



Right Ventricular Myocardial Infarction

Rodolfo Pike, RN, BN

Right ventricular myocardial infarction (RVMI) rarely occurs in isolation. Guidelines recommend that practitioners should evaluate for the presence of an RVMI in a patient with an inferior infarction who is hemodynamically compromised. Diagnostic and treatment modalities for an RVMI can vary from treatments used to treat patients presenting with a left ventricular infarction. This column

includes information about the diagnosis and management of a patient presenting with an RVMI.

Address for correspondence: Rodolfo Pike, RN, MN, Clinical Educator, Inpatient Cardiology/Cardiac Surgery and Cardiac Catheterization Laboratory, Health Science Centre, St. John's, NL

Caring for patients who experience myocardial infarctions (MIs) can prove to be challenging even for the most experienced cardiovascular nurse. While most MIs affect the left ventricle (LV), right ventricle myocardial infarction (RVMI) can occur in isolation. RVMIs are usually associated with inferior-posterior infarctions with the right coronary artery (RCA) as the primary lesion (Goldstein, 2002). Diagnosing and managing an RVMI can come with its own unique challenges for the practitioner caring for the patient. Early detection and management of an RVMI is important, especially when paired with an inferior MI. Mortality from inferior MI alone has been estimated at about 6%, whereas inferior MI with right ventricular involvement carries a mortality of about 30% (Carter & Ellis, 2005; Goldich, 2006).

Physiology

Despite the inherent anatomical and workload differences between the left and right ventricle (RV) both chambers have the same cardiac output, which is directly related to pulmonary vascular resistance being less than the systemic vascular resistance encountered by the LV (Kinch & Ryan, 1994). Coronary circulation to the RV is through the RCA and its branches and occurs during systole and diastole (Kinch & Ryan). It is the combination of these factors that make the RV less susceptible to myocardial ischemia (Dima, Pershad, Coven, & Desser, 2008; Levin, 2008). The RV is a thinned walled, low-pressure chamber that is sensitive to changes in preload and afterload, especially in the presence of impaired contractile forces of the ventricular myocardium (Carter & Ellis, 2005).

Incidence

The incidence of RVMI is dependent on whether the RVMI is in isolation or associated with an inferior-posterior or anterior MI. The incidence of an isolated RVMI has been estimated at less than 3%, while RVMI paired with an inferior-posterior MI ranges from 10% to 84% and at about 13% for those experiencing an anterior MI (Kinch & Ryan, 1994).

Pathophysiology

An RVMI is usually the result of occlusion of the proximal RCA, but other arteries, such as the left dominant circumflex or left anterior descending artery, can be the culprits. When this occurs, the RVMI usually presents with either an anterior or posterior-lateral MI (Kinch & Ryan, 1994).

Depending on the extent of myocardial damage from the infarction, a number of problems can arise from the injury to the RV. The impaired contractility can decrease ventricular compliance and stroke volume, which will negatively impact cardiac output (Carter & Ellis, 2005). Impaired RV systolic function is responsible for the decrease in cardiac output despite having an intact LV (Goldstein, 2002). Due to the inability to handle venous return, the RV is unable to pump blood to the pulmonary circulation, thus affecting the amount of oxygenated blood being delivered to the LV. The impaired contractility and decreased ventricular compliance will lead to venous pooling and increased central venous pressure (Carter & Ellis, 2005).

It is the impairment of the RV coupled with RV dilatation that subsequently cause the interventricular septum to bulge into the already volume-depleted LV

resulting in impaired LV filling leading to a further reduction in cardiac output (Carter & Ellis, 2005; Goldstein, 2002). In an attempt to compensate for the decreased cardiac output, tachycardia may be present. The tachycardia, although initially adaptive, may shorten ventricular filling time resulting in an even further decrease in cardiac output (Carter & Ellis, 2005).

Presentation

Typical presentation of a patient with a hemodynamically significant RVMI is a triad of symptoms: jugular venous distension and systemic hypotension in the absence of pulmonary congestion (Goldich, 2006; Levin, 2008) and, occasionally, shock (Levin). The jugular venous distension is a result of the RV's inability to handle venous return, a consequence of impaired RV contractility and compliance. Systemic hypotension is due to the impaired filling of the LV, and the absence of pulmonary congestion is related to the ability of the LV to pump blood forward in addition to the decreased output that the pulmonary circulation receives from the RV (Goldich, 2006). The degree of patient symptoms depends, in part, on the infarction size.

