PREVALENCE OF LEFT VENTRICULAR CLOT AND ITS CORRELATION WITH EJECTION FRACTION AMONG CARDIOMYOPATHY PATIENTS IN L.R.H PESHAWAR

(CROSS SECTIONAL STUDY)



BS CARDIOLOGY Technology

By

MUHAMMAD SULAIMAN

INSTITUTE OF PARAMEDICAL SCIENCES
KHYBER MEDICAL UNIVERSITY
PESHAWAR, PAKISTAN

Session 2012-2016

PREVALANCE OF LEFT VENTRICULAR CLOT AND IT'S CORRELATION WITH EJECTION FRACTION AMONG CARDIOMYOPATHY PATIENTS IN L.R.H PESHAWAR (CROSS SECTIONAL STUDY)

A THESIS SUBMITTED IN THE PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

BECHLOR OF SCIENCE

In

CARDIOLOGY TECHNOLOGY

MUHAMMAD SULAIMAN

2012/KMU/IPS-1/038

INSTITUTE OF PARAMEDICAL SCIENCES

KHYBER MEDICAL UNIVERSITY

PESHAWAR, PAKISTAN

Session 2012-2016

CERTIFICATE

This research project report by Muhammad Sulaiman is accepted in its present form, by the Department of Cardiology, Institute of Paramedical Sciences, Khyber Medical University Peshawar, as satisfying research project report requirements for award of degree of BS in Cardiology

Supervisor: _____
Mr. Muhammad Imran

Co- Supervisor _____

Mr. Saeed

External Examiner: _____
Mr Sana ullah

Head of Department: _____
Mr.Muhammad Imran

Head of IPMS _____
Dr Fazal Mehmood

Date: ____/___/_____

DEDICATION

This is dedicated to My Parents who always guided, supported and helped me to complete my BS program. This success is achieved because of my parents' prayers. Without their support I would have unable to achieve anything. I thank Allah Almighty for blessing me with such kind and loving Parents.

DECLARATION

I hereby declare that the work accomplished in this research project report is my own research effort carried out in Echocardiography Department in Lady Reading Hospital Peshawar and Institute of paramedical Sciences, Khyber Medical University Peshawar. The research project report has been written and composed by me.

The work in this research project report has neither been previously submitted for examination leading to the award of a degree nor does it contains any material from the published resources that can be considered as the violation of the international copyright law.

I also declare that I am aware of the terms ‘copyright’ and ‘plagiarism’. I will be solely responsible for the consequences of violation to these rules (if any) found in the research project report. The research project report has been checked for plagiarism by turnitin software.

Name: Muhammad Sulaiman

Signature: _____

Date: May 12, 2016

ACKNOWLEDGMENT

Thanks to Allah, Almighty for all his blessing on me throughout my life and who enable me to complete my research project report because of His blessing I am able to achieve this goal. I sincerely pay my humble and heartedly thanks to my most affectionate parents who supported and encouraged me throughout my life time in completing my education. My success is fruit of their devoted prayers.

I am deeply obliged to my supervisor Mr.Muhammad Imran who guided me during my research and research project report. His keen interest and valuable suggestions made this work to end. Thanks to my co supervisor Sir, Saeed Lecturer in Cardiology Khyber Medical University Peshawar.

I am also thankful to Sir, Shahid who is master in statistics and he gave her precious time to calculate statistical analysis regarding research project report.

TABLE OF CONTENTS

ACKNOWLEDGMENT ..... **I**

LIST OF TABLES ..... **IV**

LIST OF FIGURES ..... **V**

LIST OF ABBREVIATIONS ..... **VI**

ABSTRACT ..... **VII**

1. INTRODUCTION **1**

1.2 EVALUATION OF CLASSIFICATIO 1

1.3 DILATED CARDIOMYOPATHY 2

1.4 PREVELANCE 3

1.5 FAMILIAL (GENETICS) DILATED CARDIOMYOPATHY 3

2. PATHOLOGY **5**

2.1 MICROSCOPIC EXAMINATION 5

2.2 HISTOLOGICAL EXAMINATION 5

2.3 CLINICAL MANIFESTATION 5

2.4 ELECTROCARDIOGRAPHY 6

2.5 ECHOCARDIOGRAPHY 6

2.6 CARDIAC CATHETERIZATION 7

2.7 ECHOCARDIOGRAPHIC MEASUREMENT EF 7

2.8 LV THROMBUS 8

3. METHODS AND MATERIALS **11**

3.1 STUDY DESIGN AND SETTING 11

3.2 PARTICIPANTS 11

3.3 SAMPLING TECHNIQUE 11

3.5 ELECTROCARDIOGRAPHY 12

3.6 ECHOCARDIOGRAM 12

3.8 INCLUSION 12

3.9 EXCLUSION 13

3.10 STATISTICAL ANALYSIS 13

4. RESULTS 14

5. DISCUSSION 20

6. CONCLUSION 22

7. REFERENCES.....23

LIST OF TABLES

Table 1: Baseline characteristic of Participants. 14

Table 2: Gender frequency Table 15

Table 3: Frequency of Cardiomyopathy 15

Table 4: Frequency of LV Clot 16

Table 5: LV CLOT Cardioyopathy cross tabulation 17

Table 6:Chi-square test 19

LIST OF FIGURES

Figure 1: Pie chart of gender frequency.....15

Figure 2 16

LIST OF ABBREVIATIONS

ECG	Electrocardiography
ECHO	Echocardiography
DCM	Dilated Cardiomyopathy
ICM	Ischemic Cardiomyopathy
COCM	Congestive cardiomyopathy
AV	Atrio ventricular
LVEDP	Left Ventricular end diastolic pressure
MV	Mitral Valve
EF	Ejection Fraction
LVC	Left Ventricular Clot
PCI	Percutaneous Coronary intervention
AMI	Acute Myocardial infarction
TTE	Transthoracic echocardiography
RV	Right Ventricle
RCM	Restrictve cardiomyopathy
ESC	European Society of Cardiology

ABSTRACT

Background : To find the prevelance of left ventricular clot and correlate it with ejection fraction among patients of cardiomyopathy**.**

Objectives: To determine the prevelance of LV Clot and its coorelation with ejection fraction among cardiomyopathy.

