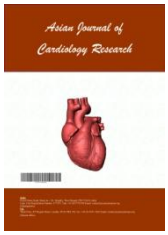

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Arrhythmia Induced by Myocardial Infarction

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Ventricular arrhythmias (VA) usually appear early in ischemia and remain a common cause of sudden death in acute myocardial infarction. Thrombolysis and the period of primary percutaneous coronary intervention caused changes in the natural course of myocardial infarction and subsequent osteoarthritis. The presence of VA can independently affect the mortality of patients recovering from a myocardial infarction. A proper risk assessment and further treatment are appropriate for these patients. Prevention and treatment of significant hemodynamic VA in the post-infarction period and sudden cardiac death outside the event remain areas of ongoing research. The aim of the review article is to show methods of treatment in cases of ventricular arrhythmia induced by myocardial infarction.

Conclusion: When studying automated VF during acute myocardial infarction (AMI), several important factors need to be taken into account: an animal with its own cardiovascular anatomy and electrophysiology; Anesthesia and its effect on heart rate and autonomic control; And the effect of coronary occlusion, size and location, and ischemic area.

Keywords: Acute myocardial infarction; ventricular arrhythmias; risk assessment; management.

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1. INTRODUCTION

Ventricular tachyarrhythmias (VA) usually occur early in ischemia and are at increased risk for patients with severe MI and ventricular arrhythmias. Primary percutaneous coronary intervention (PCI) thrombolytic therapy and the use of beta blockers alter the course of natural infarction, while increasing the incidence of ventricular tachycardia (VT) or ventricular fibrillation (VF) over several hours. Reduce the onset of acute coronary syndrome (ACS) decades ago. Hemodynamically important VA prophylaxis and treatment during the postoperative period and sudden cardiac death (SCD) outside the event are still being studied. Rapid regeneration and dosing of blood vessels, such as antiplatelet drugs, statins, angiotensin converting enzyme (ACE) inhibitors, and beta blockers, significantly reduced VA. However, about 10% of MI survivors remain at high risk of death in the first months or years after admission (death after 2 years> 25%). Sudden cardiac death after VT or progressive VF accounts for approximately 50% of all deaths in these high-risk patients [1].

There are still three classes of patients at risk: those who appear after long-term chest pain, those who have undergone partial rehabilitation only, and those who have an erythematous substrate. VA is most common in people with cardiogenic shock associated with infarct size. The genetic predisposition of VA may be present in an ischemic situation. The detection rate of repolarization (ER) changes in unconscious VF survivors and their relatives has increased significantly, as did the ECG in patients with coronary artery disease and ST-segment elevation myocardial infarction (STEMI) VA. It may be showing. There is a temporary distribution of VA after acute MI: premature or severe stages, up to 48-72 hours, which is a highly variable period of ischemia and recurrence. Endless phase with 72 hours to weeks to monthly post-events and continuous updates. Premature ventricular contractions (PVCs) are common in the first stages. The importance of this and the emergence of unsustainable or continuous VA associated with short-term and long-term forecasts have been debated for years. The combination of biochemical, electrophysiological, autonomic, and currently unknown genetic factors must lead to the so-called "complete storm" that leads to post-MI arrhythmias. Patients who develop VF or persistent VT 48 hours after the MI index show a much higher causal mortality rate. However, the relationship between VF / VT and death prematurely (within 48 hours of the MI index) is controversial. Other data suggest that persistent ventricular arrhythmia during the first period after MI may be associated with a 30-day increased mortality rate, but without long-term risk [2].

Unstable VT at a later stage does not contribute to the MI risk assessment in this group of patients. The acute myocardial infarction (horizon-AMI) trials studied the predictive effect of VA post PCI in concordance of outcomes with revascularization and stents. In this case, 5.2% of patients developed VA post primary PCI with 85% VA in the first 48 hours. They reported that sustained VT/VF was not significantly associated with 3-year mortality or serious clinical events [3].

2. MECHANISMS OF ISCHEMIA INDUCING ARRHYTHMIA

Acute MI is diagnosed histopathologically as coagulative necrosis of the myocardium. In the case of unstructured MI, it is seen within 30 to 40 minutes of progressive ischemia, the changes can only be seen on the resolution of the electron microscope. After 2 weeks, the scar grows from the surrounding area to the center and after another month its formation is complete. Any attempt to change the perfusion can reverse the process. Thus, in the post-MI period, the origin of ventricular arrhythmias and the temporal division of the mail pathways occur. The first stage, which can return within 30 minutes of the apical cardiac coronary artery closing, has been identified in animal studies. This is followed by an irreversible phase of 90 minutes to 72 hours, during which the embedded tissue components become increasingly flexible. Regeneration contributes to profound electrophysiological changes [4].

Severe ischemia causes hypoxia, which leads to a decrease in adenosine triphosphate and the accumulation of adenosine diphosphate and anaerobic glycolysis products, which lead to intracellular acidosis. This decrease in pH stimulates the exchange channels of Na + / H + and Na + / Ca +++ by releasing hydrogen ions instead of sodium, entering the cell and exchanging calcium, causing cellular Has inflammation and saturation. - Calcium. It is associated with the extracellular structure of potassium, catecholamines and lysophosphatidylcholine. This causes the cell membrane to rupture and a rapid decrease in the internal sodium current and an increase in the

sodium present at the end of the potential time (APD). Finally, the acronym APD during ischemia results from the current development of ATP-sensitive potassium, a decrease in intracellular calcium channels (blocked by acidosis) and a decrease in intracellular ATP after hypoxia. Low transmembrane relaxation, low intracellular calcium intake, and modest performance differences are also affected. Lack of anaerobic glycolysis indicates low glycogen and high intracellular lactic acid content, low ATP levels below 10%, sodium and calcium saturation, and persistent accumulation of extracellular potassium. Cells of autoimmune calcium first initiate ectopic cavity and then depolarize the deficiency [5].

