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Review

Right Ventricular Infarction—Diagnosis and Treatment

SHOWKAT A. HAJI, M.D., AND ASSAD MOVAHED, M.D., FACP, FACC

Section of Cardiology, Department of Medicine, East Carolina University School of Medicine, Greenville, North Carolina, USA

Summary: Right ventricular infarction (RVI) as assessed by various diagnostic methods accompanies inferior-posterior wall myocardial infarction (MI) in 30 to 50% of patients. Recognition of the syndrome of RVI is important as it defines a significant clinical entity, which is associated with considerable immediate morbidity and mortality and has a well-delineated set of priorities for its management. Patients may clinically present with hypotension, elevated jugular venous pulse (JVP), and occasionally shock, all in the presence of clear lung fields. The ST-segment elevation of ≥ 0.1mV in the right precordial leads V_4R is a readily available electrocardiographic sign used for diagnosis of RVI. Other diagnostic approaches for assessing RVI include echocardiography, radionuclide ventriculography, technetium pyrophosphate scanning, and hemodynamic measurements. The proper management of RVI includes volume loading to maintain adequate right ventricular preload, ionotropic support, and maintenance of atrioventricular synchrony. Reperfusion therapy should be initiated at the earliest signs of right ventricular dysfunction. Finally, complete recovery over a period of weeks to months is a rule in a majority of patients, suggesting right ventricular "stunning" rather than irreversible necrosis has occurred.

Key words: right ventricle, myocardial infarction, diagnosis, electrocardiogram, hemodynamics, treatment, reperfusion

Introduction

Although right ventricular infarction (RVI) was described earlier in the autopsy studies,[1–3] Cohn *et al*.[4] in 1974 gave the initial description of the clinical syndrome of right ventricular failure in patients of RVI. Since then RVI has been recognized more frequently and continues to be a diagnostic and therapeutic challenge. Acute myocardial infarction (MI) involving only the right ventricle is a rare event.[1,5–7] Right ventricular involvement in the setting of an acute inferior wall MI is much more common. Recognition of the syndrome of RVI is important as it identifies a significant clinical entity, which is associated with considerable immediate morbidity and mortality[8–11] and has a well-delineated set of priorities for its management. Its presence defines a high-risk subgroup of patients with acute inferior left ventricular infarction who should be considered high-priority candidates for reperfusion. Ischemia or infarction of the right ventricle results in decreased right ventricular compliance, reduced filling, and decreased right ventricular stroke volume. In turn, these changes lead to diminished left ventricular filling and drop in cardiac output that could result in systemic hypotension and shock. Frequent accompaniments may include atrial infarction, sinus bradycardia, atrial fibrillation, and atrioventricular block. Hence the presence of RVI should raise a clinical alert for its potential immediate life-threatening consequences.

Acute occlusion of the right coronary artery, proximal to the right ventricular branches, results in right ventricular dysfunction.[12–16] However, many right coronary artery occlusions do not result in significant right ventricular necrosis.[11,17,18] This may be due to lesser right ventricular myocardial oxygen demands (the right ventricle has much smaller muscle mass than the left ventricle); coronary perfusion of the right ventricle occurs both in systole and diastole; and the presence of more extensive collateral vessels from left to right. Right ventricular hypertrophy may predispose a patient to RVI when coronary artery disease is present.[13,19–21]

The incidence of RVI is variable depending on the criteria used for detection.[1,7,13,22] Autopsy studies[1,13] suggest that right ventricular infarction accompanies fatal inferior left ventricular infarction in 24 to 34 percent of cases. Noninvasive studies[22–25] suggest that RVI occurs in more than 30 percent of patients with acute inferior-posterior left ventricular MI. Anatomic evidence of RVI is more common than expected hemodynamic pattern.[15,16,22,23]

A spectrum of right ventricular systolic and diastolic dysfunction in the setting of inferior wall MI has been recognized. Although there are potentially life-threatening acute hemodynamic and clinical consequences in some, most patients with

Address for reprints:

Assad Movahed, M.D.
Section of Cardiology, Department of Medicine
East Carolina University School of Medicine
Greenville, NC 27858, USA

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right ventricular dysfunction after MI have spontaneous recovery of right ventricular function, leading some clinicians[26] to believe that the term "right ventricular infarction" is a misnomer and represents viable but "stunned" myocardium. The following review gives an insight into the various diagnostic modalities and treatment of RVI.