Materials and Methods: A cross sectional study performed at Lady Reading Hospital Peshawar. The patients come to cardio-pulmonary laboratory for echocardiography from wards, OPDs and CCU and fulfilling the criteria of our study were included in the study. All patients included in the study were informed about study purpose and their ECG, history and echocardiography parameters were entered in a pre-formed Performa. SPSS version 22 was used for Statistical analysis.

Results: Total of 365 patients were included in this study. 149 (48.8%) participants were male subjects. Mean age of the participants was 33.06±10.1 and a range of 18-56 Years. Ejection fraction mean was 36.65±6.1 and range of 25-45%. Among 365 patients congestive cardiomyopathy was present 238 (65.2 %), Ischemic cardiomyopathy was present in 41 (11.2%), Paripartum cardiomyopathy was noted in 86 (23.6%) participants. Clot was present in 20 patients out of 238 participants Congestive cardiomyopathy, 3 Clots was present out of 41 patients of Ischemic cardiomyopathy, while five clots were noted in patients of Paripartum cardiomyopathy.

Conclusion:.1. LV clot was mostly present in COCM.2.Reduced EF is the main cause of LV clot.3.Clot mostly present in LV apex

Keywords: Left ventricle, Hypertrophic cardiomyopathy, ischemic cardiomyopathy, Left ventricular end diastolic pressure.

1. INTRODUCTION

Cardiomyopathies are basically disease of myocardium which potentially and practically unusual without unequivocal sickness skilled to bring about the myocardial pathology. Cardiomyopathies are further classified into four major classes

- Dilated cardiomyopathy (DCM)
- Hypertrophic cardiomyopathy (HCM)
- Restrictive cardiomyopathy (RCM)
- Arrhythmogenic cardiomyopathy (ARVC/D)

Among all these the most common is DCM ranges upto 60 % of cases. that can manifest as LV enlargement and globally impaired LV function .

1.2 EVALUATION OF CLASSIFICATIO

As classification of cardiomyopathy is concerned .this may be either or primary or secondary cardiomyopathies.Primary cardiomyopathy is mostly idiopathic.

Cardiomyopathy can leads to several adverse outcome like organ damage and systemic effect (1) .According to WHO in 1980 they introduce the word of cardiomyopathy as disease of cardiac muscle having unknown cause.this definition was later on disapproved after the introducing of ARVCM/RCM (2,3).

Another definition was put forward by AHA that is, cardiomyopathy are the heterogeneous disease of myocardium related to both as electrical or mechanical dysfunction,which as a rule (however not constantly) show wrong ventricular hypertrophy or dilatation, because of an assortment of etiologies that every now and 1 again are hereditary. Cardiomyopathies can

cause systemic issue or may have generalized reduced pump capacity. and regularly prompt to cardiovascular passing/dynamic heart disappointment related disability (1).

However there is no clear pathophysiology or etiology is define still now but in modern decades the concept of this pathophysiology or etiology clearly define by AHA and ESC (4).AHA classified this into two major gathering in light of overwhelming organ association Primary cardiomyopathy are specific to heart muscle with abnormal muscular morphology.Auxillary cardiomyopathy related to necrotic heart muscle inclusion as a major aspect of few number f systemic pathologies (5).

World heart federation in 2013 introduce the phenotype-genotype terminology of cardomyopathies.according to this cardiomyopathies are further attributes into 5 group known as MOGE(S).the MOGE(S) characterization has a few focal points with respect to synchronous maximal portrayal of malady from clinical and hereditary focuses. In any case this grouping cannot related to symptomatic criteria of cardiomyopathy along with clinical practice. then again the characterization in light of deliberately hereditary testing and observing may bring about over diagnostic states without clinically apparent indications of CM and non appearance practical phenotype.MOG(S) characteristic of cardiomyopathies are multicellular registries.

1.3 DILATED CARDIOMYOPATHY

The most common cardiomyopathies is dilated cardiomyopathies that can be characterized by LV enlargement and severly reduced LV function it can be hereditary or acquired. This illness can be determined in relationship to have perceived cardiovascular infection that can be characterized as DCM in which the function of myocardium is being arrested. Like a sustain increase blood pressure or coronary heart disease (7,4).

These muscular disease and systemic infection leads to globally impaired LV function and the dilation of left ventricular however no definite cause of cardiomyopathies is still not found. This can also called as idiopathic dilated cardiomyopathies.

1.4 PREVELANCE

The incidence in this disease is found in old age patients. Commonness at all inclusive community stays unclear however this disease can be found at any age and sex.DCM is more common in male as compare to female. among 100000 kids zero point five seven percent of cases identified in this group of patient. The young age are found as sixty six percent of patient with unknown cause (4).in USA among 100000 patients thirty six patients were identified as DCM (8).also known as familial cardiomyopathies. Approximately twenty to fourty eight percent of DCM is identified as familial cardiomyopathy (8).

1.5 FAMILIAL (GENETICS) DILATED CARDIOMYOPATHY

Well known advance has been made in investigations of the hereditary qualities of DCM. The majority of the qualities required in the improvement of DCM encode basic components of the cardiomyocytes, especially dystrophy related glycoprotein complex or parts of the sarcomeric complex. Hereditary inclination may have a conclusive part in the advancement of essential and optional DCM. Right now, > 30 autosomal and 2-X-connected qualities have been appeared to incline to DCM and the quantity of these qualities will keep on increasing. There are adequate information that with new finding of IDC the clinical screening of first-degree relatives will uncover familial (hereditary) DCM in 20%-35% of those relatives. Late rules prescribe that hereditary testing ought to be given in families in whom familial DCM is associated for early finding with cardiomyopathy in family members(4).

