Right ventricular myocardial infarction: pathophysiology, diagnosis, and management

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ABSTRACT

Right ventricular (RV) ischaemia complicates up to 50% of inferior myocardial infarctions (MIs), though isolated RV myocardial infarction (RVMI) is extremely rare. Although the RV shows good long term recovery, in the short term RV involvement portends a worse prognosis to uncomplicated inferior MI, with haemodynamic and electrophysiologic complications increasing in-hospital morbidity and mortality. Acute RV shock has an equally high mortality to left ventricular (LV) shock. Identification of RV involvement, particularly in the setting of hypotension, can help anticipate and prevent complications and has important management implications which are distinct from the management of patients presenting with LV infarction. Reperfusion therapy, particularly by primary percutaneous coronary intervention, hastens and enhances RV functional recovery that occurs to near normality in most patients. The diagnostic methods for RVMI are discussed, including clinical, electrocardiographic, and various imaging modalities as well as the RV pathophysiology that underpins the specifics of RVMI management.

INTRODUCTION

Despite the clinical observation of right ventricular (RV) infarction by Sanders almost 80 years ago,[1] this condition had received little clinical attention until recent years, as early animal studies suggested it to be of low haemodynamic significance.[2] It was first described as a distinct clinical entity by a seminal paper by Cohn *et al* 36 years ago.[3] Post-mortem studies reveal that there is RV involvement in 14–60% of patients dying with acute infero-posterior myocardial infarctions (MIs).[4 5] Non-invasive studies also suggest the presence of RV ischaemic dysfunction in about 50% of patients with acute inferior MI[6 7] and in ≤10% of patients with anterior infarcts, whereas isolated RV infarction is rare, accounting for <3% of all cases of fatal infarction.[8]

The prognosis of right ventricular myocardial infarction (RVMI) is generally thought to be good—a view corroborated by the observation that the outcome of inferior myocardial infarction is more favourable than that of anterior infarction. Multiple studies have, however, described increased acute mortality in patients with acute inferior infarcts complicated by RV involvement.[9–12] In a recent meta-analysis of 22 studies involving a total of 7136 patients with acute MI, the presence of RVMI was associated with a 2.6-times increased risk of mortality as well as statistically significant increases in secondary end points of morbidity such as ventricular arrhythmias, high grade atrioventricular (AV) block, and mechanical complications.[13]

The early recognition of RVMI in a patient with acute MI is of prime importance, not only for prognostication purposes, but also because it can guide specific therapy, including aggressive primary percutaneous coronary intervention (PCI), with particular attention to RV branch revascularisation, in order to limit the deleterious effects of this diagnosis.

In this review we first discuss the pathophysiology of RVMI, as this underpins the specific management decisions for RVMI patients. We then aim to provide an overview of clinical, electrocardiographic, and several imaging diagnostic techniques, their strengths and limitations as well as the applicability of each modality in the various clinical settings of RVMI presentation. Finally, we discuss the management implications of RVMI.

RV PATHOPHYSIOLOGY

The RV has about one sixth the muscle mass and performs about one quarter of the work of the left ventricle (LV) yet, with the exception of small physiologic shunts, provides the same cardiac output.[14] This is achieved through the pulmonary circulation providing a much lower afterload than systemic resistance.[15 16] RV myocardial oxygen consumption is approximately half that of the LV and oxygen extraction at rest is lower than the LV, with capacity to increase on stress.[17] Anatomic blood supply is dual with predominant supply from the right coronary artery (RCA) and nearly one third of RV free wall blood flow derived from the left coronary branches.[18] Unlike the LV, RV coronary perfusion occurs in systole as well as diastole in the absence of severe RV hypertrophy,[19] and there is also potential for more extensive subacute collateral formation from left to right coronaries.[20] Consequently, the supply–demand profile of the RV is more favourable than that of the LV.

The RV acts as a volume pump by its free wall (RVFW) longitudinally shortening and contracting towards the interventricular septum from apex to RV outflow tract.[20] The septal contraction contributes about one third of RV stroke work even under physiological conditions,[21 22] with the LV assisting further by providing traction on the RV free wall at their attachment points.[23] The RV pressure–volume loop has relatively brief periods of isovolumic contraction and relaxation and a sustained ejection period during pressure development but also pressure decline. This low pressure, volume pump system can adapt well to volume overload states such as tricuspid regurgitation or atrial septal defect,[24] but is very sensitive to

increases in afterload to which the RV can respond only by compensatory dilatation to maintain stroke volume, albeit at a reduced ejection fraction.[14 25]

OCCURRENCE OF RVMI

RVMI occurs principally due to occlusion of the RCA proximal to the major RV branches in the context of an inferior MI.[26] It may also occur by occlusion of the left circumflex artery in patients with a left-dominant circulation but also, less commonly, in anterior infarcts as the anterior part of the RV free wall is supplied by collaterals from the left anterior descending artery.[18] The conus artery, that has a separate ostium to the RCA in 30% of cases, supplies the infundibulum—which explains the sparing of this region even in proximal RCA occlusions.