Heavy purified fibers with reduced APD or amplitude, reduced depolarized membrane strength, and reduced Vmax are believed to be responsible for VA autofocus. Re-entry of intermediate and intermediate distributions affects unidirectional conduction block regions, cytopotential segmentation, and abbreviated APD-based reentry pathology. Tissue heterogeneity is prominent mainly around the infarct or in the "boundary area" and is thought to cause erythema. In particular, both human and animal studies have shown anomalous susceptibility activity in these border areas. These nerve centers are more susceptible to ischemic damage than myositis [6].

It is generally accepted that re-entry into a stable cycle with red infarct tissue is the most probable form of persistent monomorphic ventricular tachycardia. In acute myocardial ischemia in which there is no previous scar, slowly moving areas and obstructions can cause recurrence. These patients are more likely to have large areas of infarction and a large area of acute ischemic stroke may create conditions for a stable zone of relapse that may support monomorphic reentry tachycardia. Alternatively, mechanical stretching of the insufficient ventricle in the vicinity of the highly sensitive drive associated with MI may lead to VA due to underlying causes (Figs. 1, 2) [7].

2.1 COVID-19-Related Cardiac Arrhythmias

SARS-CoV-2, which has features typical of the coronavirus family and is classified in the beta-coronavirus, belongs to the same genus as human severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus, according to full-genome sequencing and phylogenic analysis (MERS-CoV) [8].

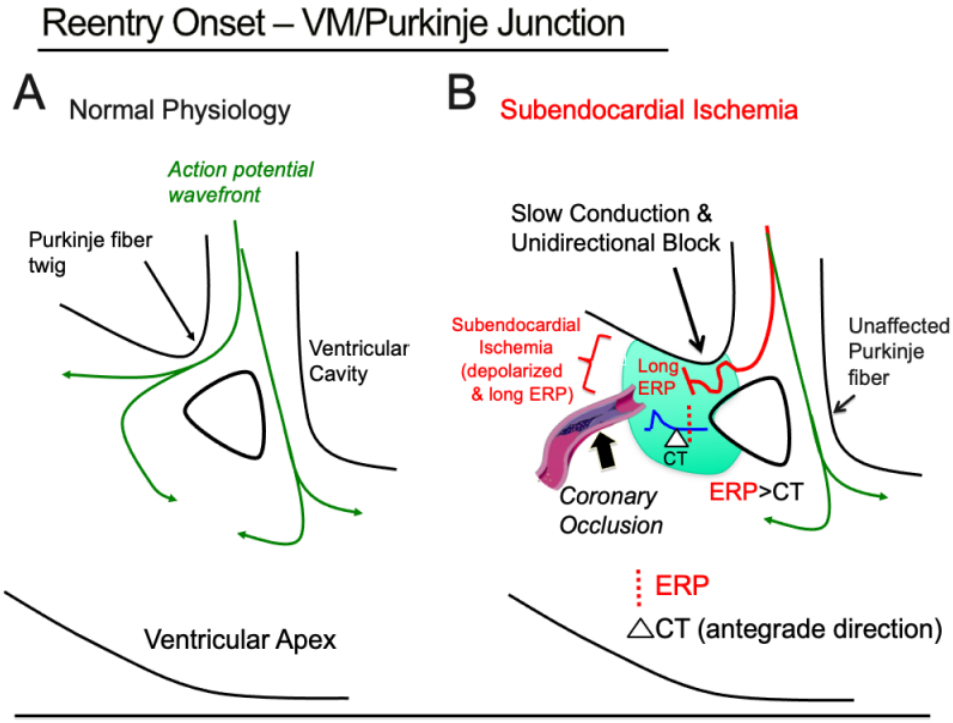

Fig. 1. Steps A, B of Ischemia inducing arrhythmia [7]

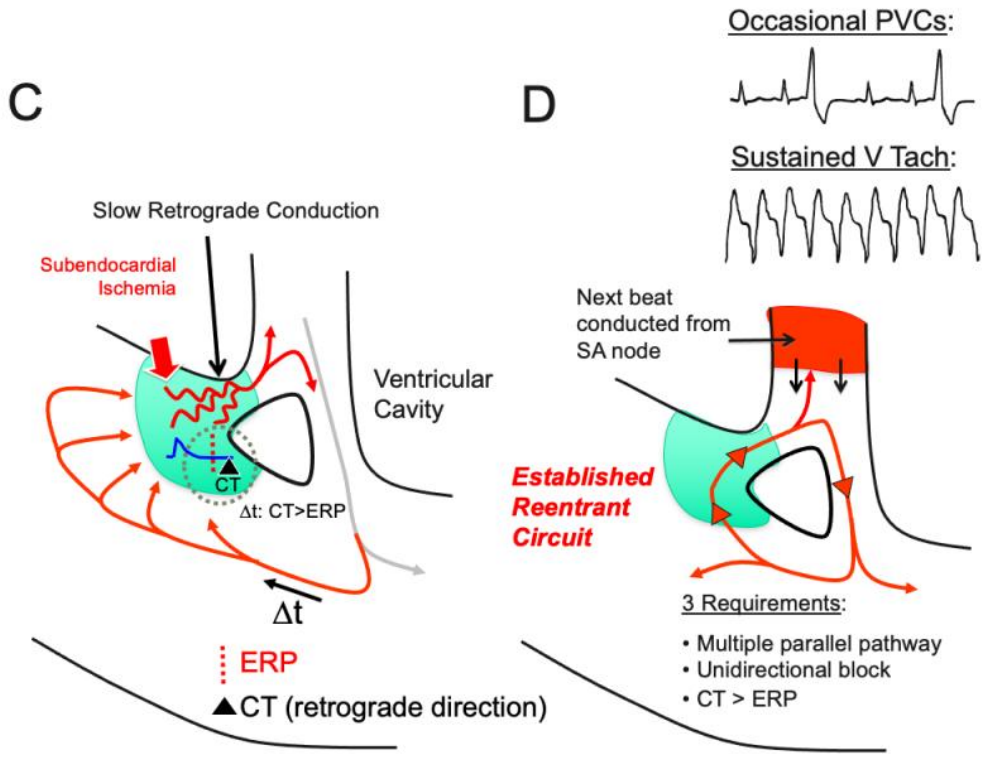

Fig. 2. Steps C, D of Ischemia inducing arrhythmia [7]

Tachycardia was the most prevalent ECG abnormality in SARS patients, but it was usually self-limiting, with an incidence of 72 percent; bradycardia was less common, with an incidence of 2 percent to 15 percent. SARS patients also had ST-T alterations and cardiac arrhythmias like branch block, atrial fibrillation (AF), premature beats, QT interval prolongation, and even sudden cardiac death (SCD). Cardiac arrhythmias were seen in 16 percent (11/70) of MERS cases, including varied tachyarrhythmias and severe bradycardia necessitating temporary pacemaker installation. In 2014, two MERS patients with tachyarrhythmia were documented; one had hyperkalemia with concomitant ventricular tachycardia and ultimately cardiac arrest, while the other developed supraventricular tachycardia later in the course of illness, both of whom died. Arrhythmias are common in SARS and MERS patients, and they can have serious consequences [9].