Clinical Presentation

Clinical recognition of acute RVI is extremely important, as appropriate therapy for hypotension and shock must be started prior to consideration of noninvasive tests or invasive monitoring. Right ventricular infarction should be suspected in any patient with acute inferior wall MI. Even in patients who do not present with hypotension, the potential of RVI should be recognized to avoid therapy that will further lower right heart preload. Ischemia or infarction of the right ventricle results in decreased right ventricular compliance, reduced filling, and decreased right ventricular stroke volume. These changes lead to diminished left ventricular filling and drop in cardiac output. In addition, acute right ventricular dilatation causes a leftward shift of interventricular septum, increasing left ventricular end-diastolic pressure with a decrease of left ventricular compliance and cardiac output.[8, 27–29] These changes in left ventricular compliance are further aggravated by increased intrapericardial pressure[27, 30–32] as a result of right ventricular dilatation. In a recent study, Brookes *et al*.[33] demonstrated that the geometric changes in the left ventricle, caused by right ventricular dilatation due to RVI, resulted in a significant impairment of left ventricular contractile function in addition to the diastolic filling abnormalities and changes in compliance. Therefore, although the patient has clinical signs of increased right-sided pressure, the left ventricular filling and systolic function may be below normal. The symptoms of RVI may be more pronounced in the presence of combined right atrial infarction with associated rate and rhythm disturbances.[16] When the culprit coronary artery lesion is distal to the right atrial branches, augmented right atrial contractility enhances right ventricular performance and offsets some of the hemodynamic consequences of RVI.

The triad of hypotension, elevated jugular venous pressure (JVP), and clear lung fields has been recognized as marker of RVI in acute inferior-posterior wall MI.[4, 34, 35] Pulsus paradoxus (decrease in size, or even momentary disappearance of the pulse during inspiration) and Kussmaul's sign[36](an inspiratory increase in JVP) have also been reported in patients with RVI. The presence of elevated JVP and Kussmaul's sign in the setting of an acute inferior wall infarction indicate a hemodynamically significant RVI[35] (sensitivity = 88% and specificity = 100%), particularly when it is associated with significant damage to the left ventricle and/or interventricular septum. Elevated JVP alone was found to be more sensitive (88%) but less specific (69%) in the same study in detecting a hemodynamically significant RVI. Therefore, a careful bedside examination of the jugular venous pulse serves as an important diagnostic tool in determining the severity of RVI and raising the clinical alert of its acute hemodynamic consequences and the caution for the judicious use of drugs like morphine and nitrates. Patients with intact right atrial perfusion manifest augmented atrial contraction resulting in enhanced A wave and X descent, but diminished Y descent in the jugular venous pulsations. In contrast, patients with depressed right atrial function have higher right atrial and systemic venous pressures, but depressed A wave, X descent, and Y descent.[16] Finding of diminished A wave in patients with hemodynamically important RVI, signifying right atrial infarction, has been proven to be a bad prognostic indicator even in the presence of preserved left ventricular function.[16]

Auscultation may reveal a right-sided S_3 and S_4.[36] Tricuspid regurgitation may be identified because of dilatation of right ventricular chamber, which may be severe when related to papillary muscle dysfunction.[37,38] Right ventricular infarction occasionally may be accompanied by ventricular septal defect,[39,40] identified by a systolic murmur that increases with inspiration, and is accompanied by profound hemodynamic compromise. It is important to recognize that findings of RVI may be masked by global left ventricular systolic dysfunction with hypotension and pulmonary congestion. Finally, high-grade atrioventricular blocks may occur and result in loss of atrial ventricular synchrony with exacerbation of hypotension and shock.[41]

Noninvasive Diagnosis of Right Ventricular Infarction

Since right ventricular function can improve rapidly, diagnostic tests should be performed soon after presentation. Various noninvasive methods have been used for diagnosis of right ventricular infarction, including chest x-ray, electrocardiogram, two-dimensional echocardiogram, radionuclide ventriculogram, and technetium-99m pyrophosphate myocardial scintigram.

Chest Roentgenogram

The chest x-ray is not particularly helpful in the diagnosis of RVI. The suggested radiographic findings of right atrial and ventricular enlargement have a very low sensitivity and specificity for right ventricular infarction.[42]

Electrocardiogram

Electrocardiography is recognized as the most simple and readily available diagnostic tool for identification of RVI. Therefore, an increasing number of electrocardiographic criteria for RVI[24, 43–53] have been studied (Table I) with variable sensitivity and specificity.[24, 48] The right ventricular involvement can be diagnosed with a predictive accuracy well above 80% by the presence of ST-segment elevation of ≥ 1 mm in the right-sided precordial lead, V_4R, in the presence of an acute inferior or inferioposterior MI.[24,25,43–47,54] The ST-segment elevation in V_4R is a strong independent predictor of major complications and in-hospital mortality.[48, 55–58] Zehender e*t al*.,[48]

TABLE I Electrocardiographic criteria for diagnosing right ventricular infarction