Although ischaemic dysfunction occurs initially, the ischaemic RV usually recovers its function in the long term even in many non-revascularised patients.[27] Recovery is so common as to have led some clinicians to believe that the term 'right ventricular infarction' is a misnomer and that RV 'stunning' with viability is more appropriate.[28] As might be expected, RV hypertrophy with consequent increased oxygen demands may predispose to more RV infarction.[5 29 30]

RVMI is associated with clinically evident haemodynamic manifestations in <50% of affected patients. Nonetheless, even in the absence of evident haemodynamic compromise, the potential of RV ischaemia should be recognised so as to avoid treatment that will further lower RV preload and compromise the patient's condition.

RV ischaemia causes depressed RV contractility and RV dilatation as well as impaired relaxation. This leads to decreased RV compliance, reduced filling, and decreased RV stroke volume, resulting in reduced transpulmonary delivery of LV preload that leads to lower total cardiac output despite intact (or mildly reduced) LV contractility. RV dysfunction, however, has a more pronounced effect on cardiac output than would simply be expected by the reduced LV preload. The elevated RV volume and RV end diastolic pressure (RVEDP) also displaces the septum towards the volume deprived LV, further impairing LV compliance and limiting LV filling.[20] Moreover, RV dilatation within the non-compliant pericardium leads to elevated intrapericardial pressure, causing further restraint of LV filling and to equalisation of diastolic pressures.[31] Notably, the early animal studies, that had found no significant haemodynamic compromise from experimentally induced isolated RV damage, had used an open pericardial model.[2 32]

Associated ischaemia of the interventricular septum, with loss of its significant contribution to global RV systolic function, may further exacerbate haemodynamic compromise.[33] In the acute setting, coexisting LV MI may cause pulmonary capillary wedge pressure to rise, increasing RV afterload and further embarrassing RV function.

If the right atrium (RA) is spared, it may demonstrate increased contractility through increased preload. This leads to enhanced atrial relaxation, facilitated atrial inflow and may enhance RA performance to the extent of offsetting some of the haemodynamic consequences of RV ischaemia. The forceful RA contractions can cause the pulmonary valve to open before RV systole, a phenomenon initially detected by M mode echocardiography.[34] The increased loading conditions on the RA, however, increase RA oxygen demands while the increased intra-atrial pressure tends to diminish transmural perfusion. These features, in addition to the RA blood supply being related to that of the RV and consequently likely to be compromised, make RA ischemic involvement not uncommon in RVMI; it is documented in up to 20% of RVMI cases on autopsy studies.[20 35]

Right atrial ischaemia may further compound RV ischaemia by leading to rate and rhythm disturbances that impair RA and RV function as well as AV synchrony in as many as 50% of RVMIs, especially in patients with proximal RCA occlusion.[36 37] High degree AV block is often due to AV nodal ischaemia and is associated with poorer prognosis,[38] whereas atrial fibrillation (AF) may occur secondary to atrial wall stretch by the elevated RA pressures.

Afferent vagal stimulation by receptors on the inferior and posterior walls of the heart, commonly affected in RVMI, as well as baroreceptors from the RV lead to enhanced parasympathetic tone and the cardioinhibitory Bezold–Jarisch reflex. Reperfusion of the acutely occluded RCA by thrombolysis or primary PCI may paradoxically lead to severe yet transient bradycardia hypotension, which is thought to be due to this right heart reflex mechanism. AV nodal block occurring beyond the first 24 h of infarction tends to be atropine insensitive; it has been suggested to be secondary to adenosine release by the ischaemic myocardium[39 40] and may therefore respond to aminophylline.[41] Ventricular tachyarrhythmias from the dilated RV are common in the acute setting, complicating up to a third of cases[10 11 42 43] particularly in the absence of coronary reperfusion,[28] though late scar-related arrhythmias are unlikely.[20]

DIAGNOSIS OF RVMI

The diagnosis of RVMI is commonly made from the physical examination, electrocardiography, echocardiography, and haemodynamic measurements. Chest radiography may be useful in determining the presence or absence of pulmonary oedema but is not useful for the detection of RVMI related chamber dilatation, as the RV is an anterior cardiac structure occupying little of any heart border.[44] Radionuclide angiography was previously considered the gold standard (next to autopsy) for detection of haemodynamically significant RV dysfunction,[4] but has now been superseded by cardiac MRI (CMR). Electrocardiography and echocardiography remain the most readily available and simplest of these techniques in the acute setting.

