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REVIEW

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ABSTRACT

Introduction: Acute right ventricular myocardial infarction (RVMI) is observed in 30–50% of patients presenting with inferior wall myocardial infarction (MI) and, occasionally, with anterior wall MI. The clinical consequences vary from no hemodynamic compromise to severe hypotension and cardiogenic shock depending on the extent of RV ischemia.

Areas covered: The pathophysiological mechanisms, diagnostic steps, and novel therapeutic approaches of acute RVMI are described.

Expert commentary: Diagnosis of acute RVMI is based on physical examination, cardiac biomarkers, electrocardiography, and coronary angiography, whereas noninvasive imaging modalities (echocardiography, cardiac magnetic resonance imaging) play a complementary role. Early revascularization, percutaneous or pharmacological, represents key step in the management of RMVI. Maintenance of reasonable heart rate and atrioventricular synchrony is essential to sustain adequate cardiac output in these patients. When conventional treatment is not successful, mechanical circulatory support, including right ventricle assist devices, percutaneous cardiopulmonary support, and intra-aortic balloon pump, might be considered. The prognosis associated with RVMI is worse in the short term, compared to non-RVMI, but those patients who survive hospitalization have a relatively good long-term prognosis.

1. Introduction

Coronary artery disease remains the main cause of morbidity and mortality globally [1]. Acute coronary syndrome occurs when there is a decreased blood flow or complete cessation of flow in one of coronary arteries. Acute right ventricular myocardial infarction (RVMI) was first described in the literature in 1974 in a series of six patients [2]. RVMI occurs in one-third to one-half of patients presenting with inferior myocardial infarction (MI) [3–5], and it significantly contributes to the clinical and hemodynamic instability that these patients are presented with [6–8]. Occasionally, RVMI can accompany anterior wall MI, and very rarely it can occur in isolation [9]. Right ventricle (RV) involvement in the setting of inferior MI increases the inhospital morbidity and mortality [10]. Almost, half of RVMI patients have poor outcomes secondary to electrical or hemodynamic instability [11]. Effective fluid resuscitation aiming to restore the preload, and subsequently maintain adequate cardiac output, along with percutaneous or pharmacological revascularization is first-line therapy of acute RVMI [12]. It is very important to early recognize the RV involvement in a patient presenting with acute MI, not only for prognosis, but also to choose the specific therapy, including aggressive primary percutaneous coronary intervention (PCI), with particular attention to RV branch revascularization, all in order to avoid any unwanted detrimental complications associated with this diagnosis.

In this review, we aim to discuss the (1) pathophysiology of RVMI, (2) diagnostic approach, (3) therapeutic management, including fluid- and pharmacotherapy, revascularization approaches, and mechanical support, and finally (4) short- and long-term prognosis. Figure 1 provides a comprehensive illustration of the pathophysiology and the key management steps of RVMI.

2. Pathophysiology

Acute RVMI can occur when there is occlusion of the right coronary artery (RCA), proximally to the takeoff of RV branches [3,5,13,14].′ The RV has unique physiological and structural characteristics compared to the left ventricle (LV), which account for the reduced prevalence and faster recovery of RVMI. More specific (1) the RV has thin walls requiring less oxygen, (2) the RV is a 'low-pressure chamber' and hence perfusion occurs both during systole and diastole, (3) the ability of RV to extract oxygen is increased during hemodynamic stress, (4) the RV may have rich collaterals from the left coronary artery, and (5) the RV has direct blood supply from RV cavity through the thebesian veins [15,16].

