Myocardial Wall Thickness Predicts Recovery of Contractile Function After Primary Coronary Intervention for Acute Myocardial Infarction

Thesis

Submitted for partial fulfillment of master

Degree in cardiology

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2007

سمك عضلة القلب يتنبأ استعادة الوظيفة الإنقباضية بعد معالجة الإحشاء الحاد لعضلة القلب بالقسطرة الأولية التداخلية

الرسالة

مقدمة للتوطئة للحصول على درجة ماجستير القلب والاوعية الدموية

بواسطة

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2007

Introduction

Coronary artery disease is nowadays, considered one of the leading causes of death in the developed countries ***(Murray and Lopez, 1997)***. After myocardial infarction, limitation of the infarct size exhibit an improvement of left ventricular function and long term mortality ***(Holmes, 2003)***.

Modification of the infarct size can be carried out by several approaches including primary Percutanous Coronay Intervention (PCI) ***(Antman, 2005)***.

Early restoration of epicardial coronary blood flow reduces infarct size in patients with acute myocardial infarction and has a beneficial effect on post infarction myocardial infarct healing and left ventricular remodeling ***(Keely et al., 2003)*.**

However in some patients successful PCI does not result in recovery of contractile function in the infarct territory ***(Simes R J, 1995)***, apparently because of subsequent left ventricular remodeling despite spontaneous recovery of regional and global left ventricular (LV) function suggesting that the incidence of post myocardial infarction (MI) LV dilatation is independent of which reperfusion strategy has been used ***(De Luca et al., 2006).***

Because myocardial necrosis is associated with myocardial thinning, preserved end diastolic wall thickness (EDWT) may provide a simple index of myocardial viability that is readily available from the resting echocardiogram ***(Biagini et al., 2004).***

Aim of the Work

We are seeking to determine whether end-diastolic wall thickness can predict recovery of left ventricular contractile function after primary percutanous coronary intervention (PCI).

Echocardiography in Ischemic Heart Disease

Echocardiography and Coronary artery disease

Ischemic Heart Disease (IHD) remains a major health problem and number one killer world wide. With the aging population and increasing survival rate of acute Myocardial Infarction, overall burden of chronic IHD is on the rise. It has been reported that for every recognised heart attack there are 30 cases of chronic IHD. In fact stable angina is the principal manifestation of IHD in 60% of cases.

Such cases are commonly evaluated by the resting ECG and chest radiograph. This does not give a detailed insight into the quantitative aspect of disease, functional status, complications and prognostic considerations. Echocardiography could be very useful for the evaluation of chronic IHD and this modality needs to be more utilised in such clinical condition ***(Ahmad R et al., 2009).***

Pathophysiology of Coronary Syndromes

Normal left ventricular wall motion consists of simultaneous wall thickening and endocardial excursion so that the cavity decreases in size in a relatively symmetric manner. Interruption of contraction, due to ischemia, infarction, or other process, results in regional abnormalities of motion.

There is a well-defined hierarchy of functional abnormalities that occur as a consequence of interruption in coronary blood flow. Resting blood flow to the myocardium is preserved until a coronary artery stenosis approaches 90% diameter narrowing. It should be emphasized that simple diameter narrowing is only one component of the anatomic

abnormality that results in reduced coronary flow, and lesion eccentricity, length, and number of sequential lesions all play crucial roles. At lesser degrees of stenosis, rest flow is preserved, but coronary flow reserve may be reduced, and at times of increasing demand such as exercise, a supply-demand mismatch occurs. Creation and detection of a supply-demand mismatch in the presence of an otherwise non-flow obstructive lesion are the underlying principles of stress echocardiography and other stress-testing techniques designed to unmask occult coronary artery stenosis ***(Feigenbaum et al., 2005)***.

Depending on the presence or absence of collaterals and the duration of occlusion, a series of identifiable wall motion abnormalities can be noted. Obviously, if flow is not restored, myocardial necrosis occurs and a wall motion abnormality persists as a permanent feature of ventricular function. If flow is restored before the onset of myocardial necrosis, variable degrees of recovery of function can be expected. In most instances, a total occlusion of 4 to 6 hours will result in irreversible loss of transmural myocardium. Total interruption for less than 60 minutes will result in lesser degrees of loss of myocardium. In between these two extremes of flow interruption, varying degrees of nontransmural necrosis occur. This predominantly involves the subendocardial layers of the myocardium. As is discussed subsequently, the severity, nature, and extent of wall motion abnormalities depend on the amount of transmural versus nontransmural infarction present in a given segment ***(Feigenbaum et al., 2005).***

If a substantial period of ischemia has occurred, as may be seen in transient occlusion of 40 to 120 minutes, recovery of function may not be immediate but rather delayed due to myocardial stunning. Myocardial stunning is a phenomenon easily demonstrated on echocardiography and represents persistent wall motion abnormalities after restitution of coronary blood flow. These abnormalities recover over a

variable time period. Typically, with brief occlusions of 5 minutes or less, recovery of function occurs within 60 to 120 seconds. With coronary occlusions of 30 to 120 minutes, there may be a 48- to 72-hour delay in recovery of function. There is a substantial degree of variability in the time course over which stunning recovers, and in clinical practice, recovery of function occasionally is delayed for weeks to months ***(Feigenbaum et al., 2005)***.

