Title: Dysrhythmias and heart failure complicating acute myocardial infarction: An Emergency Medicine Review

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1. Abstract

1.1 Introduction: Patients with acute myocardial infarction (AMI) may suffer several complications after the acute event, including dysrhythmias or heart failure (HF). These complications place patients at risk for morbidity and mortality.

1.2 Objective: This narrative review evaluates literature and guideline recommendations relevant to the acute emergency department (ED) management of AMI complicated by dysrhythmia or HF, with a focus on evidence-based considerations for ED interventions.

1.3 Discussion: Complications occurring acutely after AMI include dysrhythmia and HF. Limited evidence exists for dysrhythmias in AMI due to relatively low prevalence and frequent exclusion of patients with active cardiac ischemia from clinical studies. Management decisions for bradycardia in the setting of AMI are determined by location of infarction, timing of the dysrhythmia, rhythm assessment, and hemodynamic status of the patient. Atrial fibrillation is common in the setting of AMI, and caution is warranted in initiating rate control given the possibility of compensation for decreased ventricular function. Regular wide complex tachycardia in the setting of AMI should be assumed to be ventricular tachycardia in the majority of cases. Management directed towards HF in AMI consists of noninvasive positive pressure ventilation, early cardiac catheterization, and nitroglycerin therapy if right ventricular

involvement is not suspected. Norepinephrine is the first line vasopressor for patients with cardiogenic shock and hypoperfusion on clinical exam. Early involvement of a multi-disciplinary team is recommended when caring for patients in cardiogenic shock.

1.4 Conclusions: This review discusses considerations of ED management of dysrhythmias and HF associated with AMI.

Keywords: acute myocardial infarction; dysrhythmia; heart block; atrial fibrillation; bradycardia; ventricular tachycardia; heart failure; cardiogenic shock

2. Introduction

Early complications associated with acute myocardial infarction (AMI) have decreased in the era of reperfusion therapy but remain a source of morbidity and mortality among emergency department (ED) patients. Among 10 million annual ED visits for chest pain, 625,000 patients are diagnosed with acute coronary syndrome (ACS) of which 30% are STEMI.[1] The prevalence of ST elevation myocardial infarction (STEMI) decreased from 133 to 50 cases per 100,000 patient admissions from 1999 to 2008, respectively.[2] The risk of dysrhythmia from AMI varies by infarct location. In a multinational registry of patients with ACS, 4.6% developed cardiogenic shock.[3] Historically, ventricular arrhythmias were the leading cause of death in AMI, but cardiogenic shock is the leading cause of death among patients hospitalized for AMI in the modern era.[4]

Patients with AMI complicated by dysrhythmia or heart failure represent a subset of patients at high risk for morbidity and mortality. Attentive supportive care addressing volume optimization, electrolyte replacement (especially hypokalemia and hypomagnesemia), and avoidance of hypoxia are important elements in each disease process.[5] Early cardiology consult with urgent or emergent coronary angiography is indicated for the majority of these patients.

The objective of this narrative review is to provide emergency providers with an evidence-based review of the prevalence and prognosis of dysrhythmias or heart failure in the setting of AMI. This manuscript aims to provide data and recommendations relevant to patients presenting to ED with AMI or following recent discharge for AMI complicated by dysrhythmia, pulmonary edema, or cardiogenic shock. Application of evidence-based medicine is particularly challenging for the acute management of this patient population given the low prevalence of these conditions and frequent exclusion of patients with AMI or concern for active coronary ischemia from studies of these disease processes. Mechanical, inflammatory, and embolic complications of AMI are covered in a separate review. This review will not cover treatment issues for cardiac arrest, general treatment for ACS, ECG interpretation and rhythm recognition, and treatment considerations beyond acute stabilization in the ED.