Patients with COVID-19 have also been documented to have cardiac involvement, which can appear as ECG abnormalities. Wang and colleagues were the first to disclose COVID-19-related cardiac arrhythmias, reporting a 17 percent (23/138) incidence of arrhythmias, 16 of whom were admitted to the intensive care unit (ICU), representing 44 percent of the total number of ICU patients. According to a recent study, just 26% of patients had normal electrocardiograms. According to Lei et al, 24% of COVID-19 patients got arrhythmias, and 33% of ICU patients developed arrhythmias. 60 percent of COVID-19 fatal cases showed arrhythmia, and cardiac arrhythmias were independently linked to a higher risk of in-hospital death (11.5 percent, vs 5.6 percent among those without arrhythmia; odds ratio, 1.95; 95 percent CI, 1.33-2.86). As a result, arrhythmia should be considered one of COVID-19's key consequences, and vigilant arrhythmia monitoring and management are required [10].

2.2 Tachycardia

In sinus rhythm, COVID-19 patients had faster heart rates (HR) ranging from 80 to 88 beats per minute (bpm). Patients admitted to the ICU had a quicker heart rate than those admitted to the normal ward. On admission, non-survivors had much quicker baseline heart rates than survivors. Another study looked at the heart rates of 17 COVID-19 patients and found tachycardia in three of them (17.6%), one of whom was a severe case and the other two were critical cases. Furthermore, two patients in the critical group developed AF with ventricular rates ranging from 123 to 160 beats per minute. One

of the patients experienced persistent AF, whereas the other had never had AF before. COVID-19 claimed both of their lives. In COVID-19 patients, the rise in HR is disproportionate to the rise in body temperature [11].

A subsequent 12-lead ECG revealed atrial tachyarrhythmias in 19 COVID-19 patients (19/115, 16.5 percent), all of whom were admitted to the MICU (27.5 percent of MICU patients), including atrial fibrillation in 12 patients, atrial flutter in 6 patients, and atrial tachycardia in 1 patient. Patients with COVID-19 who needed to be admitted to an intensive care unit frequently experienced atrial tachyarrhythmias, which were frequently followed by hemodynamic deterioration [12].

In short, we must pay close attention to tachycardias in COVID-19 patients who are severely ill. SARS-CoV-2 may cause electrophysiological abnormalities in patients with no previous history of heart illness through a variety of pathways, in addition to aggravating existing cardiomyopathy and conduction issues and causing arrhythmia episodes [13].

2.3 Atrioventricular/Intraventricular Conduction Block

According to a recent study, the incidence of cardiac arrhythmias in COVID-19 patients ranged from 17% to 30%. The most common arrhythmia was atrioventricular/ventricular block (11.8 percent), which outnumbered sinus tachycardia (7.5 percent), sinus bradycardia (8 percent), atrial arrhythmias (7 percent), and ventricular arrhythmias (7 percent) (4 percent) A child with COVID-19 infection had the complete cardiac block and significant left ventricular dysfunction. Another case from Iran described transitory total heart block in a patient with COVID-19, with an ECG showing nonspecific intraventricular conduction delay and several premature ventricular complexes in a 21-year-old female patient [14].

2.4 ST-T Changes

ST-T alterations were the most common ECG abnormality in COVID-19 patients, accounting for roughly 41% (38/93), according to our findings. Acute myocardial infarction was diagnosed in five of these patients (AMI). In a recent case study, 13 (72%) of 18 COVID-19 patients with ST-segment elevation in their ECG died in the hospital (acute ST-segment elevation myocardial infarction: n = 4; noncoronary myocardial injury: n = 9). A 61-year-old Hispanic man presented with a Brugada-type pattern ECG in the right precordial leads, followed by a brief episode of atrioventricular nodal reentrant tachycardia two days later (AVNRT). Within 24 hours after the onset of multifocal ventricular tachycardia (VT) and ST-segment elevation, a patient died [15].

2.5 QT Interval Prolongation

In our study, 13 percent (12/93) of COVID-19 patients exhibited a prolonged QT interval, with a mean QT interval of 431 milliseconds (414-454 milliseconds). QT prolongation has been linked to a variety of illnesses in the past (eg, inherited arrhythmia syndromes, myocarditis toxicity, metabolic disorders, certain drugs). Several antimicrobials used as prospective COVID-19 therapeutics have questionable efficacy and may cause electrocardiographic QT prolongation with potential ventricular pro-arrhythmic effects. Chloroquine (CQ), hydroxychloroquine (HCQ), azithromycin, and lopinavir/ritonavir are the drugs in question. Recent evidence suggests that patients with COVID-19 who are given HCQ have considerable QT prolongation [16].

Borba and colleagues, for example, conducted a parallel, double-blind, randomized clinical trial to assess the safety of CQ in various dosages and discovered that prolongation of the QTc interval occurred in 4 of 36 patients (11.1 percent) in the low-dose group (ie, 450 mg twice daily on day 1 and once daily for 4 days) and 7 of 37 patients (18.9%) in the high-dose group (ie, 600 mg CQ twice daily for 10 days). Furthermore, patients who got HCQ and azithromycin at the same time had a higher risk of QTc alterations; 13% of them had serious QTc prolongation, and the combination caused more prolongation than either drug alone. Chorin et colleagues reported QTc prolongation from a baseline average of 435 24 milliseconds to 463 32 milliseconds (P.001) 3.6 1.6 days following HCQ + azithromycin medication. QTc was substantially extended to >500 milliseconds in a subset of those individuals (9/84, 11%), a known ECG indication of high risk of malignant arrhythmia and sudden cardiac death. Patients receiving HCQ+azithromycin had a higher rate of cardiac arrest (15.5%) and abnormal ECG findings (27.1%) than those getting HCQ alone (13.7 percent and 27.3 percent, respectively) than those receiving azithromycin alone (6.2 percent and 16.1 percent, respectively) [16].

