CARDIOLOGY Rounds®

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Cardiogenic shock from right ventricular infarction

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Cardiogenic shock associated with right ventricular infarction (RVI) is a challenging clinical entity with important therapeutic implications. Unlike cardiogenic shock due to left ventricular (LV) pump failure, shock associated with RVI may be associated with good long-term survival if recognized and managed appropriately. Acute RVI can result in severe hemodynamic compromise associated with high in-hospital morbidity and mortality.[1] However, unlike the left ventricle where prolonged ischemia results in irreversible myocardial necrosis associated with permanent LV dysfunction, the right ventricle (RV) is considerably more resistant to ischemic insult and may recover normal function over time.[2,3] Thus, aggressive intervention to improve hemodynamics, manage complications, and restore coronary perfusion is essential to help facilitate RV recovery and improve long-term clinical outcome. Fortunately, the development of cardiogenic shock due to RVI is infrequent: only 2.8% of cardiogenic shock cases in the SHOCK registry were due to RVI.[4]

Normal physiology

The RV is a pyramidal-shaped chamber, comprised of the right ventricular free wall (RVFW) and the interventricular septum. RV systolic pressure and flow are generated by RVFW shortening and contraction in a peristaltic wave towards the septum from apex to outflow tract. Interventricular septal contraction further contributes to RV performance.[5]

The RVFW is a thin structure with a small tissue mass generating low pressures, and with relatively low oxygenation demands. The RV has an extensive collateral coronary circulation from the left coronary artery and, in the absence of significant RV hypertrophy, receives coronary perfusion during both systole and diastole.[6] Consequently, the RV has more favourable oxygenation supply-demand characteristics and is relatively resistant to ischemia when compared to the left ventricle.

Ischemia and the right ventricle

Ischemia or infarction of the RV occurs in up to 50% of patients with acute transmural inferior-posterior left ventricular myocardial infarction (MI).[7] Proximal occlusion of the right coronary artery (RCA) prior to the origin of the RV branch is nearly always responsible for hemodynamically significant RVI,[8] although circumflex occlusion with a left-dominant coronary circulation may also be responsible.[9] However, RV function is preserved whenever there is adequate collateral circulation from the left coronary system or spontaneous reperfusion of the occluded RCA.[8]

Pathophysiology of cardiogenic shock in RV infarction

Although RV infarction occurs frequently in association with inferior wall MI, hemodynamic impairment is uncommon.[6,10] Several important mechanisms contribute to the characteristic

