University Heart Journal
Vol. 13, No. 2, July 2017

REVIEW ARTICLE

Echocardiographic Assessment of Patient with Ischemic versus non-ischemic Dilated Cardiomyopathy

MOSTASHIRUL HAQUE, JAHANARA ARZU, CHAUDHURY MESHKAT AHMED, MSI TIPU CHOWDHURY,CHAYAN KUMAR SINGHA,FAISAL IBN KABIR, MD RASUL AMIN, RAYHAN MASUM MANDAL, MD. FAKHRUL ISLAM KHALED, ARIFUL ISLAM JOARDER, SM EAR-E-MAHBUB

Department of Cardiology, Bangabandhu sheikh Mujib Medical University (BSMMU), Dhaka

Address of correspondence: Dr. Mostashirul Haque, Assistant Professor, Department of Cardiology, University Cardiac Center, Bangabandhu Sheikh Mujib Medical University, Bangladesh. Email:mustashir.haque@yahoo.com

University Heart Journal 2017; 13(2): 62-64

Introduction:

Ischemic and non-ischemic cardiomyopathy (ICM and NICM) both cause heart failure, but the different etiologies may result in differences in management and outcome. Multiple trails and epidemiologic surveys have demonstrated that patients with ICM have decreased survival compared to patients with non-ischemic dilated cardiomyopathy (NIDCM). Increased age, multivessel arteriopathy, potent neurohormonal stimulation and arrhythmias associated with sudden death predispose patients with ICM to greater morbidity and mortality compared to patients with NIDCM.[1]

Discussion:

LV may be enlarged and show dysfunctional in both ischemic heart disease and dilated cardiomyopathy (DCM). Patients with ICM may benefit from a revascularization treatment strategy.[2] In clinical practice distinguishing between two types of conditions can be very challenging. In some situations, a diagnosis can be inferred from history and physical examination(e.g. Postpartum or chemotherapy induced NICM).Definition of ICM requires the identification of significant coronary artery disease (stenosis of any epicardial vessels more than 75%) or a history of myocardial infarction or previous revascularization of the coronaries in the presence of depressed LV ejection fraction (LVEF<45%).[3]

Coronary angiogram remains gold standard method of evaluating coronary artery disease. Nonetheless, coronary angiography is not without risks. It is invasive, operative dependent and may be associated with adverse events. A noninvasive imaging(e.g. echocardiogram) modality particularly in patients with low to intermediate pretest probability for coronary artery disease is often recommended.[4]

Two-dimensional echocardiography:

- Increased in ventricular chamber sizes with reduced indices of systolic function(LVEF <45% or Fractional shortening <25%) in the setting of normal or reduced LV thickness(Fig).
- An end systolic volume index > 60 ml/m^2 is associated with adverse outcomes in patients withICM.[5]
- RV dysfunction A TAPSE <15 mm-poor RV systolic function associated with increased mortality in patients admitted for heart failure.[6]
- LA size: Enlarged LA defined as a LA size indexed to body surface area of at least 2.4cm/m^2.LA size have prognostic value in patients with NICM. LA volume is determined by the degree of LV dilatation, diastolic dysfunction as well as severity of MR. LA size may

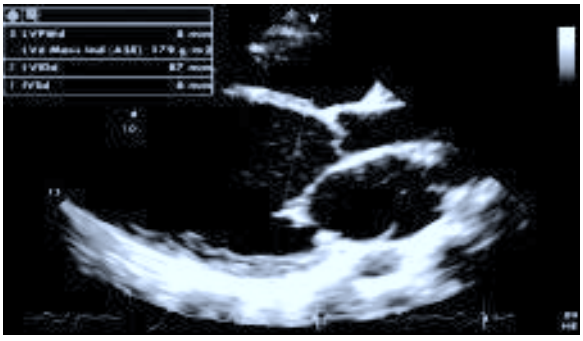

Fig: *Parasternal long axis view showing hugely dilated LV with thin interventricular septum and Increased tenting area >2.5 cm^2*

Received: 11 March, 2017 **Accepted:** 15 June, 2017

reflect the duration of MR in NICM patient with functional MR.[7]

The main mechanism of ischemic mitral regurgitation (IMR) is ventricular remodeling, which displaces the papillary muscles apically & laterally as a result of myocardial infarction of the segments underlying the papillary muscles, which in turn pulls the fixed length chordae tendinae & leaflets apically. The line of coaptation of the tethered mitral leaflets is also apically displaced & prevents normal valve closure. Remodeling begins within hours after Ml & may continue to progress if not arrested, culminating in heart failure. LV remodeling is defined as an increase in end diastolic volume (EDV) or end systolic volume (ESV) of 20% from baseline at the 6 month follow up. A large infarct size, transmurality (the evidenced by cardiac enzymes, extent of wall motion abnormalities & duration of ischemia) & infarct related vessel patency are the most important predictors for chronic LV remodeling.

LV ESV has en shown to have greater predictive value for survival as compared with EDV or EF in a large study of acute MI.[3]

M-mode echocardiography:

- Sphericity index(SI) is a surrogate marker of LV remodeling. It is the ratio of LV long axis internal dimension to LV diameter at end systole. A SI of <1.5 underscores a very severely dilated LV cavity.
- Tenting area: Patient with a tenting area >2.5 cm^2 indicating severe MR and independent predictor of mortality.
- E-point septal separation(EPSS): M-mode parameter which related to LV cavity dilatation and reduced LV systolic excursion. A normal value <6mm. EPSS of >10mm reflects severe LV dilatation and systolic dysfunction.[8]

Doppler study: Doppler measures of ventricular contractility are reduced in both ICM &NICM.