The determination of FDC is made when IDC is analyzed in two firmly related relatives. Around 20%-48% of DCM has been accounted for as familial, in spite of the fact that with inadequate and age-subordinate penetrance, and connected to a differing gathering of > 20 loci and genes(10). while hereditarily heterogeneous, the major method of inheritance forfor DCM is autosomal predominant, with X-connected autosomal latent and mitochondrial legacy being less incessant. In this way, when taking a family history, particular consideration ought to be given to a past filled with solid dystrophy, components of mitochondrial sickness (e.g., familial diabetes, deafness, epilepsy, or maternal legacy), and signs and side effects of other acquired metabolic diseases(10). A few of the mutant qualities connected to autosomal prevailing DCM encode the same contractile sarcomeric proteins that are in charge of HCM, including α-heart actin; α-tropomyosin; cardiovascular troponin T, I and C; β-and α-myosin overwhelming chain; and myosin restricting protein C. Z-circle protein-encoding qualities, including muscle LIM protein, α-actinin-2, ZASP, and titin, additionally have been recognized. DCM is additionally brought about by various changes in different qualities encoding cytoskeletal/sarcolemmal, atomic envelope, sarcomere, and transcriptional coactivator proteins. The most widely recognized of these presumably is the lamin A/C quality, likewise connected with conduction framework sickness, which encodes an atomic envelope moderate fiber protein. Changes in this quality likewise cause Emery-Dreifuss strong dystrophy (9,8). Other DCM qualities of this sort incorporate desmin, caveolin, and β-and α-sarcoglycan, and additionally the mitochondrial respiratory chain gene(1). X-connected DCM is brought on by the Duchenne strong dystrophy (dystrophin) quality, while G4.5 (tafazzin), a mitochondrial protein of obscure capacity, causes Barth disorder, which is a X-connected cardioskeletalmyopathy (8,10).

2. PATHOLOGY

2.1 MICROSCOPIC EXAMINATION

Plainly visible examination of heart uncover ventricular chamber enlargement with thickened or typical thickness walls. Typical changes are not valvular, in spite of the fact that enlargement of valvular holes might be available as optional changes because of expansion of chambers. Coronary structures is most usually typical, in spite of the fact that the nearness of non occlusive atherosclerotic plaques might be available.as the function of myocardium is being reduced leading to stasis of blood which causing thrombi in ventricles or atrium.

2.2 HISTOLOGICAL EXAMINATION

Histological examination show that cardiomyopathy are basically the fibrosis of pre vascular and interstitial (11).subendocardium is mainstay with myocardial necrosis. this examination is done by shirani methods (12), myocardial fibrosis in patients with IDC and ischemic dilatation cardiomyopathy. The rate of volumic collagen division in the LV myocardium was fundamentally higher in DCM patients contrasted with those with ischemic cardiomyopathy. Increment of collagen part connected with the level of widening of the left ventricle (13).

2.3 CLINICAL MANIFESTATION

Dilated cardiomyopathies are clinically correlates with congestive heart failure along with thromboembolic issue. Other findings are unusual finding of ECG and cardiomegaly with radiological examination. as the illness advances, distinct side effects of congestive heart disappointment show. Mid-section uneasiness may happen sometimes, symptoms may not be releved by nitroglycerin.JVP is raised with CHF.the clinical treatment of DCM should be variable both moderate movement with quickly dynamic more than a while. Cachexia and

fringe edema ordinarily emerge late in the course. Sudden passing, probably because of ventricular fibrillation might be the main indication.myocarditis may have a history of viral overload.an intense systemic febrile irresistible sickness, (for example, flu) is trailed by an inert period amid which time the patient might be asymptomatic.20-25 percent of patient presented with new onset of symptoms (14). A few clinical, lab and instrumental variables may have prognostic criticalness in DCM patients. These components are symptomatic ventricular arrhythmias, tenacious dash musicality, determined jugular venous expansion, systemic hypotension, relentlessly lifted B-sort natriuretic peptide, left package branch piece, pneumonic slender wedge weight > 20 mmHg, heart file < 2.5 L/min per square meter, seriously lessened launch division, prohibitive diastolic filling design, and extreme mitral regurgitation(15).

2.4 ELECTROCARDIOGRAPHY

ECG finding septal wave appearance ,BBB ,A:V prolongation ,SVT or atrial fibrillation,20-30 percent patients have non sustained VT with few numbers present with maintained VT. ECG is a first-line screening and indicative instrument used for distinguishing situations connected with abrupt passing. Idiopathic DCM personnel with a delayed QRS have altogether more terrible existence compare toother personnel (16).

2.5 ECHOCARDIOGRAPHY

Echocardiography in DCM has distinguishing configurations, despite the fact that it is impractical to conclude distinctive conclusion by echocardiography amongst ICM and further optional LV enlargement with impairment. M-mode echocardiography indicates LV widening with 7 global reduce walls thickening (figure1). Despite the fact that cardiomyopathy is diffuse disease, there might be regional dissimilarities level of hypokinesis

uncovered by two-dimensional echocardiography, leading to make it difficult for the operator to differentiate from ischemic cardiomyopathy. Ventricular expansion typically not joined to adequate hypertrophy, leading to increment of volume-to-mass ratio (17). Doppler echocardiography indicates mostly diastolic dysfunction by putting it on mitral and tricuspid valve, depending upon seriousness of intra- cardiac hemodynamic irregularities

2.6 CARDIAC CATHETERIZATION

Catheterization for prohibition of coronary supply route ailment may be critically taking after administration of DCM patients. Catheterization likewise uncovers expanded LVEDP and PA supply route wedge pressure with left ventriculography may demonstrate ventriclular widening with global hypokinesis.

2.7 ECHOCARDIOGRAPHIC MEASUREMENT EF

There are three important views for measuring EF:

1. Parasternal long-axis
2. Apical four-chamber
3. Apical long axis.

It is considered that lateral and anterior wall must be cleared so that apical long axis is converted into 2 chambers view that is LA and LV. .Measurements was obligated at end-diastole and end systole of all LV short-axis dimensions.In the 4 chamber and long-axis windows the measurement start from the tip of MV tip and all of the LV is traced and meet at the tip of other MV leaflet. Entire measurement was done from the endocardial marks opposing walls to an imagined conceive axis intersecting the LV from mitral valve to apex(20).