The hemodynamic compromise and the volume overload following RVMI depend primarily on the location of the culprit lesion, in that the more proximal the RCA occlusion, the larger the RV infarction [17–19] and subsequently on the extent of the ischemic injury. The consequent systolic and diastolic RV dysfunction decreases the RV output and increase the right atrial pressure (RAP). In the context of reduced preload and/or
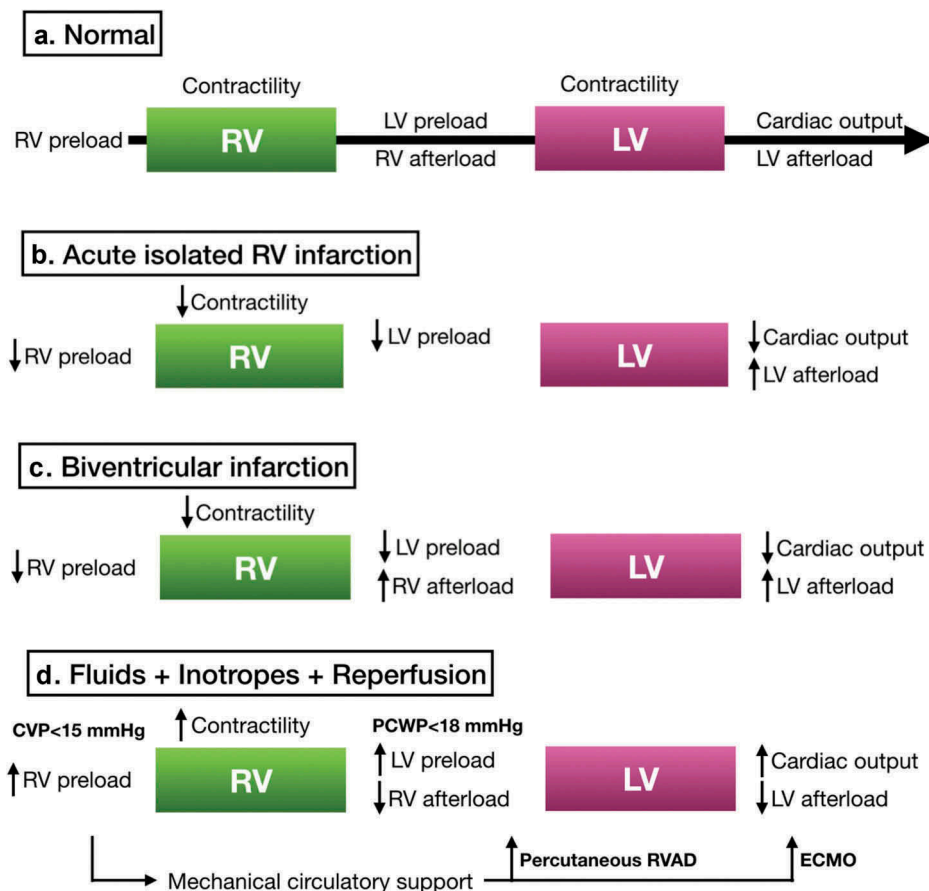
Figure 1. Schematic illustration of the physiology (a) of the right ventricle (RV) and the left ventricle (LV), the pathophysiological alterations during isolated RV infarction (b), biventricular infraction/failure (c), as well as the appropriate management steps (d). CVP: central venous pressure, PCWP: pulmonary capillary wedge pressure.

loss of atrioventricular synchrony, left ventricular dysfunction emerges [11,20,21,22]. In the setting of acute RV dysfunction, the RV free wall is usually unable to contribute to stroke work, resulting in failure to maintain forward flow into the pulmonary artery (PA), which leads to reduced LV preload. Subsequently, RV dilation shifts the interventricular septum toward the LV, which further worsening of LV preload, an effect further exacerbated by elevated intrapericardial pressure. In case of acute RVMI, RV systolic pressure and global work are generated by LV septal contractile contributions mediated via the interventricular septum [23–25]. If this cascade of events is not managed promptly and urgently, it will lead to hypotension, shock, and death.

3. Diagnostic approach

3.1. Clinical features

While not pathognomonic, the presence of hypotension, elevated jugular venous pressure without pulmonary congestion, Kussmaul sign, tricuspid regurgitation murmur, and atrioventricular dissociation might be suggestive of RVMI [26–28]. RVMI tends to be associated more frequently with vagal symptoms, such as bradycardia, nausea, vomiting, diaphoresis, and pallor. Tachycardia can also occur and is often due to increased sympathetic discharge secondary to anxiety or as a compensatory mechanism to decreased cardiac output. In few

occasions, a ventricular septal defect may accompany acute RVMI. This usually presents with a holosystolic murmur and often leading to severe acute hemodynamic compromise and cardiogenic shock [29]. This happens when the left-to-right shunt decreases effective forward LV output, leading to hypotension and precipitating pulmonary edema. Although with high mortality if left untreated, surgical repair or percutaneous device closure is imperative [30]. Elevated right heart pressures in the context of acute RVMI may also stretch open a patent foramen ovale or cause a right-to-left shunt via an atrial septal defect, which is clinically evident as oxygen-resistant systemic hypoxemia or paradoxic emboli [31,32].

