Review

Right ventricular myocardial infarction: pathophysiology, clinical implications and management

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Right ventricular myocardial infarction (RVMI) and right ventricular (RV) failure are complications from an acute occlusion of a dominant right coronary artery (RCA) or left anterior descending (LAD) artery. Although some patients have good long-term RV recovery, RVMI is associated with high rates of in-hospital morbidity and mortality driven by hemodynamic compromise, cardiogenic shock, and electrical complications. As such, it is important to identify specific clinical signs and symptoms, initiate resuscitation and commence reperfusion therapy with fibrinolytic therapy or percutaneous coronary intervention. This review will discuss RVMI pathophysiology, describe the current diagnostic measures, highlight current therapies, and explore future management options.

Keywords

Acute coronary syndrome; Right ventricular myocardial infarction; Shock

1. Introduction

For many years, post-myocardial infarction clinical research has focused on the left ventricle (LV), overshadowing conditions involving the right ventricle (RV). Right ventricular myocardial infarction (RVMI) was first described by Cohn *et al.* [1] over 45 years ago, however the importance of right ventricular function was not initially appreciated. RVMI typically occurs in conjunction with left ventricular myocardial infarction (LVMI) from either an acute occlusion of a dominant right coronary artery (RCA) or left anterior descending (LAD) artery although the resultant infarct size in the latter cases is typically small [2–4]. Less commonly, isolated RVMI can occur from occluding the RV marginal artery such as occurs with an iatrogenic occlusion during coronary intervention or from an acute occlusion of a non-dominant RCA. Overall, RVMI has a high rate of in-hospital mortality driven by hemodynamic compromise, cardiogenic shock, and electrical complications. In this review, we describe the pathophysiology, the diagnostic parameters, contemporary therapies, and long-term prognosis of RVMI.

2. Right ventricular anatomy, function and arterial blood supply

The RV is a thinned wall cardiac chamber with a complex geometrical shape. It has a three-part structure: (1) the inlet; (2) the trabeculated apex; and (3) the smooth myocardial outflow tract or conus [5]. Anatomically, the RV myocardial wall is approximately 3–4 mm thick and weighs about 15% of the LV. Although the RV has a larger volume, the RV and LV have the same stroke volume. Under normal hemodynamics, the RV has a low-impedance, highly distensible pulmonary vascular system with low vascular resistance allowing the RV to have only about 25% of the stroke work of the LV [6, 7].

The RV receives arterial blood from both the RCA and the LAD. Typically, the RCA arises from the right coronary sinus and travels along the right atrio-ventricular (AV) groove. The conal or infundibular artery is the first branch after the ostium and runs anteriorly to provide blood to the muscular right ventricular outflow tract. In 30% of people the conal artery has a separate ostium which explains why some patients with a proximal RCA occlusion maintain function of the infundibular region [6]. The right ventricular marginal branch, there can up to two branches supplies blood to the RV lateral wall. Additionally, the LAD artery supplies branches that perfuse the antero-septal region and anterior right ventricular free wall. In right dominant circulation, the RCA also supplies the posterior descending and posterolateral branches while in left dominant circulation, the non-dominant RCA supplies blood to the RV lateral wall and right atrium (RA) only. About 80–85% of people have a dominant RCA anatomy, 5–10% have a dominant left circumflex (LCx) and 5–10% are co-dominant (CD) or have a "balanced circulation" [8] (Fig. 1). Importantly, the proximal posterolateral branch supplies the AV node.

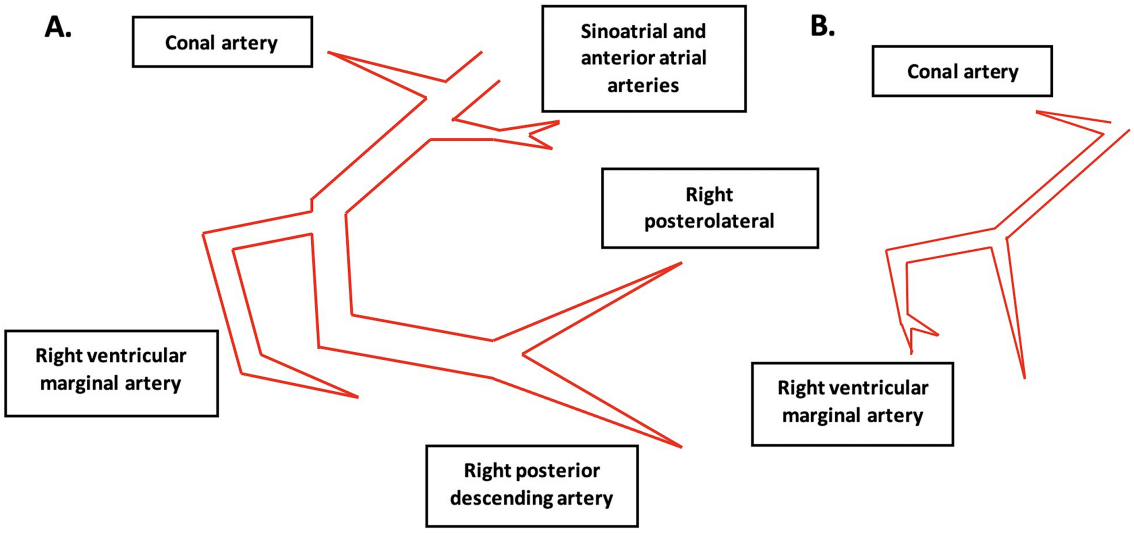

Fig. 1. Right coronary artery anatomy. (A) Dominant right coronary artery anatomy. (B) Non-dominant right coronary artery anatomy or left dominant coronary artery anatomy.

Table 1. Causes of acute right ventricular myocardial infarction.