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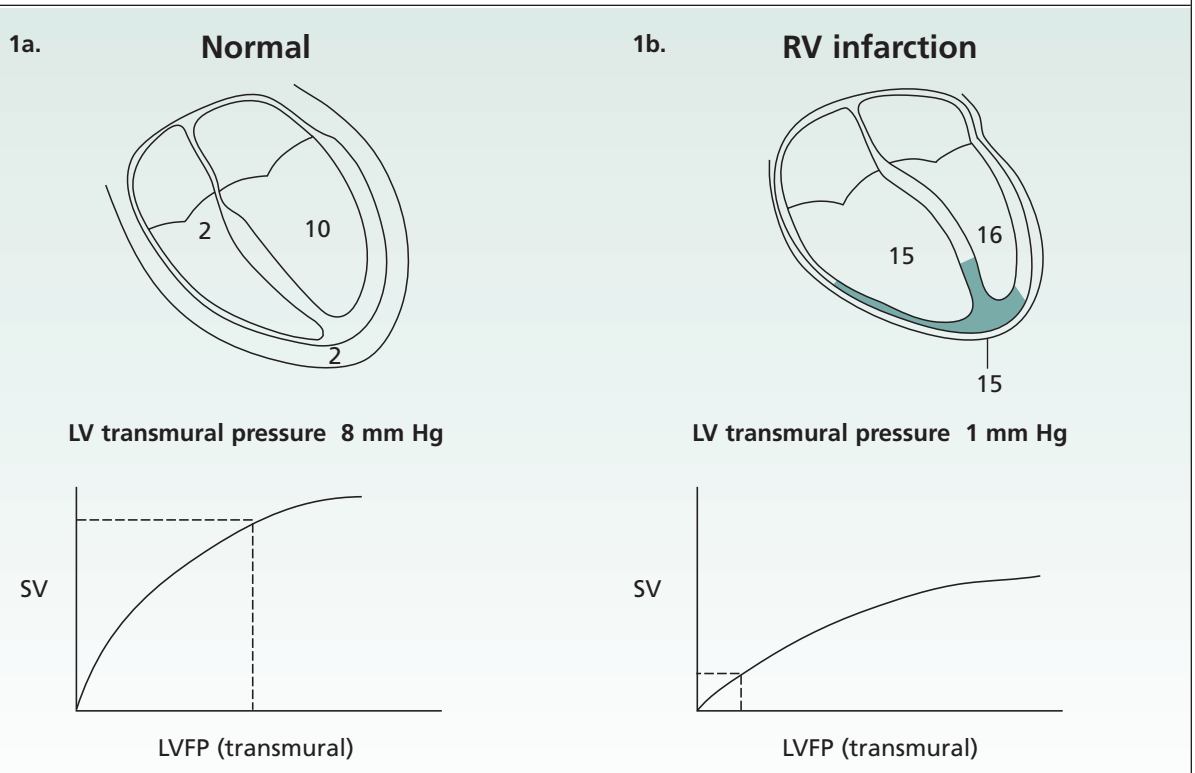
Figure 1: Hemodynamic consequences of right ventricular infarction.
An example of normal diastolic hemodynamics is shown in Figure 1a. The intra-pericardial pressure (2 mm Hg) is largely determined by the right heart filling pressures (2 mm Hg). The LV filling pressure is the LV transmural pressure (8 mm Hg), which is the difference between LV cavitary pressure (10 mm Hg) and the intrapericardial pressure (2 mm Hg). The LV stroke volume is a function of the LV transmural pressure. When an RV free wall infarction occurs, the chamber dilates and RV filling pressures increase (15 mm Hg) (Figure 1b). Although intracavitary LV pressures have risen to 16 mm Hg, the increase in pericardial pressure (15 mm Hg) results in a substantial reduction of LV transmural pressure (1 mm Hg). Consequently stroke volume is markedly reduced as a consequence of inadequate preload.



LV = left ventricular, RV = right ventricular, LVFP = left ventricular filling pressure, SV = stroke volume,

hemodynamic impairment observed with cardiogenic shock consequent to RVI:

• Acute RV free wall ischemia causes an immediate dilatation of the RV, which is limited by the constraints of the pericardial sac.

• Right heart filling pressures and intrapericardial pressures increase as a result of increased splanchnic venous tone associated with a falling cardiac output. In the normal heart, pericardial pressures and RV diastolic pressures are low and equal. Consequently, there is an adequate LV transmural pressure (LV intracavitary pressure – pericardial pressure) to facilitate LV diastolic filling (Figure 1a).

• Acute ischemic dilatation of the RV results in a rapid increase in intrapericardial pressure and a consequent decrease in LV transmural pressure (Figure 1b). As transmural diastolic pressure is a major determinant of ventricular preload, a marked reduction of preload results in a profound reduction of LV stroke output. Furthermore, if there is any significant LV impairment, the LV performance will be even more dependent on adequate diastolic filling.

• When RV and pericardial pressures equilibrate with LV filling pressures, further increases in RV filling pressures (eg, by fluid administration) will increase LV filling pressures by the same amount with no improvement in LV transmural pressure. The hemodynamic consequences of acute RV dilatation and impaired LV filling are similar to those observed in cardiac tamponade.