3.2. Electrocardiography

All patients presenting with inferior ST segment elevation should have electrocardiographic assessment of potential RV involvement. Only lead V1 and possibly V2 may provide a partial view of the RV free wall especially where there is ST deviation; however, greater ST elevation in lead III than lead II is usually suggestive of RV involvement [33]. Assessment of right precordial leads (i.e. rV1 through rV6) is particularly helpful for the diagnosis of RV involvement and the localization of the culprit lesion. ST elevation >1.0 mm in lead rV4 is highly suggestive of proximal RCA occlusion and RVMI [34,35]. However, the ST elevation in rV4 is transient and its absence cannot exclude the occurrence of RVMI. Nevertheless, ST elevation in rV4 is also associated with other

cardiac diseases including acute anteroseptal MI, previous anterior MI with aneurysm, LV hypertrophy, and acute pulmonary embolus, and may mimic Brugada syndrome [36]. From the prognosis point of view, the greater the ST elevation in rV4, the more significant the RV dysfunction and the higher inhospital morbidity and mortality [8,37].

3.3. Coronary angiography and hemodynamic study

The gold standard diagnostic modality for RVMI is coronary angiography. In the majority of RVMI, the RCA is the culprit artery in right dominant systems when there is an occlusion proximal to the major RV branches in the setting of inferior MI (Figure 2). Occasionally, the left circumflex or left anterior descending artery can be the culprit for RVMI [5,38]. The conus artery, which has a separate ostium to the RCA in 30% of cases, supplies the infundibulum – in this to some extends, explains the sparing of this region even in proximal RCA occlusions.

Despite the initial functional abnormalities associated with RVMI, the ischemic RV usually recovers its function in the long term even in many non-revascularized patients [39].

Hemodynamically significant RVMI is associated with increased RAP (>10 mmHg), RAP to pulmonary capillary wedge pressure (PCWP) ratio > 0.8 (normal value < 0.6), RAP within 5 mmHg of the PCWP, and reduced cardiac index. However, in the setting of concomitant LV dysfunction, the RAP to PCWP ratio can change depending on the magnitude of change of right atrial and PCWPs [40]. Another important hemodynamic measure is the pulmonary artery pulsatility index (PAPi), which is equal to (systolic PA pressure – diastolic PA pressure)/mean RA, pressure with a value ≤ 0.9 which provides 100% sensitivity and 98.3% specificity for predicting outcomes for high-risk patients with acute RVMI [41]. Cardiac power output is the single most predictive marker of prognosis in cardiogenic shock, which could be another tool used to assess hemodynamic status in cardiogenic shock complicated by acute RVMI [42].

Additional hemodynamic findings of RVMI include prominent y-descent of the RAP, increased RAP and drop of systemic arterial pressure >10 mmHg with inspiration, 'dip and plateau' morphology and equalization of the diastolic filling pressures, and pericardial pressure due to increased right ventricular volume.

3.4. Echocardiography

Two-dimensional transthoracic echocardiography can show RV dilation, as well as depressed RV systolic function and regional wall motion abnormalities associated with RVMI [23,43–46]. It can also detect elevated pulmonary pressures, pulmonary regurgitation, tricuspid regurgitation, and increased RAPs in hemodynamically unstable RVMI patients [47,48]. RV akinesia or dyskinesia was found to be surrogate marker of hemodynamically significant RVMI [49]. Despite being widely available and accessible imaging modality for the evaluation of patients with RVMI, echocardiographic assessment of RV can be challenging, due to the geometrical complexity of RV and the transient nature of ischemic RV dysfunction [50]. Three-dimensional echocardiography is a promising alternative modality for the volumetric assessment of RV [51].

More recently, the assessment of RV free wall longitudinal strain using speckle tracking images with a cutoff value ≥−19.7% was found to be a useful tool for RV involvement and an independent predictor to rule in proximal RCA culprit lesion in inferior wall MI patients presenting to the emergency department [52,53]. Nevertheless, noninvasive imaging modalities remain complementary modalities and in the acute setting reperfusion therapy should never be delayed by complementary imaging.

3.5. Cardiac magnetic resonance (CMR)

Although the role of CMR in the diagnosis of acute RVMI is not well investigated, CMR is considered the standard imaging technique for the evaluation of RV function and structure [54]. Late gadolinium enhancement appears to be more sensitive in detecting RV involvement compared to echocardiography. CMR studies showed that RVMI coexists in 47–57% of inferior wall MI (Figure 3) [55–57]. As stated above, CMR is reserved for more non-emergent, nonurgent assessment of RV function.