Acute thrombotic occlusion
Catheter induced dissection
Stent-jailing of the right ventricular marginal branches during percutaneous coronary intervention
Coronary artery thrombo-emboli due to atrial fibrillation
Spontaneous coronary artery dissection
Prolonged occlusion of the right ventricular marginal branches during retrograde intervention
Aortic dissection with retrograde involvement of the right coronary artery
Dislodgment or mal positioned cardiac device (MitraClip or transcatheter aortic valve)

3. Pathophysiology of right ventricular myocardial infarction

The RV has unique characteristics that make it less susceptible to myocardial infarction. First, the RV myocardium is thin and has a lower oxygen requirement than the LV [9]. Second, the RV is perfused during both systole and diastole with an increased ability to extract oxygen during hemodynamic stress [10]. Ultimately, this may provide greater oxygen transportation that may potentially limit the degree of complications. Despite these structural features and an ability to function at low oxygen pressures, the RV is still susceptible to ischemia and myocardial infarction. In fact, studies have demonstrated a direct correlation between the location of RCA occlusions and the extent of right ventricular infarction. Specifically, occlusions proximal to the right ventricular marginal artery have been shown to result in larger infarction sizes, a higher rate of cardiogenic shock and cardiac death [11, 12] (Fig. 2).

Causes of acute RVMI include acute thrombotic occlusion, catheter induced dissection, 'stent-jailing' of the RV marginal branch during percutaneous coronary intervention (PCI), thrombo-embolism from atrial fibrillation, spontaneous dissection, dislodgement/mal-positioning of a newly inserted cardiac device (MitraClip embolization or transcatheter aortic valve implantation induced coronary occlusion) or prolonged occlusion of the marginal branches during retrograde PCI of a LAD chronic total occlusion (CTO) [13, 14] (Table 1). In one case report, mitral regurgitation was treated with three MitraClips but six weeks after the procedure, the patient presented with chest pain and inferior ST-elevation caused by embolization of one of the MitraClips to the right coronary cusp obstructing the ostium of the RCA [15]. Prompt recognition with a 12-lead electrocardiogram (ECG) and hemodynamic data is pivotal to prevent idiopathic ischemia, infarction, and long-term complications. RVMI may also occur from an occlusion of the LAD artery with impaired perfusion of the RV anterior wall and anteroseptal region [2–4, 16].

4. Diagnosis

4.1 Clinical presentation and symptoms

Although the clinical sequalae can be variable and the diagnosis can be challenging, early recognition of RVMI is critical in preventing morbidity and mortality [17]. Table 2 outlines some diagnostic criteria of RVMI based on clinical signs and investigations.

Clinically RVMI is defined by a triad of physical signs: hypotension, jugular venous congestion, and clear lungs. An elevated jugular venous pressure (JVP) alone is highly sensi-

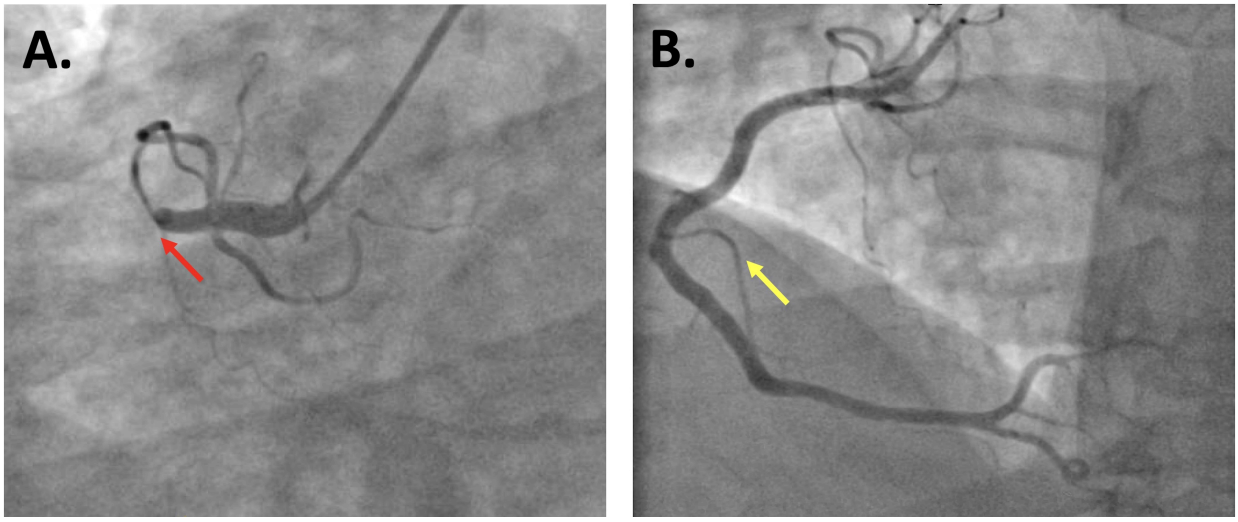

Fig. 2. Coronary angiogram images in the left anterior oblique view of a 63-year-old male who presented with chest pain and an inferior ST-elevation myocardial infarction. (A) Image of occluded right coronary artery, proximal to the right ventricular marginal branch (red arrow). (B) Image of dominant right coronary artery post percutaneous intervention with a drug eluting stent confirming patency of right ventricular marginal branch (yellow arrow).

Table 2. Diagnostic features of right ventricular myocardial infarction.