Electrocardiographic criteria	No. of patients	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Reference Nos.
ST elevation					
$V_3R > 0.05$ mV	43	79	68	—	47
$V_3R > 0.1$ mV	110	59	90	89	47,53
$V_4R > 0.05$ mV	181	81	76	75	44,46,47
$V_4R > 0.1$ mV	255	80	88	87	24,43,45,46,48
$V_5R > 0.1$ mV	110	72	88	86	24,49
$V_6R > 0.1$ mV	110	67	90	88	24,49
$V_4R–V_6R > 0.1$mV	33	90	91	82	50
Q wave					
V_3R	43	37	87	83	49
Q /QS V_4R	71	85	59	—	46,47
QS $V_3R–V_4R$	28	78	100	94	46
ST elevation					
$V_1 > 0.05$ mV	115	38	40	27	47,51
$V_1 > 0.1$mV	183	28	65	47	24,50,51
$V_2 > 0.05$ mV	115	33	18	20	47,51
$V_2 > 0.1$mV	105	20	38	19	45,50
ST elevation V_2 / ST depression aVF < 50%	68	79	91	82	52
ST elevation >0.1mV:					
In II+ III/II >1	24	63	88	91	53
In III+ III/II >1	24	65	57	79	53

in a prospective study of 200 patients, demonstrated that the ST-segment elevation in lead V_4R identified patients with increased hospital mortality and major complications, including cardiogenic shock, ventricular fibrillation, and third-degree atrioventricular block. It is important to recognize that ST-segment elevation in right precordial leads is transient[24,44] and may be absent in one half of patients with RVI after 12 h of onset of chest pain. Furthermore, conditions other than RVI may produce ST-segment elevation in lead V_4R, including acute pulmonary embolism, left ventricular hypertrophy, acute anterior septal myocardial infarction, pericarditis, and previous anterior myocardial infarction with aneurysm formation.

There are reports of RVI presenting as ST-segment elevation in leads $V_1–V_5$ mimicking anterior wall MI.[59–63] It is of note that the distribution of ST-segment elevation in the anterior leads is greater in leads $V_1–V_2$ and decreased toward $V_5–V_6$ in RVI compared with anterior wall MI.[59] The two infarcts can be separated when vector concepts are used.[64] As explained in a recent article by Hurst, in the case of RVI the ST-segment vector is directed anteriorly and is more than +90° to the right (producing a downward displacement of the ST-segment in lead I), while in the case of anteroseptal left ventricular infarction the vector is also anterior, but is usually located from −30° to −90° to the left in the frontal plane (producing an elevation of the ST segment in lead I).[64]

Right bundle-branch block and complete atrioventricular block are the most frequent conduction abnormalities associated with RVI.[54] Disturbances of rhythm such as atrial fibrillation[65] and sinus bradycardia[16,66,67] may be present.

Thus electrocardiogram serves as a very important tool for the diagnosis of RVI, and it is imperative to record right-sided precordial leads in all patients with inferior wall MI as soon as possible.

Echocardiogram

As in other fields of cardiology, echocardiography has expanded its implications in the diagnosis of RVI. Abnormal findings include right ventricular dilatation, right ventricular wall akinesis or dyskinesis, reversed septal curvature caused by the reversal of the transseptal pressure due to increased right ventricular end-diastolic pressure, and the presence of severe right atrial enlargement. The presence of interatrial septal bowing indicating a concomitant right atrial infarction is an important prognostic marker and is predictive of more hypotension, atrioventricular blocks, and higher mortality in RVI.[68] In a study on 53 patients with inferior wall MI, Dell'Italia *et al*.,[22] using right ventricular akinesis or dyskinesis as a marker for RVI, found that echocardiography was a highly sensitive technique for detecting hemodynamically significant RVI. However, it also detected wall motion abnormalities in patients who never developed hemodynamic evidence of RVI. This reduced specificity may be due to a superior sensitivity of echocardiography in detecting right ventricular ischemia or infarction that may or may not be associated with hemodynamic derangement. Lopez-Sendon *et al*.[69] obtained similar results.

The addition of Doppler echocardiography has increased the diagnostic utility of this technique by detecting complica-

tions of RVI such as tricuspid regurgitation, ventricular septal defect, and shunt flow across a patent foramen ovale. Doppler echocardiography can also detect premature opening of the pulmonic valve, indicating a noncompliant right ventricle.[70]

Radionuclide Angiogram

Radionuclide angiography is a useful technique for determination of right ventricular ejection fraction. Right ventricular dilatation or right ventricular ejection fraction of < 45% has been demonstrated in 40 to 50% of patients with inferior wall MI using both first pass and gated equilibrium technique.[71,72] However, because of a wide range of normal values for right ventricular ejection fraction, a reduction of right ventricular ejection fraction alone cannot be used as a specific marker of RVI. When low ejection fraction is combined with right ventricular wall motion abnormalities, together they had a sensitivity of 92% and a specificity of 82% in detecting hemodynamically important RVI.[22]

Technetium-99m Pyrophosphate Scintigram

In various studies using this technique, the incidence of RVI associated with acute inferior wall MI is variable, ranging from 20 to 40%.[73] Although highly specific, technetium-99m pyrophosphate scintigraphy is not very sensitive, especially for detecting hemodynamically important RVI. The sensitivity of this method to detect this clinically important presentation of RVI approaches 25%.[22] Also, application of this technique is limited in the acute setting because scans should be obtained 24 to 36 h after the onset of the infarct.