3.6. Radionuclide myocardial imaging

Radionuclide myocardial imaging used to play an important role in the assessment of both end systolic and end diastolic volumes to calculate RV ejection fraction (RVEF). It is known that the assessment of radionuclide count density is not geometry dependent [58]. Segmental RV wall motion abnormalities in association with reduced RVEF (to < 40%) with segmental RV wall motion abnormalities on first-pass ventriculography are highly sensitive and specific for RVMI or RV ischemia [49]. With the widespread use of CMR given no radiation and accurate assessment of function and morphology, these radionuclide techniques have become less popular nowadays [54].
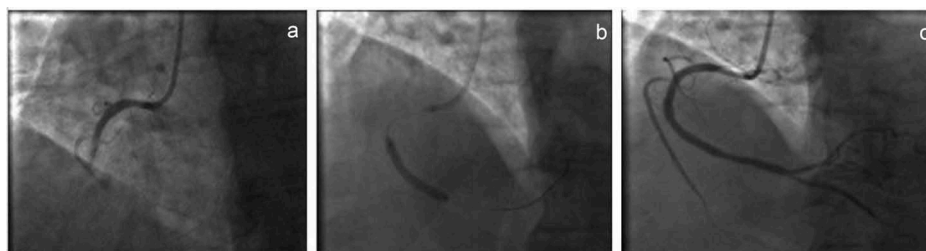

Figure 2. Cardiac catheterization demonstrating acute thrombotic occlusion of mid-RCA (a). After crossing the lesion and deploying drug-eluting stent (b), TIMI flow 3 was restored (c).
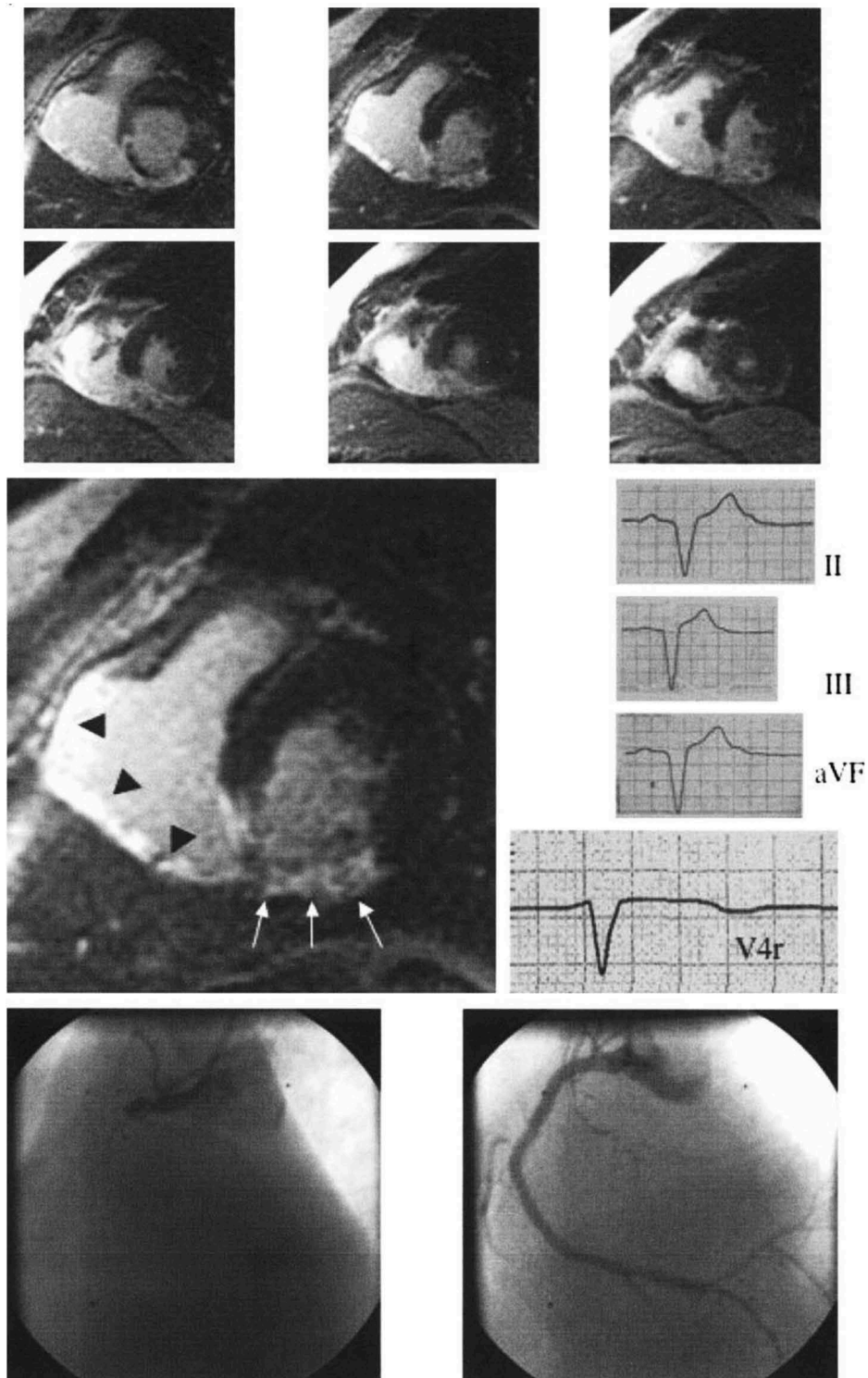
Figure 3. Patient with acute inferior and right ventricular (RV) infarction on late enhancement cardiovascular magnetic resonance imaging (LE-CMR). (Upper panels) Short-axis LE-CMR images showing contrast enhancement of the RV wall. (Middle panels, left) Enlarged short-axis view with infarction of the RV wall (black arrowheads) and the inferior left ventricle (white arrows). (Middle panels, right) Electrocardiogram with ST-segment elevation in V4R. (Lower panels) Culprit right coronary artery lesion in a right dominant perfusion pattern before (left) and after (right) angioplasty. Echocardiography revealed RV hypokinesis and dilatation. Reproduced with permission from [55].

