Review Article

The Role of Echocardiography in Coronary Artery Disease and Acute Myocardial Infarction

Maryam Esmaeilzadeh, MD, FACC, FCAPSC, Mozhgan Parsaee, MD, Majid Maleki, MD, FACC, FCAPSC*

Echocardiography Research Center, Rajaie Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran.

Received 07 November 2011; Accepted 21 October 2012

Abstract

Echocardiography is a non-invasive diagnostic technique which provides information regarding cardiac function and hemodynamics. It is the most frequently used cardiovascular diagnostic test after electrocardiography and chest X-ray. However, in a patient with acute chest pain, Transthoracic Echocardiography is essential both for diagnosing acute coronary syndrome, zeroing on the evaluation of ventricular function and the presence of regional wall motion abnormalities, and for ruling out other etiologies of acute chest pain or dyspnea, including aortic dissection and pericardial effusion.

Echocardiography is a versatile imaging modality for the management of patients with chest pain and assessment of left ventricular systolic function, diastolic function, and even myocardial and coronary perfusion and is, therefore, useful in the diagnosis and triage of patients with acute chest pain or dyspnea.

This review has focused on the current applications of echocardiography in patients with coronary artery disease and myocardial infarction.

J Teh Univ Heart Ctr 2013;8(1):1-13

This paper should be cited as: Esmaeilzadeh M, Parsaee M, Maleki M. The Role of Echocardiography in Coronary Artery Disease and Acute Myocardial Infarction. J Teh Univ Heart Ctr 2013;8(1):1-13.

Keywords: *Echocardiography • Diagnosis • Coronary artery disease • Myocardial infarction*

Background

A large number of patients present daily to the emergency department with the chief complaint of chest pain. The challenge for the physician is to identify patients who require intervention, particularly when acute coronary syndromes (ACSs) present with atypical symptoms or non-diagnostic electrocardiogram (ECG) changes or normal cardiac enzyme levels. Since the mortality and morbidity of ischemic heart disease improves following early treatment, timely diagnosis is of vital importance not only to help the patient but also to reduce hospital stay and economic costs.

The backbone for the diagnosis of myocardial infarction comprises the patient's history, ECG findings, and cardiac enzyme (Troponin, CKMB) levels,[1] but it should be taken into account that it takes cardiac enzymes some time to elevate above the normal range after the onset of chest pain. In ACSs not associated with myocardial necrosis, these markers do not tend to rise. Enzyme levels are also likely to elevate even in the absence of ACSs, although this increase depends on renal function and underlying disease. ECG changes are sometimes non-specific and non-diagnostic.

Given the limitations in various clinical settings, echocardiography is now deemed an important and

*Corresponding Author: ***Majid Maleki,*** *Professor of Cardiology, Echocardiography Research Center, Rajaie Cardiovascular, Medical and Research Center, Vali-Asr Street. Adjacent to Mellat Park, Tehran, Iran. 1996911151. Tel: +98 21 2392192. Fax: +98 21 22055594. E-mail: majid33@yahoo.com.*

resourceful imaging technique for the management of patients with chest pain. Assessment of left ventricular systolic function, diastolic function, and even myocardial and coronary perfusion may be of value in the diagnosis and triage of patients with acute chest pain or dyspnea. Echocardiography is also very useful when it comes to rule out the possibility of other etiologies of acute chest pain or dyspnea such as aortic dissection and pericardial effusion.

This review will concentrate on the current applications of echocardiography in patients with known or suspected coronary artery disease.

Echocardiography in the Emergency Department

Echocardiography is a non-invasive diagnostic technique for the provision of information on cardiac function and hemodynamics and is the most frequently utilized cardiovascular diagnostic test after electrocardiography (ECG) and chest X-ray. Still, in a patient with acute chest pain, Transthoracic Echocardiography (TTE) takes precedence in order to diagnose acute coronary syndrome (ACS) and to rule out other etiologies, with an emphasis on the evaluation of ventricular function and the presence of regional wall motion abnormalities. The study should also take heed of non-ischemic causes of chest pain. Be that as it may, in a patient suspected of aortic dissection or pulmonary thromboemboli, a negative TTE is not sufficient to rule out the diagnosis.

Regional wall motion should be assessed on multiple image views at the parasternal long-axis and short-axis views and the apical four-chamber, two-chamber, and three-chamber views. Subcostal views can prove extremely helpful, especially when parasternal or apical views are of poor quality, and off-axis or foreshortened views should be avoided in that they render the interpretation of regional wall motion difficult and increase the likelihood of error. Second harmonic imaging with high signal-to-noise ratio can augment the clarity of the images.

The Role of Echocardiography in the Diagnosis of Coronary Artery Disease and Localization of Acute Myocardial Infarction

Evaluation of Myocardial Wall Motion Abnormalities

In clinical practice, visual (eye ball) assessment via Two-Dimensional (2D) Echocardiography provides a rapid evaluation of regional systolic function. In each imaging plane, the left ventricle (LV) is divided into several segments, with each segment being scored a numerical value to signify the degree of contraction. The American Heart Association recently recommended a 17-segment model (Figure 1).[2] The locations of the segments follow the territory of the coronary arteries to expedite the evaluation of ischemia. The severity of contractile dysfunction is, accordingly, scored visually in each segment as 1 for normal contraction or hyperkinesia, 2 for hypokinesis, 3 for akinesia, 4 for dyskinesia, and 5 for aneurysmal segments,[3] and the global wall motion score is thereafter calculated by averaging the readings in all the segments. A normal LV has a wall motion score index of 1, and the index increases as wall motion abnormalities increase in severity. There is a good correlation between the wall motion score index and functional impairment: a wall motion score index of 1.1-1.9 can predict a small infarct size, and an index equal to or greater than 2.0 can predict the occurrence of complications.

Accuracy of the qualitative assessment of wall motion is influenced by image quality and imaging plane in conjunction with the experience of the observer.[4-7] Thickening is preferred for regional systolic function evaluation because of the counterclockwise rotation of the LV around its long axis during systole.[8] In addition to wall motion abnormality assessment, Hand-Held Echocardiography is also applicable with a moderate correlation by comparison with standard 2D echocardiography and the device can be utilized by cardiology trainees with limited experience in echocardiography. Nevertheless, Hand-Held Echocardiography should be drawn upon only in combination with a standard examination.[9] Regional myocardial contractility is quantifiable by strain imaging. Strain signifies deformation and constitutes an excellent parameter of regional function, independent of cardiac translation. Strain values enable the differentiation of normal from abnormal contraction in the ischemic myocardium. In a study in patients receiving coronary angioplasty, systolic strain was reduced and post-systolic strain was elevated.[10] Strain can be measured with Tissue Doppler Imaging (TDI), but it is angle-dependent.[11] Speckle Tracking Echocardiography (STE) is a 2D-based strain imaging modality and has been validated recently for the measurement of regional function in long- and short-axis views.[12] An angle-independent strain imaging modality, STE confers direct measurement of strain, whereas TDI calculates strain by integrating the strain rate. In contrast to Doppler-based strain, STE enables the measurement of radial and circumferential strains from the LV short-axis and longitudinal strain from apical views.

Localization of Infarction

The potential value of 2D Echocardiography as a diagnostic tool in acute myocardial infarction (AMI) was discovered very early,[13, 14] and a large number of studies reported its high sensitivity, both qualitatively and quantitatively. As a general rule, for the differentiation of normal from infarcted