3.Methods

Authors searched PubMed and Google Scholar using a variety of keywords, including myocardial infarction, acute coronary syndrome, electrical complications, atrioventricular block, bradycardia, atrial fibrillation, ventricular tachycardia, cardiogenic shock, and acute heart failure for production of this narrative review. Authors also reviewed guidelines and supporting citations for emergency management of acute complications of AMI related to electrical complications, heart failure, or cardiogenic shock. The literature search was restricted to studies published in English. Inclusion was based on relevance to emergent care of electrical complications and heart failure in the setting of AMI based on author review. This is a narrative review so no pooling of study data was performed.

4. Discussion

4.1 Perfusion of the cardiac conduction system

An understanding of coronary perfusion in terms of the cardiac conduction system can assist in predicting the occurrence and prognosis of different dysrhythmias based on infarct location. Table 1 displays the most common sources of perfusion for the cardiac conduction system in terms of the right coronary artery

(RCA), posterior descending artery (PDA), left circumflex artery (LCX), and left anterior descending artery (LAD), and Figure 1 depicts the cardiac conduction system.[6,7] RCA is the most common culprit artery in inferior MI (80% of cases) followed by LCX lesions.[6] LAD is the most common culprit artery in anterior MI and the proximal LCX is the most common in lateral MI.[6,8]

Table 1. Perfusion of the cardiac conduction system.

Conduction location	Right coronary artery	Left circumflex artery	Left anterior descending artery
Sinoatrial node	60%	40%	0%
Atrioventricular node	90%	10%	0%
Right bundle branch block*	33%	13%	52%
Left bundle branch block*	17%	0%	43%

*The location of culprit lesions in patients in GUSTO I and TAMI trials with new onset right and left bundle branch block in AMI.[7]

Figure 1. Cardiac Conduction System. From https://commons.wikimedia.org/wiki/File:2018_Conduction_System_of_Heart.jpg

4.2 Bradydysrhythmias

Etiology

Bradydysrhythmias are present in 25-30% of AMI. Sinus bradycardia (SB) is the most common (40%), followed by junctional bradycardia (20%) and idioventricular rhythm (15%).[9,10] Compromising bradycardia due to AMI is rare, occurring approximately once per 10,000 ED visits and accompanying 14% of compromising bradycardia presenting to the ED in a European registry.[11] Among patients with unstable bradycardia associated with AMI, third-degree atrioventricular block (AVB) is most common (40%), followed by sinus bradycardia (25%) and junctional rhythm (20%).[9] Sinus bradycardia is the most common bradydysrhythmia associated with AMI, but there is no increase in mortality compared to the general population of AMI patients.[10]

Two mechanisms are responsible for the majority of bradydysrhythmias in the setting of AMI. First, the physiologic changes associated with AMI result in a state of autonomic instability. Increased

parasympathetic input to the cardiac system can trigger bradydysrhythmias. Second, AMI may cause ischemia or infarction of the cardiac conduction system. Many bradydysrhythmias are likely the result of a combination of these two mechanisms. The type of bradydysrhythmia, timing of onset after AMI, and location of coronary infarct can assist determining which mechanism is most likely dominant for an individual patient.

Atrioventricular Block (AVB)

AVB associated with AMI can be divided into early-onset AVB developing within six hours of AMI onset versus late-onset AVB developing after six hours. Early-onset AVB is more likely to be due to autonomic imbalance from AMI resulting in increased parasympathetic input.[12] Myocardial reperfusion may play a role in triggering this reflex.[13] Early-onset AVB is likely to present with very slow ventricular rates and be more responsive to atropine compared to late-onset AVB.[12] Late-onset AVB presenting more than six hours after AMI onset is more likely to be due to ischemia of the cardiac conduction system. Late-onset AVB tends to present with wide QRS complexes and higher ventricular rate than early-onset AVB. Patients with AVB associated with AMI tend to be younger and experience chest pain and hypotension when compared to all comers with AVB.[14]

Although first-degree AVB is typically a benign finding, weak evidence suggests that first-degree AVB carries an increased risk of progression to high-grade AVB (HAVB) in the setting of AMI. Among 14 patients identified with HAVB >6 hours after AMI, 12 of these were preceded by a hemodynamically stable period with first-degree AVB.[12] Cardiac monitoring for patients with first-degree AVB in the setting of AMI is recommended, in order to evaluate for progression to HAVB.[15]

