this context [2, 9–12, 27]. Esmolol is an ideal agent for RVF because it is an ultra-short-acting beta-1 selective adrenergic receptor blocker. Esmolol is very cardiac selective and has a short onset of action of 90 s with a half-life of 9 min, which means that it can be quickly turned off after termination of the RVF without having the negative inotropic effects after resuscitation [4]. Blocking beta-adrenergic receptors with esmolol has been shown to counteract the cardiac effects of epinephrine (increased heart rate and contractility and lowered VF threshold), thereby increasing the VF threshold in both ischemic and nonischemic conditions [8]. This possible effect could break the cycle of RVF. Upon literature review, no specific examples of β -blocker use in blunt myocardial injury were found; however, it does seem consistent that arrhythmias caused by cardiac injury are managed with typical antiarrhythmic agents [18], and CA in trauma patients without hemorrhage is managed according to ACLS protocols. In addition to the cardiac effects of esmolol, there seems to be an additional component of neuroprotection

afforded by the medication. A recent swine model showed improved neurological outcomes in RVF with the administration of esmolol with epinephrine. The thought is that esmolol counteracted the impairment of cerebral microcirculatory blood flow [13].

The patient in our case was, by definition, in RVF and met criteria for esmolol use. Our hospital's current protocol is to use esmolol IV bolus, with subsequent esmolol drip if ROSC is not achieved. Advanced Trauma Life Support protocol was followed during resuscitation, and a decision to administer esmolol was made after there was no sign of exsanguination on primary survey and a negative eFAST exam. In our case, mainstay ACLS treatment was ineffective in terminating the VF arrhythmia, so esmolol was administered at a dose of 50 mg IV bolus (500 mcg/kg) with subsequent ROSC. The literature on the use of esmolol in RVF has yet to cross into the realm of use in a traumatic RVF. As this case report discusses, there are many possible benefits of esmolol for patients in RVF. Our case, however, challenges the notion that esmolol is not suitable for use in an undifferentiated traumatic injury when massive hemorrhage or hypovolemia is unlikely. Treatment with esmolol may be a reasonable option in traumatic CA patients that are in RVF with no signs of exsanguination and a corresponding negative eFAST.

Conclusion

More prospective studies of beta-blockade in CA are warranted. Beta-blockade with esmolol should be considered in patients with RVF, resistant to standard therapy prior to cessation of resuscitative efforts, and should be considered in patients even with a possible traumatic history.

Statement of Ethics

The authors have no ethical conflicts to disclose. Written informed consent was obtained from the patient for publication of this case report. There are no patient identifiers in the case report which may link the patient to the report. The study is exempt from ethics committee approval due to being a retrospective chart review of <3 patients and therefore did not meet the DHHS definition of research.

Conflict of Interest Statement

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Author Contributions

K.R. conceived and designed the study, collected the data, wrote the manuscript, and performed final edits. E.W. and M.S. collected the data and assisted with writing the manuscript and performing final edits.

Data Availability Statement

All data generated or analyzed during this case report are included in this article. Further enquiries can be directed to the corresponding author.

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