Clinical and haemodynamic findings

Clinically, the triad of hypotension, elevated jugular venous pressure (JVP), and clear lung fields is recognised as a marker of RVMI in patients with acute inferoposterior wall infarction, with high specificity (96%) but low sensitivity (25%).[45] Similarly, the haemodynamic correlate of RA pressure (RAP) ≥10 mm Hg with an RAP: pulmonary capillary wedge pressure (PCWP) ratio of ≥0.86 is highly specific (97%) for RV necrosis determined on postmortem examination, and persists after diuresis or use of inodilators.[34]

Pulsus paradoxus and Kussmaul's sign may also occur with RV ischaemia.[46] The combination of elevated JVP and Kussmaul's sign in a patient with acute inferior wall MI is highly specific and sensitive for RV ischaemia.[45] Auscultation may reveal a right-sided S3 and S4 gallop.[46] If the atrial perfusion is not compromised, the a-wave and x descend waveforms of the JVP are enhanced but y descent is blunted due to pandiastolic RV dysfunction, giving rise to a 'W' pattern waveform.[47–49] In patients with associated RA infarction, the RA and central venous pressures are higher but with depressed a-wave, and x and y descent, forming an 'M' pattern.[48] Dilatation of the RV may lead to functional tricuspid regurgitation such that the RA pressure tracing reveals a systolic wave that precedes and may fuse with the venous filling wave.[50] Severe tricuspid

regurgitation may occur in cases of ischaemic papillary muscle dysfunction or rupture.[51] In these cases the RA waveform progressively approximates the RV waveform.

Occasionally, a ventricular septal defect may accompany RV infarction, causing a holosystolic murmur and often leading to severe acute haemodynamic compromise.[52] The left-to-right shunt reduces effective forward LV output and further overloads the dysfunctional RV leading to hypotension and precipitating pulmonary oedema. Surgical repair or percutaneous device closure is imperative, albeit high risk.[53] Elevated right heart pressures due to ischaemia may also stretch open a patent foramen ovale or cause a right-to-left shunt via an atrial septal defect, clinically evident as oxygen resistant systemic hypoxaemia or paradoxic emboli.[54–56]

Electrocardiography

The precordial leads of the classic 12 lead ECG provide a wealth of information on the LV, but yield limited information on the electrical activity of the right heart. Only lead V1 and possibly V2 may provide a partial view of the RV free wall as shown in figure 1. 'Right precordial' leads are obtained by placing the precordial electrodes over the right chest in positions mirroring their usual arrangement (figure 1). The presence of acute ST segment elevation, Q waves or both in the right precordial leads (V3R to V6R), is highly reliable in the diagnosis of RVMI.[57–59] ST segment elevation ≥0.1 mV in the right precordial leads, especially V4R, is observed in 60–90% of patients with acute RVMI.[58 60–64] It correlates with reduced RV ejection fraction and is strongly associated with major complications and in-hospital mortality.[43 65 66] Nonetheless, right precordial ST segment elevation is a transient event that may be absent in up to half of patients with RV infarction 10–12 h after the onset of pain,[64 67] and 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.[68]

ST elevation from the RV free wall may also be detected by ST elevation in lead III being more than that in lead II or by reciprocal ST depression in leads I and aVL >2 mm in total (figure 2).[69 70] The positive predictive value of both these findings is about 70–80%.[71 72] Similarly, reciprocal ST depression in the lateral leads is a sensitive predictor of RV ST elevation, though specificity is understandably low.[73]

In inferior infarction involving the LV posterior wall in addition to the RV, right precordial ST elevation or R wave loss have low sensitivity and only moderate specificity. The opposing vectors generated by the thicker LV posterior wall dominate, creating prominent R waves and reciprocally depressed ST segments on the right precordial leads. The individual contribution by each of these vectors may be resolved by comparing ST elevation in aVF with ST depression in V2, which is roughly orthogonal to the standard limb plane and reflects posterior wall contribution. More specifically, elevation in aVF exceeding the depression in V2 is suggestive of additional RV involvement. Similarly a ratio of <0.5 between ST depression in lead V3 and ST elevation in lead III has approximately 90% sensitivity and specificity in diagnosing infarction related to occlusion of the proximal RCA.[74]

It has been suggested that slurring of the 'r' wave in either right precordial leads or lead aVR is both sensitive and specific for RV involvement in cases of inferoposterior infarction (70% and 94%, accordingly[75]). This is thought to arise by focal conduction block and depolarisation heterogeneity in the RV free wall caused by RV ischaemia. This conduction block can also lead to the development of isolated islands of viable myocytes, delayed depolarisation of which can lead to the post-excitation ECG phenomenon called 'epsilon wave', more commonly recognised as a major diagnostic criterion of arrhythmogenic RV dysplasia.[76]