Antifungal medications, glucocorticoids, and antiarrhythmic medicines can all cause QT intervals to lengthen. If these drugs are administered, clinicians should keep an eye on the patient for adverse effects, particularly a prolonged QTc interval, which can be detected through continuous ECG monitoring [17].

2.6 Malignant Arrhythmias

COVID-19 individuals are more likely to have comorbidities that put them at risk for significant arrhythmias. Guo and colleagues were the first to report malignant arrhythmias in COVID-19, reporting a 5.9% (11/187) incidence of malignant arrhythmias, including VT/ventricular fibrillation (VF). In addition, Du et al reported a large series of COVID-19-related deaths, finding that cardiac arrest (8.64%) was the most common cause of death in 7 of the 81 patients, followed by acute coronary syndrome (4.94%) and malignant arrhythmias (4.94%) (2.47 percent). Cardiac arrest was more common in individuals getting HCQ + azithromycin in adjusted models compared to those receiving HCQ or azithromycin (adjusted OR, 2.13 [95 percent CI, 1.12-4.05]; E-value = 1.31). SARS-CoV-2, like SARS-CoV, can induce a range of ECG abnormalities. SARS-CoV-2, on the other hand, is more likely to cause atrioventricular/intraventricular conduction block, QT interval prolongation, and malignant ventricular tachyarrhythmias [18].

The ECG data of COVID-19 patients is currently sparse, and there are inconsistencies amongst studies in terms of the reported rates of various arrhythmia types. This is most likely due to the nonhomogeneous characteristics of selected cases across studies, as well as the limited sample size, origin region, and lack of continuous ECG monitoring data. A single ECG examination is insufficient. To further determine the kind of arrhythmia, dynamic ECG monitoring is essential. According to the available data, COVID-19 has a quick clinical course, and ECG abnormalities observed during hospitalization can be utilized as a prediction of disease severity. Patients with pathophysiological alterations similar to fulminant myocarditis or ECG abnormalities such as conduction block, QT interval prolongation, or ventricular arrhythmia have a dismal prognosis. It is therefore recommended that clinicians should perform a comprehensive evaluation through combining cardiac injury biomarkers, ECG dynamic changes, and cardiac imaging, and be alert to life-threatening ventricular arrhythmia storms [19].

3. RESULTS

The incidence of VA is directly proportional to the size of the infarct and is related to the contrast of LVEF. Delayed arrival or failure to properly patent a broken ship poses the greatest risk. In addition, more than one-third of STEMI patients, and most complex STEMI patients with cardiogenic shock, suffer from serious coronary artery disease and appear to be at high risk for VA. This is a valid argument for early vascular resuscitation. Transthoracic echocardiography is commonly used to assess infarct levels and rule out patients based on LVEF. When measuring only LVEF, operators are more mobile with echo than with MRI. Regardless of how LVEF is determined, its use in identifying people at risk for VA and SCD is limited [19].

The multicenter automated defibrillator implantation trial (MADIT) identified an advantage in death from ICD in 5.6% of patients with a post-infarction LVEF of 30% or less for 27 months from the baseline event. Sudden Cardiac Death on Heart Failure Test (SCD-HeFT) identified a 7.3% mortality benefit over 60 months for people with LVEF of 35% or less. In addition to being modest numbers, most patients with heart disease after a myocardial infarction have an LVEF greater than 35%. When examining the long-term risk of AV and SCD, it is important to note that less than 20% of ICD recipients in previous studies received appropriate ICD treatments during their follow-up periods [19].

In addition to the incidence of chronic coronary artery disease, analysis of the Multicenter Non-Sustained Tachycardia Study (MUSTT) study draws attention when calculating LVEF-based risk-only complications. People who experience non-persistent ventricular tachycardia (NSVT), recruitment status, and 30% to 40% of LVEF have a risk of traumatic death or heart failure compared to LVEF, or less than 30%. Major predictors of predictable variability include functional class, history of heart failure, non-NSVT surgical bypass surgery, EF, age, left ventricular dysfunction, progressive ventricular tachycardia, patient enrollment, and atrial fibrillation. Noninvasive screening of scar tissue, ventricular conduction and reabsorption, and independent pressure have been evaluated in risk prediction. Magnetic resonance of the heart is very high in showing implanted tissue and

examining wound load compared to other diagnostic imaging modalities. Increased tissue diversity and enlargement of the peri-infarct or "border area" was found to be associated with a higher risk of death, which can be better diagnosed by MRI. Big data is lacking to determine how ICD treatment can be directed toward primary prevention through MRI without LVEF screening. In a study of 48 patients with coronary artery disease at PES, infarct area and weight were measured by cardiac MRI in patients with monomorphic VT substrate compared to LVEF. This is recognized. Patients were examined more than a month after MI. DETERMINE-ICD is a large randomized clinical trial for the treatment of prophylactic post-MI CDI in patients with 35% LVEF and multiple MRI-diagnosed scans (Fig. 3) [19].

The current data only support the use of PES in patients with myocardial infarction and whose LVEF is 40% or less. The duration of programmed electrical stimulation (PES) is a matter of debate. In MADIT I, it was found that patients with inducible VA and LVEF 35% or less after MI had significant benefits in ICD mortality. Finding similarities in the implantable cardioverter defibrillator (BEST + ICD) in addition to the beta-blocker strategy in the month following MI suggested that initial relief after the event could do the same, but did not really predict benefits. Can After an acute myocardial infarction test (CARISMA), however, 6 weeks after MI, an incredible VT was found to be a strong predictor of future arrhythmic events. Despite the fact that a change in rehabilitation protocol is a factor that could lead to a change in the results of the study, PES is still less sensitive than that shown in more than a quarter of 35% or less of the patients. Has gone There are significant events compared to LVEF and the continuation of a negative study [20].

