Management of Refractory Ventricular Fibrillation (Prehospital and Emergency Department)

Sean M. Bell, MD^a, David H. Lam, MD^{a,b}, Kathleen Kearney, MD^{a,b}, Ravi S. Hira, MD^{a,b,c,*}

KEYWORDS

- Refractory ventricular fibrillation Urgent coronary angiography Out-of-hospital cardiac arrest
- Extracorporeal membrane oxygenation Mechanical cardiopulmonary resuscitation

KEY POINTS

- Refractory ventricular fibrillation carries a high mortality rate, and often does not respond to conventional therapy.
- Most patients presenting with out-of-hospital cardiac arrest and refractory ventricular fibrillation have an acute thrombotic coronary artery lesion; urgent coronary angiography with revascularization is critical.
- Extracorporeal membrane oxygenation allows providers additional time to diagnose and treat potentially reversible causes of ventricular fibrillation-associated cardiac arrest.
- Novel multifaceted approaches for the management of refractory ventricular fibrillation have provided encouraging results.

Video content accompanies this article at www.cardiology.theclinics.com.

INTRODUCTION

Ventricular fibrillation (VF) is a potentially fatal, lifethreatening cardiac arrhythmia that can lead to loss of cardiac function and sudden cardiac death. VF is characterized on an electrocardiogram (ECG) as irregular and disorganized electrical activity without any discernible pattern. With an annual incidence of 12.1 per 100,000 people, VF remains the leading cause of sudden cardiac death and out-of-hospital cardiac arrest (OHCA).¹ Prompt treatment with resuscitation and defibrillation can be life saving and, therefore, recognition of VF by first responders and medical professionals is essential. However, an estimated 4% to 5% of OHCA is characterized by VF that does not respond to multiple attempts at defibrillation, a phenomenon known as refractory VF. The incidence of refractory VF is estimated at 0.5 to 0.6 per 100,000 people.²

Despite improvement in survival rates of patients experiencing OHCA, survival in the subset of patients with refractory VF remains poor.² Mortality rates among patients with refractory VF



Disclosure: R.S. Hira is a consultant for Abbott Vascular, Inc. S.M. Bell, D. H. Lam, and K. Kearney have nothing to disclose.

^a Department of Medicine, University of Washington, 1959 Northeast Pacific Street, Seattle, WA 98195, USA;

^b Division of Cardiology, University of Washington, 1959 Northeast Pacific Street, Seattle, WA 98195, USA;

^c Department of Medicine, Division of Cardiology, Harborview Medical Center, 325 Ninth Avenue, Seattle, WA 98104, USA

^{*} Corresponding author. 325 Ninth Avenue, Box 359748, Seattle, WA 98104.

E-mail address: hira@uw.edu

range between 85% and 97%, and the proportion of patients with neurologically intact function at 1 month after refractory VF is estimated at only 5.6%.^{2,3} By contrast, patients who present with OHCA and an initial shockable rhythm have a survival to hospital discharge rate of 33.0%, with survival with good neurologic function of 30.1% as reported in the Cardiac Arrest Registry to Enhance Survival (CARES) in 2015.⁴ This finding highlights a need for early detection and improved intervention for patients who suffer from refractory VF.

Normal cardiac electrophysiology requires intricate, spontaneous, automatic, and synchronized signaling within the cardiomyocytes. Typically, the electric signal originates from pacemaker cells in the sinoatrial node in the right atrium. The signal then continues toward the atrioventricular node and, ultimately, through the ventricles producing synchronized muscular contraction. Under normal conditions, atrial and ventricular myocytes do not spontaneously depolarize. If, however, ventricular cardiomyocytes have endured damage in some form, they can become prone to electrical hyperexcitability and depolarization without a signal from the sinoatrial node. This scenario is most applicable to patients who experience acute myocardial injury, usually from ischemic events. Without synchrony, there is a loss of coordinated contraction and reduced cardiac output. This situation, in turn, leads to a vicious cycle of continued ischemic damage and further deterioration of the electrophysiologic integrity of the myocardium. By ECG, this cycle manifests as increased fibrillation cycle length of the ventricles (ie, VF) and the usual P, QRS, and T wave morphologies are replaced with an erratic rhythm. This pattern results in mechanical dyssynchrony without adequate ventricular ejection and stroke volume. Without intervention, the VF rhythm eventually progresses to asystole and death.

In this review, we summarize therapeutic interventions and guidelines to provide guidance to clinicians managing patients with OHCA and refractory VF, both in the prehospital and emergency department (ED) setting. The approach to VF focuses on initiation of aggressive resuscitation and supportive care, which includes advanced cardiac life support (ACLS), restoration of normal electrical activity by defibrillation and/or medical therapy, followed by appropriate postresuscitation care after return of spontaneous circulation (ROSC). This process includes hemodynamic stabilization, prompt investigation for the underlying etiology of VF, and targeted temperature management (TTM) when indicated. Investigation of the underlying etiology is done with a history and physical examination, laboratory results, imaging, and

ancillary investigation, which may include coronary angiography. In patients with refractory VF who do not have ROSC, however, there is currently no accepted consensus on management after failed defibrillation and resuscitation attempts. Here, we discuss a strategy of limited duration of resuscitative efforts in the field followed by prompt transfer to a facility capable of immediately initiating use of venoarterial extracorporeal membrane oxygenation (ECMO) in these patients followed by cardiac catheterization and revascularization.

CURRENT GUIDELINES

Current management guidelines for VF emphasize the importance of early, high-quality cardiopulmonary resuscitation (CPR) at a rate of 100 to 120 compressions per minute.⁵ Ventilatory support is provided at a rate of 2 breaths for every 30 compressions or 1 breath every 6 seconds if an advanced airway is in place. After the initiation of CPR, use of an automated external defibrillator (AED) should be implemented rapidly with the defibrillation pads placed and cardiac rhythm assessment performed. If this process identifies a shockable rhythm, the shock should be delivered as soon as possible. If a biphasic AED is in use, the initial shock energy delivered is based on the manufacturer's recommendation. If the manufacturer's recommendation is unknown, then the highest energy level available is recommended (typically 200 J). For a monophasic AED, the recommended shock energy is 360 J. CPR is then resumed immediately for 2 minutes, during which time vascular access should be attained. CPR is continued in 2-minute intervals, with rhythm checks performed every 2 minutes. If ROSC is achieved, the patient is then immediately transitioned to post-cardiac arrest care. If ROSC is not achieved, and the rhythm check continues to show VF, a shock should be delivered with immediate resumption of CPR. After the second defibrillation attempt, 1 mg of epinephrine either intravenous or intraosseous every 3 to 5 minutes is recommended. After the third defibrillation attempt, an initial amiodarone load of 300 mg administered intravenously or intraosseously is recommended, followed by doses of 150 mg. If the patient remains in VF despite 3 defibrillation attempts and optimal medical therapy, we would define this as refractory VF with a significant decrease in anticipated survival.⁶

CARDIOPULMONARY RESUSCITATION

Early recognition and intervention of patients with OHCA is critical for patient survival. In a

Download English Version:

https://daneshyari.com/en/article/8657585

Download Persian Version:

https://daneshyari.com/article/8657585

Daneshyari.com