This process first done in end diastole when the MV is open frame by frame, that is a large cavity, and then it is measure in end systole when mitral valve is closed the small cavity. That permit one to ratiocinate frequent dimensions in part of cardiac cycles and frame by frame movement is checked in End systole and end diastole, thus gaining well relationship of endocardial marks. This all process can be done in just 5 minutes. The inter observer variability of the rule of thumb was assessed in 30 of 55 studies by two investigators who interpreted the same diamond in the rough and measured the EF independent of each other. Intra-observer variability was evaluated by such of the investigators repeating the measurements in 20 of 55 studies occupied at random at antipodal 1 month at the heels of his as a matter of choice interpretation and without knowledge of a matter of choice measurements. To runs it up a mast the reproducibility of the manner, 15 additional patients (four wise, nine by the whole of coronary blood vessel disease and LV dyssynergy and two by the whole of cardiomyopathy) were studied in two graded mornings. All 30 tapes were coded and if randomly to a psychiatrist to interpret and study the EF.A by the numbers M-mode echocardiogram was obtained in each static and when technically full, shortening 50 percent was calculated per standard deviation (20).

2.8 LV THROMBUS

The tendency of progressive ventricular thrombus (LVT) is a well-known difficulty in distinctive cardiac conditions mutually the highest lay out observed in brilliant anterior heart attack and congestive heart failure (CHF) as a explain of easier said than done left ventricular (LV) systolic dysfunction(21)(22).

The power of LVT, particularly in settings where coming down the percutaneous coronary intervention (PCI) facilities are accessible, is seemingly reducing by all of estimates ranging

surrounded by 5% and 15%.(22)(23). Solheim et al., declared an incidence of 15% within 3 months of brilliant myocardial infarction (AMI) in occupied patients managed by the whole of primary PCI(24). The constellation, of endothelial grudge, hypercoagulability, and flesh stagnation, which are sufficiently described by the anticipate mentioned as Virchow's trinity, is responsible for the laying down of thrombus(25). In AMI, disparate predisposing factors are lavish infarct period of time, difficult apical asynergy, LV aneurysm and myocardial infarction (MI)(26). The speedily and wary recognition of LVT is carrying a lot of weight to prevent the distasteful sequelae of systemic thromboembolic events (21) (25) (27). Transthoracic echocardiography (TTE) is too widely having a full plate modality in diagnosis of LVT seeing of its beg borrow or steal, preservation, and convenience. It has been demonstrated to have tiff of 90–95% and specificity of 85–90% in a furnishings of efficient imaging(26)(28). when compared to findings at surgery and autopsy. However, assistance must be exercised to blackball false convinced results occasioned by endocardial elastosis, trabeculae, false tendons near-field perplexity, and artefacts amid others(29). The detection price tag can be enhanced by off-axis imaging planes in situations to what place substrates for the arts and science of LVT one as abnormal obstruct motion and dilated ventricles mutually markedly reduced ejection fraction (EF) exist. Improved LV dead end assessment and thrombi detection by the activity of TTE correlate studies were outstanding to be top than non contrast TTE, specially for mural (laminar) and low thrombus(30). This attracted to to the advice that feature contrast should be having a full plate when non contrast images are suboptimal for clear diagnosis(31). Additional advantages of echocardiography hook up with the desire of LV size, for show of regional traverse motion abnormalities, and detection of Doppler-derived transmitral hover changes. Lower mitral E rotate deceleration time and abnormal traverse motion perform index were both associated by all of LVT conception(32)

In this retrospective design, we reviewed demographic announcement and aetiology of LVT in patients undergoing echocardiography at the University of Maiduguri Teaching Hospital around a three-year period.

3. METHODS AND MATERIALS

3.1 STUDY DESIGN AND SETTING

A cross sectional study was performed at echocardiography section of medical teaching institute Lady Reading Hospital Peshawar, the biggest hospital of Khyber Pakhtunkhwa (KPK). The study was conducted after approval of head of department of cardiology Lady Reading Hospital Peshawar.

3.2 PARTICIPANTS

The sample size was 365, calculated after the prevalence of LV clot in cardiomyopathy patients in a stud(33). A preformed Performa was filled from all patients selected for the said study. This study was conducted from August 2016 to December 2016.

A written consent was obtained from each patient who was agreed to take part in the study and it was approved by the research and ethical committee of Lady Reading Hospital Peshawar.

3.3 SAMPLING TECHNIQUE

Convenient sampling technique was used

3.4 Sample size

$$\mathbf{N=Z^2xp(1-p)/E^2}$$

$$\mathbf{N=1.96^2x0.39(1-0.39)/0.5^2}$$

$$\mathbf{N=3.84x0.39(0.61)/0.0025}$$

$$\mathbf{N=365}$$

3.5 ELECTROCARDIOGRAPHY

ECG was performed by a senior technician of the ECG department of cardiology department of LRH using 3-chnne Nihon kohden ECG machine. The ECG was observed by a senior cardiologist and looked for the diagnosis of Left Ventricular Hypertrophy, P wave width, Right Ventricular Hypertrophy, heart rate, arrhythmias, Left Bundle Branch Block, ST segment & T-wave changes, previous or new Myocardial Infarction.

3.6 ECHOCARDIOGRAM

Echocardiogram was performed and observed by a senior echocardiography technician and all parameters are taken carefully. The M-mode measurement was taken in 2D, by putting cursor on the tips of Mitral valve. Taking Left Ventricular end diastolic diameter, Left Ventricular end systolic diameter, inter ventricular septum and Left Ventricular Posterior Wall in diastole, RV in diastole, Left Atria in end diastole and aortic root in end systole.

Patient echocardiogram regurgitation was also observed by sonographer and graded it according to its severity, and wall motion abnormality was seen in 2D, in views of short axis at the mitral valve level, papillary muscle level, and apical level and then in apical 4 chambers and 2 chamber views.

3.8 INCLUSION

Those patients were included in the study that have dilated and paripartum cardiomyopathy and reduced ejection fraction.

3.9 EXCLUSION

- All cardiomyopathy except Dilated and paripartum cardiomyopathy were excluded from the study.
- Patients having normal ejection fraction,
- Those who were not agree to take part in the study.

3.10 STATISTICAL ANALYSIS

SPSS version 22 was used to analyze the data. Z-testing and chai square were used. We also used linear and simple regression for correlation of reduced EF and LV clot in dilated Cardiomyopathy.