3.1 Medication

3.1.1 Anti-arrhythmic drugs

There is no strong evidence for antidepressant (ADA) use in the first phase of ischemia and recurrence within the first 48 to 72 hours after MI compared to use in the chronic phase. Despite the discovery of an initial revascularization and the use of beta blockers, 6% of patients still have persistent VA in this first phase. Although the rapid treatment of VA with hemodynamic collapse remains DC cardioversion, VA continues where vascular regeneration is not required and normal electrolytes generally require some form of drug therapy. DAAs have no side effects other than their effects on transmembrane tension and heart rate, all of which can worsen instability [21].

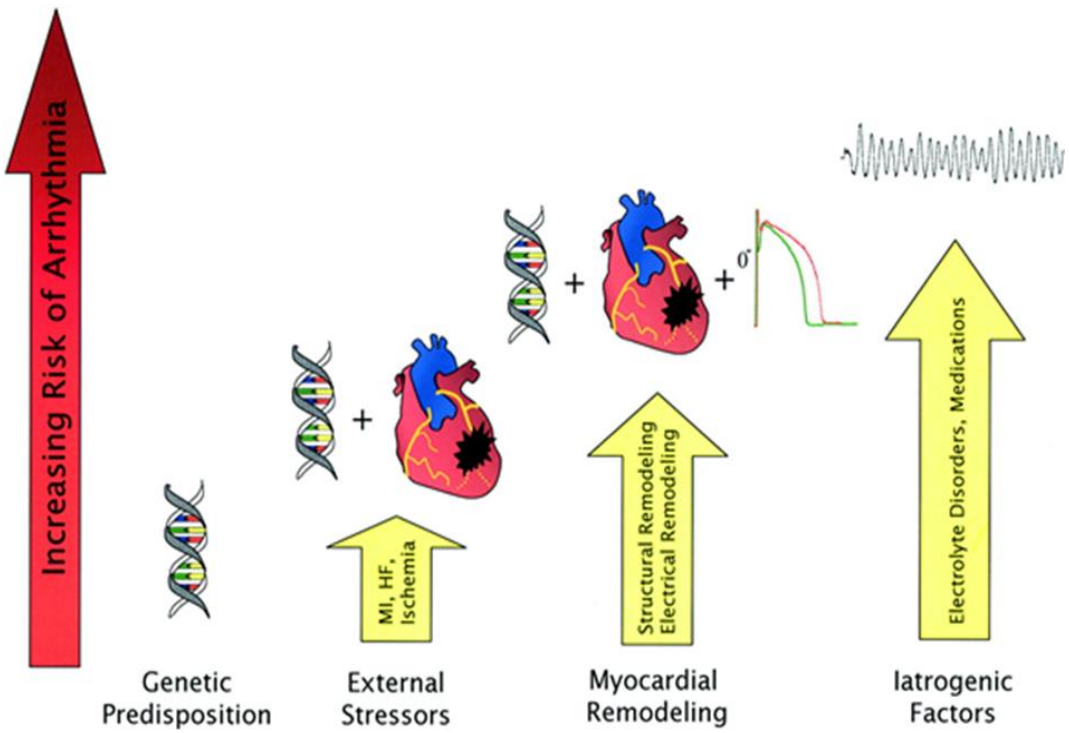

Fig. 3. Factors increasing risk of arrhythmia [19]

Beta-blockers have been used successfully in patients with acute coronary heart disease, reducing major cardiovascular events including SCD. In a meta-analysis conducted by Huang et al., The use of beta-blockers was associated with a reduction in all causes of death in patients with malignant MI undergoing PCI. Benefits are limited to those with reduced EF, low use of other secondary prevention drugs or non-STEMI. The correlation between beta-blocker use and improved survival rate was noticeable only <1-year follow-up period. They concluded that there was a lack of evidence to support the common use of beta-blockers in all AMI patients who received PCI. In carvedilol post-infarct survival control in a left ventricular dysfunction study (CAPRICORN), Carvedilol has been shown to have a significant anti-arrhythmic effect after AMI. It suppressed both atrial and ventricular arrhythmias in these patients [21].

There have been conflicting reports about the Ib class of the drug lidocaine or a significant trend toward a lower risk of death during the first myocardial infection, as well as lower VA and the benefit of avoiding cardiac arrest when used as a precaution. Neutral effect. Death or overeating. The use of prophylactic lidocaine is strongly discouraged, although it is still a potential treatment for developing VA post MI veins that are still ongoing. IC-class drugs such as flecainide and propafenone cause a significant slowdown in driving, which can increase MI VA after MI and should not be used [21].

Amiodarone remains the most widely used DAA after myocardial infarction and is particularly useful in severe structural disease. However, it takes time to reach the level of healing. Its use after out-of-hospital cardiac arrest in patients with refractory VF shock was associated with a survival advantage compared to lidocaine. In patients who survived more than 3 hours after myocardial infarction, the use of amiodarone was associated with increased short-term mortality (30 days) and a longer duration (6 months) compared to lidocaine for its use in SCA condition. No additional survival benefit has been demonstrated with repeated use of amiodarone from beta-blocker therapy after myocardial infarction and its significant side effect profile has been shown to increase long-term mortality. There is no data to support the use of dronedarone in the post-MI period. Some phase III medications, such as dofetilide, increase heart rate and suppress VA, but there are no data to show additional beneficial effects on their use. All phase III drugs prolong the QT interval and cause compromise of polymorphic VT, although the incidence of polymorphic VT is lower with amiodarone [22].

Ranolazine is the new entry into the arena. Metabolic efficacy with ranolazine for minor ischemia in acute coronary syndrome without ST segment elevation (MERLIN) - TIMI tests showed no significant differences in end-stage heart failure, MI, or ischemic recurrence, but significantly reduced the incidence of unsupported compared to placebo VT. More research is needed, especially when compared to current drug therapy. Similarly, Eplerenone 25 mg / day, in addition to conventional therapy, significantly reduced LVEF ≤ 40% and occasional mortality at 30 days after randomization of patients with cardiac symptoms. Failure. There is a 37% reduction in the risk of sickle cell anemia in patients receiving eplerenone. If you are considering drug therapy, you should not forget about deep sedative use. In addition to the use required when an alert patient encounters DC cardioversion, a reduction in responsive drive associated with subsequent MI VA makes it an effective treatment option (Fig. 4) [23].