4. Differential diagnosis

There are certain diagnoses that could be confused with RVMI and these include pulmonary embolism (PE) (with classical ECG changes such as a large S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III ($S_1Q_3T_3$) and ST elevation in the right-sided precordial leads caused by 'strain'), pericarditis with pericardial tamponade (with widespread saddle shape ST elevation, including right-sided leads), and anteroseptal MI (ST elevation in leads V1 and V2 may be seen with an RV injury pattern). Of these, PE and RVMI are most often

confused. Both can present with chest pain and findings of clear lung fields and hypotension (including shock) on examination. The nature of the chest pain (ischemic versus pleuritic) may be helpful in making a distinction. The electrocardiogram is usually crucial to discriminate between the two: ST elevation in the inferior leads is rarely present in patients with PE, whereas elevation of serum troponin may be present with either diagnosis. On echocardiography, right ventricular systolic dysfunction may be seen with both diagnoses. Sparing of the right ventricular apex ('McConnell's sign') has been suggested to be specific for a large PE in some reports [52,53]. If there is uncertainty in the diagnosis, additional testing, such as pulmonary CT angiography or ventilation/perfusion scanning, may be necessary to establish the correct diagnosis of PE.

5. Complications

The most common complications of RVMI are summarized in Table 1. Conduction disorders and arrhythmias are more commonly encountered in RVMI compared to other MIs [10]. High-grade atrioventricular block is observed in almost 50% of the patients presenting with RVMI and is associated with poor prognosis [59]. Atrial fibrillation attributed to atrial infarction or distention is found in one-third of the patients, followed by rapid clinical deterioration. Ventricular tachyarrhythmias occur more frequently with RVMI compared to left-sided MI [60]. Other complications include rupture of the interventricular septum, tricuspid valve regurgitation, thrombus formation in the RV and subsequent PE, acute post-MI pericarditis, and cardiogenic shock.

6. Therapeutic approaches

The following considerations are critical for the management of acute RVMI: (1) revascularize emergently, (2) maintain adequate RV preload, (3) optimize rhythm, (4) support the RV with inotropes, and (5) support mechanically.

6.1. Reperfusion

Early and complete reperfusion with thrombolysis or percutaneous coronary revascularization improves outcomes in RVMI patients [61–63]. Incomplete or partial revascularization is

Table 1. RVMI complications.

Complications of RVMI
1. Atrioventricular blocks
2. Arrhythmias (bradyarrhythmia or tachyarrhythmia)
3. Vasovagal symptoms
4. Hypotension
5. Cardiogenic shock
6. Ventricular septal defect
7. Pericarditis +/− pericardial effusion
8. RV thrombus
9. Tricuspid regurgitation
10. Pulmonary hypertension
11. Right heart failure
12. Atrial fibrillation

associated with ventricular tachyarrhythmias, persistent hypotension, and higher mortality [64].