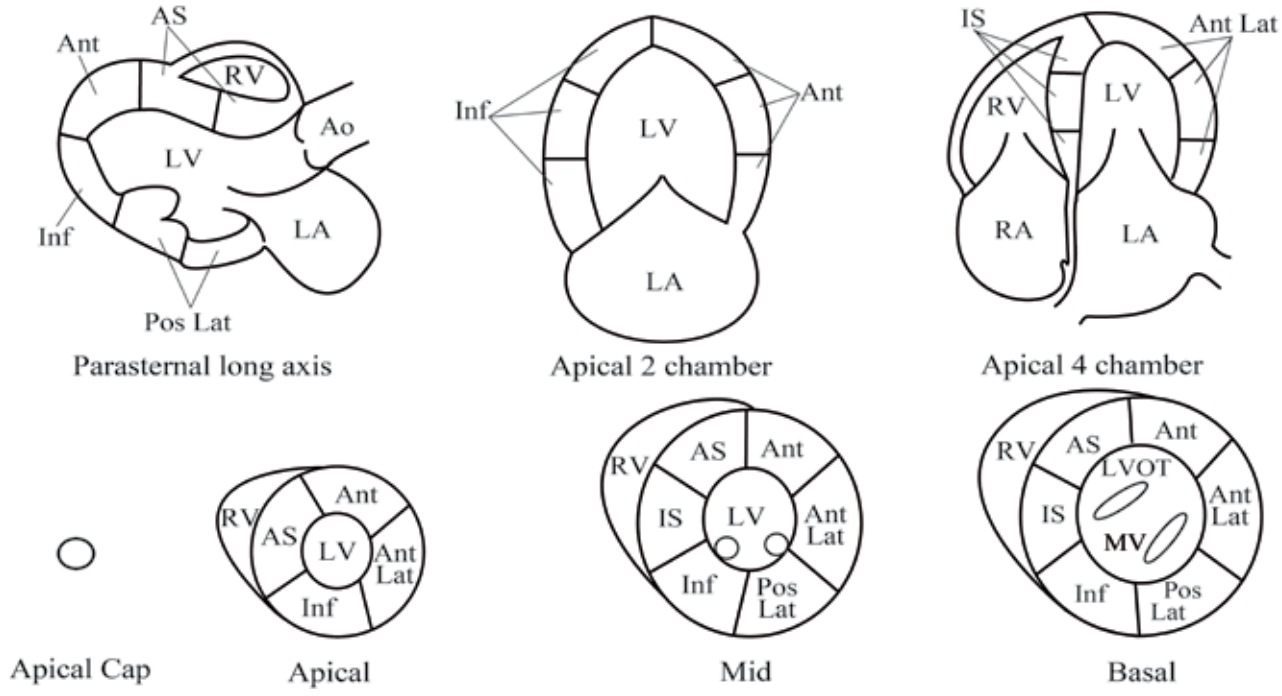


Figure 1. Segmental model for regional wall motion analysis. The left ventricle (LV) is divided into three levels: 6 basal; 6 mid; and 4 apical segments
LA, Left atrium; LV, Left ventricle; RA, Right atrium; RV, Right ventricle; Ao, Aorta; LVOT, Left ventricular outflow tract; MVO, Mitral valve orifice; AS, Anteroseptum; Ant, Anterior; Ant-Lat, Anterolateral; Post-Lat, Posterolateral; Inf, Inferior; IS, Inferoseptum

myocardium, wall thickening is preferred to wall motion.[15, 16] Transmural infarctions are also more optimally recognized than subendocardial infarctions involving less than 20% of the wall thickness.[17] Moreover, 2D Echocardiography is extremely accurate for the localization of the infarction. Exceptions are multi-vessel disease, previous infarction, and overlap between the perfusion territories of the right and circumflex coronary arteries. There is a significant relationship between infarction and contractile dysfunction; consequently, the absence of wall motion abnormality or wall thinning rules out a clinically significant infarction.[18]

Assessment of Infarct Size

As much as the pattern of dysfunction may be a reflection of the extent of an infarction, the circumferential extent of dysfunction could be more than the extent of the infarction,[19-23] which might be in consequence of multi-vessel disease or a previous infarction. Ordinarily, wall motion analysis, in comparison with wall thickening, tends to overestimate the infarct size, but both parameters offer similarly close estimates of the infarct size.[16, 24]

Right Ventricular Infarction

Echocardiography is the imaging method of choice for the diagnosis of right ventricular (RV) infarction.[25] It is also helpful in excluding cardiac tamponade, which may be hemodynamically mistaken with RV infarction. The hemodynamic profile of acute RV infarction can also be diagnostic of an acute pulmonary embolism in the absence of an ischemic event. The 2D Echocardiographic findings of RV infarction encompass RV dilation, RV systolic dysfunction, segmental wall motion abnormalities, and paradoxical septal motion.[26] In many cases, inferior wall motion abnormality is likely to be subtle with a preserved overall LV function. TDI may provide complementary evidence of RV infarction. A case in point is a recent study of 60 patients with a first acute inferior MI, where a tricuspid valve annulus peak systolic velocity smaller than 12 cm/s had a sensitivity of 81%, specificity of 82%, and negative predictive value of 92% for RV infarction.[27]

The characteristic echocardiographic features of RV dysfunction in the setting of an RV infarction are increased right atrial pressure, begetting an interatrial septum shift toward the left atrium (LA), and dilation of the inferior vena cava with decreased (or lack of) inspiratory collapsibility, which correlates perfectly with the clinical status and prognosis.[28] Echocardiography also plays an important role in detecting the complications of RV infarction, including ventricular septal rupture and severe tricuspid regurgitation (as a result of papillary muscle ischemic dysfunction or rupture and functional regurgitation resulting from annular dilatation). Indeed, severe hypoxemia caused by a right-

to-left shunt across a patent foramen ovale, which can be confirmed simply through an injection of agitated saline solution into the arm vein, may occur in tandem with large RV infarction associated with high right atrial pressure.[29]

Hemodynamic Assessment of Patients with Acute Myocardial Infarction Using Doppler Echocardiography

Patients with an AMI were classified into several groups based on the cardiac index and pulmonary capillary wedge pressure (PCWP) by Forrester and colleagues in 1976:[30] Group I: normal hemodynamics (cardiac index [CI] > 2.2 L/min/m^2, PCWP ≤ 18 mmHg); Group II: pulmonary congestion (CI > 2.2 L/min/m^2, PCWP > 18 mmHg); Group III: peripheral hypoperfusion (CI ≤ 2.2 L/min/m^2, PCWP ≤ 18 mmHg); and Group IV: pulmonary congestion and peripheral hypoperfusion (CI ≤ 2.2 L/min/m^2, PCWP > 18 mmHg).[31] This classification is capable of predicting in-hospital mortality, irrespective of the patient's age, gender, precipitating factors, and location of the infarction. Despite the fact that invasive measurement of the cardiac output and PCWP remains common in patients with AMI complicated by cardiac failure or cardiogenic shock, the use of invasive catheters carries the risk of increased mortality in critically ill patients.

In contrast, a meticulously performed Doppler echocardiographic examination can provide sufficient information to determine the hemodynamic category after an infarction without increased mortality. This non-invasive measurement of the cardiac output and PCWP in patients with AMI and cardiac failure can guide therapy and predict prognosis. In post-infarction patients with a left ventricular ejection fraction (LVEF) smaller than 35%, a mitral deceleration time less than 120 msec is deemed highly predictive of a PCWP greater than 20 mmHg.[31, 32]

Several studies have shown the value of pulmonary venous flow Doppler in the estimation of PCWP. The deceleration time of the diastolic component of the pulmonary venous flow enjoys a better correlation with PCWP than with mitral deceleration time. The sensitivity and specificity of a diastolic component pulmonary venous deceleration time smaller than 160 msec in predicting a PCWP equal to or greater than 18 mmHg were reported to be 97% and 96%, respectively.[31, 32] In subjects older than 40 years of age with normal filling pressures, pulmonary venous peak velocities and velocity time integrals are higher during systole than during diastole, and the duration of pulmonary venous A wave is less than that of mitral A wave. With high filling pressures, diastolic filling predominant and A wave duration exceeds that of mitral A wave. In patients with AMI, a systolic fraction of the pulmonary venous flow smaller than 45% was highly correlated with a PCWP greater than 18 mmHg.[31, 32]

TDI measurement of mitral annular velocities is a well-validated method for PCWP estimation. The measurement of peak mitral early diastolic filling velocity / velocity of propagation (E/Vp) by color M-mode Doppler in patients with AMI is strongly allied with PCWP. An E/Vp equal to or greater than 2 is believed to predict a PCWP equal to or greater than 18 mmHg with a respective sensitivity and specificity of 95% and 98%.[31, 32] The Tei index, defined as the sum of isovolumic contraction and relaxation times divided by ejection time, denotes the global LV function. In AMI patients, the Tei index correlates significantly with PCWP and the cardiac index. A Tei index equal to or greater than 0.60 can diagnose impaired hemodynamics (PCWP ≥ 18 mmHg and/or cardiac index ≤ 2.2 L/min/m^2) with a sensitivity, specificity, and accuracy of 86%, 82%, and 83%, respectively.[31, 32] The Tei index confers a swift and practical non-invasive prediction of complications in ST-elevation MI (STEMI) patients insofar as it rises significantly in patients with complications compared with those without them (> 0.66 predicts complications).[33]

Elevated pulmonary artery pressure is allied to increased mortality in AMI patients. Doppler Echocardiography can estimate systolic pulmonary artery pressure by using tricuspid regurgitation and the Bernoulli equation. Pulmonary artery pressure can also be measured based on the size and the respiratory variation of the inferior vena cava using 2D imaging. Needless to say, hemodynamic information obtained from an echocardiographic examination only at a single point in time should be complemented by continued invasive monitoring in patients with ongoing instability.[29, 31]

The Role of Echocardiography for Detection of Complications of Acute Myocardial Infarction

Echocardiography is a vital, non-invasive, and readily available tool in the diagnosis and evaluation of patients with the mechanical complications of AMI and can be used repeatedly at the bedside. Unfortunately, the mechanical complications of AMI are more often than not fatal and, without immediate surgery, they shortly lead to sudden death. The major mechanical complications of AMI are ventricular free wall rupture, ventricular septal rupture, and papillary muscle rupture with severe mitral regurgitation, each of which is capable of triggering cardiogenic shock.