Isolated ST segment elevation in V1 to V3 (and possibly V4), with decreasing levels of elevation from V1 onwards and without Q wave formation, has also been reported in a few cases of RV infarction.[77–80] This seems unrelated to septal damage, but is rather a correlate to the rare entity of isolated RV infarction. In the clinical setting, isolated RV infarction has been reported after acute occlusion of the RV branches after angioplasty,[81–84] occlusion of a rudimentary or non-dominant RCA,[80] and good left coronary supply to the inferoposterior LV[85 86] or if the inferior wall has already previously been infarcted.[82 87 88] In patients with RV infarction, in contrast to basal interventricular septal infarction, ST segment elevation in V4R is greater than that in V1 to V3.[78] The relatively small representation of this isolated RVMI ECG entity in the literature may be partly due to misdiagnosis as anteroseptal infarction, and stresses the importance of performing right precordial ECG recordings in patients with isolated V1 to V3 ST elevation.

Echocardiography

Two dimensional echocardiography provides an assessment of RV function, wall motion abnormalities, valve lesions and LV

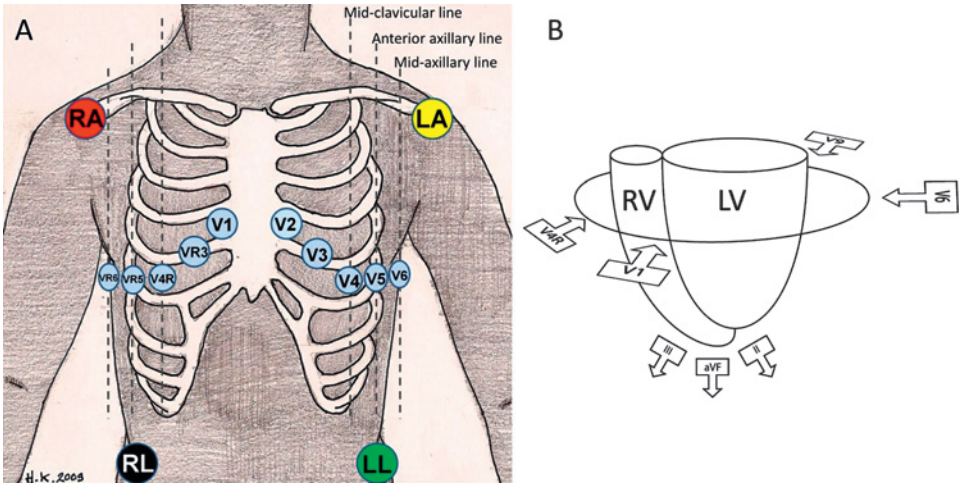
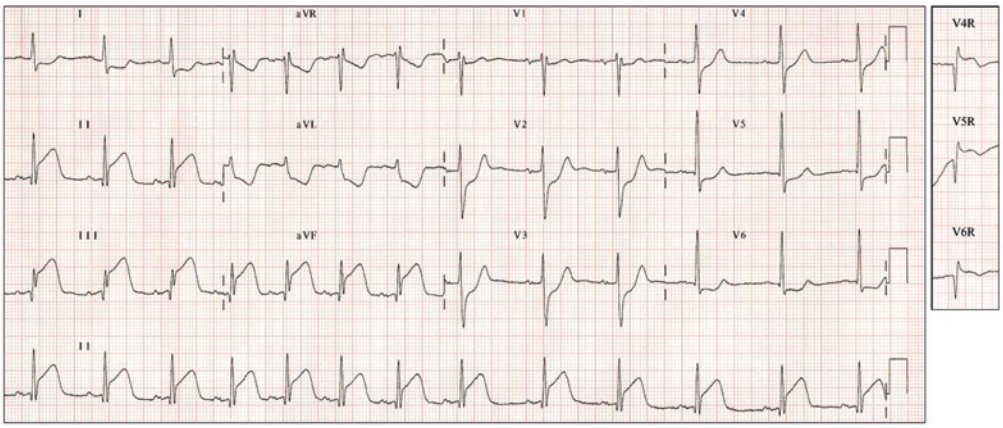


Figure 1 (A) Placement of right precordial leads in a mirror arrangement to the left precordial leads, and (B) simplified schematic demonstrating the relation of the leads to the ventricles. Note that, of the common left precordial leads, V1 is best placed to view any right ventricular free wall injury currents. LV, left ventricle; RV, right ventricle.