6.2. Maintenance of RV preload

Maintenance of adequate RV preload is what distinguishes the therapy of RVMI from the left ventricular MI. There is significant hemodynamic variability in patients with RVMI, related to the intravascular volume state, inter-ventricular dependence, and status of left ventricular function. Preload reducing agents, such as nitrates, diuretics, and morphine, should be avoided in RVMI due to the increased risk to induce hypotension and cardiogenic shock. The first-line therapy of RVMI-induced hypotension without pulmonary congestion involves intravenous administration of fluids (i.e. N/S 0.9% at 40 ml/min for 2 L), trying to maintain a central venous pressure (CVP) <15 mmHg and the PCWP between 18–24 mmHg [65–68].

It is not until 1981 when Lopez-Sendon and group were among the first physicians to describe the concept of fluid replacement in ischemic RV dysfunction, and this was very much apparent especially with the development of PA catheterization (Swan-Ganz catheter). Following this, many studies complemented this work and showed the usefulness of volume resuscitation in patients presenting with RV dysfunction in the setting of RVMI [49,69].

Earlier studies showed that using normal saline with the failing RV was sufficient enough to maintain adequate RV preload to resolve accompanying hypotension and subsequently improve cardiac output [70]. One study showed that flow-driven volume resuscitation with special attention to measurement of CVP and cardiac index (CI) significantly influenced clinical decision in the setting of hypovolemia with RVMI [71]. Previous reports have shown that maximal RV stroke index has been achieved when the right-sided filling pressure was 10–14 mmgHg, in contrary a mean RAP of > 14 mmHg was seen, almost always, in case of reduced RV stroke index. In an study involving 41 patients, it was found that patient's volume status and degree of LV involvement were important factors in hemodynamic instability in those presenting with acute RVMI, and that the optimal PCWP, which was an indirect measure of maximum LV stroke work index, was 16 mmHg [67]. For this reason, someone must pay a great attention that over administration of fluid will worsen the function of the failing RV which leads to LV preload reduction and hence worsening of cardiac output [72].

6.3. Rhythm optimization

Maintaining adequate atrioventricular synchrony is a basic component in optimizing right ventricular preload. It is important to know that the infarcted RV and, consequently, the preload-deprived LV have a fixed stroke volume, and in this circumstance, cardiac output mainly depends on the heart rate [73,74]. Atropine can increase heart rate to some extent, but patients with profound bradyarrhythmias likely need a pacemaker [75]. Patients with RVMI and atrioventricular block should have a temporary dual-chamber pacemaker implanted, as it helps increasing the cardiac output and preventing the

development of cardiogenic shock to a greater extent than a single chamber pacemaker [76]. Transcutaneous pacing can be also considered when the transvenous pacing cannot be adequately sensed by the infarcted RV.

6.4. Inotropic support

Inotropic support of RVMI patients plays a pivotal role in the improvement of both RV function. Administration of dobutamine along with fluids improves the systolic capacity of RV, thereby increasing the systolic performance of LV and systemic pressure [24]. Furthermore, dobutamine seems to reduce the pulmonary vascular resistances and the RV afterload, as well as improving AV conduction. Dobutamine appears to be particularly helpful in RVMI patients with interventricular septum involvement [77,78]. However, its utility is limited by arrhythmias, systemic vasodilation, and hypotensive response.

Other inotropes that can be used in RV failure/cardiogenic shock in the setting of RVMI include milrinone, levosimendan (approved only in Europe), and norepinephrine. Milrinone may further reduce preload and exacerbate hypotension, while lowering RV afterload by reducing pulmonary resistance. Levosimendan, a calcium sensitizer inotropic agent, appears to improve RV contractility in patients with chronic LV failure without worsening diastolic function or an obvious increase in myocardial oxygen demand [79]. Levosimendan works by activation of adenosine triphosphate (ATP)-sensitive potassium channels in the pulmonary vasculature, leading to dilatation hence RV afterload reduction, while reducing LV afterload and improving coronary perfusion by a similar mechanism on systemic and coronary vessels. In term of other agents such as dopamine and phenylephrine, their role in RVMI is questionable [66].

6.5. Mechanical circulatory support

In patients with cardiogenic shock secondary to RVMI, mechanical circulatory support maybe achieved with (1) direct RV support, (2) indirect RV support, or (3) biventricular support, depending on the degree of support needed [80].