AVB was more common in posterior MI (7.3%) than anterior MI (3.0%) in one study of elderly patients with AMI.[16] HAVB consists of type II second-degree AVB and third-degree AVB (also known as complete heart block). The vast majority of HAVB in AMI is third-degree AVB. Among patients with STEMI in a recent prospective registry, 2.2% of patients had HAVB at the time of admission.[17] A large

registry of ACS found 1.3% prevalence of HAVB on presentation.[18] This is decreased from prior studies noting a 2.7-14% prevalence of HAVB among patients admitted with STEMI.[17] Risk factors for developing HAVB among STEMI patients included RCA culprit lesion lesions, age >65 years, hypertension, diabetes, and female gender.[19] Hospital mortality for patients with HAVB is increased compared to all comers with STEMI (29% vs 18%), although no mortality difference was found on multivariable analysis.[17] Mortality and risk of hemodynamically instability are highly affected by the location of myocardial infarction, with inferior MI having a more favorable prognosis than anterior MI. HAVB in the setting of inferior MI is usually due to ischemia proximal to the His bundle.[20] The escape rhythm tends to have a narrow QRS, and many patients tolerate this rhythm without hemodynamic compromise.[6] HAVB from anterior MI is usually the result of ischemia to the trifascicular conduction system distal to the AV node. The infarct size of anterior MI causing HAVB is usually larger than HAVB associated with posterior MI.[21] The escape rhythm tends to be slow, have a wide QRS, and has a greater risk of hemodynamic compromise than the escape rhythm in inferior MI.[6,9]

Management

Atropine increases automaticity of the SA and AV nodes by inhibiting parasympathetic input. Indications for atropine include sinus bradycardia with hypoperfusion, symptomatic second-degree AVB, or third-degree AVB associated with a narrow QRS complex (Class IIa, LOE B).[9,22] In a retrospective review of prehospital patients with hemodynamically unstable bradycardia (n=86) or AVB (n=35) who received atropine in the prehospital setting, 47.3% of patients had clinical improvement, defined as systolic blood pressure (SBP)>90 mm Hg and heart rate (HR)>60, following atropine administration. Approximately half of these responses were transient, while the remainder were sustained throughout the prehospital phase. A minority of patients had AMI (34.4%), but the response to atropine was similar in the AMI subgroup to those without AMI.[23] Potential adverse effects of atropine include worsening of HAVB, increased risk of dysrhythmia, and progression of infarction. In the same prehospital study, 4 patients (3.1%) developed the following dysrhythmias: frequent premature ventricular contractions (2), ventricular

tachycardia (1), and ventricular fibrillation (1).[23] This risk of dysrhythmia was lower than a study of 56 patients in the critical care unit receiving atropine for SB in which 5% of patients had increased premature ventricular contractions and 5% had ventricular dysrhythmias.[24]

Pacer pads should be placed prophylactically in patients presenting with bradycardia. In cases of bradycardia with hemodynamic compromise unresponsive to atropine (or if atropine is contraindicated), then transcutaneous pacing (TCP) should be initiated as a bridge to transvenous pacing or definitive management. A recent prospective, observational study in the ED found increased systolic blood pressure and HR with the use of TCP in patients with bradycardia unresponsive to atropine.[25] Although data is limited by small sample size for transcutaneous pacing for symptomatic bradycardia in the PH setting, two randomized controlled trials (RCTs) found improved survival in the patients with a pulse on EMS arrival to the scene who received TCP versus those who did not receive TCP.[26,27]