Ventricular Free Wall Rupture and Pseudoaneurysm Formation

A free wall rupture is the most catastrophic complication of STEMI and involves the tearing or rupture of the acutely

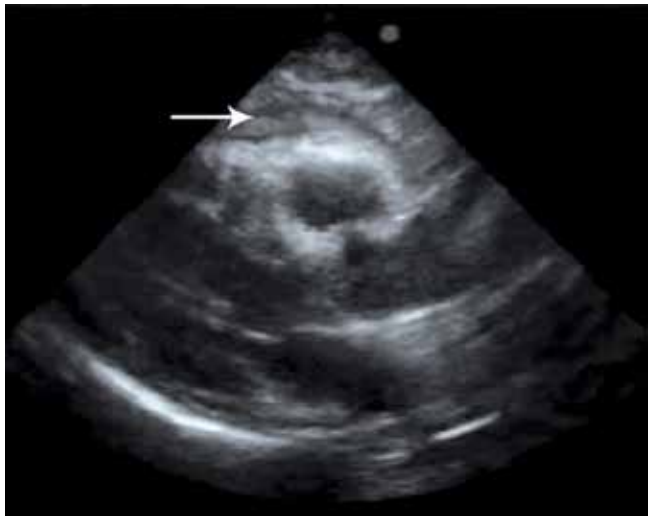
infarcted tissue. The rupture of the free wall of the LV occurs in 1- 4.5% of AMI patients and accounts for 10-15% of early AMI deaths.[33, 35] Some features suggestive of this serious complication of STEMI are as follows:[34] 1) Commonly occurs in old-aged, female gender, and hypertensive patients; 2) Occurs more frequently in the LV than in the RV and seldom occurs in the atria; 3) Occurs commonly in the anterior or lateral wall of the ventricle in the distribution areas of the left anterior descending coronary (LAD) artery; 4) Occurs together with a relatively large transmural infarct involving at least 20% of the LV; and 5) Occurs between 1 day and 3 weeks, but most commonly within 1-4 days in the wake of infarction, usually in close proximity of the junction of the infarct and the normal muscle and mostly happens in first MI.

A free wall rupture tends to happen at three distinct intervals and with three distinctive pathologic subsets.[34] Type I occurs early (within 24 hours) and is a full-thickness rupture, with its frequency rising owing to fibrinolytics. Type II happens 1-3 days post-MI and is in consequence of the erosion of the myocardium at the site of the infarction. Type III occurs late and is situated at the border zone of the infarction and the normal myocardium.[36] It has been hypothesized that type III ruptures may happen in the wake of increased wall stress secondary to dynamic LV outflow tract obstruction.[37] The rupture usually presents with a sudden profound shock, often promptly leading to electro-mechanical dissociation (pulse-less electrical activity) created by cardiac tamponade. If the patient's condition is relatively stable, echocardiography may help diagnose hemopericardium and tamponade[34] (Figure 2a).

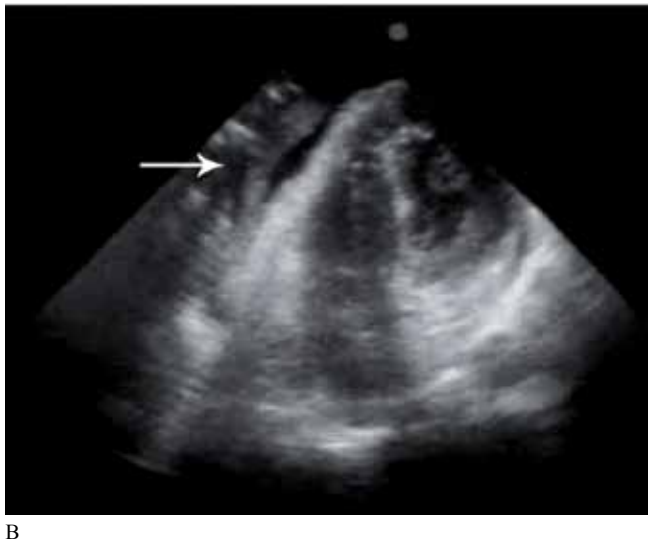
Direct visualization of the rupture is often problematic as it may be only a "slit" in the myocardium, and the location of the pericardial fluid may not directly correlate with the area of the rupture[37] (Figure 2b). However, an intra-pericardial thrombus is often present and is very characteristic. It is rare to show the flow into the pericardium. When created by abrupt intra-pericardial hemorrhage, the characteristic echocardiographic signs of tamponade may be absent and the clinical status should be drawn upon for diagnosis.[38]

Pseudoaneurysms are begotten by the contained rupture of the LV free wall. An incomplete rupture of the heart may take place when both thrombus and hematoma, along with the pericardium, seal the LV rupture and thus thwart the development of hemopericardium. This area of organized thrombus and pericardium is called a pseudoaneurysm, which communicates with the LV cavity through a low velocity bidirectional flow (Figure 3).

In contrast to a true aneurysm, which always contains some myocardial elements, a pseudoaneurysm is composed of organized hematoma and pericardium and lacks any elements of the original myocardial wall. Pseudoaneurysms can increase in size, even equaling the true ventricular cavity in size, and communicate with the LV cavity via a narrow neck. The diagnosis of pseudoaneurysms can usually be made by echocardiography. TTE is the fastest, albeit a partially sensitive, diagnostic tool for the detection of a cardiac rupture and even unexpected diagnosis is reported in an outpatient in the echo-lab.[39] An LV free wall rupture is also a possibility, even after coronary artery bypass grafting.[40]



A



B

Figure 2. Indirect evidence of a free wall rupture as the presence of hemopericardium, the small arrow in parasternal long-axis view (A), and the presence of a thrombus in the pericardial sac in the subcostal four-chamber view (B), the large arrow

The differentiation between true aneurysms and pseudoaneurysms can be difficult by any imaging technique. The crucial diagnostic finding is moderate to large pericardial effusion with clinical and echocardiographic signs of impending pericardial tamponade.[41] The absence of pericardial effusion on echocardiography carries a high negative predictive value. It is advisable that patients without initial cardiac tamponade be followed up because a late

rupture may still occur.[42] Pseudoaneurysms are sometimes distinguishable from true aneurysms thanks to an abrupt interruption in the LV wall (contrasted with the smooth curve of true aneurysms) and a narrow neck[37] (Figures 4 and 5).

If TTE is not feasible in patients receiving mechanical ventilation, Transesophageal Echocardiography (TEE) can aid to confirm a free wall rupture. Cardiovascular Magnetic Resonance (CMR) can also prove helpful when there is a cardiac rupture.[43, 44] CMR offers outstanding image quality and permits the identification of the site and anatomy of a ventricular pseudoaneurysm. Nonetheless, CMR is of limited use in the acute setting because it is time-consuming and non-portable.

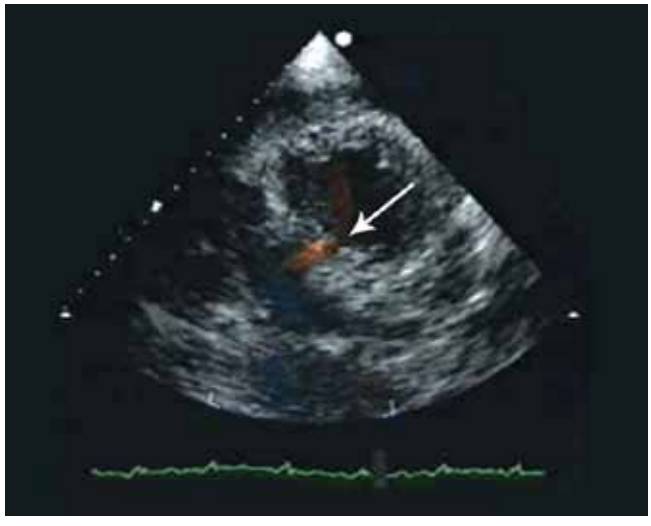


Figure 3. Direct visualization of a free wall rupture (arrow), detected via Color Doppler Echocardiography in the subcostal view

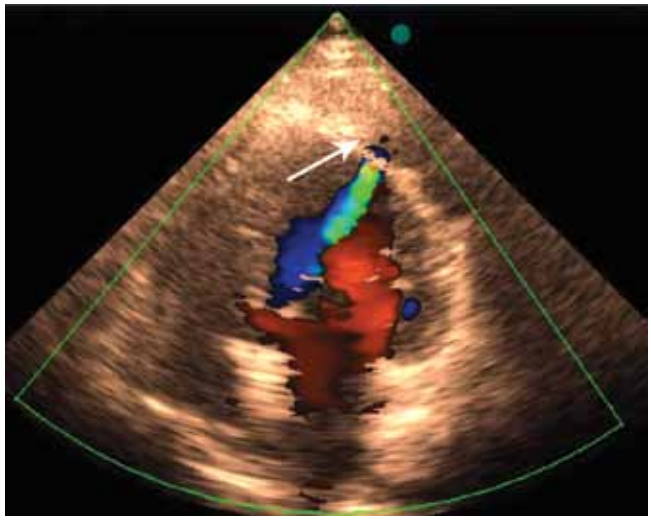


Figure 4. Two-dimensional echocardiogram with Color Flow Doppler in the apical four-chamber view in a patient with a history of anterior myocardial infarction. There is a large pseudoaneurysm (spiral arrow) with a narrow neck in the apicolateral segment of the left ventricle, and the flow passes through its entrance.