ST depression I + aVL > 2mm	I + aVL >2mm	ST elevation in aVF > ST depression in lead V2	aVF > V2
ST elevation ≥1mm in V4R	≥1mm V4R	ST depression V3 < ½ ST elevation lead III	V3 < ½ III
ST elevation lead III > elevation at lead II	III > II	QRS prolongation in R precordial leads	V4R > V6
Reciprocal depression at lateral leads	V6	Epsilon waves in R precordial leads	V4R

Figure 2 Summary of ECG features of right ventricular myocardial infarction (MI) complicating inferior MI. Coronary angiography confirmed proximal occlusion of the right coronary artery with minor left anterior descending artery disease.

function.[27] Nonetheless, heavy RV trabeculations make endocardiac surface definition difficult and volume calculations are hindered by the complex geometry of the RV, while echocardiographic imaging windows can be limited by the retrosternal position of the chamber.

Several different echocardiographic features have been used as surrogates of global RV function. Assessment of RV free wall for hypokinesia or akinesia, performed qualitatively by operator real-time evaluation, is a sensitive assessment for detecting RV dysfunction.[89 90] Nonetheless, this visual assessment leads to underestimation of dysfunction due to a visually misleading asymmetric contraction of the RV walls toward its centre, and is best combined with demonstration of RV dilatation in order to define RVMI accurately.[91] A semiquantitative method based on RV wall motion score indexing (assigning points to parts of the wall) has been found to correlate well with radionuclide derived RVEF.[92]

Additional features of RV involvement include paradoxical septal motion due to increased RV end diastolic pressure,[91 93] tricuspid regurgitation which can be assessed by Doppler echocardiography, and severe RA enlargement.[94] The increased RA pressure may cause deviation of the interatrial septum with convexity toward the left atrium in up to 80% of RVMI patients.[93] Doppler echo may also detect flow across a patent foramen ovale opened by the increased right heart pressures as well as an acute ventricular septal defect.

M mode measurements of the systolic displacement of the lateral portion of the tricuspid annular plane (TAPSE: tricuspid annular plane systolic excursion) have been advocated as a measure of RV base-to-apex shortening during systole and a correlate of RV ejection fraction, with the rationale that, in contrast to the LV, the RV contraction involves predominantly longitudinal shortening. TAPSE has been shown to be highly reproducible and of prognostic value in patients with congestive cardiac failure.[95 96] TAPSE as an estimate of RVEF has been shown to correlate well with RVEF measured by first pass ventriculography and CMR.[95 97 98] There are limited data on the application of TAPSE in the early diagnosis and assessment of RVMI. In a study by Kidawa *et al*, patients with RCA occlusion on angiography who presented >2 h after onset of symptoms had significantly reduced TAPSE and worse long term prognosis compared with patients who presented earlier than 2 h.[99] In a recent study by Engstrom *et al* of patients with ST elevation MI (STEMI) complicated by cardiogenic shock, a low TAPSE (≤14 mm) was an independent predictor of long term mortality.[12] It is noteworthy, however, that TAPSE is not only affected by RV systolic function but may also be reduced by impaired LV systolic function.[100]

Echocardiography: tissue Doppler imaging

Tissue Doppler imaging (TDI) can provide information on myocardial wall motion during the cardiac cycle. Annular velocities towards the apex reflect the contraction and relaxation of longitudinal myocardial fibres in both ventricles. As most RV muscle fibres run in an oblique or longitudinal direction,[101] this is a good measure of global RV function.[97 102–104]

TDI has been used to assess RV function in patients with RVMI complicating inferior infarction. Significantly reduced systolic lateral tricuspid velocities are found in patients with concomitant RV infarction.[94 105–107] The TDI derived RV systolic strain also correlates with RVEF, is lower in patients with RVMI, and has prognostic value in AMI patients.[108–110]

Lateral tricuspid annulus TDI can also detect ischaemic RV diastolic dysfunction.[111] In particular, the early diastolic annular velocities (Em) show a significant and steady decrease with progressive diastolic dysfunction, which appears unaffected by preload state.[107 112] Reduced early diastolic tricuspid annular velocities have been demonstrated in patients with RV infarction complicating inferior MI.[94 105–107]

Echocardiography: myocardial perfusion index

The concept of myocardial perfusion index (MPI), initially described by Tei in 1995, has also been applied to assess RV function.[113 114] This simple index of combined systolic and diastolic myocardial performance is defined as the sum of the isovolumetric contraction and relaxation periods divided by ejection time which can be derived from pulse wave Doppler interrogation of the tricuspid inflow and RV outflow or from the TDI signal of the tricuspid annulus.[107 115]

As MPI encompasses the energy dependent processes of RV relaxation, contraction and ejection, it is well poised to be a sensitive marker of RV ischaemia. RV MPI is higher in patients with inferior acute MI and RV involvement than in patients with no RV involvement,[93 115] such that a TDI derived MPI of >0.70 is both highly sensitive and specific in detecting RV ischaemia in the setting of an acute inferior MI.[107] Notably, RV MPI may be increased by left heart disease, cor pulmonale, pulmonary valve stenosis, and thromboembolism which need to be excluded. We have recently demonstrated that combining the peak lateral tricuspid annulus systolic velocity (S') data with MPI, as the novel S'/MPI index, yields good sensitivity and specificity in detecting RVMI both in the acute and late phase of RVMI.[116]