The decision to start transvenous pacing is multifactorial based on the presenting rhythm, hemodynamic stability, and hospital setting/consultant support. While placing transcutaneous pacing pads can be accomplished easily, the decision to institute transvenous pacing is more challenging. In a study of 277 ED patients with bradycardia and hemodynamic compromise, 20% of patients required transvenous pacing. The risk of requiring transvenous pacing was doubled (43%) among the 40 patients with bradycardia secondary to AMI.[11] When considering the risk of progression to complete heart block and development of hemodynamic compromise, the location of infarction is a key consideration. Anterior infarctions are more likely than inferior infarctions to progress to higher grade AVB and develop hemodynamic compromise. AVB in setting of anterior MI are associated with infranodal ischemia.[9] The presence of any AVB, including proximal AVB, in the setting of MI may increase the risk of progression to high grade AVB. Patients with first-degree or type I second-degree AVB should have cardiac monitoring to evaluate for progression to HAVB. Patients with second-degree type 2 are at significant risk to progress to third-degree AVB. The presence of a new bundle branch block indicates a greater risk of needing transvenous pacing. Patients with third-degree AVB, particularly in the setting of anterior

infarction, are likely to develop hemodynamic instability requiring transvenous pacing.[28] In summary, concern for hemodynamic instability warrants consideration of ED placement of a transvenous pacemaker.

4.3 Atrial fibrillation

Etiology

Reported prevalence of atrial fibrillation (AF) complicating AMI has varied between 2% and 21% over the last 30 years.[29] A recent large retrospective study of patients with ACS reported 7.6% prevalence of new AF during hospitalization for AMI.[30] Multiple factors likely play a role in the development of AF during AMI.[16] A commonly proposed mechanism is left ventricular dysfunction leading to increased atrial pressures.[31] This is supported by the association with increased risk of AF if heart failure is present on AMI presentation.[29] Other possible contributing factors include ischemia of the atria, metabolic abnormalities, or autonomic instability associated with AMI.[16]

Risk Factors

The strongest risk factor for the development of AF in the setting of AMI is acute heart failure on presentation, with an odds ratio of 1.58 for cardiogenic shock.[29] Additional risk factors include tachycardia on presentation, advanced age, and male gender.[29,30] Patients with AF in the setting of ACS had increased risk of mortality (adjusted hazard ratio [HR] 1.59), repeat MI (HR 1.14) and ischemic stroke (HR 2.29) at 90 days compared to patients in sinus rhythm in a large retrospective study.[30]

Management

Before initiating interventions to specifically address AF in the setting of AMI, an assessment is needed as to whether AF is a primary cause or significant contributor to the patient's hemodynamic condition.[32] Tachycardia may be a compensatory mechanism to the AMI, and adverse events are common following

rate control interventions. Among 15 patients receiving rate control in the setting of ACS, 4 developed significant hypotension with 2 patients requiring vasopressors.[33]

Synchronized cardioversion is indicated to treat AF in the setting of AMI if any of the following conditions are present: 1) hemodynamic instability, 2) uncontrolled rapid ventricular rate (RVR) despite pharmacotherapy, or 3) evidence of ongoing cardiac ischemia despite pharmacotherapy.[34] A recent expert opinion manuscript described stability as a continuum rather than dichotomous state, and AMI coexisting with AF should decrease the threshold for a patient to be considered unstable.[35]

Pharmacotherapy recommendations for acute rate control depend on clinical evidence of heart failure or hemodynamic instability. Amiodarone is recommended in patients presenting with reduced ejection fraction or hemodynamic instability if pharmacotherapy is used (Class IIb, LOE B).[34] In a retrospective study of 38 patients with atrial dysrhythmias in the intensive care unit (ICU) refractive to conventional therapy, patients receiving amiodarone had acute decreases in HR and increases in SBP, while the majority of patients receiving diltiazem, esmolol, or digoxin had decreases in SBP without slowing the HR.[36] The choice of rate control agent in hemodynamically stable patients is less clear. The American Heart Association/American College of Cardiology (AHA/ACC) and European Society of Cardiology (ESC) guidelines recommend intravenous beta blockers (Class I, LOE C) over non-dihydropryidine calcium channel blockers (Class IIb, LOE C) for acute rate control of hemodynamically stable patients in the setting of ACS.[31,34] Notably, this recommendation appears largely based on expert opinion, as studies evaluating rate control agents in the ED for AF with RVR usually do not include patients with AMI.[37–39] The same guidelines recommend avoiding non-dihydropryidine calcium channel blockers if any evidence of heart failure or hemodynamic instability is present (Class IIb, LOE C).[34]