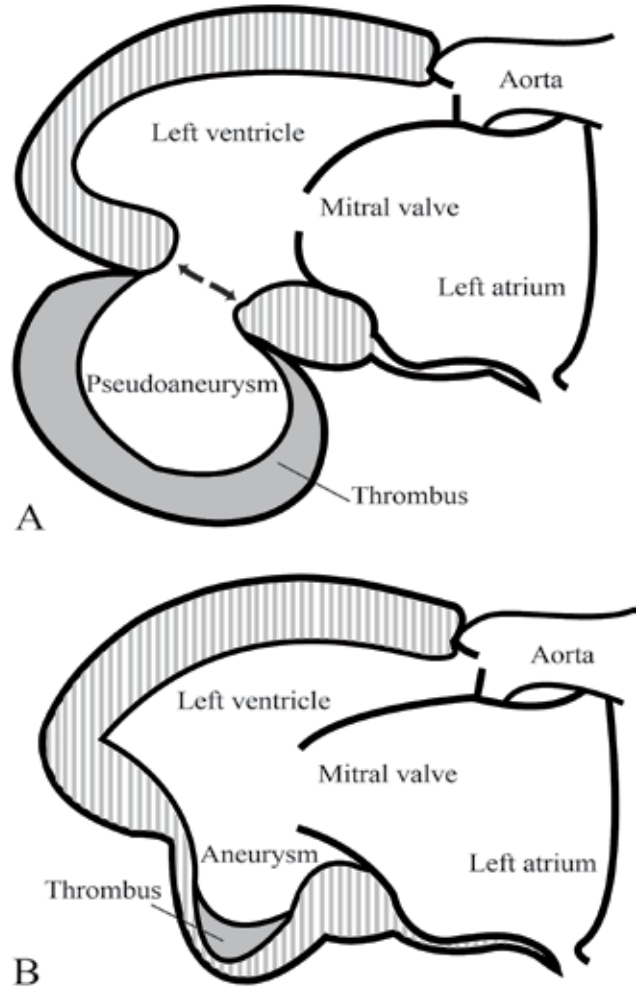


Figure 5. A posterior wall pseudoaneurysm (A) versus a true aneurysm (B) LV, Left ventricle; LA, Left atrium; AO, Aorta

Ventricular Septal Rupture

Before the widespread use of cardiac intervention and in the era prior to reperfusion therapy, a ventricular septal rupture causing a ventricular septal defect (VSD) occurred in 3-5% of transmural or Q-wave MIs.[45] Over time, the rate decreased through the introduction of thrombolytic therapy.[46, 47] Post-MI VSDs after STEMI are associated with high 30-day mortality rates. The perforation can range from one to several centimeters in length. The rupture of the septum with an anterior infarct tends to be apical in location (Figure 6), whereas an inferior infarct is associated with the perforation of the basal inferior septum and has a worse prognosis than those in an anterior location (Figure 7). The likelihood of survival depends on the degree of the impairment of the ventricular function and the size of the defect. Biventricular failure generally happens within hours to days. The defect can be recognized via echocardiography

with Color Flow Doppler Imaging.[34] It is categorized as simple or complex, depending on the length, course, and location. In a simple rupture, the perforation is at the same level on both sides of the septum, and a direct through-and-through communication is present across the septum, while a complex rupture is characterized by extensive hemorrhage with irregular, serpiginous tracts in the necrotic tissue in 30-40% of patients. The diagnosis can usually be made by TTE; experience is essential as the most useful views depend on the location of the defect. Subcostal views are particularly useful in the critically ill, supine patient with inferior infarction. Small defects may not be visible, but Color Doppler is very sensitive. Because these defects are often not discrete, the evaluation of the degree of a left-to-right shunt may be difficult. A large shunt is typified by the hypercontractility of non-infracted LV segments with a low forward stroke volume, high pulmonary artery flow velocity, and elevated pulmonary artery systolic pressure.[38]

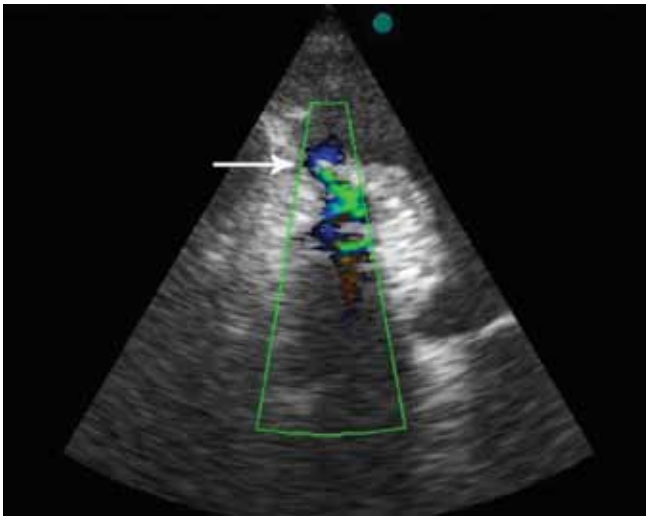


Figure 6. Modified two-dimensional echocardiogram with Color Flow Doppler in the apical four-chamber view, revealing a breach in the interventricular septum and a free communication between the ventricles through an apical septum ventricular septal defect in a patient who recently had an anterior myocardial infarction (spiral arrow)

A post-MI VSD is an infrequent but life-threatening complication of AMI. An extensive infarct size and RV involvement are known risk factors. Septal ruptures are most common in patients with large anterior MI due to the occlusion of the LAD artery. These ruptures are generally apical and simple. Septal ruptures in patients with inferior MI occur relatively infrequently and involve the basal inferoposterior septum and are often complex. At the present time, it seems that the occurrence rate of a post-AMI VSD is lower than that reported by earlier studies, but the mortality rate is still high. Because non-apical VSD and AMI in other walls, except for the anterior wall, are allied with poorer survival, surgical repair is deemed superior to conservative therapy in such scenarios.[48]

An extremely rare case is a partial closure due to thrombus formation.[49] In rare instances, AMI can be complicated by two distinct kinds of rupture named as a ventricular double rupture. The most common form of a ventricular double rupture consists of a ventricular septal rupture in combination with a free-wall rupture.[50, 51] Echocardiography with Color Flow Doppler Imaging is the preferred diagnostic tool to identify a ventricular septal rupture[51] (Figures 6 and 7) with reported sensitivity and specificity of as high as 100%. This modality can also be used for the definition of the site and size of a septal rupture, assessment of the LV and RV functions, estimation of the RV systolic pressure, and quantification of a left-to-right shunt.

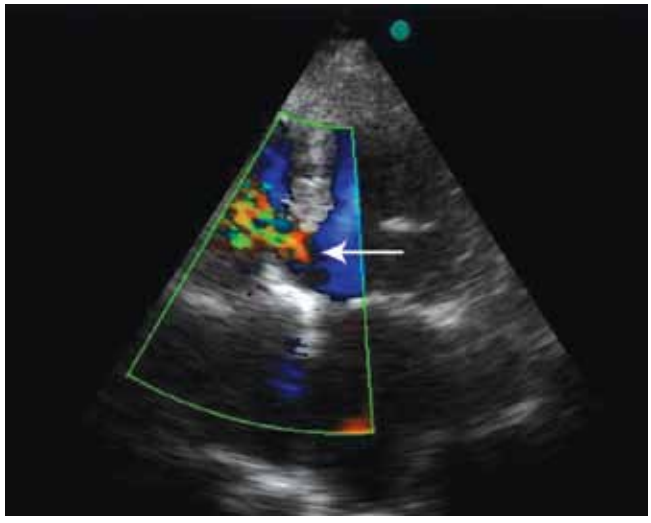


Figure 7. Modified two-dimensional echocardiogram with Color Flow Doppler in the apical four-chamber view, illustrating a basal inferoseptal ventricular septal rupture (ventricular septal defect) (arrow) in a patient who recently had an inferior myocardial infarction

Acute Mitral Regurgitation Secondary to Papillary Muscle Rupture

Severe mitral regurgitation begotten by a papillary muscle rupture accounts for about 5% of deaths in AMI patients.[35] A partial or total rupture of a papillary muscle is a rare but often fatal complication of a transmural MI. An inferior wall infarction can lead to the rupture of the posteromedial papillary muscle (Figure 7), which occurs more commonly than the rupture of the anterolateral muscle (Figure 8), a consequence of an anterolateral MI. The rupture of an RV papillary muscle is unusual but can cause massive tricuspid regurgitation and RV failure. A complete rupture of an LV papillary muscle is fatal because the sudden massive mitral regurgitation that has developed cannot be tolerated. The rupture of a portion of a papillary muscle, usually the tip or head of the muscle, resulting in severe, albeit not necessarily overwhelming, mitral regurgitation, is much more frequent

and not immediately fatal. Unlike the rupture of the ventricular septum, which occurs with large infarcts, a papillary rupture occurs with a relatively small infarct (approximately in half of the cases). Up to half of the patients may be burdened by single-vessel disease.[35] The diagnosis of choice is 2D Echocardiography with Doppler and Color Flow Imaging. Therefore, an echocardiogram should be done promptly on any suspected patient because hemodynamic deterioration can happen rapidly. Mitral regurgitation caused by the rupture of a papillary muscle can be easily recognized by Color Flow Doppler Imaging, which is particularity helpful in distinguishing acute mitral regurgitation from a VSD in the setting of STEMI.