Hemodynamic Measurements

Invasive hemodynamic measurements provide a reliable information about the extent and severity of right heart involvement. The diagnosis of RVI can be confirmed by hemodynamic data when the right atrial pressure exceeds 10 mmHg and the ratio of right atrial pressure to pulmonary capillary wedge pressure exceeds 0.8 (normal value < 0.6).[22,23] Cohn *et al*.[4] observed an elevated right atrial pressure in excess of pulmonary artery wedge pressure in 6 (8%) of the 78 patients with acute myocardial infarction who underwent hemodynamic monitoring as part of their clinical management. Lorell *et al*.[34] described 12 (4%) of the 306 patients with inferior wall MI who had elevated right heart filling pressures compared with left heart filling pressures. Lopez-Sendon *et al*. reported that right atrial pressure > 10 mmHg and within 1–5 mmHg of pulmonary artery wedge pressure had a sensitivity of 73% and a specificity of 100% in identifying hemodynamically important RVI.[23]

A prominent Y descent in the right atrial waveform was considered a hallmark of RVI in earlier studies.[23,43,45,46] These studies did not correlate their observations with the extent of concomitant left ventricular infarction, and as these waveforms were related to electrocardiographic criteria the results were confounded by atrioventricular dyssynchrony. Goldstein *et al*.,[16] by timing right atrial waveforms to right ventricular mechanical events, demonstrated that the predominant descent was the X descent. Patients with intact right atrial perfusion manifest augmented atrial contraction resulting in enhanced A wave and X descent, but diminished Y descent, reflecting reduced right ventricular compliance and increased impedance to ventricular filling throughout diastole. In contrast, patients with depressed right atrial function have depressed A wave, X descent, and Y descent. Other workers also observed a diminished Y descent in RVI.[15,74,75] Patients who develop tricuspid incompetence have prominent V wave and a rapid Y descent. The right ventricular waveform shows a diminished peak systolic pressure that appears broad and sluggish. Elevated right ventricular end-diastolic pressures, right ventricular "dip and plateau," and equalization of diastolic filling pressure indicate right ventricular diastolic dysfunction. This equalization of pressure is partly due to elevated intrapericardial pressure caused by dilatation of the right ventricle.[27,30] The elevated intrapericardial pressure may be partly responsible for the low output syndrome of RVI.

Treatment

Earlier experiments on animal models suggested that the right ventricle functions as a passive conduit and its contraction was not important in the maintenance of circulation.[76–79] In 1974, Guiha *et al*.,[80] working on a canine model of RVI, demonstrated an increase in right heart filling pressures and a decrease in cardiac output in experimentally induced right ventricular damage, and that progressive volume loading produced an increment in right heart filling pressures, as well as an increase in systolic arterial pressure and cardiac output. This study provided the rationale behind volume loading in the treatment of RVI. In the absence of any other pathogenic mechanisms, volume loading, by restoring left ventricular filling pressures, should improve the cardiac output. However clinical studies failed to show uniform benefits with volume loading,[8,15,29,81] indicating that other mechanisms might be responsible for the hemodynamic alterations in RVI.

In 1983, Goldstein *et al*.[27] demonstrated that right ventricular infarction in dogs caused a fall in cardiac output which could be improved by volume loading and further augmented by pericardiotomy, highlighting the role of an intact pericardium in hemodynamic changes of RVI. It is thought that acute right ventricular dilatation as a result of RVI in a closed pericardial space is responsible for decreased left ventricular compliance due to pericardial restraining effects. In addition, acute right ventricular dilatation causes a leftward shift of interventricular septum, increasing left ventricular end-diastolic pressure with a decrease of left ventricular compliance and cardiac output.[8,27–29] Furthermore, recent experiments on pigs by Brookes *et al*.[33] showed that the alteration of septal curvature as a result of acute right ventricular dilatation in RVI changes the left ventricular geometry and that this has a direct impact on left ventricular contractile function, which is independent

of the effects of left ventricular inferior wall ischemia or left ventricular compliance.