3.1.2 Overdrive pacing

If the above measures fail to compress the VA in the initial MI period, temporary overdrive pacing can be used. Automatic attention can be captured and suppressed, or an exit block is achieved by reversing the surrounding myocardium. Tachycardia caused by reentry machine can be eliminated by rejection due to change of direction and speed. This measure can be used in antagonistic AV to reduce the need for repeat cardioversion while waiting for drug treatment to work, or before resuscitation or catheter ablation continues (Fig. 5) [24].

3.1.3 Radiofrequency ablation

Withdrawal of the VA catheter in the critical phase is seldom performed. The severe success rate is 70% and is associated with 3% short-term mortality in unstable patients and 18% long-term mortality due to reduced heart failure. The ablation occurs predominantly subendocardially and is located in the border area. Targeted circuits invade various myocardia as well as post-depolarizations and automatic foci emanating from the Purkinje fibers. Activation of the card can be done before conventional PVCs.

Quick assembly can be done against pre-registered PVCs if these are in short supply. The final points include the pressure of the PVC trap and the loss of strength from Purkinje. In cases where PVCs are not available, general guidelines including PES or drug irritation may not be clear and may not be clear during the severe period. In these cases, a voltage-controlled substrate extraction can be carried out. This group of patients is often hemodynamically unstable, and the complexity of the procedure means it is best performed at very high altitudes by skilled electrophysiologists using 3D electroanatomical mapping systems and possibly supportive treatment that includes the ability to mechanically rotate. support if necessary (Fig. 6) [25].

3.1.4 Mechanical circulatory support

VA often occurs in complex cardiogenic MI shock and is associated with high temporal mortality. In patients with selected cardiogenic shock and acute myocardial infarction who undergo thrombolysis or primary PCI, prolonged VT occurs in 17–21% and VF occurs less frequently (24–29%). Revascularization improves survival. The use of inotropes can worsen the VA and reduce the amount required when used with mechanical assistance. In addition to supporting regenerative processes, mechanical support can help maintain adequate cardiac output in the post-MI period. The most common form is the intra-aortic balloon pump (IABP). This counter-pulsation device significantly reduces posterior stress, increases diastolic coronary congestion, and contributes to cardiac output. It cannot support VF while other types of devices can. The use of an impla-assisted device in this patient population was associated with a reduction in tissue hypoxia, hemodynamic stabilization, and improved neurological outcome. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) PCI has been shown to improve survival in patients with cardiogenic shock and VT / VF resistance compared to IABP. These supporting systems can act as a bridge for reconstruction or final replacement of the heart transplant (Fig. 7) [26].

3.1.5 The implantable cardioverter-defibrillator

The current guideline recommends the inclusion of ICD from 40 days after MI in patients with 35% LVEF or less New York Heart Association (NYHA) in class 1, 2 or 3. Defibrillator on Acute Myocardial Infarction Trial (DINAMIT) and Emergency Risk Stratification Improves Survival (IRIS) has shown that there is no survival benefit in inclusion less than 40 days from the event. days) compared with conventional treatment. This was the main prevention study and did not include people who had VA 48 hours after MI. The DINAMIT study included the HRV measurement, with an EF of <35% as study inclusion criteria. Studies have shown a reduction in arrhythmic death, which was largely measured by an increase in non-arrhythmic

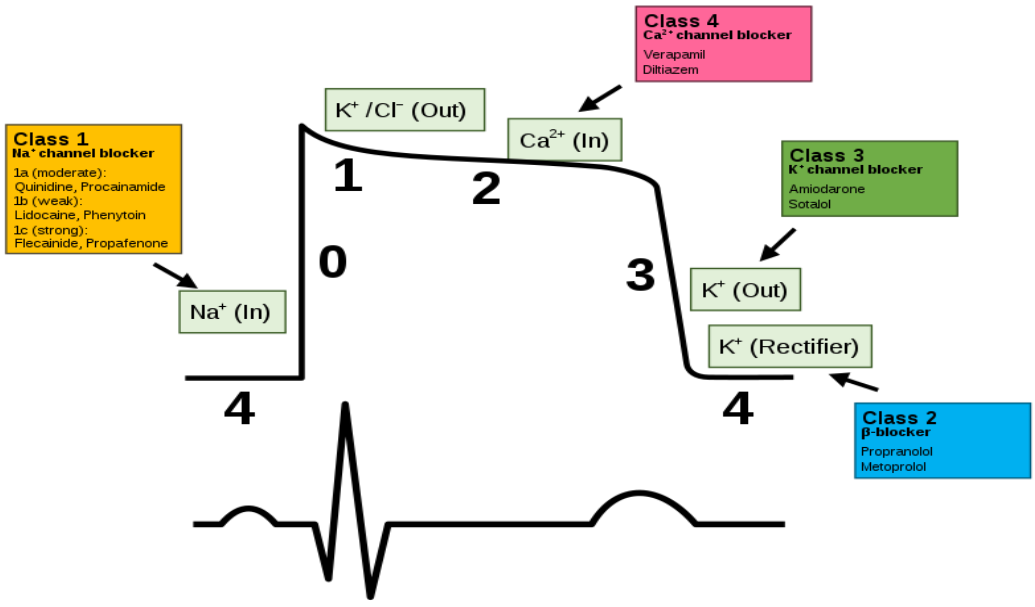

Fig. 4. Anti-arrhythmic drugs [23]