Clinical features	Clear lung fields plus Hypotension plus Distended neck veins In addition, there may be: New tricuspid regurgitation Right-sided third heart sound
Electrocardiogram	ST-segment elevation in the right-sided leads (V4R-V6R) ST-segment depression in lead I + aVL; I + aVL >0.2 mV ST-segment elevation in lead III > II ST-segment elevation in V1 and ST depression in V2 Atrio-ventricular block and bradycardia
Hemodynamic findings with pulmonary artery catheterization	Right atrial pressure (RAP) ≥10 mmHg RAP: PCWP ≥0.8 PAPi <0.9
Echocardiography	TAPSE ≤16 TDI-MPI ≥10.7 S' ≤12.3
Cardiac magnetic resonance	Myocardial Oedema Left ventricular +/- right ventricular LGE

RAP, right atrial pressure; PCWP, pulmonary capillary wedge pressure; PAPi, pulmonary artery pulsatility index; TAPSE, tricuspid annular plane systolic excursion; TDI-MPI, tissue Doppler imaging-derived myocardial performance index; LGE, late gadolinium enhancement.

tive but not specific for a hemodynamically significant RVMI (88% and 69%). As a consequence of RV ischemia and myocardial infarction, the chamber dilates leading to tricuspid regurgitation resulting in an increase in inspiratory central venous pressure and pulsus paradoxus [6]. Together, these three factors are highly predictive of significant RVMI (sensitivity = 88%; specificity = 100%) [18]. In the acute setting, it is important to recognize that the findings of RVMI may be masked by global left ventricular systolic dysfunction associated with hypotension and pulmonary congestion. Additional investigations, described in the subsequent sections, may aid in this diagnosis.

4.2 Electrocardiography

A 12-lead ECG is a mandatory investigation in any case of suspected myocardial infarction. There are several diagnostic features suggestive of RVMI [19, 20]. First, ST-segment elevation ≥1 mm in the right precordial leads (V4R to V6R) correlates with a proximal RCA occlusion and strongly predicts in-hospital mortality (sensitivity of 100%, specificity of 87% and a positive predictive value of 92%) [20]. Second, ST-

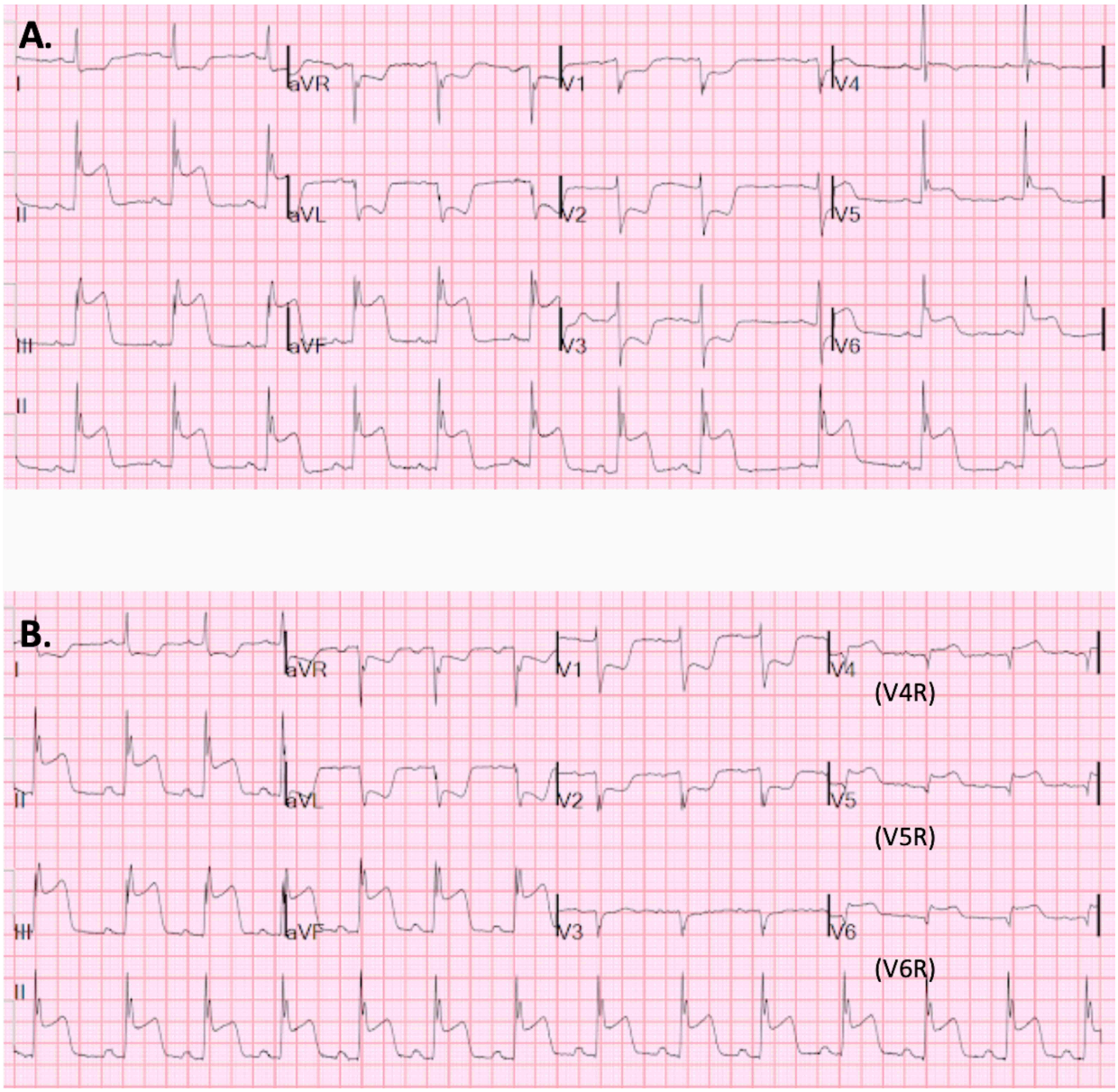

Fig. 3. 12-lead electrocardiograms from a 45-year male with chest pain. (A) Standard 12-lead electrocardiogram shows ST-segment elevation in leads II, III and aVF and ST-segment depression in leads I, aVL, aVR, V1, V2 and V3. (B) 12-lead electrocardiogram with right-sided precordial leads (V4R to V6R) demonstrating ST-segment elevation consistent with a right ventricular myocardial infarction.