Echocardiography: three dimensional

Three dimensional echocardiography (3DE) has been available for some years, from 3D reconstruction of 2D images and more recently with real-time 3D echo imaging, and can provide volumetric images of the RV in most patients. Although early results suggested poor correlation with CMR volumes,[98] there have since been significant improvements in hardware and modelling software.[117] In a recent study by Leibundgut *et al*, although the 3DE derived volumes were lower than CMR, the two modalities correlated very well.[118] Nonetheless, despite these improvements, the technique applicability remains limited in dyspnoeic patients and irregular cardiac cycles whereas new hardware as well as expertise in the semi-automated post-processing is required. Currently, the utility of 3DE in the acute setting of RVMI remains unknown.

Radionuclide techniques

Radionuclide angiography used to be the gold standard for the assessment of RV end diastolic and end systolic volumes and calculation of RV ejection fraction, as assessment of radionuclide count density is not geometry dependent.[119] Segmental RV wall motion abnormalities in association with a reduced RVEF (to <40%) on first pass ventriculography are highly sensitive and specific for RVMI or RV ischaemia.[89] These radionuclide techniques have now been superseded by CMR which is accurate and does not require radiation exposure.

CMR

CMR is a volumetric technique based on visualisation of the anatomy of the RV that can directly evaluate RV size, mass, morphology, and function in an accurate and reproducible manner.[44 120] It can additionally detect other right sided myocardial disease such as arrhythmogenic RV dysplasia, complex congenital disease, pericardial disease, as well as mediastinal pathology. Manual or semi-automated volume measurements by CMR show high reproducibility and very good agreement between calculated LV and RV stroke volumes.[119] CMR is now considered the gold standard for non-invasive assessment of RV function, particularly as it provides additional information on RV anatomy and myocardial mass.

Gadolinium late hyperenhancement of RVFW on CMR has been demonstrated in cases of RVMI[121–123] and may persist at least 6 months after the ischaemic event.[124] Advances in technology including shorter acquisition times and more widespread availability of equipment and expertise is likely to lead to the increased utilisation of this modality.

CT

CT has been used in the assessment of RV function in the setting of pulmonary embolism, with better sensitivity and specificity than echocardiography when compared with a 40% pulmonary occlusion standard.[125] Delayed enhancement multislice cardiac CT (MSCT) has been shown to have good agreement with delayed enhancement cardiac MRI and postmortem evaluation in determining myocardial infarct size at 3–7 days in an animal model.[126] Despite high spatial resolution, soft tissue contrast is poor and radiation exposure remains significant, rendering its utility in the diagnosis of RVMI very limited above the previously discussed imaging techniques. Notably, however, MSCT angiography can provide increasingly higher quality coronary images which can be helpful, particularly for congenital malformations such as anomalous RCA origin.[127]

OUTCOME OF RVMI

Successful intervention by primary PCI in patients with RVMI has been shown to normalise the RV ejection fraction and is associated with improved in-hospital mortality compared with patients in whom angioplasty is unsuccessful.[28 128] Reperfusion within 1 h of occlusion leads to immediate recovery of RVFW function and consequent improved LV filling and performance.[20] Delayed reperfusion after 4–8 h is associated with a higher degree of RV dysfunction and complications, but still results in significant, yet slower, recovery of RV function.[129 130] Failure to reperfuse the RCA, and in particular the RV branches, leads to lack of recovery in the acute setting and consequent haemodynamic compromise, which is associated with high in-hospital mortality. Similarly, thrombolysis leads to functional RV recovery and imparts a survival benefit in patients sustaining an inferior MI with RV involvement, but only as long as it is successful in achieving RCA patency.[131 132] This is an important caveat as the proximal RCA lesion, which frequently presents with high clot burden in association with reduced coronary delivery of fibrinolytic agent due to associated hypotension, leads to a higher incidence of primary thrombolytic failure as well as a higher incidence of reocclusion.[131] A study of unselected patients who presented with acute MI, and received thrombolysis with tissue plasminogen activator, found the infarct related artery more likely to be occluded in patients with RVMI than in those without. In view of the above, thrombolysis is not an appropriate treatment modality in this group of patients if primary angioplasty is available.