4. RESULTS

Total of 365 patients were included in this study. One forty nine (48.8%) participants were male subjects. Mean age of the participants was 33.06±10.1 and a range of 18-56 Years. Ejection fraction mean was 36.65±6.1 and range of 25-45%. Among 365 patients congestive cardiomyopathy was present 238 (65.2 %), Ischemic cardiomyopathy was present in 41 (11.2%), Paripartum cardiomyopathy was noted in 86 (23.6%) participants. Clot was present in 20 patients out of 238 participants Congestive cardiomyopathy, 3 Clots was present out of 41 patients of Ischemic cardiomyopathy, while five clots were noted in patients of Paripartum cardiomyopathy.

Table 1: Baseline characteristic of Participants.

Age mean	33.06±10.1 (18-56 Years)
EF mean	36.65±6.1 (25-45%)
Male	149 (48.8%)
Congestive Cardiomyopathy	238 (65.2%)
Ischemic Cardiomyopathy	41 (11.2%)
Paripartum Cardiomyopathy	86 (23.6%)
LV clot	28 (7.7%)

Table 2: Gender frequency Table

	Frequency	Percent	Valid Percent	Cumulative Percent
Male	149	40.8	40.8	40.8
Female	216	59.2	59.2	100.0
Total	365	100.0	100.0	

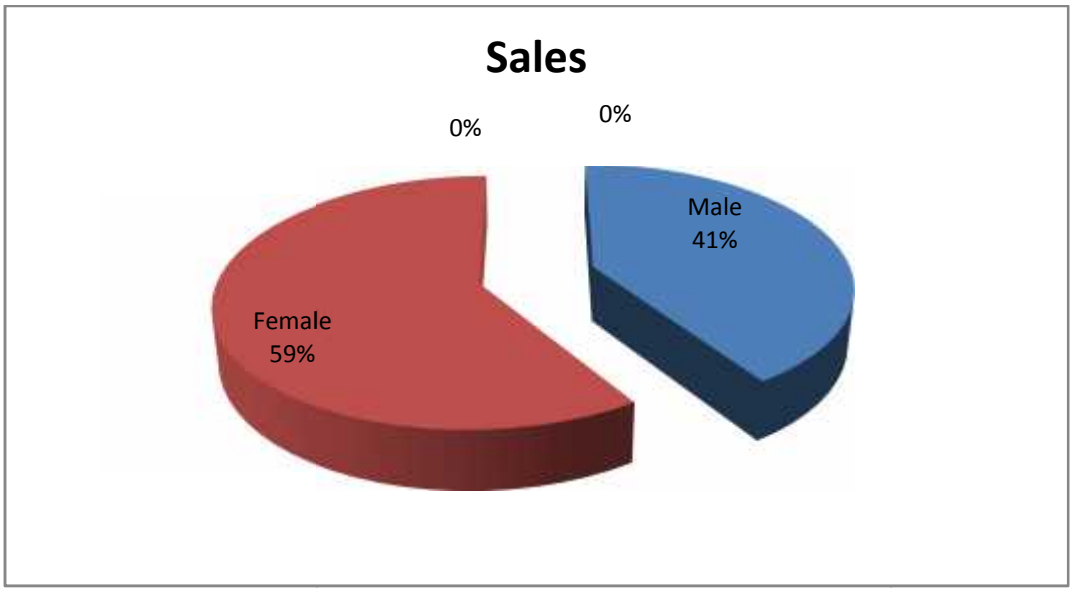


Figure 1: Pie chart of gender frequency

Table 3: Frequency of Cardiomyopathy

Cardiomyopathy

	Frequency	Percent	Valid Percent	Cumulative Percent
Cocm	238	65.2	65.2	65.2
Isch.CM	41	11.2	11.2	76.4
Pari.CM	86	23.6	23.6	100.0
Total	365	100.0	100.0	

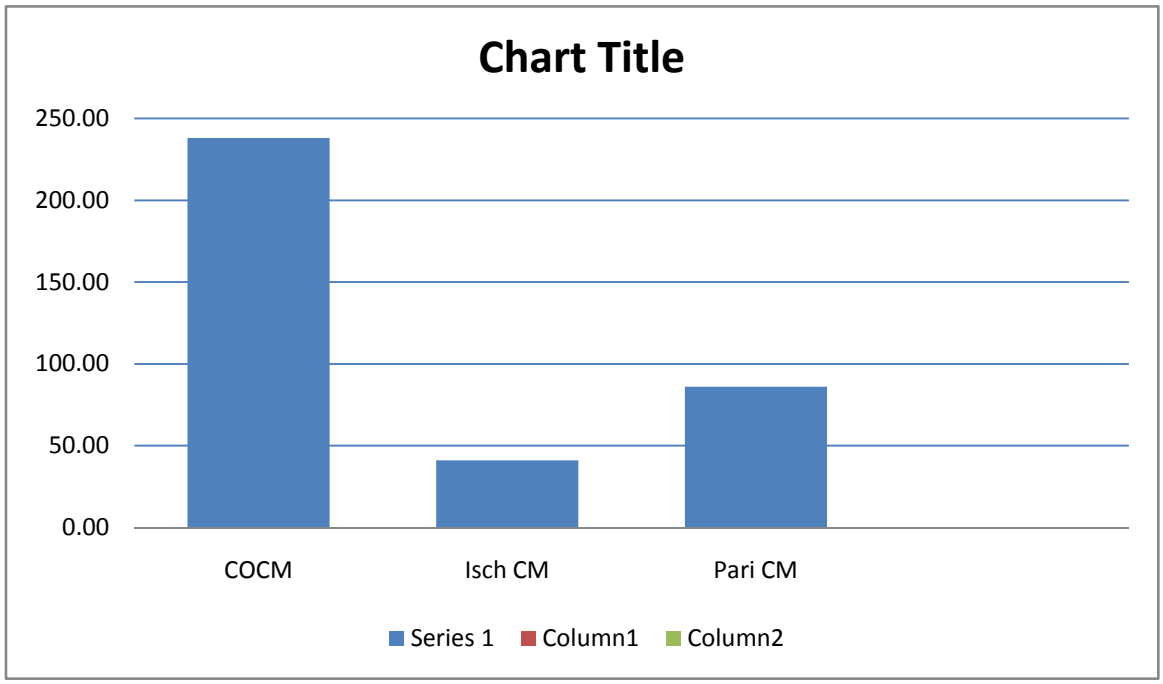


Figure-2

Table 4: Frequency of LV Clot

	Frequency	Percent	Valid Percent	Cumulative Percent
present	28	7.7	7.7	7.7
Absent	337	92.3	92.3	100.0
Total	365	100.0	100.0	