A phenomenon of repetitive stunning has also been well described. In this scenario, the myocardium is subject to repetitive, brief episodes of ischemia. No single episode of ischemia is sufficient to result in postischemic dysfunction; however, the combined effect of multiple episodes over time may result in prolonged postischemic dysfunction that mimics myocardial hibernation ***(Feigenbaum et al., 2005)***.

After complete coronary occlusion and transmural infarction, a series of events known as remodeling occurs. Over a period of roughly 6 weeks, the necrotic myocardium is replaced by fibrosis and scar tissue, which is thinner and denser than normal myocardium but which has similar tensile strength, rendering it unlikely to rupture. There may be regional dilation in the area of the scar that results in a ventricular aneurysm (Figs. 1 and 2). An aneurysm is defined as a regional area of akinesis or dyskinesis and scar that has abnormal geometry in both diastole and systole. This is in contrast to a regional wall motion abnormality that has normal geometry in diastole and the distortion occurs exclusively in systole ***(Feigenbaum et al., 2005)***.

On occasion, there can be acute remodeling in an infarct segment that results in expansion of the myocardium in that area. Myocardial expansion occurs typically in the first 48 hours after extensive transmural myocardial infarction and represents acute thinning of the infarcted myocardium. Because expansion occurs acutely, there is no time for scar

formation or gradual remodeling, and, as such, the wall in the area of myocardial expansion consists of relatively thin necrotic myocardium with reduced tensile strength. Myocardial infarct expansion typically is heralded by new electrocardiographic changes and pain but without enzymatic evidence of further necrosis. It is the anatomic substrate for free-wall rupture, ventricular septal defect, and other mechanical complications of myocardial infarction ***(Feigenbaum et al., 2005)***.

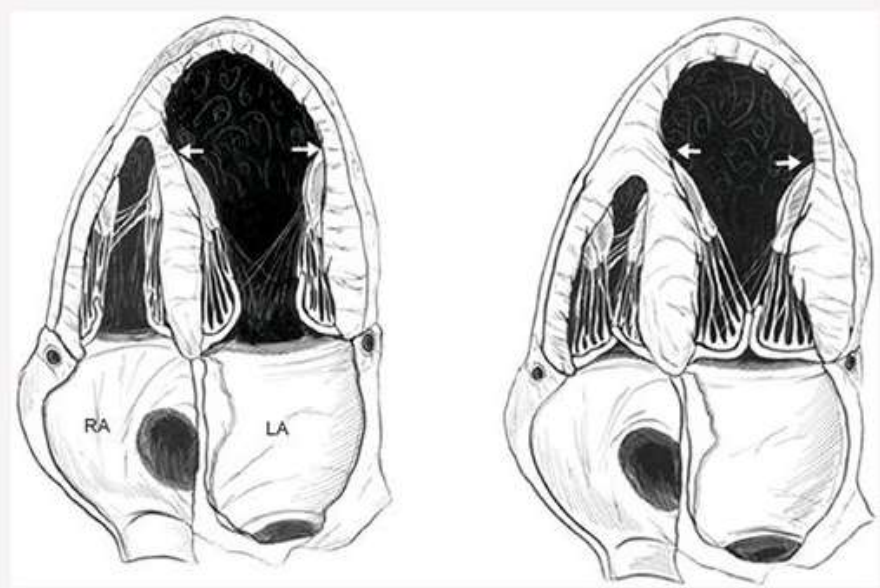

Fig (1): Anatomic rendering in the four-chamber view depicts a left ventricular apical aneurysm. Left: Diastole. Right: Systole. Note in diastole the abnormal geometry of the apex with localized apical and septal dilation and the relative thinning of the wall compared with the thickness in the proximal walls. Right: The preserved thickening of the proximal walls and a lack of thickening in the aneurysmal segment in all segments distal to the arrows are shown. This abnormal geometry in both diastole and systole with wall thinning is the hallmark of true vetricular aneurysm. LA, left atrium; RA, right atrium. ***(Feigenbaum et al., 2005).***