(1) Direct RV support: the available devices include Impella RP (Abiomed, Inc., Denvers, MA, USA) and TandemHeart (TandemLife, Pittsburgh, PA, USA) and ProtekDuo (CardiacAssist Inc., Pittsburgh, Philadelphia, PA, USA). Impella RP is the only device currently approved for the management of acute RV failure. It is an axial flow pump which is inserted via the femoral vein with the inflow portion is placed in the inferior vena cava and the outflow is placed in the main PA. The device can generate a cardiac output up to 4 L/min [81]; however, more studies are needed to assess its efficacy and safety. RECOVER-RIGHT study included 30 patients with acute RV failure managed with Impella RP [82]. In this cohort, survival to 30 days or hospital discharge was 83.3%. In contrast, in the subset of patients treated with Impella RP for acute RV failure due to cardiotomy or acute MI, survival was 58.3%. It was noted that the most common adverse events were bleeding and hemolysis. In contrast, TandemHeart is a centrifugal flow pump which is inserted through the two femoral veins or one of them and one internal jugular vein. The ProtekDuo is another centrifugal flow pump, but the inflow and outflow cannulas are combined into one double-lumen catheter that is inserted via the internal jugular vein. An oxygenator can be fitted to either TandemHeart or ProtekDuo, and this may be useful in the setting of RV failure with hypoxemia [80].

(2) Indirect RV support: VA-extracorporeal membrane oxygenation (ECMO) is one of the most effective therapies in providing mechanical circularity support for the failing RV [83,82]. It is a centrifugal pump that pumps blood via a cannula inserted into the central venous circulation, then this blood got mixed with O_2 through a membrane oxygenator and later ejected to the central arterial circulation. It causes a reduction in both RV preload and RV cardiac output, while increasing systemic arterial pressure and LV afterload. It is important to pay an attention when there is a biventricular failure while using ECMO device, as the LV needs to be unloaded to prevent further worsening of LV failure and pulmonary edema. This could be achieved by combined VA-ECMO with Impella or with intra-aortic balloon pump (IABP). IABP can be considered in acute RV failure as it improves cardiac hemodynamics by increasing coronary artery perfusion during diastole and reducing LV afterload [64]. In a series of 32 patients with cardiogenic shock secondary to RVMI managed with revascularization of culprit RCA and IABP support, survival was 81% [85]. Moreover, the failing RV depends on the interventricular septum to maintain stroke volume, so reducing LV afterload with IABP may indirectly improve RV performance. This may be especially beneficial in patients with RVMI and concomitant LV failure [86].

(3) Biventricular support: direct RV support devices in combination with either direct LV support devices (Impella, TandemHeart) or IABP have been used successfully in this situation [87–92].

Last, left ventricular assist devices may be also useful in RVMI-induced cardiogenic shock by increasing coronary perfusion, especially when the culprit vessel has already been revascularized, and improving the contractility of the interventricular septum.

7. Prognosis

RV involvement in the setting of inferior wall MI is an independent risk factor for increased mortality (17% vs. 6.3%) [93]. Refractory cardiogenic shock is the major determinant of poor outcomes in those patients. Percutaneous revascularization has improved overall short-term prognosis compared to fibrinolysis (7% vs. 9%) [12,94]. RVMI patients end up with a spectrum of inhospital complications especially arrhythmias. However, those who survive hospitalization have a relatively good long-term prognosis [95]. In the SHOCK (Should we emergently revascularize Occluded coronaries for Cardiogenic shock) trial registry, the cardiac index was depressed to the same degree in patients with RV shock similar to those with LV shock, albeit with higher RA pressures

and lower PA pressures for a similarly elevated LV filling pressure [96]. Furthermore, there was almost equivalent rate of mortality due to cardiogenic shock in the setting of RVMI as compared to cardiogenic shock in the setting of LV infarction (55% and 60% inhospital mortality, respectively) despite the patients' younger age, lower rate of anterior MI, and higher prevalence of single-vessel coronary disease [97].

In the majority of patients suffering from RVMI, RV function returns to normal [98]. Clinical improvement and normalization of hemodynamic parameters is witnessed even in patients with persistent RV dysfunction.

8. Conclusion

In summary, one-third to one-half of inferior MIs are complicated by RVMI and this varies from mild asymptomatic RV dysfunction to severe hypotension, cardiogenic shock, and death. The diagnosis of RVMI can be challenging; the 12 lead ECGs with supplemental right precordial recordings remain the principal diagnostic tool in the acute setting, but the findings may be transient. High clinical suspicion is required for accurate diagnosis and assessment of RVMI by integrating clinical, imaging, hemodynamic study, and angiographic data to avoid any detrimental complications associated with RVMI. The pathophysiology of the RV makes it resistant to infarction, but acute ischemia can lead to severe hemodynamic consequences. Fluid resuscitation to maintain an adequate RV preload is the first-line therapy. Emergent revascularization, with preference to primary PCIs, is the cornerstone of RVMI management. When refractory hypotension or cardiogenic shock emerges, mechanical circulatory support confers survival benefit. Patients who survive from the acute phase exhibit an overall favorable long-term prognosis.