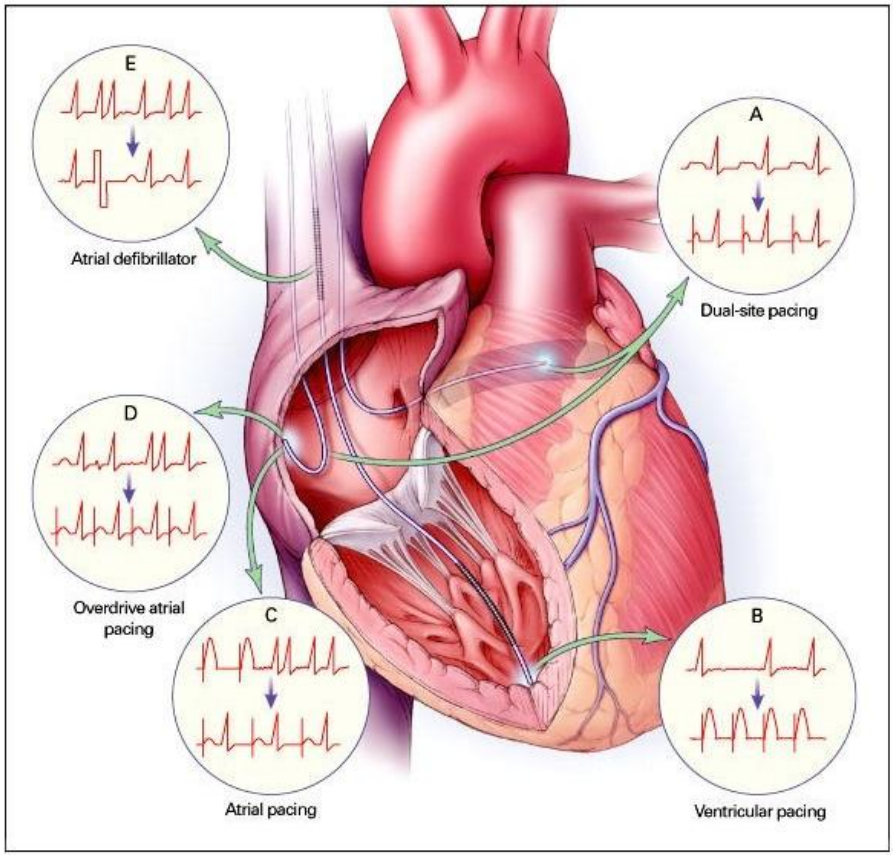

Fig. 5. Overdrive pacing [24]

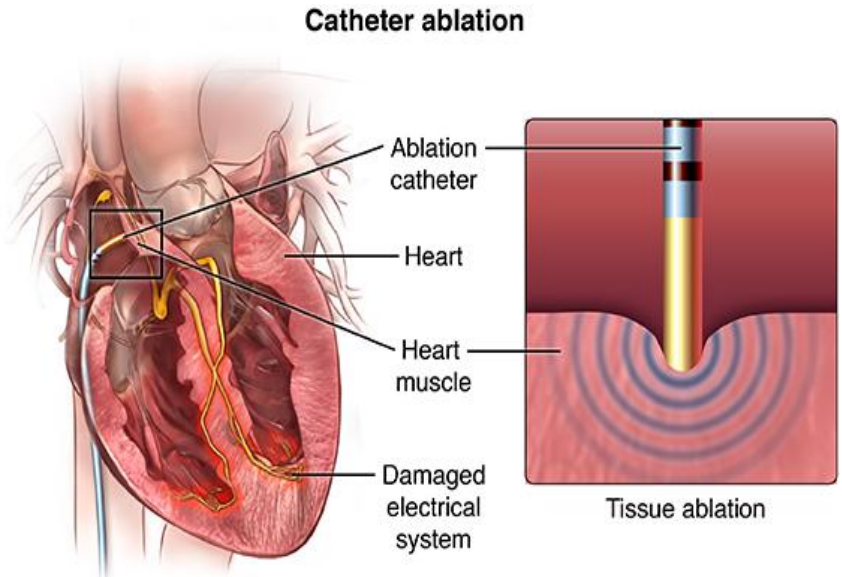

Fig. 6. Radiofrequency ablation [25]

cardiac death in the ICD arm compared with the control group, but there was no reduction in the number of deaths. Similarly, the IRIS trial enrolled patients, 5-31 days after MI. The insertion procedure includes reduced LVEF (≤40%) and a heart rate of 90 or more. This was also a major prevention study and failed to show any benefit of prophylactic ICD in this group of patients, although the rate of arhythmic mortality was lower in patients with ICD [27].

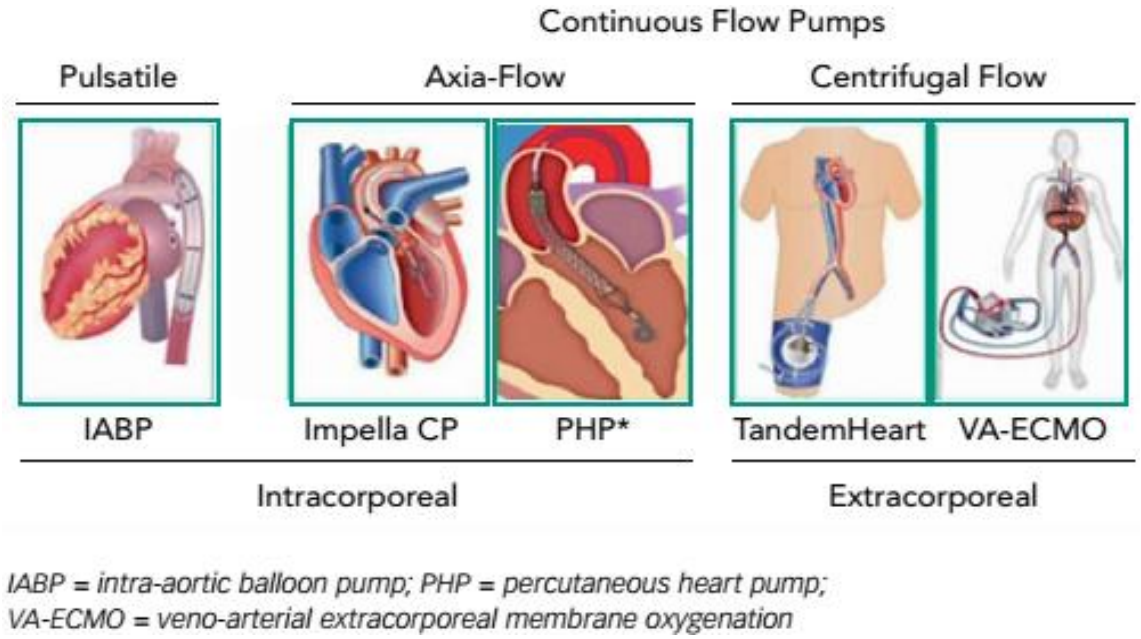

Fig. 7. Mechanical circulatory supports [26]