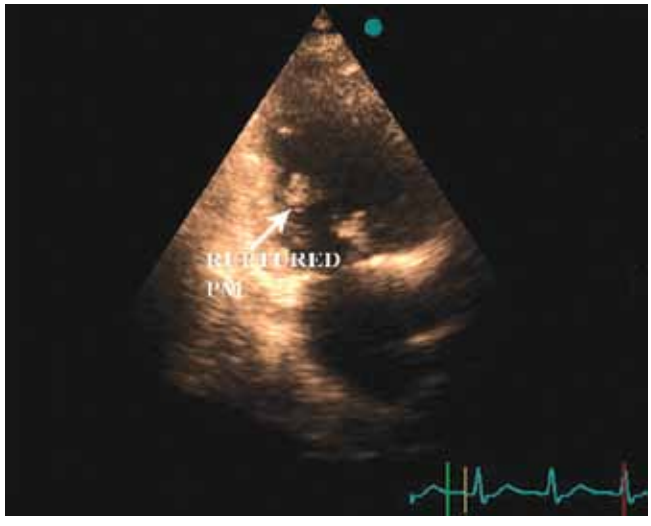


Figure 8. Modified two-dimensional echocardiogram in the apical two-chamber view, depicting the rupture of a posteromedial papillary muscle (arrow) in a patient who recently had a posterior myocardial infarction

Echocardiography can also differentiate between a papillary muscle rupture and other, generally less severe, forms of mitral regurgitation that occur with STEMI.[34] The LV function is hyperdynamic, as a result of the severe regurgitation into the low-impedance LA; this finding alone, in a patient with severe congestive heart failure, should hint at the diagnosis.[35] TTE is often suboptimal for evaluation because the papillary muscles views are often limited, the mitral regurgitation jet is eccentric, and Color Doppler is influenced by the low LV/LA gradient in acute severe mitral regurgitation. TEE is a particularly appropriate imaging modality and should be carried out immediately if this diagnosis is suspected because it provides high resolution images and accurate assessment of mitral regurgitation.[38] The LA is often normal in size.[45] In severe mitral regurgitation, the mitral valve leaflet is usually flail or prolaptic.[45]

Pericardial Effusion and Tamponade

The incidence of post-infarction pericardial effusion as detected by 2D Echocardiography is reported to be from 30-40% in patients with STEMI. The amount of effusion is minor, and the peak time for effusion to occur is three days after the infarction. The resolution is slow; effusion may still be present six months after the infraction. Pericardial effusion occurs more frequently in patients who have had ananterior MI and in those with heart failure. The cause of pericardial effusion is epicardial inflammation. Tamponade is rare in an uncomplicated MI. Larger effusion or effusion with a hemorrhagic appearance should always be considered a myocardial rupture.[45, 52]

Infarct Expansion and True Aneurysm Formation

Infarct expansion is defined as the acute thinning of the ventricular wall with aneurysmal dilation, occurring twenty-four to seventy-two hours after a transmural MI. It represents an acute remodeling phenomenon and carries significant prognostic implications. This complication is not seen in a non-transmural MI. The diagnosis of the infarct expansion is made through echocardiography, showing a typical aneurysmal bulge of the myocardium without a dense scar. The wall consists of a necrotic myocardial tissue only 3-5 mm in thickness (rather than the normal 8-10 mm), which is the antecedent of the majority of the mechanical complications of MI.[45]

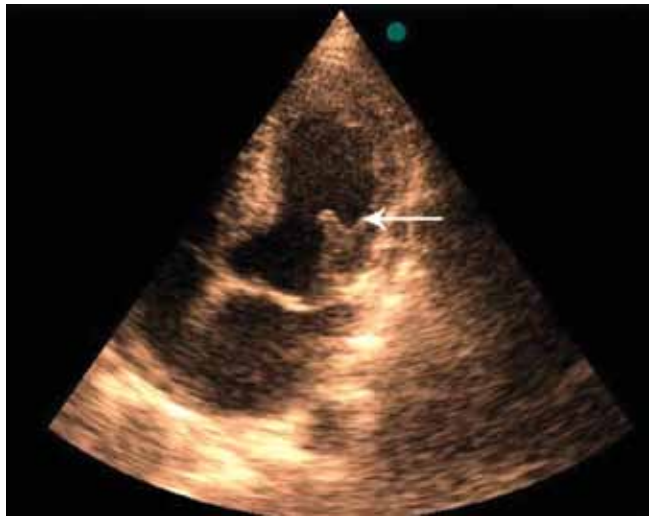


Figure 9. Modified two-dimensional echocardiogram in the apical four-chamber view, showing the rupture of an anterolateral papillary muscle (arrow) in a patient who recently had a lateral myocardial infarction

True aneurysms complicate transmural infarction and are caused by the dilatation of an area of scar. An aneurysm is defined as the deformation of both the diastolic and systolic LV contours with dyskinesis in systole (Figure 9). TTE is a sensitive tool for the diagnosis; nonetheless, when the aneurysm involves a small part of the apex or the basal anterolateral wall, occasional false negatives may occur.

Scar formation requires approximately six weeks. Aneurysm formation is a poor prognostic sign and is associated with congestive heart failure, arrhythmias, and thrombus formation.[38] Mostly, a true aneurysm has a relatively wide mouth, communicating with the aneurismal cavity, compared with a narrow neck observed in a pseudoaneurysm.[45]

LV aneurysms generally range from 1-8 cm. Echocardiography is 93% sensitive and 94% specific for the detection of LV aneurysms, but cardiac catheterization remains the standard for establishing the diagnosis.

Left Ventricular Thrombi

Before the reperfusion era, ventricular thrombi were reported in 25-40% of patients after an anterior MI. They were frequently reported with anteroapical MI and relatively extensive areas of abnormal wall motion. LV thrombus formation occurs in the regions of blood stasis (most commonly in the apex), but it may also be seen within aneurysms at the lateral and inferior walls. The peak timing of early thrombus formation is seventy-two hours; nevertheless, in larger MIs with large areas of akinesis and stagnant flow, thrombus formation tends to occur even within hours. Certain echocardiographic characteristics (pedunculated and mobile thrombi) are associated with higher risk of embolization. TTE remains the imaging modality of choice and is 92% sensitive and 88% specific for detecting LV thrombi. TEE may not be able to visualize the apex as clearly as TTE[38, 45] (Figures 10 and 11).

Intravenous sonicated albumin microbubbles, as left-heart contrast agents, which enhance the definition of the LV border definition and quality of the image may have indications in patients with suspected LV thrombi and poor image views and also for ruling out the other etiologies of chest pain such as hypertrophic cardiomyopathy.[53]

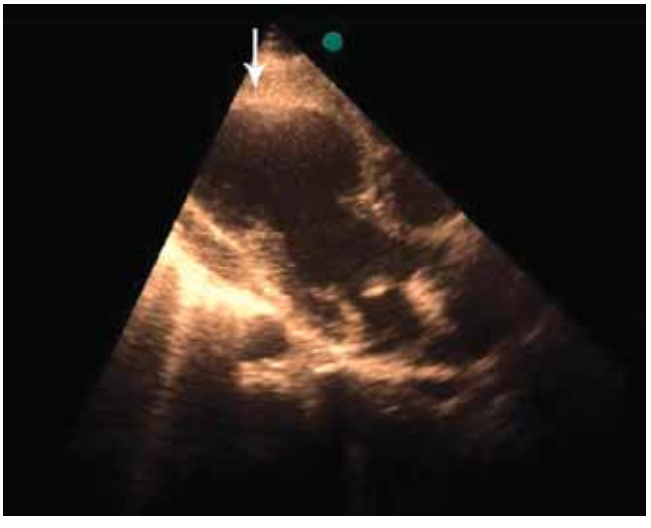


Figure 10. Two-dimensional echocardiogram in the parasternal long-axis view, demonstrating a large true aneurysm in the left ventricle apex (arrow) in a patient who had a relatively old anteroapical myocardial infarction