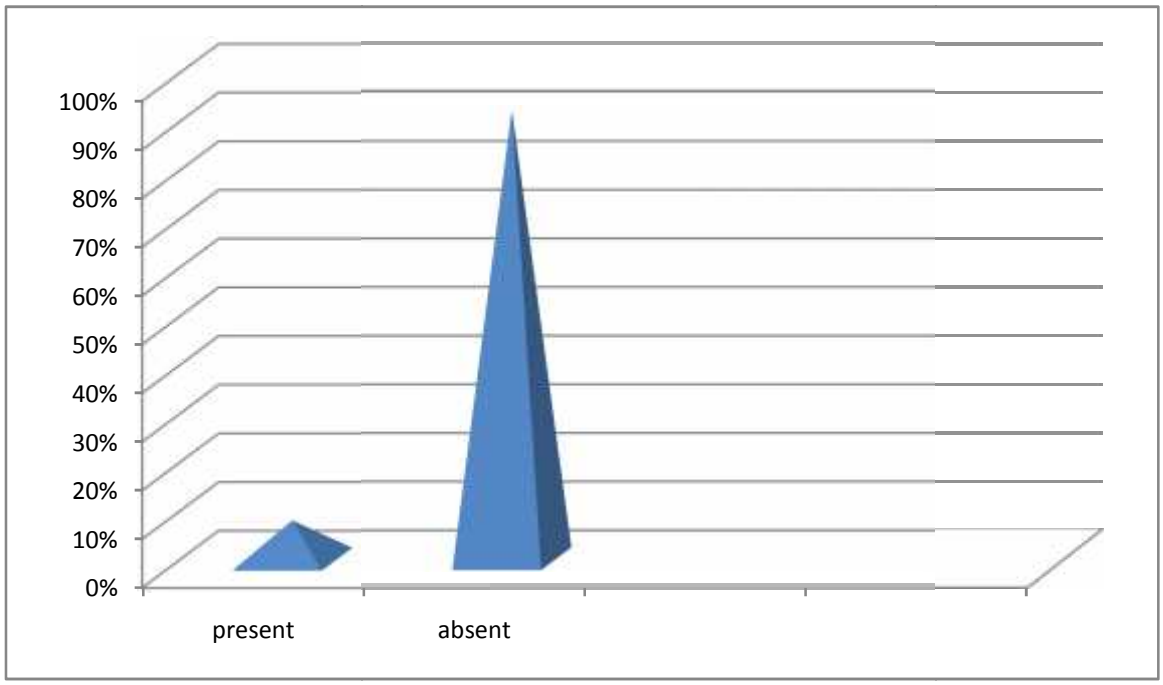


Figure-3

Table 5: LV CLOT Cardioyopathy cross tabulation

LV Clot * Cardiomyopathy Crosstabulation

		Cardiomyopathy			
		Cocm	ICM	PCM	Total
LV Clot	present	20	3	5	28
	Absent	218	38	81	337
Total		238	41	86	365

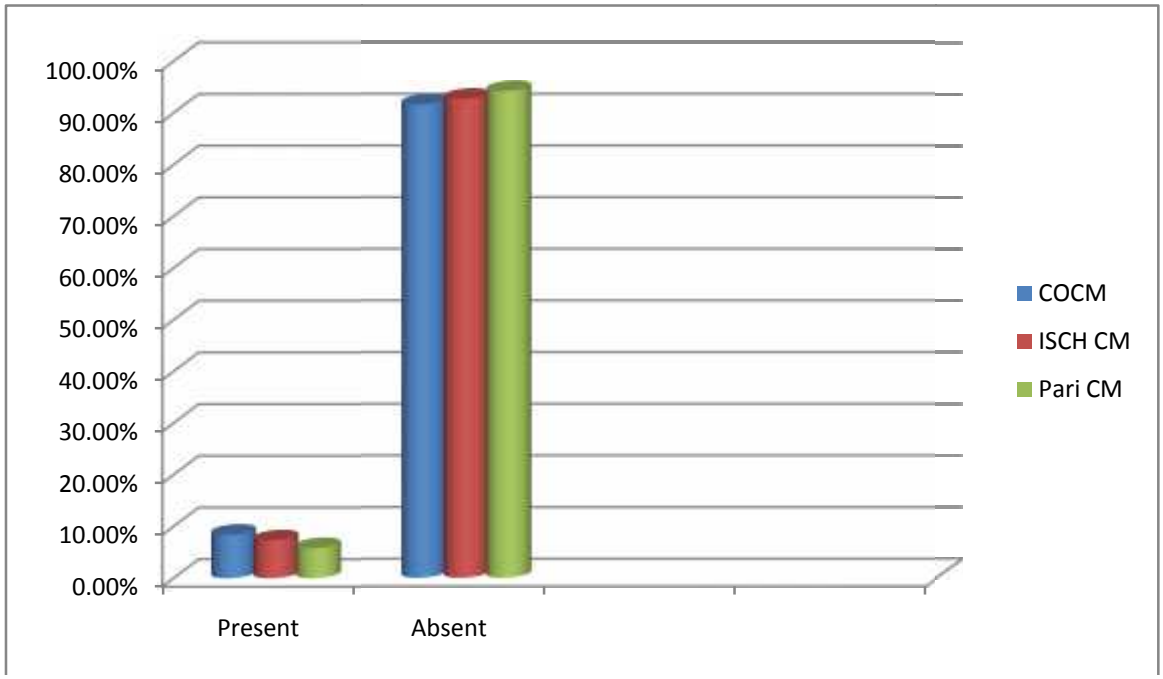


Figure-4

From this study we have concluded that LV clot had strongly correlated with reduced EF with a P=0.0001 and r=139.9 table 6, while there is no correlation in patients having cardiomyopathy with a P=0.739 and r=0.608 tabe 7,

Table 6:Chi-square test

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.606[a]	2	.739
Likelihood Ratio	.637	2	.727
N of Valid Cases	365		

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	139.930[a]	8	.000
Likelihood Ratio	107.646	8	.000
N of Valid Cases	365		

5. DISCUSSION

We report a pervasiveness of 7.7% for left ventricular thrombus among patients with anomalous. The greater part of LVT are included in the apical sections, with regards to reports from different reviews. The summit is the most widely recognized area required in LVT in patients with MI and non-ischemic cardiomyopathy.