4.4 Accelerated idioventricular rhythm

Accelerated idioventricular rhythm (AIVR) is characterized by a ventricular rhythm with a heart rate of 60 to 120 beats per minute.[40] A rate greater than 120 beats per minute is concerning for VT. This rhythm

is seen most commonly during therapy either with thrombolytic medications or PCI, but may rarely be seen in the setting of AMI. The presence of AIVR does not affect prognosis. AIVR is a stable escape rhythm, and antidysrhythmic treatment is not recommended.[41]

4.5 Ventricular tachycardia

Etiology

VT is a ventricular rhythm with a QRS duration greater than 120 milliseconds, a rate of at least 120 beats per minute, and loss of association between atrial and ventricular depolarization.[42] Sustained VT is estimated to occur following 1% of AMI.[43] This is a decrease from historical estimates of 3-5%, presumably secondary to improved cardiac care to reduce infarct size in AMI.[43] VT usually occurs remotely from the AMI event as a result of reentry pathways at the site of myocardial scar.[43] VT in the setting of AMI is usually due to abnormal automaticity causing focal activity at the border of the infarction. In early ischemia, mild hyperkalemia causes greater excitability of cardiac myocytes. As ischemia progresses, worsening hyperkalemia results in slowed conduction and decreased excitability. This mismatch may result in depolarization-induced automaticity in cells bordering the infarct. This focal activity can promote VT.[44] Notably, this is a different mechanism from the majority of VT due to reentry pathways in areas of scar from prior MI.[43]

Risk factors

Risk factors for VT include large infarct size and reduced ejection fraction.[43] Multiple algorithms exist for distinguishing ventricular tachycardia from other causes of regular wide complex tachycardia.[45] The occurrence of wide complex tachycardia during or shortly after AMI strongly suggests VT.[46]

Management

Immediate cardioversion is recommended for VT with a pulse in the setting of ischemic chest pain or hemodynamic instability.[47,48] Stable monomorphic VT is typically defined as the prevalence of VT in the

setting of minimal patient symptoms. The threshold to categorize a patient with AMI as 'unstable' VT should be low, with consideration of immediate cardioversion.[42] For patients with persistent VT despite attempted direct current cardioversion, intravenous amiodarone is recommended (Class I, LOE A) along with further attempts at cardioversion.[49] Among 18 patients receiving amiodarone in the setting of unstable VT (8 had AMI), 78% had termination of VT with a protocol involving repetitive shocks and amiodarone boluses.[50]

For hemodynamically stable patients with regular wide complex tachycardia, the 2017 AHA guidelines for ventricular arrhythmias include procainamide (Class IIa, LOE A), amiodarone (Class IIb, LOE B), and sotalol (Class IIb, LOE B).[48] The trials supporting procainamide did not include patients with AMI or severe anginal symptoms.[51,52] The PROCAMIO trial found improved termination of VT (67% vs 38%) and fewer major adverse events (9% vs 41%) for procainamide versus amiodarone, respectively.[52] A retrospective cohort of 97 infusions of amiodarone or procainamide for stable sustained VT in the ED included 9 patients with STEMI or NSTEMI. Among patients receiving procainamide, 19% had discontinued infusions secondary to hypotension.[53] In summary, most patients with VT in the setting of AMI should undergo electrical cardioversion. Although procainamide has recently been shown to terminate stable VT more frequently than amiodarone, there is little evidence to support procainamide use in the setting of AMI.