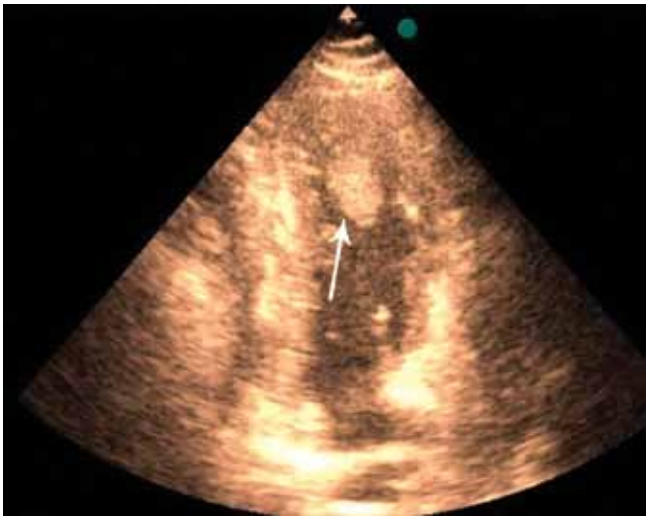


Figure 11. Modified two-dimensional echocardiogram in the apical two-chamber view, revealing a large left ventricular apical clot (arrow) following a recent anterior myocardial infarction

Acute Dynamic Left Ventricular Outflow Tract Obstruction

A dynamic LV outflow tract obstruction is an uncommon complication of an acute anterior STEMI, as was first described in a case report by Bartunek and associates.[54] The occurrence of a dynamic LV outflow obstruction has been reported in numerous reports; still, its true incidence is not obvious, in spite of the fact that it may be significantly unrecognized. It is dependent on the compensatory hyperkinesis of the basal and mid segments of the LV in patients with apical infarcts. The increased contractile force of these regions decreases the cross-sectional area of the LV outflow tract. The resulting increased velocity of the blood through the outflow tract can produce a decreased pressure below the mitral valve and lead to the anterior displacement of the leaflet toward the septum (Venturi effect). This creates a further outflow tract obstruction as well as mitral regurgitation because of the systolic anterior motion of the anterior mitral leaflet. It has been postulated that this complication can play a role in a free wall rupture secondary to increased end-systolic intraventricular pressure.

Echocardiography is the method of choice for the diagnosis and accurately reveals the hyperkinetic segment and the LV outflow tract obstruction as well as the systolic anterior motion of the mitral leaflet (Figure 12).[39, 55]

Post-Infarction Pericarditis

Post-infarction pericarditis most often occurs between three and ten days in the wake of a Q wave MI with a mean incidence of 25% and is less common when thrombolytic

therapy has been employed.[32] An echocardiogram is often ordered in patients in whom pericarditis is suspected. No echocardiographic feature is diagnostic of the disease. Echocardiography is a sensitive technique for the diagnosis of pericardial effusion along with pericarditis; the absence of fluid, however, does not exclude pericarditis.[38]

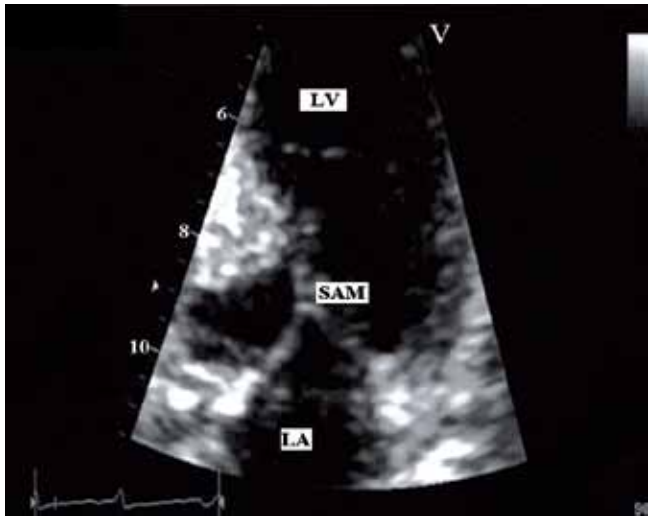


Figure 12. Two-dimensional echocardiogram in the apical four-chamber view, showing left ventricular outflow tract obstruction and systolic anterior motion after an acute anterior myocardial infarction
LV, Left ventricle; SAM, Systolic anterior motion; LA, Left atrium

Post-myocardial Infarction Risk Stratification

Left Ventricular Remodeling

After a transmural MI, alterations in the LV structure and function bring about the progressive dilation of the LV and the impairment of the systolic function, which is commonly referred to as LV remodeling. As the LV dilates and becomes more spherical, the LVEF reduces, and the papillary muscles are displaced more apically and laterally, permitting a significant rise in the degree of mitral regurgitation, all of which result in the exacerbation of heart failure and a rise in the mortality rate.

The early phase of LV remodeling consists primarily of infarct expansion and is only limited to the infarct zone, whereas in the late phase, which may persist for several months, the entire myocardium is liable to change. The magnitude and duration of the remodeling are determined not only by the size and location of the initial infarct, but also by the patency and restoration of the flow in the infarct-related artery and the ability to form stable scars.[56, 57]

Echocardiography provides crucial information for predicting ventricular remodeling and functional recovery, LV size and volume, regional wall motion abnormality, myocardial viability, LV filling pressures, severity of mitral regurgitation, and systolic pulmonary artery pressure.

Ischemic Mitral Regurgitation

Functionalmitral regurgitation occurs when the leaflets and chordae are relatively normal, but systolic coaptation and apposition of the leaflets are lessened[57, 58] (Figure 13). In patients with coronary artery disease, the posterior leaflet motion in systole is restricted or tethered secondary to the inadequate contraction of the posterolateral wall. The resulting malcoaptation and malapposition is allied to a posteriorly directed mitral regurgitation jet.[60-63] The dilation of the mitral annulus also may contribute to the development of mitral regurgitation. In patients with ischemic heart disease, functional mitral regurgitation is associated principally with an inferior MI and the lateral displacement of the posterior papillary muscle.[58] It is also worthy of note that significant ischemic mitral regurgitation is correlated with a poor outcome.[64]

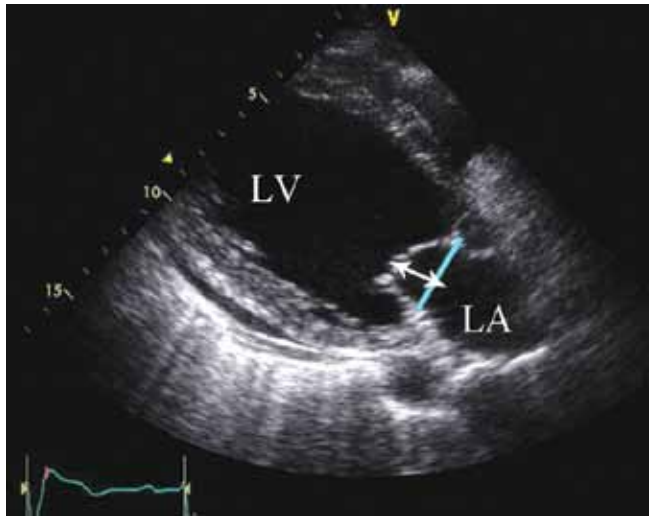


Figure 13. Illustration of the mechanism of functional mitral regurgitation. Echocardiographic images in the parasternal long-axis view at end-systole in a patient with dilated cardiomyopathy and severe mitral regurgitation. The mitral annulus plane is indicated by the line. Notice that the leaflets are "tented" at end-systole in the patient with dilated cardiomyopathy, with a greater distance between the annulus plane and leaflet closure line (arrow)
LV, Left ventricle; LA, Left atrium

Stress Echocardiography

Echocardiography is a valuable tool for the assessment of the cardiac structure and function in patients with coronary artery disease. Regional wall motion abnormalities correlate well with the significant stenosis of the coronary arteries; this becomes more evident during stress.

Another supplement to the routine exercise stress test is Stress Echocardiography. Stress (exercise or pharmacological)

Echocardiography can be employed to demonstrate the presence of coronary artery disease by the induction of wall motion abnormalities. Stress Echocardiography enhances our interpretation of the Exercise Stress Test and has been utilized for the evaluation of the functional importance of coronary artery disease[65] and for risk stratification in patients with known or suspected coronary disease.

Both exercise and pharmacologic stress echocardiographic examinations are well accepted and well tolerated by patients. Stress Echocardiography is suitable for symptomatic patients with an intermediate pretest probability of coronary artery disease and contraindication to regular treadmill stress testing.[65]

Choosing the type of a stress test is based upon the patient's ability to perform the exercise protocol, presence of baseline electrocardiographic abnormalities that could interfere with the interpretation of the exercise ECG, preoperative risk stratification prior to non-cardiac surgery, and whether or not we want to localize ischemia or assess myocardial viability. Still, many patients are unable to exercise maximally for stress testing due to a variety of conditions, including arthritis, severe lung disease, severe cardiac disease, orthopedic conditions, and diseases of the nervous system. In such patients, pharmacological stress testing is often employed.