segment depression in lead I and aVL (I + aVL >0.2 mV) was found to have a 94% sensitivity and 90% specificity for RVMI [21]. Finally, greater ST-segment elevation in lead III than lead II is linked to a high in-hospital mortality but has a low specificity and often found in patients without RVMI [22] (Fig. 3). Three high risk groups have been identified based on ECG criteria: those with ST-segment depression in the precordial leads, ST-segment elevation in the right precordial leads or third-degree AV block [23]. Although lead V4R is not routinely performed, it should be considered in all patients with inferior ST-segment elevation as it may improve the diagnosis of RVMI.

4.3 Chest radiograph

Although routinely performed in the acute setting, a chest x-ray has not been found to be helpful in the diagnosis of RVMI. In fact, the radiographic findings of RA and RV enlargement have poor diagnostic accuracy in patients with RVMI [24].

4.4 Echocardiography

Trans-thoracic echocardiogram (TTE) plays an important role in the diagnosis of RVMI due to its wide-spread availability and diagnostic utility. In studies with autopsy or surgery confirmed RVMI, five TTE features were found to be clinically significant: RV dilation, RV free wall impairment, diastolic reversed septal curvature, systolic paradoxical sep-

tal motion and decreased tricuspid annular plane systolic excursion (TAPSE) which is a correlate of RV ejection fraction and an excellent measure of RV systolic function [25, 26]. In the emergency setting however, imaging may be limited and some of these parameters may not be attainable. It should be noted that the specificity of these features may be reduced by pre-existing pulmonary disease and pulmonary artery systolic hypertension, particularly with systolic pressures >45 mmHg. In one study, three features were found to be useful in predicting proximal RCA stenosis: TAPSE, myocardial performance index by tissue Doppler imaging (MPI-TDI) and velocity of the tricuspid annular systolic motion (RV S'). TAPSE ≤16 mm, MPI-TDI ≥10.7 and S' ≤12.3 cm/second were all associated with sensitivities >90% and specificities >93% [27]. In a study of 53 patients with inferior MI, right ventricular akinesia or hypokinesia was a highly sensitive marker for hemodynamically significant RVMI. Unfortunately, this feature was not specific as wall motion abnormalities were detected even in patients who did not develop hemodynamic compromise [6]. Finally, right atrial infarction and intra-atrial septal bowing were predictive of cardiogenic shock, significant bradycardia, and a higher mortality rate [28].

4.5 Invasive hemodynamic monitoring with pulmonary artery catheterization

Invasive measurements with pulmonary artery catheterization (PAC) can provide important information about the prognosis of RVMI. In the acute setting, a disproportionate elevation of right-sided filling pressures compared to pulmonary artery and left-sided filling pressures represents the hallmark of RVMI. RVMI is defined by RA pressure greater than 10 mmHg and RA pressure to pulmonary capillary wedge pressure greater than 0.8 [6, 29]. RA pressure greater than 10 mmHg and within 1–5 mmHg of the pulmonary artery wedge pressure has a sensitivity of 73% and specificity of 100% in diagnosing significant RVMI [29]. More recently, pulmonary artery pulsatility index (PAPi) defined as the ratio of pulmonary artery (PA) pulse pressure (PA systolic – PA diastolic) to RA pressure has emerged as a predictor of right ventricular failure in patients with RVMI. In one study of patients with angiographically confirmed proximal RCA occlusion, PAPi ≤0.9 was found to be a simple and accurate hemodynamic measurement that predicts the need for percutaneous right ventricular support and in-hospital mortality [30]. In another study, PAPi was associated with clinical, echocardiographic, and hemodynamic signs of right ventricular failure [31]. Despite the potential benefit, data supporting the use of PAC in patients with acute right ventricular myocardial infarction and cardiogenic shock (RVMICS) is variable. In a large retrospective study of 364,001 patients with acute myocardial infarction and cardiogenic shock, PAC was used in only 8.1% of cases with a 75% decrease during the study period (13.9% to 5.4%) [32]. In addition, the PAC cohort had a higher in-hospital mortality (adjusted odds ratio 1.07, 95% confidence interval 1.04–1.10), longer length of stay, higher hospitalization costs and fewer discharges home. In a randomized study of 433 patients with heart failure, there was no difference in mortality, or the length of hospital stay between those who had PAC and those who did not [33]. Most recently, the authors of the National Cardiogenic Shock Initiative (NCSI) presented the results of the study involving 406 patients with RVMICS and emphasized the need for rapid identification of cardiogenic shock (door to support time of less than 90 minutes), early placement of mechanical support, invasive hemodynamic monitoring, and revascularization [34, 35]. In the study, 71% of patients survived to discharge and 68% were alive at 30 days. Although, the results were not isolated to RVMI, it does highlight the need for a standardized protocol and expedited support.