4.6 Acute heart failure associated with AMI

Myocardial ischemia was identified as the precipitating factor in 15% of heart failure admissions in the OPTIMIZE-HF trial.[54] In-hospital mortality was independently higher (adjusted odds ratio 1.2) on multivariate analysis for patients with ACS as the precipitating factor compared to all HF admissions.[54] The degree of HF on admission is commonly characterized by physical examination using the Killip classification.[55] Patients with Killip class I have no evidence of HF on physical examination. Patients with Killip class II and III have preserved SBP of at least 90 mmHg with pulmonary edema involving less than a third (class II) or greater than a third (class III) of the lung fields on physical examination. Patients

with Killip class IV have cardiogenic shock, with evidence of pulmonary edema and SBP less than 90 mmHg.[55] In-hospital mortality is greater for patients with AMI and HF on initial presentation compared to AMI alone (OR 3.1 in one study).[56] Increasing Killip classification was associated with greater mortality in one study of non-STE ACS patients, which found 30 day mortality of 2.8% in Killip class I, 8.8% in Killip class II, and 14.4% in Killip class III or IV.[55] Risk factors for HF complicating AMI include advanced age, hypertension, diabetes, and prior MI. [55,56]

Heart failure with normal or elevated blood pressure

Management directed towards HF in the setting of LV dysfunction from AMI is similar to the ED management of HF not associated with AMI.[57] Initial resuscitation to address pulmonary edema includes noninvasive positive pressure ventilation and nitroglycerin as tolerated by blood pressure.[32] Oxygen saturation should be maintained above 90%.[58] High dose nitrate therapy with low dose furosemide led to fewer intubations and AMI compared to high dose furosemide and low dose nitrate therapy in one RCT.[59]

The presence of AMI is not a contraindication to diuretic use in acute HF management and is recommended by recent ESC guidelines (Class I, LOE C) for patients with STEMI and evidence of volume overload on clinical exam.[5] Early use of diuretics in HF has recently received a great deal of attention. In an observational study showing an association between early furosemide treatment and decreased in-hospital mortality, patients with ACS requiring urgent catheterization were excluded.[60] As ESC guidelines recommend urgent catheterization for all patients with decompensated HF in the setting of ACS, this study may not apply to the HF subgroup with AMI.[58] In summary, management directed towards HF in AMI should focus on nitroglycerin therapy and noninvasive positive pressure ventilation to address pulmonary edema. An intravenous dose of diuretic equal to the patient's home maintenance dose can be given if clinical assessment suggests volume overload.[61]

Cardiogenic Shock

Cardiogenic shock (CS) occurs in 4-9% of patients with AMI across recent studies.[62] In a study of multiple registries, prevalence of CS in AMI slightly decreased from 1995 to 2005 from 6.9% to 5.7%.[63] Risk factors for CS include advanced age, female gender, prior MI, and diabetes.[4,64,65] The majority of CS is precipitated by STEMI and typically occurs hours to days after initial presentation.[62]

The most common etiology of CS in the SHOCK registry was left ventricular failure (78%).[66] Isolated right ventricular failure and mechanical complications precipitated CS in 3% and 12% of patients respectively.[66] Half of patients had MI in multiple locations, with anterior MI (55%) and posterior MI (46%) being the most common infarct locations.[66]

Patients with AMI complicated by cardiogenic shock can rapidly decompensate due to a positive feedback loop: 1) myocardial ischemia leads to decreased ejection fraction, 2) the resulting decrease in diastolic blood pressure worsens coronary perfusion pressure, 3) decreased coronary perfusion worsens coronary ischemia. This loop provides significant challenges in management.

Management

Patients with CS in the setting of AMI require immediate resuscitation and cardiology consultation. Echocardiography is needed to evaluate for mechanical complications.[31] For management and further discussion of mechanical complications of AMI, please see part 2 of this review. Immediate cardiac catheterization is indicated if any of the following conditions persist despite medical management: ST segment deviation on electrocardiogram (ECG), anginal symptoms, or end organ hypoperfusion.[31]

The decision to initiate vasopressors and selection of a specific medication rests on a balance of benefit and risk. An important benefit is the increase in diastolic blood pressure (DBP) to improve coronary perfusion. Adverse effects of vasopressors include risk of progression of infarct, increased risk of dysrhythmias, and an increase of myocardial oxygen consumption which can worsen existing ischemia or infarction.[67]

