# IN HOUSE ANALYSIS OF MYOCARDIAL BRIDGING WITH NO FIXED OBSTRUCTION- A MULTIVARIATE ANALYSIS

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# CHAPTER I: INTRODUCTION

Myocardial bridging, a congenital abnormality, is a condition where a segment of major epicardial coronary artery goes intramurally through the myocardium below the muscle bridge (Angelini et al., 2002a). Myocardial bridging was first identified by Reyman (1737) and it was described angiographically by Portman and Iwig in 1960. A characteristic diastolic flow disruption has been documented on quantitative coronary angiography (Schwarz et al., 1996), intracoronary Doppler studies and intravascular ultrasonography (Ge et al., 1994a). The extent to which the coronary obstruction occurs depends on the location, thickness, length of muscle bridge, and the level of cardiac contractility. Estimated frequency of myocardial bridging that has been reported ranges from 1.5 to 16 % when assessment is done using coronary angiography. However, in some autopsy series, it has been found to have to be 80 % (Rossi et al., 1980b). Conventionally, myocardial bridging has been regarded as a benign condition, but complications such as ischemia, acute coronary syndrome, coronary spasm, ventricular septal rupture, exercise induced atrioventricular conduction, stunning, arrhythmias, transient ventricular dysfunction and early death after cardiac transplantation, and sudden cardiac death have been reported (Alegria et al., 2005).

Prevalence of myocardial bridging has not been reported consistently in the studies. It has been much higher at autopsy than on angiography (Somanath et al., 1989). This level of variation at autopsy can be attributable to the selection of hearts and care during preparation. Myocardial bridging has been reported to be highly prevalent in heart transplant recipients and in those with hypertrophic obstructive cardiomyopathy (HOCM) (Achrafi, 1992). In the case of HOCM, more intense contraction may reveal the bridges which are otherwise undetectable.

Epicardial course of coronary arteries is not pre-requisite in mammals as in rodents and lagomorpha, principal vessels are found to be embedded in myocardium under the epicardial surface (Type1) (Polacek & Zechmeister, 1968). Small ruminants, carnivores, and primates are the animals with a mostly epicardial course of arteries (Type 2). Major coronary arteries in the gorilla take the epicardial course but in chimpanzee they tend to take the mural course. Myocardial bridges are more frequent in goats and sheep than in humans (Giampalmo et al., 1964a) They can be also observed in seals, felines and canines. It is missing or found very rarely in horses and pigs (Type III) (Polacek & Zechmeister, 1968). Myocardial bridges which are congenital in origin and shows the possibility of possessing an evolutionary

remnant in the genetic code. They are most commonly found in the mid left anterior descending coronary artery (LAD) (Polacek & Zechmeister, 1968). When two parallel LAD branches are present, one often takes the intramural course (Geiringer, 1951).

Myocardial bridging was classified into two types of bridging by Ferreira et al. (1991). First type is superficial bridges which are found in 75 % of the cases. They cross the artery perpendicularly or at an acute angle toward the apex. The second type is a muscle bundles that emerge from the right ventricular apical trabeculae which cross the LAD diagonally, circuitously or spirally before ending in the interventricular septum. Atherosclerosis in relation with myocardial bridging has principally been studied in the left anterior descending artery. The portion proximal to the bridge normally shows atherosclerotic plaque formation. However, the myocardial segment is not affected by atherosclerotic plaque formation (Ishii et al., 1998a). Nonsignificant stenosis near the myocardial bridge and systolic compression of the tunneled segment have not been found to be sufficient evidence for association of myocardial bridging with ischemia. But, the likelihood of ischemia aggravates with the increase in the depth of the intramyocardial tunneled segment. Clinical presentations of myocardial bridging include angina, myocardial infarction, myocardial ischemia, stunning, left ventricular dysfunction, paroxysmal atrioventricular block besides exercise-induced ventricular tachycardia and sudden cardiac death (Hort, 2000; Noble et al., 1976). Nevertheless, these complications are rare considering the rate of prevalence of myocardial bridging. Patients have been found to present atypical or angina like chest pain with no stable association between severity of symptom and the length or depth of the myocardial segment or the degree of systolic compression (Ferreira et al., 1991). Perfusion defects have been noticed on myocardial scintigraphy but they are not a necessary condition even in even in deep bridges with considerable systolic compression or after vasoactive stimulation (Greenspan et al., 1980).