4.6 Cardiac magnetic resonance imaging

Although not typically performed in the acute setting, cardiac magnetic resonance (CMR) imaging has been shown to be highly accurate in the diagnosis of RVMI. As TTE and nuclear imaging techniques have difficulty assessing the complex RV anatomy, CMR has increasingly been used to evaluate patients with RVMI. It has been reported that a significant discrepancy exists between the frequency of RVMI detected by ECG, TTE or hemodynamic monitoring and RVMI diagnosed at autopsy [3]. On the other hand, CMR is considered the gold standard for right ventricular evaluation and volumetric assessment [36]. CMR provides imaging with very high spatial resolution, obviating the need for any geometric assumptions, has larger fields of view with unrestricted imaging planes and uses contiguous slices to assess ventricular volumes. In addition, late gadolinium enhancement (LGE) has been shown to be more specific for the detection of RVMI than physical examination, ECG and TTE [37]. Importantly, LGE is safe in patients with acute coronary syndrome (ACS) and has very good inter-observer consistency. Two observational studies comparing identification of RVMI in acute MI found CMR more frequently detected right ventricular involvement than ECG or TTE [38, 39]. In a study by Masci *et al.* [40], the authors concluded that in patients with RV ischemia, patients with persistent RV LGE had lower right ventricular function at follow-up. In patients with a dominant RCA ST-elevation myocardial infarction (STEMI), there may be LGE in the RV free wall or inferior wall and LV inferior wall (Fig. 4). Although CMR can be extremely useful, there is limited availability in some areas.

4.7 Post-mortem

Myocardial infarction is the most common cause of sudden cardiac death in patients over the age of 35 [41]. Post-mortem assessment with conventional autopsy can identify causes of sudden death due to coronary artery disease with a high degree of accuracy by finding (sub-)total coronary artery thrombotic occlusion, extensive three vessel atheroma or coronary artery disease and a large myocardial scar. In fact, at autopsy, the presence of a totally occlusive thrombus can be

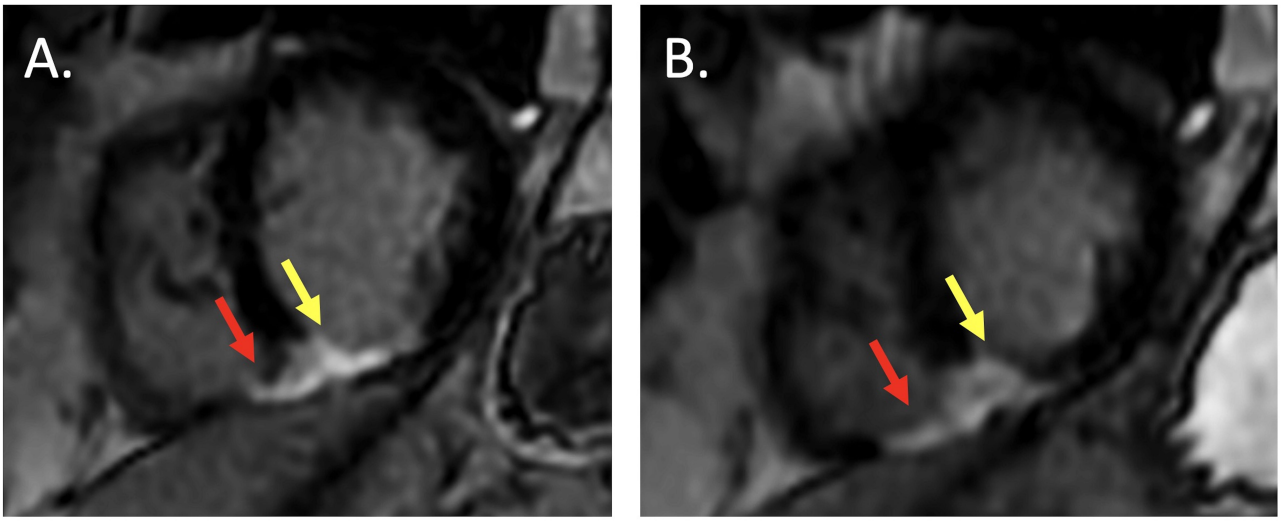

Fig. 4. Short-axis late gadolinium enhancement images of a 51-year-old male with an inferior ST-elevation myocardial infarction and occluded dominant right coronary artery. (A,B) Late gadolinium enhancement (LGE) in the infero-septal wall of the right ventricle (red arrows) and infero-septal wall of the left ventricle (yellow arrows).

observed in approximately 50 to 70% of these cases [42, 43]. Histologically, the early stages of myocardial infarction are defined by mitochondrial swelling and sarcolemmal disruptions followed by interstitial oedema, neutrophils infiltration, coagulative necrosis, and hyper-eosinophilia [44, 45]. This early stage is followed by infiltration of other mononuclear cells, phagocytosis of necrotic myocytes by macrophages and the onset of a fibrovascular response. After the first week, there is capillary sprouting and ingrowth of fibroblasts with deposition of collagen fibers, plasma cells and macrophages. Beyond several weeks, granulation tissue is replaced by dense collagen leading to fibrotic scars.

More recently, post-mortem CMR has been shown to be an accurate non-invasive alternative to conventional autopsy [46]. During the acute phase (day 1 to 7), the necrotic core is hypointense while the marginal regions are hyperintense on T2-weighted imaging [47]. In one study, the authors found that areas of autopsy proven acute myocardial infarction had significantly higher T2-weighted signal intensity than normal myocardium of patients who died from other non-cardiovascular causes [48]. In the later phases (>1 week), the necrotic core becomes hyperintense while the marginal regions become isointense [47]. In situations where conventional autopsy cannot be performed, these CMR changes can provide important information and help identify myocardial infarction as the cause of sudden cardiac death.