Dobutamine Stress Echocardiography for Evaluation of Hibernating Viable Myocardium

Echocardiography is useful for the evaluation of myocardial viability and the demonstration of the magnitude of recovery after revascularization. Pharmacological Stress Echocardiography examines the «inotropic reserve» of the dysfunctional but viable myocardium through the administration of an inotropic agent. (Dobutamine is the most frequently used agent.) In response to inotrope administration, the viable myocardium exhibits an improved global or regional contractile function (inotropic reserve), which is assessed via simultaneous TTE.[66] For a contractile response to Dobutamine, at least 50% of the myocytes in a segment should be viable.[67] In patients with chronic coronary artery disease and LV dysfunction, Sadeghian et al.[68] suggested that a higher degree of myocyte functional integrity was required for a positive inotropic response to adrenergic stimulation than that responsible for Tc99m-Sestamibi uptake.[68]

Conclusion

Echocardiography is an accurate tool for the evaluation of patients with known or suspected coronary artery disease. Every patient should be evaluated clinically for pretest probability of coronary artery disease and risk of future cardiac events. Patients with intermediate and high pretest probability could potentially benefit from testing for either diagnosis or prognosis.

Acknowledgment

We sincerely thank Miss Esmaeili Darabi for drawing the figures.

References

1. Kontoyannis DA, Anastasiou-Nana MI, Kontoyannis SA, Zaga AK, Nanas JN. Intravenous amiodarone decreases the duration of atrial fibrillation associated with acute myocardial infarction. Cardiovasc Drugs Ther 2001;15:155-160.
2. Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. Circulation 2002;105:539-542.
3. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, Gutgesell H, Reichek N, Sahn D, Schnittger I. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989;2:358-367.
4. Bhatnagar SK, Al-Yusuf AR. Significance of early two-dimensional echocardiography after acute myocardial infarction. Int J Cardiol 1984;5:575-584.
5. Loh IK, Charuzi Y, Beeder C, Marshall LA, Ginsburg JH. Early diagnosis of nontransmural myocardial infarction by two-dimensional echocardiography. Am Heart J 1982;104:963-968.
6. Deutsch HJ, Curtius JM, Leischik R, Sechtem U, Maschler C, de Vivie ER, Hilger HH. Reproducibility of assessment of left-ventricular function using intraoperative transesophageal echocardiography. Thorac Cardiovasc Surg 1993;41:54-58.
7. Otto CM, Stratton JR, Maynard C, Althouse R, Johannessen KA, Kennedy JW. Echocardiographic evaluation of segmental wall motion early and late after thrombolytic therapy in acute myocardial infarction: the Western Washington Tissue Plasminogen Activator Emergency Room Trial. Am J Cardiol 1990;65:132-138.
8. Urheim S, Edvardsen T, Torp H, Angelsen B, Smiseth OA. Myocardial strain by Doppler echocardiography. Validation of a new method to quantify regional myocardial function. Circulation 2000;102:1158-1164.
9. Giusca S, Jurcut R, Ticulescu R, Dumitru D, Vladaia A, Savu O, Voican A, Popescu BA, Ginghina C. Accuracy of handheld echocardiography for bedside diagnostic evaluation in a tertiary cardiology center: comparison with standard echocardiography. Echocardiography 2011;28:136-141.
10. Behar V, Adam D, Lysyansky P, Friedman Z. The combined effect of nonlinear filtration and window size on the accuracy of tissue displacement estimation using detected echo signals. Ultrasonics 2004;41:743-753.
11. Notomi Y, Lysyansky P, Setser RM, Shiota T, Popović ZB, Martin-Miklovic MG, Weaver JA, Oryszak SJ, Greenberg NL, White RD, Thomas JD. Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. J Am Coll Cardiol 2005;45:2034-2041.
12. Amundsen BH, Helle-Valle T, Edvardsen T, Torp H, Crosby J, Lyseggen E, Støylen A, Ihlen H, Lima JA, Smiseth OA, Slørdahl SA. Noninvasive myocardial strain measurement by speckle tracking echocardiography: validation against sonomicrometry

and tagged magnetic resonance imaging. J Am Coll Cardiol 2006;47:789-793.
13. Sabia P, Abbott RD, Afrookteh A, Keller MW, Touchstone DA, Kaul S. Importance of two-dimensional echocardiographic assessment of left ventricular systolic function in patients presenting to the emergency room with cardiac-related symptoms. Circulation 1991;84:1615-1624.
14. Peels CH, Visser CA, Kupper AJ, Visser FC, Roos JP. Usefulness of two-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. Am J Cardiol 1990;65:687-691.
15. Lieberman AN, Weiss JL, Jugdutt BI, Becker LC, Bulkley BH, Garrison JG, Hutchins GM, Kallman CA, Weisfeldt ML. Two-dimensional echocardiography and infarct size: relationship of regional wall motion and thickening to the extent of myocardial infarction in the dog. Circulation 1981;63:739-746.
16. O'Boyle JE, Parisi AF, Nieminen M, Kloner RA, Khuri S. Quantitative detection of regional left ventricular contraction abnormalities by 2-dimensional echocardiography. Comparison of myocardial thickening and thinning and endocardial motion in a canine model. Am J Cardiol 1983;51:1732-1738.
17. Ideker RE, Behar VS, Wagner GS, Starr JW, Starmer CF, Lee KL, Hackel DB. Evaluation of asynergy as an indicator of myocardial fibrosis. Circulation 1978;57:715-725.
18. Horowitz RS, Morganroth J, Parrotto C, Chen CC, Soffer J, Pauletto FJ. Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography. Circulation 1982;65:323-329.
19. Kerber RE, Marcus ML, Ehrhardt J, Wilson R, Abboud FM. Correlation between echocardiographically demonstrated segmental dyskinesis and regional myocardial perfusion. Circulation 1975;52:1097-1104.
20. Wyatt HL, Meerbaum S, Heng MK, Rit J, Gueret P, Corday E. Experimental evaluation of the extent of myocardial dyssynergy and infarct size by two-dimensional echocardiography. Circulation 1981;63:607-614.
21. Pandian NG, Koyanagi S, Skorton DJ, Collins SM, Eastham CL, Kieso RA, Marcus ML, Kerber RE. Relations between 2-dimensional echocardiographic wall thickening abnormalities, myocardial infarct size and coronary risk area in normal and hypertrophied myocardium in dogs. Am J Cardiol 1983;52:1318-1325.
22. Hutchins GM, Bulkley BH, Ridolfi RL, Griffith LS, Lohr FT, Piasio MA. Correlation of coronary arteriograms and left ventriculograms with postmortem studies. Circulation 1977;56:32-37.
23. Nieminen M, Parisi AF, O›Boyle JE, Folland ED, Khuri S, Kloner RA. Serial evaluation of myocardial thickening and thinning in acute experimental infarction: identification and quantification using two-dimensional echocardiography. Circulation. 1982;66:174-180.
24. McGillem MJ, Mancini GB, DeBoe SF, Buda AJ. Modification of the centerline method for assessment of echocardiographic wall thickening and motion: a comparison with areas of risk. J Am Coll Cardiol 1988;11:861-866.
25. Kozàkovà M, Palombo C, Distante A. Right ventricular infarction: the role of echocardiography. Echocardiography 2001;18:701-707.
26. Cecchi F, Zuppiroli A, Favilli S, Di Bari M, Vannucci A, Pini R, Marchionni N. Echocardiographic features of right ventricular infarction. Clin Cardiol 1984;7:405-412.
27. Ozdemir K, Altunkeser BB, Içli A, Ozdil H, Gök H. New parameters in identification of right ventricular myocardial infarction and proximal right coronary artery lesion. Chest 2003;124:219-226.
28. Goldberger JJ, Himelman RB, Wolfe CL, Schiller NB. Right ventricular infarction: recognition and assessment of its hemodynamic significance by two-dimensional echocardiography. J Am Soc Echocardiogr 1991;4:140-146.
29. Goldstein JA. Pathophysiology and management of right heart ischemia. J Am Coll Cardiol 2002;40:841-853.
30. Forrester JS, Diamond GA, Swan HJ. Correlative classification of clinical and hemodynamic function after acute myocardial infarction. Am J Cardiol 1977;39:137-145.
31. Gerber IL, Foster E. Echocardiography in the coronary care unit: management of acute MI, detection of complication, and prognostic implications. In: Otto MC, ed. The Practice of Clinical Echocardiography. 3rd ed. Philadelphia/China: Elsevier; 2007. p. 305-315.
32. Hozumi T, Yoshida K, Mori I, Akasaka T, Takagi T, Kaji S, Kawamoto T, Ueda Y, Morioka S. Noninvasive assessment of hemodynamic subsets in patients with acute myocardial infarction using digital color Doppler velocity profile integration and pulmonary venous flow analysis. Am J Cardiol 1999;83:1027-1032.
33. Rahman N, Kazmi KA, Yousaf M. Non-invasive prediction of ST elevation myocardial infarction complications by left ventricular Tei index. J Pak Med Assoc 2009;59:75-78.
34. Antman EM, Morrow DA. ST-segment elevation myocardial infarction: management. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald›s Heart Disease. 9th ed. Philadelphia/China: Elsevier. executive publisher: Natasha Andjelkovic; 2012. p. 1147-1150.
35. Hass EE, Yang EH, Gersh BJ, Robert A. O'Rourke. ST-segment-elevation MI: clinical presentation, diagnostic evaluation, and medical management. In: Foster V, Walsh R, Harrington R, eds. Hurst's the Heart. 13th ed. New York/Chicago/San Francisco/Lisbon/London/Madrid/Mexico City Milan/New Delhi/SanJuan/Seoul/Singapore/Sydney/Toronto: McGraw-Hill; 2011. p. 1354-1385.
36. Becker RC, Gore JM, Lambrew C, Weaver WD, Rubison RM, French WJ, Tiefenbrunn AJ, Bowlby LJ, Rogers WJ. A composite view of cardiac rupture in the United States National Registry of Myocardial Infarction. J Am Coll Cardiol 1996;27:1321-1326.
37. Hrovatin E, Piazza R, Pavan D, Mimo R, Macor F, Dall'Aglio V, Burelli C, Cassin M, Canterin FA, Brieda M, Vitrella G, Gilberto C, Nicolosi GL. Dynamic left ventricular outflow tract obstruction in the setting of acute anterior myocardial infarction: a serious and potentially fatal complication? Echocardiography 2002;19:449-455.
38. Greaves SC. Role of echocardiography in acute coronary syndromes. Heart 2002;88:419-425.
39. Dencker M, Tasevska G, Grubb D, Stagmo M, Gustafsson R. Unexpected rupture of the left ventricular free wall in the echo-lab. Eur J Echocardiogr 2008;9:92-94.
40. Morimoto H, Mukai S, Obata S, Fujiwara Y. Left ventricular free wall rupture after coronary artery bypass grafting. Eur J Cardiothorac Surg 2012;41:707.
41. Esmaeilzadeh M, Mirdamadi A, Kiavar M, Omrani G. Timely diagnosis of left ventricular posterior wall rupture by echocardiography: a case report. J Teh Univ Heart Ctr 2010;5:36-38.
42. Figueras J, Barrabés JA, Serra V, Cortadellas J, Lidón RM, Carrizo A, Garcia-Dorado D. Hospital outcome of moderate to severe pericardial effusion complicating ST-elevation acute myocardial infarction. Circulation 2010;122:1902-1909.
43. Matoh F, Satoh H, Shiraki K, Odagiri K, Saitoh T, Urushida T, Katoh H, Takehara Y, Sakahara H, Hayashi H. The usefulness of delayed enhancement magnetic resonance imaging for diagnosis and evaluation of cardiac function in patients with cardiac sarcoidosis. J Cardiol 2008;51:179-188.
44. Shiozaki AA, Filho RA, Dallan LA, de Oliveira SA, Nicolau JC, Rochitte CE. Left ventricular free-wall rupture after acute myocardial infarction imaged by cardiovascular magnetic resonance. J Cardiovasc Magn Reson 2007;9:719-721.
45. Armstrong WF, Ryan T. Echocardiography and coronary artery disease. In: Armstrong WF, Ryan T, eds. Feigenbaum's Echocardiography. 7th ed. Philadelphia/Baltimore/New York/London/Buenos aires/hong kong/Sydney/Tokyo: Lippincott Williams & wilkins; 2010. p. 427-472.
46. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med