Coronary angiography is considered a diagnostic technique of gold standard for diagnosing myocardial bridges with characteristic milking effect and a step down -step up phenomenon caused by systolic compression of the myocardial segment. In symptomatic patients, initiation of therapy will improve quality of life. However, there is a lack of concrete evidence for promising effect on morbidity and mortality. Medication is recommended as first line therapy. Intracoronary administration of β-blocker reduced the vascular compression and the initial diastolic blood velocity(Schwarz et al., 1996) (Schwarz et al., 1996). Systolic

flow ratio was regulated and anginal symptoms got weakened. Surgical myotomy was first reported by Binet et al. (1975) for patients not responding to medication. Surgical myotomy abolishes clinical symptoms and is related to reversal of local myocardial ischemia and increase in coronary flow (Hill et al., 1981). It is important that surgery should be advised for patients with severe angina and evidence of clinically established ischemia because there is a probability of accidently opening the right ventricle during the surgery. The risks associated with the surgery should be reviewed against the usually uneventful course even in patient with a significant level of systolic compression.

Long term prognosis in patients with isolated myocardial bridging is normally good. Myocardial bridging can occasionally be associated with clinically significant complications although it is considered a benign condition. A significant number of research studies and reports have augmented the understanding of the pathophysiological mechanisms that are related with these complications. It is important to consider myocardial bridging particularly in patients at low risk for coronary atherosclerosis, angina like chest pain or clinically relevant myocardial ischemia.

## 1.1 Problem statement

One of the main diagnoses in the case of coronary artery disease (CAD) is the myocardial bridging. This myocardial bridging can be either typical or atypical angina pectoris. Moreover, it can occasionally manifest as acute myocardial infarction (AMI) or sudden death. Among the common public, this condition of myocardial bridging is relatively common and it is a benign pathology and the patients at low risk for CAD can be affected by this. But when it is symptomatic, it can appear to be stable or unstable angina, ventricular and supraventricular arrhythmias, sudden death and AMI; however, AMI and sudden death are rare (Cesar et al., 2004). This condition is rarely diagnosed because a few patients present the symptoms and there are unavailability and constrained utilization of the diagnostic techniques. Hence, the treatment methods and physiopathological mechanisms are not completely revealed. It is reported that there is a major discrepancy in the prevalence of myocardial bridging between the conventional angiography (average 5%, range 0.5% to 16%) (Porstmann & Iwig, 1960; Angelini et al., 1983b) and the autopsy findings (average 33%, range 15% to 85%) (Reyman, 1737; Portmann & Iwig, 1960). Therefore the detection of myocardial bridging is important as it is related to the cardiomyopathy and ischemic heart disease.

## 1.2 Aim and objectives

The aim of this study is to determine the frequency, angiographic characteristics, anatomical aspects, clinical manifestations and possible associations of myocardial bridges in a large urban Indian population of adults undergoing coronary angiography in SRMC.

The following are the objectives of this study:

To study the relationship between the systolic length and the diastolic length

To study the relationship between the systolic MLD% and diastolic MLD%

To study the difference in mean diastolic and systolic between male and female

To study the difference in mean diastolic and systolic between angina

To study the association between diabetes mellitus and gender

To study the association between hypertension and gender

To study the association between diabetes mellitus and age group

## 1.3 Chapterisation

The present study follows the chapter scheme mentioned below-

**Chapter I-** The first chapter of the present research is the Introduction wherein detailed information about the topic is covered. Furthermore, the problem statement, the aim and objectives of the research are elucidated.

**Chapter II-** The second chapter is the Literature review wherein previous researches in the context related to the present study are examined and discussed. In this section, a detailed explanation about the myocardial bridging and the previous studies are fully analyzed.

**Chapter III-** The third chapter covers the Research Methodology section. This section explains the type of research methodology adopted in the present study.

**Chapter IV-** The fourth chapter covers the results section. The results of the present research are fully covered.

**Chapter V-** The fifth chapter is the discussion and conclusion section wherein the results of the present study are examined so as to shed light on the aim and objectives of the present study. Furthermore, the conclusions of the study are drawn and recommendations for future

researches are revealed.

# CHAPTER II: LITERATURE REVIEW

This chapter presents definition of terms related to myocardial bridging and a review of the existing research on the topic.

## 2.1 Concepts and Definitions

### 2.1.1 Myocardial bridging

Myocardial bridging is a congenital coronary abnormality It is defined as a segment of a major epicardial coronary artery, the 'tunneled artery', that goes intramurally through the myocardium beneath the muscle bridge (Angelini et al., 2002b, 1983a).