Therefore, volume loading in the context of right ventricular dilatation may not improve cardiac function. Volume loading may further dilate the right ventricle, causing a further decrease in left ventricular compliance and systolic function. The discrepancy in responding to fluid therapy may reflect a spectrum of initial volume status in patients with acute RVI with hypovolemic right heart infarcts who are definitely benefiting from fluid therapy.

As a result, the therapeutic options for RVI differ from those in a patient with isolated or predominant left ventricular infarction (Table II).

Optimization of Ventricular Preload

The initial therapy of a patient with RVI, who has hypotension and no pulmonary congestion, should start with volume expansion, often by infusion of isotonic saline to increase the filling of the right ventricle which in turn will increase the filling of the underfilled left ventricle and increase cardiac output. For patients who are unresponsive to initial trial of fluids, hemodynamic monitoring may be necessary, and subsequent volume challenge may be appropriate if the estimated central venous pressure is < 15 mmHg. As the right and left ventricles are preload dependent, any interventions that reduce the preload (diuretics, nitrates, and vasodilators) should be avoided even in the absence of hypotension. This treatment strategy would differ from therapy for pump failure caused by acute left ventricular infarct due to the need for maintaining the right ventricular preload in acute RVI.

Treatment of isolated or predominant left ventricular dysfunction due to acute MI in a patient with systolic arterial pressure > 100 mmHg, cardiac index < 2.5 l/min/m^2, and elevated left side pressures of > 18 mmHg would include modest diuresis and afterload and preload reduction using intravenous nitroglycerin and an angiotensin-converting enzyme (ACE) inhibitor.[82] In contrast, treatment of RVI includes early maintenance of right ventricular preload with intravenous fluid administration and avoidance of diuretics, nitrates, and ACE inhibitor. When RVI is accompanied by severe left ventricular dysfunction and pulmonary congestion, the right ventricle is further compromised by increased afterload. In this circumstance, the use of afterload-reducing agents such as sodium-nitroprusside or an intra-aortic counterpulsation device is often necessary to unload the left and subsequently the right ventricle.

Ionotropic Stimulation

Parenteral ionotropic stimulation has been shown to improve both right and left ventricular function in the setting of an acute RVI.[8, 16, 83, 84] Dell'Italia *et al*.[8] studied the effect of dobutamine in nine patients with RVI after volume loading with normal saline and found that dobutamine produced an increase in cardiac index and right ventricular stroke volume. Further animal experiments[83, 85] confirmed these results, and it was thought that ionotropic stimulation enhances right ventricular performance by increasing ventricular septal contraction. This acts in a pistonlike fashion and contributes to right ventricular systolic pressure generation and an increase in cardiac output. Recent experiments by Brookes *et al*.[33] showed that dobutamine also acts by enhancing left ventricular systolic pressure, exaggerates septal movement toward the right ventricle in systole, reducing right ventricular cavity dilatation, thus maintaining left ventricular cavity geometry and enhancing its contractile performance. Dobutamine also can diminish pulmonary vascular resistance and therefore reduce right ventricular afterload.

Thus, ionotropic support with dobutamine should be initiated if the cardiac output fails to improve after 1–2 litres of fluid administration. Although dobutamine is the preferred initial drug of choice, patients with severe hypotension may require agents with pressor effects such as dopamine for restoration of adequate coronary pressure.

Optimization of Rhythm

Even in the absence of right ventricular infarction, patients with inferior wall MI are prone to develop bradycardia, thought to be mediated by Bezold-Jarisch reflex.[86, 87] Bradyarrhythmias may precipitate severe hemodynamic compromise in patients with RVI. Bradyarrhythmias resulting in atrioventricular dyssynchrony and loss of right atrial contribution may also lead to severe hemodynamic compromise.[16, 66, 67, 85]

The development of high-degree atrioventricular block has been reported to occur in as many as 48% of patients with RVI.[41] Although atropine may restore physiologic rhythm in some patients, atrioventricular sequential pacing may be necessary for increasing the cardiac output and reversing the

TABLE II Sequels of right ventricular ischemia

RV systolic dysfunction	➡ ↓ RV cardiac output	➡ ↓ LV filling	➡ ↓ LV cardiac output
RV dilatation	➡ Septal shift toward LV	➡ ↓ LV compliance	➡ ↓ LV cardiac output
	➡ ↑ Pericardial pressure	➡ ↓ LV compliance	➡ ↓ LV cardiac output
	➡ Change in LV geometry	➡ ↓ LV systolic function	➡ ↓ LV cardiac output
Atrioventricular dyssynchrony	➡ Loss of atrial contribution to ventricular filling	➡ ↓ LV cardiac output	
Concomitant LV infarction	➡ ↓ LV systolic function	➡ ↓LV cardiac output	

Abbreviations: RV = right ventricle, LV = left ventricle.