Neither study showed significant ICD benefits in this patient population, but these patients needed further training because they did not include patients who developed VA after 48 hours of myocardial infarction. .. Risk classification tools are needed to diagnose these individuals who may be at high risk for VA. Wearable defibrillators may play a role in preventing SCD in the absence of powerful risk mitigation tools, especially in patients with impaired left ventricular function, but such strategies are available in other randomized trials. Must be evaluated at. The risk of VA and subsequent ICD treatment appears to decrease over time after infarction, and other factors play a role because most patients with primary ICD prophylaxis (and without additional ischemia) do not have progressive VA. There is a possibility to fulfill. Left ventricular function and stable scarring [27].

ICD prevents sudden death of VA, but not VA itself. Of course, this does not prevent death from continuous pump failure and can actually change the path of death. Arrhythmias treated with ATP were not, but both positive and negative shocks were associated with increased mortality. The psychological effects of CDI treatment should not be underestimated. Highly aggressive ATP, advanced detection and extraction algorithms, initial conditions, stability and morphological identification help reduce the frequency of inappropriate electric shocks. Long-term ischemia or heart attack can cause electric shock in patients who already have an ICD. In such cases, people who do not have an ICD may need to be treated, but move the device to address the increased frequency of arrhythmias and altered aspects, and temporarily take the medication the device is taking. You can also consider disabling it. To prevent excess or negative ATP and / or shock (Fig. 8) [27].

4. DISCUSSION

Chronic ventricular arrhythmia accounts for up to 20% of acute myocardial infarction (MI) presentations and is associated with severe prognosis. A significant one-year increase in hospital mortality and patients with acute myocardial infarction. Fibrillation (VF) is the period of peri-infarction, and while progress toward rapid vascular regeneration is still ongoing, ventricular arrhythmia still has a three-fold risk of 90-day mortality. Myocardial ischemia is the result of the interaction between the substrate, the stimulator, and the changing factors. These factors vary depending on the duration of ischemia, so we produce different electrophysiological mechanisms of arrhythmias. For the purposes of this discussion, we consider ventricular dysfunction (0-4 hours from baseline), critical phase (4 hours -72 hours), and chronic myocardial ischemia. (72 h +) [27].

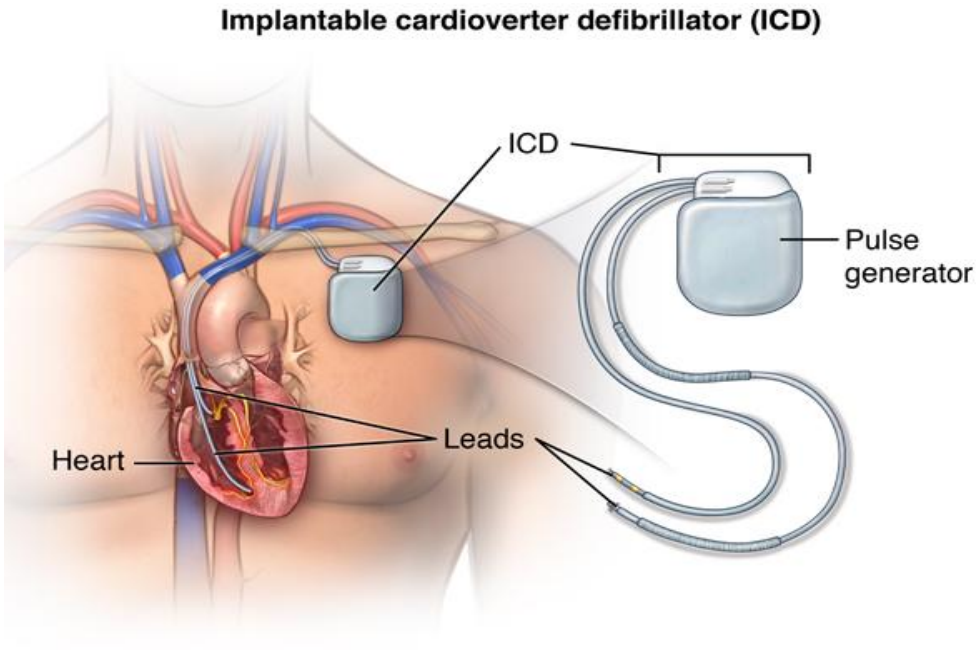

Fig. 8. The implantable cardioverter-defibrillator [27]

5. CONCLUSION

A greater understanding of cellular processes and electrophysiologic arrhythmia is needed in the setting of acute ischemia. It is clear that risk-based classification based on LVEF and PES is not sufficient and may require extensive joint investigation. Current treatments are limited to drugs with significant side effects and catheter removal techniques. The concept of genetic predisposition and therapeutic approaches to self-mutilation are interesting and require further investigation. Timely and complete rehabilitation appears to be a strategy to prevent VA death, but it is not achieved in all patients with a posterior infarction.

Perhaps the future of some lies in stem cell therapy that can repair lost or damaged myocardium. Stem cell therapy is used to treat heart failure. Early experiments in animal models (mice) and later in humans, showed that stem cell treatment increased the incidence of ventricular arrhythmias (ventricular ectopia and unstable VA), potentially leading to a lack of effective integration into the integrated network. because. Although additional studies using stem cell therapy in animal models and then in humans have reported reduced incidence of cardiac arrhythmias or no change in recurrent ventricular arrhythmias, further research Required. Ventricular arrhythmia in patients with acute coronary syndrome may contribute to rapid and long-term death in these patients. Appropriate risk assessments are needed to identify patients with an increased risk of VA and sudden cardiac death, and research is needed in this area.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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