The most highest prevelance of 8.4% was record amongst the patients with non-ischemic DCM. This is lower than prior reports by other people(34)(35). Expanded cardiomyopathy is connected with dilatation of commonly left and right ventricles with diminished systolic capacity. The significant biventricular stasis advances the design of thrombus, most regularly in the left ventricle, trailed by the correct ventricle. The danger of fringe embolization is accounted for to be hoisted in the setting of LVT(25)(27). Biventricular affiliation was exploratory in those with serious systolic brokenness (<30%). Past reviews reported expanded LVIDD and low discharge division to be free indicators of LVT formation(35)(36). This is shown in our subjects with the powerful lion's share having expanded LVIDD and low EF.

Peripartum cardiomyopathy in our grouping had LVT fame of 5.8%. This is lower to what was accounted for by Sliwa et al. among dark patients at Baragwanath Hospital (37), furthermore lower than the 12.3% reported in Sokoto, north western Nigeria (38). Karaye and Sani reported a higher predominance of 54.6% among patients with PPCM in Kano (39). The distinction in rates reported by the divergent reviews might be because of contrasts in cruelty of LV dilatation and brokenness in the populace examined. Biventricular thrombus in the setting of PPCM is to a great extent restricted to case reports, reinforcing the deficiency of such occurrence(40). This might be licensed to the propelled condition of ventricular brokenness, and in addition late presentation. The arrangement of thrombus in the setting of

PPCM is perceived to stasis resultant upon the poor myocardial contractility and in addition the hypercoagulable condition of pregnancy which may keep on for up to six weeks postpartum (41).

6. CONCLUSION

LV clot was mostly present in Congestive cardiomyopathy(COCM) ,Reduced EF is the main cause of LV clot, Clot mostly present in LV apex.

7. REFERENCES

1. Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, et al. Contemporary definitions and classification. Circulation. 2006 Apr;113(14):1807–16.

2. Report of the WHO/ISFC task force on the definition and classification of cardiomyopathies. Br Heart J. 1980 Dec;44(6):672–3.

3. Report of the 1995 World Health Organization. Circulation. 1996;93(5).

4. Jefferies JL, Towbin JA. Dilated cardiomyopathy. Lancet. 2010 Feb;375(9716):752–62.

5. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, et al. Classification of the cardiomyopathies: a position statement from the european society of cardiology working group on myocardial and pericardial diseases. Eur Heart J. 2007 Dec;29(2):270–6.

6. Arbustini E, Narula N, Dec GW, Reddy KS, Greenberg B, Kushwaha S, et al. The MOGE(S) Classification for a Phenotype–Genotype Nomenclature of Cardiomyopathy. J Am Coll Cardiol. 2013 Dec;62(22):2046–72.

7. Rosamond W, Flegal K, Friday G, Furie K, Go A, Greenlund K, et al. Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2007;115(5):e69-171.

8. Hershberger RE, Lindenfeld J, Mestroni L, Seidman CE, Taylor MRG, Towbin JA. Genetic Evaluation of Cardiomyopathy-A Heart Failure Society of America Practice Guideline. J Card Fail. 2009;15(2):83–97.

9. Arbustini E, Pilotto A, Repetto A, Grasso M, Negri A, Diegoli M, et al. Autosomal dominant dilated cardiomyopathy with atrioventricular block: A lamin A/C defect-related disease. J Am Coll Cardiol. 2002;39(6):981–90.

10. McNair WP, Ku L, Taylor MRG, Fain PR, Dao D, Wolfel E, et al. SCN5A mutation associated with dilated cardiomyopathy, conduction disorder, and arrhythmia. Circulation. 2004;110(15):2163–7.

11. Sanderson JE, Olsen EGJ, Gatei D. Dilated cardiomyopathy and myocarditis in Kenya: an endomyocardial biopsy study. Int J Cardiol. 1993;41(2):157–63.

12. Shirani J, Pick R, Guo Y, Silver MA. Usefulness of the electrocardiogram and echocardiogram in predicting the amount of interstitial myocardial collagen in endomyocardial biopsy specimens of patients with chronic heart failure. Am J Cardiol. 1992 Jun;69(17):1502–3.

13. Sisakian S, Gukasian L V, Mkrtshian LG, Kamalov GG, Avakian SA. [The role of quantitative determination of the volume fraction of interstitial collagen and fibronectin in the pathogenesis of myocardial remodeling in dilated cardiomyopathy]. Klin Med (Mosk). 2001;79(6):24–6.

14. McNamara DM, Holubkov R, Starling RC, Dec GW, Loh E, Torre-Amione G, et al. Controlled trial of intravenous immune globulin in recent-onset dilated cardiomyopathy. Circulation. 2001;103:2254–9.

15. Bart BA, Shaw LK, McCants CB, Fortin DF, Lee KL, Califf RM, et al. Clinical determinants of mortality in patients with angiographically diagnosed ischemic or nonischemic cardiomyopathy. J Am Coll Cardiol. 1997;30(4):1002–8.

16. Silverman ME, Pressel MD, Brackett JC, Lauria SS, Gold MR, Gottlieb SS. Prognostic value of the signal-averaged electrocardiogram and a prolonged QRS in ischemic ana nonischemic cardiomyopathy. Am J Cardiol. 1995;75(7):460–4.

17. Elliott P. Cardiomyopathy. Diagnosis and management of dilated cardiomyopathy. Heart. 2000 Jul;84(1):106–12.

18. Arai AE. The cardiac magnetic resonance (CMR) approach to assessing myocardial viability. J Nucl Cardiol. 2011;18(6):1095–102.