The choice of vasopressor agent must weigh multiple factors including the degree of hypotension, the patient's perfusion status on clinical assessment, emergency provider comfort with use of different agents, and preference of consultants assisting with patient care. A reasonable target blood pressure is a mean arterial pressure (MAP) of at least 65 mm Hg.[68,69]

Norepinephrine is recommended as the first line vasopressor for CS in the setting of AMI with SBP <70 mm Hg given the immediate need for improvement in blood pressure. An RCT comparing dopamine and norepinephrine use in shock found that dysrhythmias were more common with the use of dopamine (24%) versus norepinephrine (12%).[70] A subgroup analysis of patients with cardiogenic shock found decreased mortality in patients randomized to norepinephrine compared to dopamine.[70] In a pilot study of 30 patients with CS which excluded AMI, epinephrine was associated with dysrhythmias in 20% of patients compared to 0% in the norepinephrine-dopamine arm.[71]

Patients with CS and SBP 70-100 mm Hg require clinical decision making in choosing a vasopressor or inotrope. Norepinephrine is again recommended if signs of shock are present on clinical assessment.[67] This is a change from previous guidelines, which recommended dopamine as the first line agent for this patient population.[28] Norepinephrine is recommended over epinephrine as epinephrine is associated with increased oxygen consumption, lactate levels and higher mortality rates.[71,72] Dobutamine is recommended if the patient has adequate perfusion, such as warm extremities, and lack of severe end organ dysfunction by clinical assessment.[67] Dobutamine primarily acts as an agonist to beta 1 and beta 2 receptors. The net effect at low doses is increased myocardial contractility and vascular smooth muscle relaxation. Many patients will experience a decrease in blood pressure due to vasodilation. Adverse effects of dobutamine include increased myocardial oxygen consumption and increased risk of dysrhythmia, with one study finding non-sustained VT in 13% of patients during the first 24 hours of treatment.[73] Milrinone is a phosphodiesterase inhibitor which acts as an inotrope and vasodilator. An important difference from dobutamine is that parenteral milrinone has a half-life of 2-4 hours, so initiation of this medication in the ED can have prolonged clinical effects.[67] Inotropes have not been shown to improve mortality in

cardiogenic shock, and these agents are only a bridge for coronary perfusion in the acute shock state until other treatments, such as PCI or mechanical support, are performed.[67,74,75]

In cases of CS due to AMI, myocardial revascularization is the only evidenced-based therapy with proven survival benefit.[69] However, mechanical circulatory support is an essential part of the management of CS and is commonly utilized. Non-pharmacologic measures to improve organ perfusion include the use of an intra-aortic balloon pump (IABP), Impella placement, and veno-arterial extracorporeal membrane oxygenation (ECMO). [76–80] IABP decreases afterload, thereby increasing cardiac output, systemic pressures, coronary perfusion pressure and end-organ perfusion. Impella and TandemHeart devices are percutaneous left ventricular assist devices that provide mechanical circulatory support but do not alter afterload.[69] Hemodynamic optimization should not delay definitive treatment via percutaneous closure device or primary revascularization.[81–83]

Right ventricular failure

A minority of patients have CS due primarily to right ventricular (RV) failure, as seen in 2.8% of CS patients in the SHOCK registry.[66] These patients tend to be younger and present with inferior and/or posterior infarctions.[84] Causes of RV failure during an AMI can generally be divided into three main categories: insufficient myocardial contractility, excessive preload, and excessive afterload.[85] Insufficient myocardial contractility is primarily due to local myocardial ischemia or overstretching of the RV free wall. Excessive preload may be due to an acute left-to-right shunt such as a ventricular septal rupture. Excessive afterload is typically due to acute left heart failure and cardiogenic shock.[85]