5. Complications

5.1 Cardiogenic shock

The development of cardiogenic shock is dependent on the degree of free wall dysfunction, the presence of concomitant RA ischemia and the presence of left ventricular impairment. In RVMI, an acute occlusion proximal to RV marginal arteries can impair free wall perfusion leading to right ventricular dysfunction and shock [49]. Although there is no universal definition for right ventricular shock, two features are common in every contemporary trial and clinical guideline: (1) systolic blood pressure <90 mmHg after appropriate resuscitation; (2) clinical and laboratory evidence of end-organ damage (i.e., urine output <0.5 mL/kg or serum lactate >2 mmol/L) [50]. In addition, isolated right ventricular shock can be characterized by cool and clammy extremities, elevated JVP and clear lungs [51]. During RVMI, the RV chamber becomes stiff leading to an increase in diastolic pressure and a reduction in blood flow from the RA. Subsequently, the RV delivers less blood to the LV resulting in a reduction in cardiac output even in the presence of normal LV function. Finally, the dilated RV shifts the inter-ventricular septum towards the LV further impairing left ventricular filling and cardiac output [52, 53]. Although there is limited data on right ventricular shock, it is common and associated with poor outcomes. In the SHOCK (Should we emergently revascularize Occluded coronaries for Cardiogenic shock) registry, 933 patients in cardiogenic shock were evaluated: 49 (5.3%) due to predominant RV failure and 884 due to LV failure [54]. In this study, the authors found that patients with predominantly right ventricular shock were younger and had a lower prevalence of previous myocardial infarction or multi-vessel disease compared to patients with left ventricular shock. Overall, the in-hospital mortality was 53.1% for the right ventricular group.

5.2 Arrhythmias

Bradyarrhythmia and heart block are common consequences of RVMI. In fact, patients with proximal occlusions were more likely to develop bradyarrhythmia than those with distal occlusions [11]. In patients with an inferior MI, those that develop bradycardia with AV block have a significantly

higher mortality rate (22% vs. 9%) compared to those without conduction abnormalities [55, 56]. In addition, those that develop AV block early have a higher mortality then those with late development (23% vs. 7%). Two potential mechanisms have been postulated to explain AV block in these patients; occlusion of the AV branch can result in ischemia, dysfunction, and necrosis of the AV node and the Bezold-Jarisch phenomenon. Although popular, there is evidence that contradicts the idea of AV node necrosis: first, the rate of oxygen consumption in conducting tissue is 1/5 that of contractile tissue making these cells less susceptible to necrosis; second, conducting tissue has more glycogen stores and therefore are less dependent on oxidative metabolism; finally, the AV node has a rich collateral blood supply and less susceptible to direct ischemia from coronary artery disease. The Bezold-Jarisch phenomenon is characterized by stimulation of afferent nerves adjacent to the AV node resulting in an increase in vagal tone and an outpouring of parasympathetic stimulation via the vagus nerve where bradycardia occurs with or without AV block [20, 53].

Atrial arrhythmias are also common with RVMI and likely result from atrial ischemia and raised atrial pressure. The development of these arrhythmias may have significant hemodynamic consequences due to the loss of AV synchrony and its effect on both ventricular systolic and diastolic function [57]. Additionally, ventricular tachycardia (VT) and fibrillation (VF) can complicate up to 1/3 of acute RVMI cases. Of note, patients who develop ventricular arrhythmias have been shown to have larger infarct sizes, worse right ventricular function and are more likely to experience complications [58]. Recently, studies have found a genetic link between the development of VT or VF and acute myocardial infarction. In particular, mutations of the sodium channels KCNH2 and SCN5A have been linked to the development of long-QT segments and ventricular arrhythmias during acute ischemia and myocardial infarction [59, 60].

5.3 Mechanical complications

Although not common, patients with RVMI may experience mechanical complications. First, RA ischemia can lead to dilation and elevated diastolic pressure causing a patent foramen ovale to stretch open. This could cause a right-to-left shunt and systemic hypoxemia [61]. Second, severe tricuspid regurgitation (TR) may result from primary papillary muscle ischemic dysfunction, rupture or may occur secondary to right ventricular and tricuspid valve annular dilatation. TR can further impair forward right ventricular output, exacerbate right ventricular volume overload, and worsen systemic venous congestion [62]. Finally, ventricular septal or RV free wall rupture are disastrous complications that can rapidly lead to haemodynamic compromise and shock [63].

6. Management

The treatment of acute RVMI with or without shock should include hemodynamic support and urgent revascularization. Specifically, therapy should be directed at optimizing right ventricular preload, after-load, heart rate and AV synchrony. In patients that are hypotensive without pulmonary congestion, it has been suggested that right ventricular preload can be optimized with carefully monitored bolus challenges (200 to 300 mLs) of intravenous fluids. Unfortunately, fluid boluses have not been shown to improve the likelihood of preventing cardiogenic shock [64]. Importantly, nitrates, diuretics and opioid drugs should be avoided in these patients as these medications can reduce right ventricular preload. In addition, an increase in vagal tone as occurs during the insertion of a bladder catheter can acutely decrease preload and lead to cardiogenic shock. For those with ongoing hypotension who do not respond to the fluid challenge, pharmacological therapy with inotropic agents may be warranted. Although no randomized studies have examined the effects of inotropic agents in these cases, there is some evidence that dobutamine can improve cardiac index, right ventricular stroke volume and left ventricular function in patients with RVMI [5].