### 2.1.2 Atherosclerosis

Atherosclerosis is a chronic, progressive, inflammatory disease with a long asymptomatic phase. Disease progression can eventually lead to the occurrence of acute cardiovascular events such as myocardial infarction, unstable angina pectoris and sudden cardiac death (Toth, 2008).

### 2.1.3 Angina

Angina pectoris is a common symptom of Ischemic heart disease. It is used to describe clinical symptoms such as discomfort in the chest, jaw, shoulder back or arms that are caused by physical exertion and emotional stress (Elveback et al., 1986; Kannel & Feinleib, 1972).

### 2.1.4 Myocardial Ischemia

Myocardial ischemia is a disorder which is usually caused by a critical coronary artery obstruction (Heston, 2015).

### 2.1.5 Myocardial Infarction

Myocardial infarction (MI) is defined as myocardial cell death due to prolonged ischemia (Thygesen et al., 2012).

### 2.1.6 Acute Coronary Syndrome

Acute coronary syndrome comprises an array of clinical situation ranging from unstable angina pectoris to non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI) (Escardio, 2017).

### 2.1.7 Coronary Angiography

Coronary angiography is a procedure in which a contrast dye is used and x ray images to deduct to blockages in the coronary arteries caused by plaque growth (National Institutes of Health, 2016). Coronary cineangiography continues to be one the most common technique for deducting myocardial bridging. Description of bridging on angiography is characterized by systolic narrowing or "milking" of an epicardial artery with a step-down and step-up marking the affected regions. Atherosclerosis lesions are mostly identified immediately proximal to the bridged segment.

### 2.1.8 Fractional Flow Reserve (FFR)

It has proven to be one of the most important tools in the physiological assessment of myocardial bridges. It is an evidence -based diagnostic test of the physiological significance of a coronary artery stenosis (Berry et al., 2015). Manifestation of hemodynamic alterations due to myocardial bridging was a most prominent decrease in diastolic FFR from 0.88 to 0.77. But, the mean FFR dropped down from 0.90 to 0.84. It was supposed that mean FFR measurements are unrealistically elevated by exceeding systolic pressures and thus selection of diastolic FFR evaluation should be considered as a method of choice. Accuracy rate of dobutamine is seen to be more than that of adenosine for FFR evaluation of bridging. This is indicative of the importance of inotropic state in the development of vessel compression (Wang et al., 2008).

### 2.1.9 Intravascular Ultrasound (IVUS)

Intravascular ultrasound (IVUS) or intravascular echocardiography is a combination of echocardiography and a procedure called cardiac catheterization. IVUS uses sound waves to produce an image of the coronary arteries and check their condition (Texas Heart Institute, 2016). Myocardial bridging on IVUS shows the tunneled segment of the artery clearly demonstrating the systolic compression that extends into diastole. Throughout the cardiac cycle there is a highly specific echolucent "half-moon" appearance. There is no adequate

understanding of its etiology. IVUS has the potential to deduct vessel compression with coronary provocation testing even when the significant “milking” of angiography is absent (Ge et al., 1994b). IVUS is still considered an important confirmatory testing method when angiographic diagnosis is uncertain, particularly when combined with provocation testing with acetylcholine, nitroglycerin, dobutamine, or rapid atrial pacing (Pichard et al., 1981).

## 2.2 Cardiac Computed Tomography (CT) Angiography

Computed tomography is considered a tool of significant value for the analysis of coronary anatomy and patency. Computed tomography technique has been used in many studies and the results have proved that CT is much more accurate and effective than angiography in deducting myocardial segments (La Grutta et al., 2009; Konen et al., 2007). This is a definite indication of higher prevalence of anatomical bridged segments than the subset that leads to vessel compression. Nevertheless, considering the findings with the CT technique are more of structural than functional nature, there is a need for more correlation tests to evaluate its clinical relevance. Non-invasive FFR measurement aided by CT might prove to be a useful method for anatomical and hemodynamic study of myocardial bridges. But, it has not yet been reported in any of the studied so far (Taylor et al., 2013).

### 2.2.1 Intracoronary Doppler

For the first time, Doppler tipped guidewires provided precise estimation of intracoronary flow velocity (Tio et al., 1997). Myocardial bridging on intracoronary Doppler is represented by “spike-and dome” pattern and “fingertip” phenomenon with rapid initial diastolic flow acceleration, rapid mid-diastolic deceleration and a mid-tolate diastolic plateau (Klues et al., 1997). Reverse flow during the systolic phase can be deducted immediately proximal to the bridged segment, aggravated by nitroglycerine provocation, mainly in deep bridges (Hongo et al., 1999). Coronary flow reserve in these patients is weakened distal to the bridge even when being usual or slightly reduced proximal to the bridge.