TABLE III Reperfusion studies in patients with right ventricular infarction

Study First author (Ref. No.)	Patients with RVI (% of IWMI)	Time to intervention	Diagnosis of RVI	Intervention	Role of Intervention (+)	Role of Intervention (−)	Inference	Limitations
Schuler (92)	19	<4 h	Radionuclide angiography	Intracoronary thrombolytics	Improvement in RV ejection fraction	Depressed RV ejection fraction Increased mortality	Early recanalization results in swift improvement of depressed RV performance	Small number of patients
Verani (93)	19 (63%)	<6 h	Radionuclide angiography	Intracoronary thrombolytics	Improvement in RV function	Improvement in RV function	Improvement is the rule whether or not recanalization occurs	Small number of patients
Roth (94)	65	117 ± 21 min	ECG and radionuclide angiography	Early thrombolysis + PTCA in some	Recovery of RV function	Recovery of RV function	Recovery is the rule. Early thrombolysis with or without PTCA does not enhance this favorable outcome	No randomization was done
Berger (TIMI-II) (95)	58 (5%)	<4 h	Late radionuclide angiography	Thrombolytics in all patients			Thrombolysis reduces incidence of RVI	1. Delayed diagnosis of RVI 2. Only patients eligible for thrombolytics considered
Zehender (9)	107 (54%)	<6 h	ST elevation in V_4R	Thrombolytics vs. no thrombolytics	Complications 34% Mortality 10%	Complications 54% Mortality 42%	Thrombolytics reduce complications and mortality in RVI	No noninvasive studies to confirm RV or LV function
Kinn (96)	27 (19%)	<24 h	ECG and hemodynamic criteria	Primary angioplasty	Hemodynamic improvement	No hemodynamic improvement	Reperfusion in the setting of RVI leads to rapid hemodynamic improvement	1. Retrospective analysis 2. Selection bias 3. Few patients
Bowers (26)	53 (42%)	<12 h	2-D Echocardiography	Primary angioplasty	Recovery of RV function Complications 12% Mortality 2%	Lack of recovery of RV function Complications 83% Mortality 58%	Unsuccessful reperfusion is associated with impaired recovery of RV function, high complication rate and mortality	1. High mortality 2. Small number of patients (12) with incomplete reperfusion
Zeymer (97)	169 (32%)	<6 h	ST elevation in V_4R	Thrombolytics in all patients			Thrombolytics not indicated in patients with "small ST-segment"[a] elevations whether RVI is present or not	No noninvasive assessment of LV or RV function

(+) denotes either use of thrombolytics or recanalization on coronary angioplasty.

(−) denotes either no thrombolytic use or failure of recanalization on coronary angioplasty.

[a] Small ST-segment elevations = sum of ST-segment elevations of ≤ 0.8 mm on the baseline electrocardiogram.

Abbreviations: RVI = right ventricular infarction, IWMI = inferior wall myocardial infarction, RV = right ventricular, ECG = electrocardiogram, PTCA = percutaneous transluminal coronary angioplasty, LV = left ventricular.

shock associated with atrioventricular dyssynchrony in RVI.[88] There are reports that aminophylline may restore sinus rhythm in patients with acute atrioventricular block, suggesting the role of ischemia-induced adenosine.[89,90] This pharmacologic maneuver may restore atrioventricular synchrony and thereby obviate the need for transvenous pacing. In patients with atrial fibrillation, prompt cardioversion and restoration of atrioventricular synchrony should be considered at the earliest sign of hemodynamic compromise.

Reperfusion

The salutary effects of reperfusion in patients with inferior wall MI have been less impressive than in patients with anterior wall MI.[91] Whether the presence of RVI in patients with inferior wall MI is an indication for reperfusion is less clear (Table III). Some studies suggest that right ventricular function recovers only after successful reperfusion,[9, 26, 92, 95, 96, 98] whereas others report improvement even in the absence of a patent infarct-related vessel.[15,93,94,99,100]

Zehender *et al*.,[9] studied 107 patients with RVI, identified by the presence of ST-segment elevation in the right precordial lead V_4R. They found that both mortality and major in-hospital complications were lower in patients who received thrombolytics than in those who did not.

In the second phase of the Thrombolysis in Myocardial Infarction trial,[95] there was a reduction in the incidence of RVI (identified by wall motion abnormality on a predischarge radionuclide angiogram) among patients with inferior MIs who had patent infarct-related arteries, compared with those whose arteries remained occluded (14 vs. 48%). This finding suggests that successful thrombolysis may prevent RVI.

In another related study, Bowers *et al*.[26] studied the role of primary angioplasty in 53 patients with RVI (identified by echocardiographic evidence of a combination of right ventricular dilatation, depressed right ventricular global function, and wall motion abnormalities). They found that patients with right ventricular dysfunction who had incomplete reperfusion had higher mortality (58%) and a higher rate of untoward in-hospital events (83%).