The contribution of depressed RV systolic function to the low cardiac output is controversial. A reduced RV stroke volume had been considered to be primary cause of the reduction of LV filling and diminished cardiac output. However, experimental models suggest that depression of cardiac output with RV infarction is largely dependent upon an intact pericardium,[12] indicating the importance of interventricular dependence rather than depressed RV systolic function as the principal mechanism of the hemodynamic disturbance.

Another mechanism that contributes to impaired LV filling is interventricular septal shift. It has been suggested that progressive RV dilatation and elevated RV diastolic pressure shifts the interventricular septum into the LV cavity and impairs LV filling.[13] Septal shift could change LV geometry and impair LV contractile performance independently of LV preload or compliance.[12] However, reduced LV preload from decreased LV diastolic transmural pressures would also be expected to shift the interventricular septum towards the left ventricle.

A better understanding of the pathophysiology of the hemodynamic disturbance seen in RVI has important therapeutic implications. The pivotal role of the pericardium and interventricular dependence explain the poor response to fluid loading, especially when left and right heart filling pressures are equalized, and the dependence on normal atrioventricular (AV) synchrony.

Diagnosis

Unexplained hypotension or marked hypotension in response to a vasodilator such as nitroglycerin, in association with an acute inferior wall infarction, should raise a high level of suspicion for RVI as the cause of the hemodynamic disturbance. A clinical triad often observed in RVI includes arterial hypotension, an elevated jugular venous pressure (JVP), and clear lung fields.[14] Kussmaul's sign (an inspiratory increase in JVP) in the setting of acute inferior wall MI has a high sensitivity and specificity for hemodynamically severe RVI.[14] An added third (S3) or fourth (S4) heart sound originating from the right heart and a tricuspid regurgitation murmur have been observed.[15,16] Occasionally, inferior MI with RVI is complicated by a ventricular septal defect (VSD), when a loud pansystolic murmur is heard at the left sternal border and is usually accompanied by profound hemodynamic compromise.[17]

Electrocardiogram

In 1976, Erhardt et al demonstrated that the RV lead (lead V_4R) was invaluable for diagnosing RVI in the setting of acute inferior wall MI.[18] RV involvement can be diagnosed with >80% predictive accuracy if there is >1 mm ST-segment elevation present in V_4R in the setting of acute inferior wall MI.[7] Furthermore, Zehender et al demonstrated that ST-segment elevation in V_4R is a strong independent predictor of in-hospital mortality and serious complications in patients admitted with acute inferior wall MI.[19] ST-segment elevation in V_4R is a transient phenomenon that may be absent in >50% of patients with RVI after 12 hours from the onset of chest pain.

Echocardiography

Echocardiography is the most valuable initial tool in the investigation of the hypotensive patient with an acute inferior wall MI. Primary echocardiographic signs include RV dilatation, segmental wall motion abnormality of the RVFW, and decreased descent of the RV base. Secondary echocardiographic signs include paradoxical septal motion, tricuspid regurgitation, tricuspid papillary muscle rupture, dilated inferior vena cava, right-to-left interatrial septal bowing, and right-to-left shunting across a patent foramen ovale (PFO).[20] Of note, interatrial septal bowing is indicative of right atrial infarction; patients with evidence of right atrial injury have a higher incidence of hypotension, heart block, and mortality.[21]

Hemodynamics

The measurement of right heart and pulmonary pressures is important in the diagnosis and management of hypotension and suspected RVI. The characteristic finding is a marked elevation of right atrial pressure (RAP), often at the same level as the left atrial pressure when measured by the pulmonary capillary wedge pressure (PCWP) associated with low cardiac output. In addition, the RV end-diastolic pressure is elevated, RV and pulmonary arteriolar systolic pressures are normal or low, and the PCWP is normal or elevated.[22] The suspected diagnosis of RVI can be confirmed when the RAP is >10 mm Hg and the ratio of RAP to PCWP exceeds 0.8 (normal ratio < 0.6).[23] Lopez-Sendon et al reported that an RAP >10 mm Hg, within 1-5 mm Hg of the PCWP, has a sensitivity of 73% and a specificity of 100% in identifying hemodynamically-significant RVI.[24]