In the SHOCK (Should we emergently revascularize Occluded coronaries for Cardiogenic shock) trial registry, the cardiac index was depressed to a similar extent in patients with RV shock as in patients with LV shock, albeit with higher RA pressures and lower pulmonary artery pressures for a similarly elevated LV filling pressure.[133] Furthermore, mortality of cardiogenic shock associated with RVMI was equivalent to mortality of shock due to LV infarction (55% and 60% in-hospital mortality, respectively) despite the patients' younger age, lower rate of anterior MI, and higher prevalence of single vessel coronary disease.[134]

Even though significant haemodynamic compromise and arrhythmias associated with RV ischaemia lead to increased early mortality,[10 11 43] in the longer term most patients with acute RV ischaemia improve spontaneously even in the absence of reperfusion of the infarct related artery.[89 135] Clinical improvement occurs within 3–10 days whereas RV ejection fraction returns to near normal within 3–12 months.[89 132 135 136] Recovery is thought to be due the favourable supply–demand characteristics of the RV and is, predictably, less pronounced in the presence of RV hypertrophy.[29 30] The employment of right sided chest leads during stress testing may, however, reveal latent post-infarction RV ischaemia.[137 138]

FURTHER MANAGEMENT IMPLICATIONS OF RVMI

The accurate diagnosis of RVMI is vital as both the treatment and the prognosis of RV infarction substantially differ from that of LV infarction (see box 1). Early work on experimentally induced RV ischaemia found that volume loading led to an increment in RV filling pressures and increased systolic arterial pressure and cardiac output.[90] Furthermore, treatments commonly employed in predominantly LV infarction, such as the use of diuretics, intravenous nitrates, ACE inhibitors or even opiates, may reduce RV preload and cause catastrophic haemodynamic compromise. In the clinical setting, the beneficial effect of fluid loading is not universal but is dependent on the degree of RV afterload and baseline volume status of the patient. Excessive volume loading can increase wall tension, decrease contractility, and impair LV filling through interventricular dependence mechanisms discussed earlier, and lead to reduced systemic cardiac output.[139] Nonetheless, in the absence of pulmonary oedema or evidence of notably increased right sided preload, a trial of volume loading is appropriate.[20]

Should cardiac output fail to improve after fluid loading, the common inotrope of choice is dobutamine.[140 141] Dobutamine may enhance RV performance through its inotropic effect, reduce pulmonary vascular resistance and hence RV afterload, and improve AV conduction. Its utility is limited by arrhythmias, systemic vasodilation, and hypotensive response. The 'inodilator' milrinone may reduce preload and exacerbate hypotension, but also reduces RV afterload by lowering pulmonary resistance. In severely hypotensive patients the addition of a pressor (such as dopamine) to aid maintenance of coronary perfusion pressure may be beneficial.

Levosimendan, a calcium sensitiser inotrope, appears to improve RV contractility in patients with chronic LV failure without detriment to diastolic function or an apparent increase in myocardial oxygen demand.[142–144] Levosimendan may also reduce RV afterload by activation of ATP sensitive potassium channels in the pulmonary vasculature, leading to dilatation, while reducing LV afterload and improving coronary perfusion by a similar mechanism on systemic and coronary vessels.[145 146]

In an experimental animal model of acute RVMI, levosimendan improved global haemodynamics, increased RV contractility, and mildly reduced RV afterload.[147] In a recent study of 56 patients with cardiogenic shock secondary to MI, an improvement in RV function and a reduction in pulmonary vascular resistance was noted with levosimendan and persisted after the end of the infusion.[148] Although no specific RVMI criteria were applied in the study, patients with inferior MI had lower RV function indices and showed significant improvement in both right and left cardiac function in response to levosimendan. Interestingly, in an animal model of RV ischaemia/reperfusion injury, both levosimendan and milrinone appeared to have additionally cardioprotective properties.[149]

Mechanical support with intra-aortic balloon pumping may increase coronary perfusion pressure, enhance coronary fibrinolytic delivery, and improve LV performance in hypotensive patients with RVMI and depressed LV function, thus reducing RV afterload. Percutaneous cardiopulmonary support and, more recently, assist devices have also been successfully used to provide support for patients with refractory RV failure secondary to RVMI.[150–153]

Main messages

- Right ventricular (RV) ischaemia occurs in up to half of inferior myocardial infarctions (MIs).
- RVMI portends increased mortality and morbidity to acute infarction.
- Prompt revascularisation improves outcome—primary percutaneous coronary intervention (PCI) is superior to thrombolysis.
- Diagnosis of RV involvement can be challenging. Electrocardiography and echocardiography are the methods of choice in clinical practice during the acute setting assessment.
- Identification of RV involvement prompts specific management considerations (see box 1).
- The RV tends to recover to near normality in the long term.