- LVOT flow velocity or VTI is usually decreased to < 18 cm.
- The dp/dt (the change in LV pressure over time), measure from MR jet. A value of<600 mmHg/s indicates significant impairment of LV contractility. A lowdp/dt is associated with adverse CV outcomes.[9]
- The myocardial performance index(MPI) or Tei index *:* Doppler derived measure of systolic and diastolic ventricular function. A calculated MPI value of >0.60 is correlated with adverse outcomes in both patients with NICM and ICM[10.]
- Functional MR is associated with poor prognosis.

Besides WMA, identification of regional thinning of myocardial wall less than 6 mm or aneurysmal myocardial segment corresponding to coronary blood flow area increases the likelihood of the diagnosis of ICM.

Myocardial wall motion abnormality was scored e.g. hyperkinesia =-1, normal=0, hypokinesia=1, akinesia=2, and dyskinesia= 3.Patients with a LVEF <50% had a mean score of 6.9 while those with a LVEF >50% had a mean score of 1.1[12]WMA may also seen in up to two-thirds of patients with NICM. These WMA have been attributed to abnormal microcirculatory perfusion despite normal epicardial vessels.

A dilated LV chamber in the setting of normal or reduced LV wall thickness(end diastolic wall thickness of <6mm) that is global in nature is highly indicative of NICM. In ICM there may be regional variation in thickness of the myocardium. Myocardial thickness of <6 mm end diastole is highly suggestive of nonviable myocardium.[13]

Dobutamine stress echocardiogram(DSE)[14] can help differentiate between ICM and NICM. In absence of CAD, normal response to dobutamine stress test augmentation of systolic function and contractility with increasing doses of dobutamine. In presence of significant disease, an initial augmentation of LVEF and contractility followed by decline in LVEF or contractility or emergence of new WMA at higher doses of dobutamine.

DSE can also help identify patients with ICM that can benefit from revascularization. An initial augmentation at low dose followed by a decline with high dose (biphasic response) are candidates for revascularization

By using DSE assessed changes in the long axis systolic amplitude to discriminate between ICM and NICM. In the presence of LBBB, inability to increase septal systolic amplitude by >1.5 mm was highly indicative of coronary artery disease.

Conclusion:

Heart failure caused by ischemic cardiomyopathy is a chronic disease with multiple potential interventions. Echocardiography plays a key role in evaluating ICM. Consistent high-quality imaging with consistent patient positioning, transducer placement, gain settings and characterization of hemodynamics will allow the most accurate initial diagnosis.

References:

1. Ng AC, Sindone AP, Wong HS et al. Defferences in management and outcome of ischemic and non-ischemic cardiomyopathy. Int J Cardiol 2008;129:198-204.
2. Alderman EL, Fisher LD, Litwin P et al. Results of coronary artery surgery in patients with poor LV function (CASS). Circulation.1983;68(4):785-95.
3. Felker GM, Shaw LK, O'Connor CM et al. A standardized definition of ischemic cardiomyopathy for use in clinical research.J Am Coll Cardiol 2002;39(2):210-18.
4. Scanlon PJ, Faxon DP, Audet AM et al. ACC/AHA guidelines for coronary angiography. Developed in collaboration with SCAI. J Am Coll Cardiol 1999;33(6):1756-824.
5. Di Donato M, Castelvecchio S, Menicanti L. End systolic volume following surgical ventricular reconstruction impacts survival in patients with IDCM. Eurr J Heart Fail 2010;12(4):375-81.
6. Brieke A, DeNorfrio D. Right ventricular dysfunction in chronic dilated cardiomyopathy and heart failure. Coronary Artery Dis.2005;16(1):5-11.
7. Dini FL, Cortigiani L, Baldini U et al. Prognostic value of LA enlargement in patients with idiopathic dilated cardiomyopathy and ischemic cardiomyopathy. Am J Cardiol 2002;89(5):518-23.
8. Massie BM, Schiller NB, Ratshin RA et al. Mitral-septal separation: new echocardiographic index of left ventricular function. Am J Cardiol 1977;39(7):1008-16.
9. Kolias TJ, Aronson KD, Armstrong WF. Doppler derived dp/dt and –dp/dt predict survival in congestive heart failure. J Am Coll Cardiol 2000;36(5):1594-9.
10. Bruch C, Schmermund A, Marin D et al. Tei-index in patients with mild to moderate congestive heart failure. Eur Heart J 2000;21(22):1888-95.
11. Medina R, Panidis IP, Morganroth J et al. The value of echocardiographic RWMA in detecting coronary artery disease in patient with or without a dilated left ventricle. Am Heart J 1985;109(4):799-803.
12. Chen YZ, Sherrid MV, Dwyer EM Jr. Value of two dimensional echocardiography in evaluating coronary artery disease: a randomized blinded analysis. J Am Coll Cardiol 1985;5(4):911-17.
13. Schinkel AF, Bax JJ, Boersma E et al. Assessment of residual myocardial viability in regions with chronic electrographic Q wave infarction.Am Hear J 2002;144(5):865-9.
14. Sawada SG, Segar DS, Ryan T et al. Echocardiographic detection of coronary artery disease during dobutamine infusion. Circulation 1991;83(16):1605-14.