Prognosis of cardiogenic shock due to RVI

Patients with evidence of RVI have a high in-hospital mortality and cardiovascular complication rates.[10,19,25-28] In a prospective study of 200 consecutive patients admitted with acute inferior wall MI, patients with RVI had an in-hospital mortality rate of 31% as compared to 6% with no RV involvement (P <0.001). Major in-hospital complication rates (cardiogenic shock, complete heart block, and ventricular fibrillation) were 64% with RVI compared to 28% without RVI (P <0.001).[25] Elderly patients, in particular, have a 4-fold greater increase in short-term mortality if they have electrocardiographic evidence of RV involvement with an inferior wall infarction.[26]

In the majority of patients with cardiogenic shock and RVI who survive early complications, spontaneous clinical improvement may occur within 3 to 10 days after onset of the event.[2,11,23] Global RV performance improves in these patients over the following weeks, with RV ejection

fraction returning to near normal levels over a period of months.[3,29,30] The mechanisms for improved RV performance over time is thought to be due to the more favourable oxygen supply-demand characteristic of the RV and the beneficial effects of prominent left-to-right coronary collaterals.[1]

Management of cardiogenic shock due to RVI

Optimization of preload

As patients with RVI are exquisitely preload dependant, care should be taken to ensure that they have adequate cardiac filling. Thus, vasodilators and diuretics must be avoided and a small volume challenge (preferably with central venous hemodynamic monitoring) is appropriate. The objective of volume resuscitation is to ensure that the RV is not underfilled; however, hemodynamic studies have demonstrated a neutral or deleterious effect of excessive volume loading.[10,11,23,31] Excessive volume loading can result in further dilatation of the RV with the increased intrapericardial pressure decreasing the LV transmural pressure gradient and further limiting LV filling (Figure 1b). It is recommended that modest volumes of saline be given (500 ml) to achieve a central venous pressure (CVP) of up to 15 mm Hg or equilibration of RAP and PCWP. Further administration of fluids when left and right heart filling pressures are equal is not helpful and will likely have a worsening impact on hemodynamics. In addition, excessive fluids may increase the risk of bleeding by diluting circulating clotting factors .

Inotropic/pressor support

For the hypotensive patient with RVI, dopamine should be initiated immediately if there is no improvement in blood pressure following the rapid administration of a 500 ml bolus of saline. In patients with ischemic RV dysfunction, dobutamine has been shown to significantly improve myocardial performance.[31] In practice, however, dobutamine or milrinone may be poorly tolerated due to their vasodilating properties.

Optimization of rhythm

Loss of AV synchrony from AV block or atrial fibrillation may precipitate severe hemodynamic compromise in patients with RVI. Ventricular filling in the fixed constraint of a taut pericardial sac occurs when the atrium decreases in size during atrial contraction. Due to the high pericardial pressures and the interdependence of ventricular filling, a loss of synchronous atrial contraction prevents adequate ventricular filling. In patients with atrial fibrillation, prompt DC cardioversion to restore sinus rhythm should be instituted. In patients with AV dysynchrony due to a high degree AV block or complete heart block, AV sequential pacing is required to optimize hemodynamics. Several case series have demonstrated the superiority of AV sequential pacing vs. ventricular pacing to improve cardiac output and reverse cardiogenic shock.[32,33]

Reperfusion therapy

Thrombolysis: Patients with early reperfusion after thrombolytic therapy have improved RV ejection fraction and fewer complications than patients where reperfusion has been unsuccessful.[34,35] In one prospective study, patients with inferior wall infarction with RVI who received a thrombolytic had a 4.2-fold lower mortality rate and 2.4-fold lower complication rate than patients with inferior wall infarction and RVI who did not receive thrombolytic therapy.[25] Interestingly, there was no difference in mortality and major in-hospital complications in patients without RVI, whether or not they received thrombolytic therapy. The event rates in patients without RVI were low, suggesting that RV involvement in inferior wall infarction constitutes a subgroup of patients at much higher risk. Unfortunately, hypotension reduces the success of thrombolysis in patients with RVI and cardiogenic shock.