Diagnostics

A number of diagnostic tests are used in diagnosing and confirming an RVMI: electrocardiography (ECG), chest x-ray, hemodynamic monitoring, echocardiography and nuclear imaging all have their place in evaluating patients for RVMI (Carter & Ellis, 2005; Goldich, 2006; Levin, 2008).

The ECG can add valuable diagnostic information for the patient presenting with ischemic chest pain. Patients presenting with an inferior MI and hypotension should be evaluated for RV involvement. A right-sided ECG can be used to diagnose an RVMI and is a recommendation of the 2004 ACC/AHA task force on practice guidelines for the management of patients with ST-segment elevation myocardial infarction (Antman et al., 2004). The ECG leads would be placed on the right side of the chest in a similar fashion as one would place them when performing a standard 12-lead ECG. The leads would be a mirror image of those placed on the left chest wall. The single most predictive ECG finding for RVMI on a right-sided 12-lead ECG is a 1-mm ST-segment elevation in leads V1 and V4R (right precordial leads) (Antman et al., 2004). Another suggestion is that when ST-segment elevation is greater in lead III compared to lead II in the inferior leads it is also suggestive of RVMI (Levin, 2008).

Not all patients with an RVMI will be required to have hemodynamic monitoring, but if the situation should warrant its use, the information obtained will be of value. If the infarction is of hemodynamic significance

and the patient is normovolemic, the pressure in the right atrium will be > 10mmHg with a right atrial to pulmonary capillary wedge pressure ratio of 0.8 (normal value <0.6) (Levin, 2008).

Echocardiography can be used to assess RV function. Two-dimensional echocardiography can be used to measure RV ejection fraction, wall motion abnormalities and changes to RV chamber diameter (Carter & Ellis, 2005; Goldich, 2006).

The gold standard for assessment of RV ejection fraction and detecting wall-motion abnormalities is radionuclide ventriculography (Kinch & Ryan, 1994). Despite the important information that nuclear imaging studies provide, it is not considered to be a first-line diagnostic test but, rather, to be used later in the course of a patient's treatment plan to further evaluate RV function (Carter & Ellis, 2005; Levin, 2008).

Treatment

Instrumental to the treatment plan for the patient with an RVMI is early recognition and early reperfusion therapy if appropriate (Antman et al., 2004). The remainder of the treatment plan will depend on the severity of the MI and whether the infarction was limited to just the RV. There are important points to consider when caring for patients with RVMI. Since hypotension is prevalent in this patient population, fluids may be required. Administering up to a litre of IV fluid challenges is recommended, especially if there is evidence of low cardiac output and an absence of pulmonary edema (Levin, 2008). If the challenge fails to improve hemodynamics, then inotropic support such as dobutamine may be warranted. The purpose of the inotrope is to improve cardiac contractility with the hope of an overall improvement in cardiac output (Carter & Ellis, 2005; Goldich, 2006; Levin).

Cardiac output is dependent on adequate filling and emptying of the LV. In the presence of an RVMI with impaired RV function there is a decrease in the RV stroke volume, which will subsequently lead to a fall in LV filling (Levin, 2008). Thus, medications that would decrease preload (e.g., nitrates or diuretics) should be avoided.

If bradyarrhythmias or high-degree heart block should occur in the presence of RVMI it may worsen hemodynamic stability. Maintaining AV synchrony through the use of AV sequential pacing may be indicated and is recommended (Antman et al., 2004).

Despite the treatment modalities identified, a patient with RVMI may show signs of worsening hemodynamic compromise and evidence of cardiogenic shock that may warrant the insertion of an intra-aortic balloon pump for afterload reduction of the LV and, subsequently, the RV (Altman et al., 2004; Levin, 2008).

In summary, the American College of Cardiology and the American Heart Association, in collaboration with the Canadian Cardiovascular Society, made the following recommendations (Antman et al., 2004): Patients with inferior MI and hemodynamic compromise should be assessed with a V4R lead to detect ST-segment elevation and an echocardiogram to screen for RV infarction

The following principles apply to therapy for patients with STEMI and RV infarction:

- early reperfusion if possible
- AV synchrony should be achieved
- optimization of RV preload

- optimization of LV afterload
- inotropic support for hemodynamically unstable patients not responsive to fluid challenges.

Conclusion

Caring for a patient with an RVMI can prove to be challenging. Understanding the pathophysiology and rationale behind treatment modalities will enable cardiovascular nurses to provide adequate treatment to their patients through evidence-informed practice. Although it is rare to experience RVMI in isolation, clearly established guidelines do exist for diagnosis and treatment. ♥

References

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