2002;347:1426-1432.
47. Gueret P, Khalife K, Jobic Y, Fillipi E, Isaaz K, Tassan-Mangina S, Baixas C, Motreff P, Meune C; Study Investigators. Echocardiographic assessment of the incidence of mechanical complications during the early phase of myocardial infarction in the reperfusion era: a French multicentre prospective registry. Arch Cardiovasc Dis 2008;101:41-47.
48. Donoiu I, Istratoaie O, Ionescu DD. Ventricular septal rupture after acute myocardial infarction. Hellenic J Cardiol 2010;51:374-376.
49. Koh AS, Loh YJ, Lim YP, Le Tan J. Ventricular septal rupture following acute myocardial infarction. Acta Cardiol 2011;66:225-230.
50. Roberts JD, So DY, Lambert AS, Ruel M. Successful surgical repair of ventricular double rupture. Can J Cardiol 2011;27:868. e5-7.
51. Sadeghian H, Abbasi K, Moshtaghi N, Shirzad M, Majidi S, Abbasi SH, Semnani M, Haji Zeinali AM, Sahebjam M, Kassaian SE. Free wall rupture and ventricular septal defect post acute anterior myocardial infarction. J Teh Univ Heart Ctr 2007;2:111-113.
52. Wilansky S. Echocardiography in the assessment of complications of myocardial infarction. Tex Heart Inst J 1991;18:237-242.
53. Esmaeilzadeh M, Taghavi S, Bassiri HA. Contrast echocardiography for diagnosis of apical hypertrophic cardiomyopathy. Iranian Heart J 2010;11:37-39.
54. Bartunek J, Vanderheyden M, de Bruyne B. Dynamic left ventricular outflow tract obstruction after anterior myocardial infarction. A potential mechanism of myocardial rupture. Eur Heart J 1995;16:1439-1442.
55. Ballal RS, Sanyal RS, Nanda NC, Mahan EF, 3rd. Usefulness of transesophageal echocardiography in the diagnosis of ventricular septal rupture secondary to acute myocardial infarction. Am J Cardiol 1993;71:367-370.
56. Di Chiara A, Werren M, Badano LP, Fioretti PM. Dynamic left ventricular outflow tract obstruction: an unusual mechanism mimicking anterior myocardial infarction with cardiogenic shock. Ital Heart J 2001;2:60-67.
57. St John Sutton M, Scott CH. A prediction rule for left ventricular dilatation post-MI? Eur Heart J 2002;23:509-511.
58. Otsuji Y, Handschumacher MD, Schwammenthal E, Jiang L, Song JK, Guerrero JL, Vlahakes GJ, Levine RA. Insights from three-dimensional echocardiography into the mechanism of functional mitral regurgitation: direct in vivo demonstration of altered leaflet tethering geometry. Circulation 1997;96:1999-2008.
59. Kwan J, Shiota T, Agler DA, Popović ZB, Qin JX, Gillinov MA, Stewart WJ, Cosgrove DM, McCarthy PM, Thomas JD; Real-time three-dimensional echocardiography study. Geometric differences of the mitral apparatus between ischemic and dilated cardiomyopathy with significant mitral regurgitation: real-time three-dimensional echocardiography study. Circulation 2003;107:1135-1140.
60. Levine HJ, Gaasch WH. Vasoactive drugs in chronic regurgitant lesions of the mitral and aortic valves. J Am Coll Cardiol 1996;28:1083-1091.
61. Otsuji Y, Handschumacher MD, Liel-Cohen N, Tanabe H, Jiang L, Schwammenthal E, Guerrero JL, Nicholls LA, Vlahakes GJ, Levine RA. Mechanism of ischemic mitral regurgitation with segmental left ventricular dysfunction: three-dimensional echocardiographic studies in models of acute and chronic progressive regurgitation. J Am Coll Cardiol 2001;37:641-48.
62. Levine RA, Schwammenthal E. Ischemic mitral regurgitation on the threshold of a solution: from paradoxes to unifying concepts. Circulation 2005;112:745-758.
63. Gorman JH, 3rd, Jackson BM, Gorman RC, Kelley ST, Gikakis N, Edmunds LH, Jr. Papillary muscle discoordination rather than increased annular area facilitates mitral regurgitation after acute posterior myocardial infarction. Circulation 1997;96:II-124-127.
64. Grigioni F, Detaint D, Avierinos JF, Scott C, Tajik J, Enriquez-Sarano M. Contribution of ischemic mitral regurgitation to congestive heart failure after myocardial infarction. J Am Coll Cardiol 2005;45:260-267.
65. Fleischmann KE, Hunink MG, Kuntz KM, Douglas PS. Exercise echocardiography or exercise SPECT imaging? A meta-analysis of diagnostic test performance. JAMA 1998;280:913-920.
66. Main ML, Grayburn PA, Landau C, Afridi I. Relation of contractile reserve during low-dose dobutamine echocardiography and angiographic extent and severity of coronary artery disease in the presence of left ventricular dysfunction. Am J Cardiol 1997;79:1309-1313.
67. Baumgartner H, Porenta G, Lau YK, Wutte M, Klaar U, Mehrabi M, Siegel RJ, Czernin J, Laufer G, Sochor H, Schelbert H, Fishbein MC, Maurer G. Assessment of myocardial viability by dobutamine echocardiography, positron emission tomography and thallium-201 SPECT: correlation with histopathology in explanted hearts. J Am Coll Cardiol 1998;32:1701-1708.
68. Sadeghian H, Majd-Ardakani J, Lotfi-Tokaldany M, Jahangiri C, Fathollahi MS. Comparison between dobutamine stress echocardiography and myocardial perfusion scan to detect viable myocardium in patients with coronary artery disease and low ejection fraction. Hellenic J Cardiol 2009;50:45-51.