Although the location of a wall motion abnormality is an accurate marker for the site of ischemia or infarction, the extent of the wall motion abnormality often over estimates the anatomic extent of ischemia or infarction. This is in large part due to tethering. Myocardial tethering refers to the impact that an abnormal segment has on a normal adjacent border segment. Tethering occurs both on a horizontal and vertical basis. Horizontal tethering occurs when there is akinesis or dyskinesis of a segment that then results in the reduction in endocardial excursion in the adjacent functionally normal boundary tissue. The effect of horizontal or lateral tethering is for the extent of a wall motion abnormality to over represent the anatomic circumferential extent of myocardial necrosis because the detected wall motion abnormality includes not only the infarcted tissue but also a variable percentage of the immediately adjacent boundary tissue. Generally, the wall motion abnormality will overestimate the anatomic extent of a myocardial infarction by approximately 15% due to this phenomenon. The impact that tethering has on quantification of infarct size is demonstrated subsequently ***(Feigenbaum et al., 2005)***.

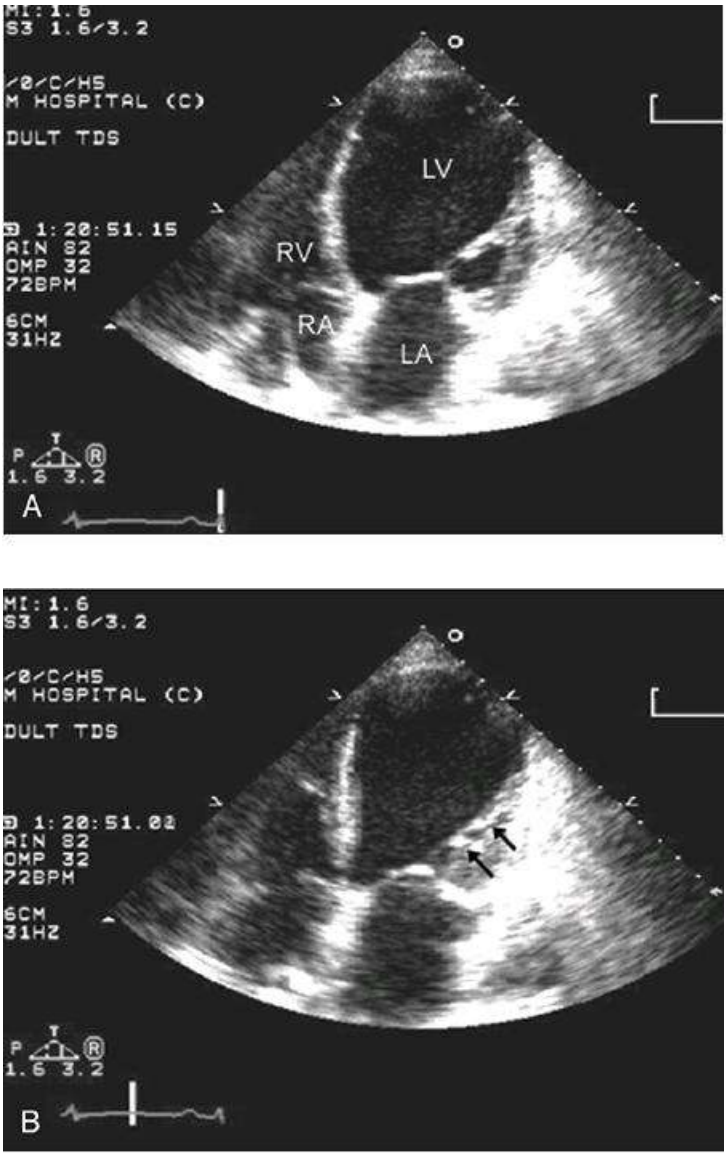

Fig. (2): Apical four-chamber view recorded in a patient with a large apical and septal aneurysm. This two-dimensional echocardiogram corresponds to the anatomic rendering in Fig. 5. As with the anatomic rendering, note the abnormal geometry of the left ventricle in both diastole (A) and systole (B) with dilation in the apical segments. In systole, only the proximal lateral wall (arrows) has preserved contraction. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle ***(Feigenbaum et al., 2005)***.

Normal contraction is heavily dependent on endocardial rather than epicardial contraction. Both the velocity and magnitude of contraction are greater in the subendocardial than in the subepicardial layers. As such, a contraction abnormality in the subendocardium has a disproportionate impact on overall wall thickening. This phenomenon is known as vertical tethering. Vertical tethering has been demonstrated both experimentally and clinically and has relevance for the determination of myocardial infarction size, based on wall motion abnormalities. In general, ischemia or infarction of the inner 20% of the myocardial wall will result in frank akinesis or dyskinesis of that segment. As such, nontransmural involvement (either infarction or ischemia) results in malfunction of the entire wall thickness, and thus the wall motion abnormality is indistinguishable from that seen with full transmural myocardial infarction or ischemia ***(Feigenbaum et al., 2005)***.