In conjunction with a reduced preload, right ventricular output may further be compromised by an elevated afterload. In patients with right ventricular dysfunction, right ventricular afterload reducing agents have not been shown to be beneficial and should not be used. However, in patients with concomitant left ventricular systolic impairment, an intra-aortic balloon pump (IABP) therapy may help improve cardiogenic shock by unloading the LV [65]. In cases with severe right ventricular cardiogenic shock, right ventricular assist devices (RVAD) such as the TandemHeart® percutaneous right ventricular assist device (RVAD) support system (TandemLife, Pittsburgh, PA, USA), the Impella RP® device (Abiomed Inc., Danvers, MA, USA) or venoarteial extracorporeal membrane oxygenation (VA-ECMO) may provide hemodynamic support in the acute setting [66–68]. Although there are no specific guidelines to help clinicians select the most appropriate RV circulatory assist device, the main indication is cardiogenic shock unresponsive to conventional resuscitation such as patients with refractory hypotension or poor cardiac index. Importantly, before selecting a device, operators must carefully assess LV function, assess the need for oxygenation and define the "exit strategy" of treatment such as a bridge to recovery, a bridge to left, right or bi-ventricular assist device (LVAD, RVAD or BiVAD), or heart transplantation. In patients with chronic RV failure, a heart team-based approach should be used to define whether mechanical devices should be used. In particular, the TandemHeart (RVAD) has been used for multiple conditions including in patients with acute RV MI and RV failure [69, 70]. This device uses an extracorporeal centrifugal-flow pump and two venous cannulas to deliver blood from the right atrium to the main pulmonary artery. Observations from the THRIVE (TandemHeart in Right Ventricular Failure) registry showed that using centrifugal flow in patients with RV failure was associated with improved haemodynamics. More recently, the Impella RP (Abiomed Inc., Danvers,

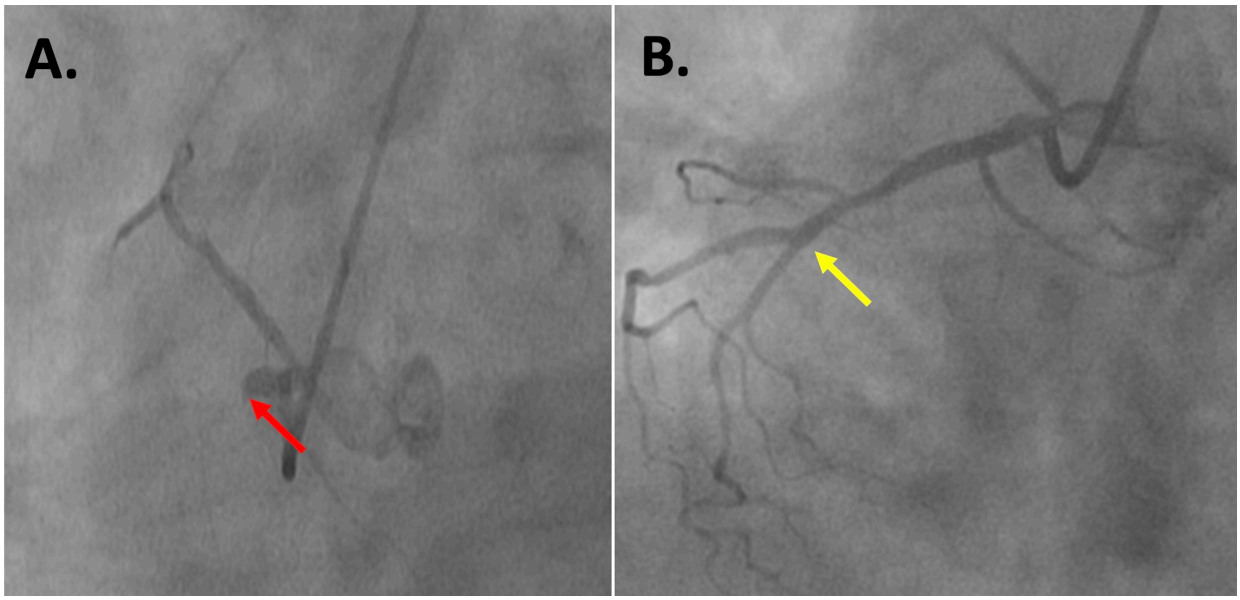

Fig. 5. Coronary angiogram images in the left anterior oblique view of a 75-year-old male who presented with chest pain and myocardial infarction. (A) Image of occluded non-dominant right coronary artery, proximal to the right ventricular marginal branch (red arrow). (B) Image of right coronary artery post percutaneous intervention with balloon angioplasty demonstrating patency of right ventricular marginal branch (yellow arrow).

MA, USA) has been described in multiple settings including RV MI. Even in cases complicated by unsuccessful revascularization and refractory right ventricular failure, the use of Impella RP device resulted in hemodynamic benefit, reversal of shock and favorable survival at 30-days [71]. More recently, the RECOVER RIGHT trial used a novel percutaneous microaxial-flow pump in two groups of patients with right ventricular failure; patients post LVAD placement and patients post cardiotomy, post-transplant or acute MI [68]. Overall, 73.3% of patients survived to discharge but 36% had significant postoperative bleeding. Finally, VA-ECMO is frequently implanted during cardiogenic shock to improve systemic oxygenation. Unfortunately, clinical data supporting the use of VA-ECMO in patients with RV failure is limited to case reports and small case series and the effect on right sided hemodynamics remains undefined [72, 73]. Although data demonstrating the efficacy of these devices is limited, there may be some benefit in patients with right ventricular failure. Further studies clarifying the optimal role in RVMI patients is required.

In situations of bradycardia, the use of atropine, temporary ventricular pacing or external pacing may be required. In fact, investigators have shown that AV sequential pacing in patients with complete AV block associated from RVMI lead to a significant improvement in cardiac output and recovery from shock [74]. Even in cases where the above strategies are successful, the most important therapy is emergency reperfusion using either percutaneous coronary intervention or fibrinolytic therapy. Extensive evidence has shown that in patients presenting within six-hours of chest pain, survival is improved with either reperfusion therapy. Less data is available for patients who present after 12-hours of pain. One study compared outcomes in patients with RVMI following reperfusion and divided them into those with right ventricular failure, those without right ventricular failure and those with cardiogenic shock. The results indicated that primary percutaneous coronary intervention (PPCI) led to significantly lower short and long-term mortality rates compared to fibrinolytic therapy in patients with right ventricular failure and cardiogenic shock (8.3% vs. 13% and 44% vs. 100%, respectively) [75]. In patients with RVMI, complete revascularization with PCI dramatically improved right ventricular function compared to unsuccessful reperfusion which was associated with persistent hemodynamic compromise and higher mortality rates. The investigators also found that revascularization led to lower right ventricular filling pressure and less right ventricular dilation, increased left ventricular filling and cardiac output.