Box 1 Right ventricular (RV) myocardial infarction management: summary

- Prompt diagnosis
  - Clinical signs, ECG (figure 2)—record right precordial leads in all patients with inferior ST elevation or isolated V1–V3 ST elevation, echocardiographic imaging
- Reperfusion therapy
  - Primary percutaneous coronary intervention preferable to thrombolysis
- Optimise RV preload
  - Avoid : morphine, diuretics, β-blockers, nitrates
  - Trial of judicious fluid administration in the absence of pulmonary oedema
- Reduce RV afterload
  - Inotropes, pulmonary vasodilators (nitric oxide, prostacycline), intra-aortic balloon pump
- Maintain chronotropic competence and atrioventricular synchrony
  - Avoid: β-blockers in patients with proximal right coronary artery occlusion
  - Consider dual-chamber temporary pacing
- If above fails consider
  - RV assist device, emergency percutaneous cardiopulmonary support

Current research questions

- Can the prompt identification of RV infarction lead to more specific treatment that can further impact on prognosis? Does aggressive primary PCI with revascularisation of the branches to the RV reduce the risk of complications?
- What is the utility of 3D echocardiography in the assessment of RV function in the acute setting of MI?
- Further trials are necessary to confirm the utility of TDI echocardiography in the identification of previous RVMI.
- What is the prognostic significance of an old RVMI (eg, as detected by late enhancement on CMR)?

The acutely ischaemic RV and consequently preload deprived LV have a relatively fixed stroke volume, and output is therefore strongly heart rate dependent.[37] Maintenance of chronotropic competence, which is frequently compromised by mechanisms discussed previously, is therefore of great importance. Atropine may restore physiological rhythm, but ventricular pacing may be required. AV asynchrony leads to loss the of the atrial transport contribution to RV filling and may precipitate tricuspid regurgitation further impairing effective forward RV output. Some of these patients may, therefore, require sequential dual chamber temporary AV pacing.[47 154] Unfortunately, transvenous pacing may prove problematic due to poor ventricular sensing from the ischaemic RV as well as difficult lead positioning in the ischaemic and often dilated RA.[20] In these cases rapid administration of intravenous aminophylline has been reported to restore sinus rhythm in patients unresponsive to atropine, presumably by reversing the negative chronotropic effects of ischaemia induced adenosine production.[155–157] The salutary effects of reperfusion therapy and the particular importance of flow restoration to the major RV branches have been discussed earlier.

CONCLUSION

The pathophysiology of the RV makes it resistant to infarction, but acute ischaemia can lead to severe haemodynamic consequences. RVMI worsens the short term prognosis of inferior MI, but has an excellent long term prognosis in the absence of associated severe LV dysfunction.

The diagnosis of RVMI can be challenging; the 12 lead ECG with supplemental right precordial recordings remains the principal diagnostic tool in the acute setting, but the findings may be transient. The previously described echocardiographic techniques, particularly the application of TDI, are most useful in the acute and later phase of assessment, whereas the utility of 3DE is unknown. In the subacute setting, modern cardiac MRI techniques may also be used to detect RV free wall scarring and have become the gold standard for assessment of RV ejection function, superseding nuclear techniques.

Identification of RVMI is important to guide initial management of fluid, inotropic, and chronotropic support with rapid reperfusion therapy, and to raise awareness of potential complications.

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SELF ASSESSMENT QUESTIONS (TRUE/FALSE; ANSWERS AFTER THE REFERENCES)

1. Patients with RVMI and high central venous pressures (elevated JVP) should be treated with furosemide even in the absence of pulmonary oedema to reduce pressure load on the ischaemic RV.
2. ST elevation ≥0.1 mV in the right precordial lead V4R can be used to diagnose concomitant RV infarction in patients with inferior MI.
3. Cardiogenic shock due to RV infarction has a better prognosis than shock due to LV infarction.
4. In patients presenting with ST elevation MI, CMR is the gold standard for the diagnosis of RV infarction.
5. Echocardiographic techniques such as TAPSE and systolic tricuspid annular velocities by TDI only assess longitudinal RV shortening and do not take into account circumferential RV function.

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ANSWERS

1. False. On the contrary, the elevated central venous pressures have the salutary effect of providing increased RV preload that helps maintain output. Treatment with furosemide or other venodilators can lead to catastrophic decompensation and haemodynamic collapse.
2. True. ST elevation in the right precordial leads and particularly V4R is a sensitive (albeit transient) diagnostic ECG finding in patients with RVMI complicating inferior STEMI.
3. False. Although inferior infarction, which RVMI usually complicates, overall has a better prognosis than anterior LV infarction, cardiogenic shock due to RVMI has a prognosis that is as poor as that due to LV infarction.
4. False. Although CMR has superseded radionuclide techniques as the gold standard for RV volume assessment, ECG and echocardiography remain best suited for the acute assessment of patients presenting with AMI.
5. True. Although this is true, both of these measures correlate very well with global RV systolic function as most RV free wall muscle fibres run in an oblique or longitudinal direction. Additionally, assessment of diastolic velocities from TDI can provide information on RV diastolic function.

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