Detection and Quantification of Wall Motion Abnormalities

Regional left ventricular wall motion and global ventricular function can be analyzed and quantified using a number of schemes. These can be classified as purely qualitative, semiquantitative, and quantitative assessments. Table 1 outlines many of the schemes that are either commonly used today or have been proposed in the past for evaluation of regional wall motion abnormalities. Although detailed quantitative schemes, which measure regional or global function as a percentage of anticipated normal, may be useful for serial studies and investigational protocols, they are not necessary for detection and localization of an ischemic event for clinical diagnosis. A compromise that allows semiquantitation and that can be employed easily is the generation of a wall motion score. The wall motion score is a unit less hierarchical number directly proportional to the

severity and magnitude of wall motion abnormalities ***(Feigenbaum et al., 2005)***.

Table (1): Wall Motion Analysis Methods

Regional
Qualitative
- Eyeballing assessment.
- Normal vs. abnormal.
- Normal hypokinetic akinetic dyskineti.
- Presence of scar/aneurysm.
- Semiquantitative.
- Wall motion score/score index
Quantitative
- Fractional shortening.
- Radial shortening.
- Cavity/fractional cavity area change.
- Chordal centerline analysis
Doppler tissue based
- Wall velocity.
- Myocardial displacement.
- Myocardial gradient.
- Strain.
- Strain rate.
- Global.
- Ventricular geometry.
- Short-axis area change.
- Left ventricular volumes.
- Diastole.
- Systole.
- Ejection fraction.
- Doppler forward flow (TVILVOT).
- Anular displacement (DTI).
- Myocardial performance index.
- Left ventricular dP/dt (from mitral regurgitation)

DTI: Doppler tissue imaging; TVI_{LVOT}: Doppler time velocity integral in the left ventricular outflow tract.

M-mode left ventricular measurements provide only limited information on patients with coronary artery disease, not only because of the regional nature of the left ventricular function but also because the M-mode dimensions are not true minor axis measurements. On the other hand, linear two-dimensional measurements still can be very valuable. The linear minor-axis dimension between the posterior left ventricular endocardium and the septum at the level of the mitral chordae provides an assessment of left ventricular systolic function at the base of the heart. The proximal septum is perfused by the first septal perforator; thus, this measurement in systole and diastole and resultant fractional shortening is a good indication of whether a left anterior descending coronary obstruction is proximal or distal to the first septal perforator ***(Feigenbaum et al., 2005)***.

Two-dimensional imaging has replaced M-mode echocardiography for evaluation of global and regional wall motion. The most commonly used method for volume determination is the Simpson rule or the rule of disks, in which the endocardial border in diastole and systole is outlined, and then mathematically a series of disks of identical height, each of which corresponds to one of the minor-axis dimensions of the ventricle, is generated. The volume of each individual disk is then summed to provide a volume. In any given view, a circular disk is assumed at each level along the ventricle. Obviously, if a regional wall motion abnormality is not visualized in the plane of examination, this technique will overestimate the ejection fraction. For this reason, when dealing with patients with coronary disease in whom regional abnormalities are anticipated, biplane methodology is necessary if precise measurements are required. Because of the regional nature of coronary disease, other methods, such as area length calculations, have had less acceptance in evaluating patients with coronary disease ***(Feigenbaum et al., 2005)***.

A two-dimensional area measurement of the short axis at the papillary muscle level has some of the same limitations as M-mode dimensions. The advantage of the short-axis area is that there is less myocardial dropout than with the apical views, and all three coronary artery territories contribute to the measurement. Thus, the resultant fractional area change is a reasonable global assessment, unless there is isolated apical dysfunction ***(Feigenbaum et al., 2005)***.

Evaluation of regional left ventricular function is substantially more complex. There are multiple schemes for regional wall motion assessment (Table 1). The assessment can be undertaken on purely qualitative terms such as an eyeballing of wall motion as being normal or abnormal or further characterized as hypokinetic, akinetic, or dyskinetic, or undertaken in detailed quantitative schemes in which shortening of multiple endocardial chords around the circumference of the ventricular cavity is undertaken ***(Feigenbaum et al., 2005)***.

With the complex nature of myocardial contraction and wall motion abnormalities in mind, one can then employ several different algorithms for quantitation of wall motion abnormalities. These were outlined in Table 1. When dealing with coronary disease, it is imperative to adopt a regional approach to the description of wall motion abnormalities, whether that description is a highly detailed quantitative scheme or a simple "eyeball". (Fig. 3) schematizes the standard segments of the left ventricle that are commonly employed for analysis as well as the coronary arteries that usually perfuse those segments. Previous schemes employed a 16-segment model. More recently, a 17-segment approach has been recommended in which the 17^{th} segment represents the true apex. This approach allows a more precise correlation with the segments visualized and analyzed by competing imaging techniques. The new segmentation schematic renames