7. Management of non-dominant right coronary artery stenosis

Presently, there is no standardized management for non-dominant RCA occlusions (NDRCA). In fact, coronary intervention is rarely undertaken as indications are unclear and data remains limited. In one study, the authors retrospectively analyzed over 43,000 patients who underwent coronary angiography and describe 35 with severe isolated stenosis of a NDRCA [42]. Of these patients, 23 were managed conservatively and 12 were managed with PCI. The most common reason for angiography in the PCI group was acute coronary syndrome. In the conservative group, none had abnormal right ventricular function on follow-up imaging assessment and 25% (3/12) of the PCI group required revascularization. The study raises several important points includ-

ing the rarity of isolated NDRCA stenosis, the difficult nature of NDRCA PCI and the benefit of conservative management. The study also draws attention to right ventricular perfusion and function as 50% (7/14) of the patients with completely occluded vessels had normal right ventricular function despite the absence of visible collaterals. Although not guideline directed, there are some potential indications for percutaneous intervention of NDRCA lesions: primary intervention for myocardial infarction with ongoing chest pain, right ventricular salvage for acute coronary syndromes or to prepare the right ventricular marginal artery as an "interventional collateral" to the apical LAD for future chronic total occlusion-PCI (Fig. 5).

8. Prognosis

In patients with acute MI, RVMI is associated with higher rates of early mortality. In fact, the rate of in-hospital mortality in patients with cardiogenic shock from RVMI and LVMI are similar (55% vs. 60%) despite the RVMI group having a younger age, lower rate of anterior MI and higher prevalence of single-vessel coronary artery disease [54]. In an early study, 19 patients with acute myocardial infarction from a proximal right coronary artery occlusion, the authors found that impairment of right ventricular systolic function was common but early reperfusion was important for recovery [76]. In this study, patients were treated with intracoronary fibrinolytic therapy and using radionuclide techniques, the authors found a significant improvement in right ventricular ejection fraction at four weeks follow-up in the 12 patients that achieved recanalization compared to the patients where the artery remained occluded (43 $\pm$ 5% vs. 32 $\pm$ 6%). In another study, the authors enlisted 135 patients with an inferior MI and found that in-hospital morality was significantly higher in those with RVMI (16% vs. 3.5%, $p = 0.019$) [77]. In a larger study of 1129 patients with an inferior MI, the authors found that those with RVMI had a higher in-hospital and 35-day mortality (7.1% vs. 5.5% and 7.5% vs. 5.6%) compared to those without RVMI, although the difference was not statistically significant [78]. In addition, studies have evaluated patients with LVMI and found that those with RVMI had higher in-hospital mortality than those without RVMI (4.1% vs. 1.0%, $p = 0.033$) [79]. The authors also pooled their results to six smaller studies and found that RV myocardial involvement was associated with an increased risk of death, shock, ventricular arrhythmias, and atrioventricular block. In the SHOCK registry, the authors found that the cardiac index in patients with RV shock was similar to those with LV shock but with higher RA pressures and lower PA pressures [54]. Overall, refractory cardiogenic shock is the major determinant of poor outcomes.

Although the RV function returns to normal in the majority of patients with RVMI after successful reperfusion, some patients may have persistent RV failure that may contribute to poor long-term outcomes. In one study, the six-month mortality was 7.8% for patients with an inferior MI compared to 13.2% in patients with an anterior MI [78]. In this group, persistent RV failure was an independent predictor of clinical outcomes after adjusting for differences in baseline characteristics. Similarly, authors found that patients with RVMI secondary to isolated RCA disease had a similar 10-year survival rate to patients with combined RCA and LAD disease (62% vs. 52%, $p = 0.21$) [80].

9. Conclusions

RVMI presents specific clinical and management issues including hypotension, bradycardia and increased short-term mortality. In these patients, early recognition, rapid resuscitation, and prompt reperfusion are recommended. In those with right ventricular shock, mechanical support and temporary pacing may be beneficial but further studies are necessary to define the efficacy of these devices.

10. Learning points

-Right coronary artery lesions proximal to right ventricular marginal arteries are associated with higher rates of acute complications such as cardiogenic shock and bradycardia as well as higher rates of in-hospital mortality.

-Right ventricular myocardial infarction is associated with a high short term mortality rate but long-term survival improves particularly in those with improvement in right ventricular function.

-In addition to traditional cardiovascular risk factors, recent studies have found links between specific sodium channel mutations (KCNH2 and SCN5A) and the development of ventricular arrhythmias in patients with myocardial infarction.

-In patients with right ventricular failure, recent evidence has shown a role for mechanical circulatory support devices such as right ventricular assist devices, Impella RP and extracorporeal membrane oxygenation.

-There are some indications for percutaneous intervention of non-dominant right coronary artery lesions such as primary intervention for myocardial infarction with ongoing chest pain, right ventricular salvage for acute coronary syndromes and in preparing the right ventricular marginal artery "interventional collateral" to the apical LAD for future chronic total.

Author contributions

GF performed the research on the topic and wrote the manuscript. JKF performed the research on the topic and wrote the manuscript. CJ performed the research on the topic and wrote the manuscript. DL performed the research on the topic and wrote the manuscript. SL performed the research on the topic and wrote the manuscript. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

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Conflict of interest

The authors declare no conflict of interest.

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