19. Stojanovska J, Garg A, Patel S, Melville DM, Kazerooni E a, Mueller GC. Congenital and hereditary causes of sudden cardiac death in young adults: diagnosis, differential diagnosis, and risk stratification. Radiographics. 2013;33:1977–2001.

20. Quinones M a, Waggoner a D, Reduto L a, Nelson JG, Young JB, Winters WL, et al. A new, simplified and accurate method for determining ejection fraction with two-dimensional echocardiography. Circulation. 1981;64(4):744–53.

21. Billingsley: Left ventricular thrombus: diagnosis,... - Google Scholar.

22. Nayak D, Aronow WS, Sukhija R, McClung JA, Monsen CE, Belkin RN, et al. Comparison of frequency of left ventricular thrombi in patients with anterior wall versus non-anterior wall acute myocardial infarction treated with antithrombotic and antiplatelet therapy with or without coronary revascularization. Am J Cardiol. 2004 Jun;93(12):1529–30.

23. Kalra A, Jang IK. Prevalence of early left ventricular thrombus after primary coronary intervention for acute myocardial infarction. J Thromb Thrombolysis. 2000;10(2):133–6.

24. Solheim S, Seljeflot I, Lunde K, Bjørnerheim R, Aakhus S, Forfang K, et al. Frequency of left ventricular thrombus in patients with anterior wall acute myocardial infarction treated with percutaneous coronary intervention and dual antiplatelet therapy. Am J Cardiol. 2010;106(9):1197–200.

25. Delewi R, Zijlstra F, Piek JJ. Left ventricular thrombus formation after acute myocardial infarction. Heart. 2012;98(23):1743–9.

26. Weinsaft JW, Kim HW, Crowley AL, Klem I, Shenoy C, Van Assche L, et al. LV thrombus detection by routine echocardiography: Insights into performance characteristics using delayed enhancement CMR. JACC Cardiovasc Imaging. 2011;4(7):702–12.

27. Nair A. Anticoagulation in patients with heart failure: who, when, and why? Eur Hear J Suppl. 2006;8(Suppl E):E32–8.

28. Visser CA, Kan G, Lie KI, Durrer D. Left ventricular thrombus following acute myocardial infarction: a prospective serial echocardiographic study of 96 patients. Eur Heart J. 1983;4(5):333–7.

29. Artham S. Feigenbaum's Echocardiography. Congenit Heart Dis. 2010;5(5):491.

30. Mansencal N, Nasr IA, Pilliere R, Farcot JC, Joseph T, Lacombe P, et al. Usefulness of contrast echocardiography for assessment of left ventricular thrombus after acute myocardial infarction. Am J Cardiol. 2007;99(12):1667–70.

31. Mulvagh SL, Rakowski H, Vannan MA, Abdelmoneim SS, Becher H, Bierig SM, et al. American Society of Echocardiography Consensus Statement on the Clinical Applications of Ultrasonic Contrast Agents in Echocardiography. Vol. 21, Journal of the American Society of Echocardiography. 2008. p. 1179–201.

32. Celik S, Baykan M, Erdöl C, Gökce M, Durmus I, Örem C, et al. Doppler-derived mitral deceleration time as an early predictor of left ventricular thrombus after first anterior acute myocardial infarction. Am Heart J. 2000;140(5):772–6.

33. Talle MA, Buba F, Anjorin CO. Prevalence and Aetiology of Left Ventricular Thrombus in Patients Undergoing Transthoracic Echocardiography at the University of Maiduguri Teaching Hospital. Adv Med [Internet]. 2014 [cited 2016 Dec 31];2014:1–8. Available from: http://www.hindawi.com/journals/amed/2014/731936/

34. Fuster V, Gersh BJ, Giuliani ER, Tajik AJ, Brandenburg RO, Frye RL. The natural history of idiopathic dilated cardiomyopathy. Am J Cardiol. 1981;47(3):525–31.

35. Sharma ND, McCullough P a, Philbin EF, Weaver WD. Left ventricular thrombus and subsequent thromboembolism in patients with severe systolic dysfunction. Chest [Internet]. 2000;117(2):314–20. Available from: http://dx .doi.org/10.1378/ chest. 117.2.314

36. Loh E, Sutton MS, Wun CC, Rouleau JL, Flaker GC, Gottlieb SS, et al. Ventricular dysfunction and the risk of stroke after myocardial infarction. N Engl J Med [Internet]. 1997;336(4):251–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/8995087

37. Sliwa K, Skudicky D, Bergemann a, Candy G, Puren a, Sareli P. Peripartum cardiomyopathy: analysis of clinical outcome, left ventricular function, plasma levels of cytokines and Fas/APO-1. J Am Coll Cardiol. 2000;35(3):701–5.

38. Isezuo SA, Abubakar SA. Epidemiologic profile of peripartum cardiomyopathy in a tertiary care hospital. Ethn Dis. 2007;17(2):228–33.

39. Karaye KM, Sani MU. Factors associated with poor prognosis among patients admitted with heart failure in a Nigerian tertiary medical centre: a cross-sectional study. BMC Cardiovasc Disord [Internet]. 2008;8:16. Available from: http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=

med5&AN=18644161
http://sfx.scholarsportal.info/uhn?sid=OVID:medline&id=pmid:18644161&id=doi:10.1186%2F1471-2261-8-16&issn=1471-2261&isbn=&volume=8&issue=1&spage=16&pages=16&date=2008&ti

40. Nishi I, Ishimitsu T, Ishizu T, Ueno Y, Suzuki A, Seo Y, et al. Peripartum cardiomyopathy and biventricular thrombi. Circ J [Internet]. 2002;66(9):863–5. Available from: http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=emed5&AN=2002320708%5Cnhttps://maastrichtuniversity.on.worldcat.org/atoztitles/link?sid=OVID:embase&id=pmid:&id=doi:10.1253/circj.66.863&issn=1346-9843&isbn=&volume=66&issue=9&spage=863&p

41. Tidswell M. Peripartum cardiomyopathy. Vol. 20, Critical Care Clinics. 2004. p. 777–88.