Management

Proper fluid management is critical for successful management of RV failure. The goal is to optimize RV preload and perfusion while avoiding fluid overload. If low intravascular volume is suspected, fluid resuscitation should be instituted as quickly as possible. In the setting of clear intravascular depletion, clinicians should use low-volume boluses, such as 250 mL of Lactated ringer's solution. Frequent, serial

assessments of end-organ perfusion, including blood pressure, urine output, and serum lactate are needed to reduce the risk of iatrogenic volume overload.[86] Care must be taken to avoid volume overloading the RV to a degree that high RV pressures cause bowing of the interventricular septum into the left ventricle, with resulting impairment of left ventricular function.[84] Additionally, volume loading and subsequent RV dilation increases free wall tension as well as oxygen demand, inducing RV ischemia. Diuresis can improve cardiac output in these settings of RV volume overload, but caution is warranted to avoid overshooting this decrease in preload beyond optimal levels with resultant decreased cardiac output.[85]

Systemic hypotension should be avoided at all costs. Cardiac output in this patient population is difficult to augment due to excessive RV afterload worsening right heart hemodynamics. Interventions aimed at reducing RV afterload should target correction of any hypercapnia, academia, and alveolar hypoxia.[85] Conversely, augmenting aortic root pressure with vasopressors may increase coronary perfusion pressure, preventing right heart ischemia.[86] Norepinephrine maintains coronary perfusion while slightly augmenting inotropy, making it an ideal first line agent.[86,87] Vasopressin may serve to decrease pulmonary vascular resistance[88] whereas phenylephrine generally increases pulmonary vascular resistance.[89] Dobutamine should be avoided in these patients, as it causes increased tachycardia and decreases systemic vascular resistance, which are both detrimental in the RV failure patient.[87,90]

Patients with acute RV failure do not tolerate arrhythmias, as they require adequate filling time as well as atrial contraction to maintain cardiac output.[86] In general, cardiodepressants such as beta blockers and calcium channel blockers should be avoided as they impair RV function. Electrocardioversion of new-onset arrhythmias should be strongly considered in this population.[86,90]

If nitrates or mechanical ventilation are used, then close monitoring for hemodynamic decompensation is needed. Positive pressure ventilation can impede RV preload by increasing intrathoracic pressure and increasing RV afterload. Acute, possibly fatal hemodynamic collapse is a well-recognized complication of intubation in RV failure patients, most likely due to impaired venous return and sedation-induced decrease in SVR. Emergency clinicians should be prepared for profound hypotension that can occur with

rapid sequence intubation in this patient population.[86] Mechanical ventilation settings for these patients parallel those for acute respiratory distress syndrome, with a goal of low tidal volumes and plateau pressures. Minimal positive end expiratory pressure should be used, and permissive hypercarbia/hypoxia should be avoided, as they may contribute to pulmonary vasoconstriction.

Patients may require mechanical circulatory support in the setting of refractory RV failure. Currently available devices include an in situ centrifugal pump (CentriMag), an axial catheter-based pump (Impella RP), and a catheter with an extracorporeal centrifugal pump (PROTEK Duo).[69] Venoarterial ECMO remains another option as a bridge to myocardial recovery, decision, durable mechanical circulatory support, heart transplant or decision for palliative therapy.[69]

5. Conclusions

Acute complications in the post-AMI period include dysrhythmias and heart failure. Dysrhythmias include bradycardia, heart block, atrial fibrillation, ventricular tachycardia, and several others. The benefits of atropine outweigh the potential adverse effects for the majority of patients with AMI presenting with symptomatic bradycardia. The decision to initiate or prepare for transvenous cardiac pacing should incorporate the presenting rhythm, location of infarction, and hemodynamic status of the patient. Rate control for AF in AMI leads to significant hypotension in a minority of patients. Regular wide complex tachycardias in the setting of AMI should be assumed to be ventricular tachycardia with a low threshold to treat by cardioversion. Patients presenting with HF associated with AMI and no right ventricular involvement should be managed with early noninvasive positive pressure ventilation and nitroglycerin as tolerated by blood pressure. Norepinephrine is the first line vasopressor for patients with cardiogenic shock and end organ hypoperfusion. Early involvement of a multi-disciplinary team is recommended for patients with cardiogenic shock. An understanding of these complications is vital in optimizing care of patients in the post-AMI period.

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