In a recent study, Zeymer *et al*.[97] studied the prognostic impact of RVI in streptokinase-treated patients with acute inferior wall MI. They found that patients with inferior wall MI and "small ST" (sum of ST-segment elevations of ≤ 0.8 mm on standard 12-lead ECG) had a very low cardiac mortality regardless of whether RVI (identified by the presence of ST-segment elevation in right precordial lead V_4R) was present or not. The authors concluded that reperfusion therapy in patients with acute inferior wall MI and "small ST," indicating a small infarction, is not indicated irrespective of the presence of RVI, unless advanced heart block or hemodynamic instability indicates a large infarct.

O'Rourke,[101] in a recent editorial, states that since different methods are used to diagnose RVI and failure to randomize patients with RVI to thrombolytic or primary angioplasty versus nonreperfusion therapy, it would be difficult to determine which patients with inferior wall MI and RVI are likely to do better with reperfusion therapy. O'Rourke recommends that patients with evidence of RVI by ECG criteria or by other noninvasive methods, who have clinical evidence of depressed right ventricular function, are candidates for coronary angioplasty (if available), thrombolytic therapy (if not contraindicated), or volume and dobutamine infusion (if necessary) when reperfusion therapy is unavailable or contraindicated.

Conclusion

Right ventricular infarction occurs in more than one-third of patients with acute inferior wall MI. A spectrum of disease from asymptomatic mild right ventricular dysfunction to cardiogenic shock has been recognized. Frequent accompaniments may include atrial infarction, sinus bradycardia, atrial fibrillation, and atrioventricular block. Only 10 to 15% of patients show hemodynamic abnormalities. Patients with hemodynamically significant RVI usually present with hypotension, elevated JVP, and occasionally shock, all in the presence of clear lung fields. All patients with inferior wall MI should have right-sided precordial leads recorded for evidence of RVI. Patients with RVI who are hemodynamically unstable should be managed with volume loading to maintain adequate right ventricular preload. Early recognition and prompt reperfusion with thrombolytic therapy or coronary angioplasty, rate and rhythm control, and ionotropic support with dobutamine may also be warranted. Patients who survive the acute phase have no long-term consequences, and complete recovery over a period of weeks to months is the rule in a majority of patients, suggesting right ventricular "stunning" rather than irreversible necrosis has occurred.

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Right Ventricular Infarction

- Author: Claudia Dima, MD, FACC; Chief Editor: Eric H Yang, MD more...

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Background

Right ventricular infarction was first recognized in a subgroup of patients with inferior wall myocardial infarctions who demonstrated right ventricular failure and elevated right ventricular filling pressures despite relatively normal left ventricular filling pressures. Increasing recognition of right ventricular infarction, either in association with left ventricular infarction or as an isolated event, emphasizes the clinical significance of the right ventricle to total cardiac function.

Interest in recognizing right ventricular infarction noninvasively has grown because of the therapeutic implications of distinguishing patients with right ventricular dysfunction from those with the more usual clinical presentation of left ventricular dysfunction. Patients with right ventricular infarctions associated with inferior infarctions have much higher rates of significant hypotension, bradycardia requiring pacing support, and in-hospital mortality than isolated inferior infarctions.[1]

For more information, see Myocardial Infarction.

Pathophysiology

The right ventricle is a thin-walled chamber that functions at low oxygen demands and pressure. It is perfused throughout the cardiac cycle in both systole and diastole, and its ability to extract oxygen is increased during hemodynamic stress. All of these factors make the right ventricle less susceptible to infarction than the left ventricle.

The posterior descending branch of the right coronary artery usually supplies the inferior and posterior walls of the right ventricle. The marginal branches of the right coronary artery supply the lateral wall of the right ventricle. The anterior wall of the right ventricle has a dual blood supply: the conus branch of the right coronary artery and the moderator branch artery, which courses from the left anterior descending artery.[2]

Interestingly, right ventricular infarction noted at necropsy usually involves the posterior septum and posterior wall rather than the right free wall. The relative sparing of the right ventricular anterior wall apparently arises from a high degree of collateralization. This collateral blood flow is thought to be derived from the thebesian veins and diffusion of oxygen directly from the ventricular cavity. A direct correlation exists between the anatomic site of right coronary artery occlusion and the extent of right ventricular infarction. Studies have demonstrated that more proximal right coronary artery occlusions result in larger right ventricular infarctions.[3] On occasion, the right ventricle can be subjected to infarction from occlusion of the left circumflex coronary artery.[4]

Because the right ventricle is considered a low-pressure volume pump, its contractility is highly dependent on diastolic pressure. Hence, when contractility and associated diastolic dysfunction are impaired attendant to right ventricular infarction, the right ventricular diastolic pressure increases substantially and systolic pressure decreases. In such a scenario, concomitant left ventricular dysfunction, with increase in right ventricular afterload, is possible. In such a setting, right ventricular output can decrease dramatically, and the only driving force remaining is elevated right atrial pressure. In such a circumstance, the right ventricle serves as a poorly functioning conduit between the right atrium and the pulmonary artery.