Primary angioplasty: A prospective study demonstrated that successful reperfusion of the RCA and RV branches with primary angioplasty leads to prompt recovery of RV function and an excellent clinical outcome.[27] Patients in this study who had unsuccessful reperfusion experienced a lack of recovery of RV function, persistent hypotension, low cardiac output, and a high mortality rate (58%) vs. the patients with successful reperfusion (2%). Consequently, there is good justification for early catheter-based reperfusion therapy in patients with hypotension due to RVI.

Mechanical circulatory support

Intra-aortic balloon counterpulsation (IABCP) may be beneficial for patients with RVI who have severe hemodynamic compromise. IABCP support improves both coronary perfusion pressure (particularly in patients with a recanalized infarct-related artery) and LV performance (particularly in patients with concomitantly depressed LV function).

Other Complications of RVI

Ventricular septal defect (VSD)

Development of a VSD in the setting of an inferior wall MI with RVI has a very poor prognosis. These VSDs tend to be complex (multiple irregular and variable interventricular connections), and are usually located at the inferobasal portion of the septum.[36] Furthermore, RV function is an important determinant of survival following interventricular septal rupture.

Hypoxemia

Unexplained hypoxemia not responsive to the administration of oxygen in the setting of RVI may be due to a right-to-left shunt through a patent foramen ovale. The shunt will be reduced with an improvement in RV function.[37]

Pulmonary embolism (PE)

PE is a rare complication that has been described in the setting of RVI.[38] This complication is thought to originate from mural thrombus formation due to extensive RVFW akinesis and dilatation.

Conclusions

Right ventricular infarction (RVI) occurs commonly in association with inferior infarction and carries an increased complication and mortality rate. Cardiogenic shock due to RVI is a relatively uncommon complication, but should be recognized early and vigorous treatment should be initiated. Early hemodynamic and mechanical support combined with percutaneous coronary intervention can lead to a good recovery and long-term prognosis. Unlike shock due to LV pump failure, near normal RV function can be restored even after prolonged ischemic insult, making cardiogenic shock due to RVI an important, but reversible clinical entity.

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Abstract of Interest

Strong predictive value of TIMI risk score analysis for in-hospital and long-term survival of patients with right ventricular infarction.

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Background: While right ventricular myocardial infarction is associated with increased in-hospital morbidity and mortality, prognostic risk factors for in-hospital and long-term mortality are poorly defined.
Objectives: To evaluate the prognostic value of TIMI (Thrombolysis in Myocardial Infarction) risk score analysis in patients with right ventricular myocardial infarction (RVI).
Design: Retrospective analysis of a community population.
Setting: Mayo Clinic Coronary Care Unit.
Patients: One hundred and two patients with RVI from 580 consecutive patients from Rochester, Minnesota admitted to the Coronary Care Unit with acute inferior or lateral wall myocardial infarction from January 1988 through March 1998.
Measurement: Combined TIMI risk score analysis with in-hospital and long-term mortality.
Results: In-hospital morbidity (RVI: 54.9% vs non-RVI: 22.2%; P<0.001) and mortality (RVI: 21.6% vs non-RVI: 6.9%;P <0.001) were increased in patients with RVI. The TIMI risk score predicted risk (per one point increase in TIMI score) for in-hospital mortality (OR 1.23, 95% CI 1.02-1.51, P=0.037) and long-term mortality (OR 1.57, 95% CI 1.25-1.96, P<0.001). Patients with RVI whose TIMI risk score was >/=4 had significantly worse long-term survival compared to those patients with RVI and TIMI score <4 (P=0.006).
Conclusions: In -hospital morbidity and mortality, and long-term mortality are increased by right ventricular infarction and can be accurately predicted by the initial TIMI risk score.
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