9. Expert commentary

Coronary artery disease is the leading cause of morbidity and mortality worldwide. RVMI is seen in up to half of inferior wall MIs, and occasionally, it can accompany anterior wall MI, and very rarely it can occur in isolation. Diagnosis of acute RVMI is based on history, physical examination, cardiac enzymes, electrocardiography, and coronary angiography, whereas noninvasive imaging such as echocardiography or MRI can play a complimentary role in the diagnosis. The clinical consequences vary from no hemodynamic compromise to severe hypotension and cardiogenic shock depending on the location of the culprit lesion, in that the more proximal the RCA occlusion, the larger the RV infarction and subsequently on the extent of the ischemic injury. Early and complete reperfusion with thrombolysis or percutaneous coronary revascularization, with the latter being the preferred choice, improves outcomes in RVMI patients; however, incomplete or partial revascularization is associated with ventricular tachyarrhythmias, persistent hypotension, and higher mortality.

Maintenance of reasonable heart rate and atrioventricular synchrony is essential to sustain adequate cardiac output in these patients. Inotropes serve an important role in the management of acute RVMI. Administration of dobutamine along with fluids improves the systolic capacity of RV, thereby increasing the systolic performance of LV and systemic pressure. Furthermore, dobutamine seems to reduce the pulmonary resistances and the RV afterload. Dobutamine appears to be particularly helpful in RVMI patients with interventricular septum involvement. When conventional treatment is not successful, mechanical circulatory support might be considered. In patients with cardiogenic shock secondary to RVMI, mechanical circulatory support with Impella RP or ECMO is one of the most effective therapies. IABP might also confer mechanical support to the failing RV by improving coronary perfusion. Surgically implanted right ventricular assist devices have been also used to support the acutely failing RV which can generate a cardia output up to 4 L/min.

RVMI worsens the short-term prognosis in the setting of inferior MI, but in the absence of LV dysfunction, there is a good long-term prognosis. The key weakness in the clinical management is mainly attributed to late diagnosis of RVMI. Current open research questions that necessitate further study data include Can accurate and prompt identification of RV infarction lead to more specific management that can further improve on prognosis? Do we have better tools of biomarkers specific for RVMI that lead to early diagnosis of the disease? Does aggressive PCI with revascularization of RV branches improve outcome and reduce risk of complications? Can the utility of 3D echocardiography further help in better diagnosis of RV function in the setting of MI? Does speckle tracking and strain analysis of free RV wall and longitudinal analysis can further help in estimating prognosis and long-term survival? Last, are there any prognostic markers such as old scar from prior MI as detected on late gadolinium enhancement on CMR that might affect the outcome? With regard to RVMI management and improved prognosis, the foremost improvement is achieved by the availability of mechanical circulatory support such as Impella devices or ECMO machines that improve the short-term prognosis.

10. Five-year view

The major advancements in the management of RVMI especially when associated with acute RVF include the use of mechanical circulatory support, including Impella family devices. The former three devices have been evaluated in randomized study and showed no major 30-day mortality difference when compared with IABP. The later was shown to provide adequate support in right-sided failure. On the other hand, more frequent use of 3D echocardiography imaging and availability of cardiac MR will assist in earlier recognition and diagnosis of RVMI and potentially leading to a better outcome in the years to come.

Key issues

- RVMI occurs in up to half of inferior myocardial infarctions.
- The majority of RVMI results from occlusion of proximal RCA.
- RVMI should be suspected in the setting of hypotension, raised JVP, clear lung fields, IWMI, and elevated ST segment in V4R.

- RVMI is associated with increased mortality and morbidity; however, those who survive the short term usually have excellent long-term prognosis.
- Early and prompt reperfusion (preferably with PCI) is the key strategy for successful management of RVMI.
- Nitrates, diuretics, opioids, beta blockers, and calcium channel blockers in the setting of RVMI should be avoided.
- Hypotension should be treated with adequate IV fluids, and inotropes are used in refractory hypotension.
- Mechanical circulatory support, e.g. IABP, ECMO, and right ventricular assist device (RVAD) should be considered in hemodynamic instable patients with acute RV failure secondary to RVMI.

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Declaration of interest

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