Elevation of right atrial pressure secondary to right ventricular infarction has been noted to serve as a stimulus for secretion of atrial natriuretic factor. Increased levels of this polypeptide can be detrimental to normal left ventricular filling pressures. This occurs by virtue of the potent vasodilating, natriuretic, diuretic, and aldosterone-inhibiting properties of atrial natriuretic factor. Inappropriately elevated levels of atrial natriuretic factor may worsen the clinical syndrome of right ventricular infarction.[5] The potential hemodynamic derangements associated with right ventricular infarction render the afflicted patient unusually sensitive to diminished preload (ie, volume) and loss of atrioventricular synchrony. These 2 circumstances can result in a severe decrease in right and, secondarily, left, ventricular output.[6, 7, 8]

Early thrombolysis or mechanical reperfusion of an occluded coronary artery resulting in right ventricular infarction is associated with prompt reduction in right atrial pressure. This is extremely important because persistently elevated right atrial pressure has been associated with increased in-hospital mortality when associated with myocardial infarction. The extent of right ventricular infarction varies greatly and is dependent on the site of occlusion of the right ventricular arterial supply. If occlusion occurs before the right ventricular marginal branches and if collateral blood flow from the left anterior descending coronary artery is absent, then the size of infarction generally is greater. Extent of infarction depends somewhat on flow through the thebesian veins.[9, 10] In general, any major reduction in blood supply to the right ventricular free wall portends an adverse prognosis in association with this disorder.

Epidemiology

Isolated infarction of the right ventricle is extremely rare; right ventricular infarction usually is noted in association with inferior wall myocardial infarction. The incidence of right ventricular infarction in such cases ranges from 10-50%, depending on the series.[11]

The frequency of right ventricular infarction, which can be detected by right-sided precordial leads, in association with non–ST-segment elevation or non–Q-wave myocardial infarction, is not known and currently is being investigated. Although right ventricular infarction is clinically evident in a sizable number of cases, the incidence is considerably less than that found at autopsy.[10, 12, 13, 14] A major reason for the discrepancy is the difficulty in establishing the presence of right ventricular infarction in living subjects. Additionally, right ventricular dysfunction and stunning frequently are of a transient nature, such that estimation of the true incidence of right venticular infarction is even more difficult.

Criteria have been set forth to diagnose right ventricular infarction; even when strictly employed, however, the criteria

lead to underestimation of the true incidence of right ventricular infarction.[15, 16, 17]

Contributor Information and Disclosures

Author
Claudia Dima, MD, FACC Interventional Cardiology

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Coauthor(s)
David L Coven, MD, PhD Assistant Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons; Director, Cardiology Outpatient Clinic, St Luke's Site, Attending Physician, Department of Medicine, Division of Cardiology, St Luke's-Roosevelt Hospital Center

David L Coven, MD, PhD is a member of the following medical societies: American College of Physicians, American Medical Association, and Massachusetts Medical Society

Disclosure: Nothing to disclose.

Kenneth B Desser, MD Clinical Professor, Director of Cardiology Fellowship, Banner Good Samaritan Medical Center, Phoenix, Arizona

Disclosure: Nothing to disclose.

Ashish Pershad, MD Consulting Staff, Heart and Vascular Center of Arizona

Ashish Pershad, MD is a member of the following medical societies: American College of Cardiology

Disclosure: Nothing to disclose.

Specialty Editor Board
George A Stouffer III, MD Henry A Foscue Distinguished Professor of Medicine and Cardiology, Director of Interventional Cardiology, Cardiac Catheterization Laboratory, Chief of Clinical Cardiology, Division of Cardiology, University of North Carolina Medical Center

George A Stouffer III, MD is a member of the following medical societies: Alpha Omega Alpha, American College of Cardiology, American College of Physicians, American Heart Association, Phi Beta Kappa, and Society for Cardiac Angiography and Interventions

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Francisco Talavera, PharmD, PhD Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy; Editor-in-Chief, Medscape Drug Reference

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Chief Editor
Eric H Yang, MD Associate Professor of Medicine, Director of Cardiac Catheterization Laboratory and Interventional Cardiology, Mayo Clinic Arizona

Eric H Yang, MD is a member of the following medical societies: Alpha Omega Alpha

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Additional Contributors
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