### 2.2.2 Special Patient Populations

There is a high prevalence of myocardial bridging among patients with hypertrophic cardiomyopathy. It has been found to be up to 80 % on angiography (Navarro-Lopez et al., 1986). This has seen a major factor for the higher level of mortality in the pediatric hypertrophic cardiomyopathy population, probably related to only ischemic or ischemic and

arrhythmic mechanisms. Several studies conducted on children with hypertrophic cardiomyopathy have reported greater levels of chest pain, ventricular tachycardia, resuscitated cardiac arrest background and positive changes of exercise testing in those with concurrent myocardial bridging (Schwarz et al., 2009). However, this kind of relationship does not appear in the case of adults. There was no variation in all-cause mortality and cardiac death at 6.8 follow up in a series of 425 adult patients with hypertrophic cardiomyopathy. Fifteen percent of the had myocardial bridging and 85 % without. A recent study reviewed the autopsied hearts in 255 patients who had died of sudden cardiac attack. The study did not find an independent contribution of myocardial bridging to mortality in patients with hypertrophic cardiomyopathy, though there was a higher level of prevalence (Basso et al., 2009).

### 2.2.3 Prognosis

Myocardial bridging is generally thought to be a benign condition, but however, it is seen to be a cause of angina-kind chest pain, coronary spasm, myocardial ischemia, which have been deducted using electrocardiography and myocardial perfusion stress testing, acute coronary syndromes, left ventricular dysfunction/ stunning, arrhythmias which includes supraventricular tachycardia and ventricular tachycardia, and sudden cardiac death. However, major events are rare and it is clear not definite as to myocardial bridging can be an independent causative factor for any of these events. Studies involving patients with myocardial bridging, tests for inducible myocardial ischemia showed a variation from 21-88 %, the wide range can be attributed to sensitivity and specificity of the tests (Tang et al., 2011). Ischemia was more closely associated with the systolic compression rate rather than lesion length and bridge location. A classification scheme for myocardial bridging was proposed by Schwarz based on the symptoms and non-invasive and invasive determination of hemodynamic and anatomical parameters (Schwarz et al., 2009). Given that there is data on long term follow up of patients with myocardial bridging, there is inadequacy in terms of studies that have established the natural history of MB in response to the intervention to corroborate the classification structure

### 2.2.4 Medical therapy

Primary medication for patients who are supposed to experiencing symptoms secondary to myocardial bridging include beta-blockers and non-dihydropyridine calcium-channel blockers (Alessandri et al., 2012).

### 2.2.5 Surgery

Surgical myotomy and coronary artery bypass graft surgery are the surgical interventions for myocardial bridging. Surgical myotomy involves resection of the overlying muscle and the procedure is recommended only if the case presents refractory symptoms despite medical therapy. Surgical myotomy has been found to eliminate symptoms and regulate coronary flow, but, the risks of this procedure include dissection into the right ventricle in patients with MB that take a deep subendocrdial course (Iversen et al., 1992). Coronary artery bypass surgery has been found to be a treatment for bridging. It involves anastomosis of the left internal mammary artery to the LAD artery (Attaran et al., 2013).

## 2.3 Background of Myocardia Bridging

It was in 1737, myocardial bridging (MB) was described anatomically for the first time by Haller and Reymann (1737). Myocardial bridging is congenital anomaly of coronary artery in a segment of epicardial coronary, mostly the middle of the left anterior descending artery takes in intramuscular path (Angelini et al., 2002b). This pattern of a segment of the artery taking an intramuscular course under the bridge of overlying myocardium frequently leads to vessel compression during systole. Despite myocardial bridging being frequently asymptomatic, the condition in many cases has been the cause for complications such as angina, myocardial ischemia (Rossi et al., 1980a), acute coronary syndrome (Tauth & Sullebarger, 1997), left ventricular dysfunction and stunning (Marchionni et al., 2002), arrhythmias and even sudden cardiac death (Tio et al., 1997). In 1960, Portsmann introduced the angiographic imaging (Porstmann & Iwig, 1960). Since then newer diagnostic applications as coronary computed tomographic angiography (CCTA), intravascular ultrasound (IVUS), intracoronary Dopplet and fractional flow reserve (FFR) have facilitated comprehensive analysis of the anatomic and hemodynamic consequences of the systolic compression including pathological effects of myocardial bridging on coronary flow. Notwithstanding the heightened understanding, treatment options for MB are limited. Beta-blockers and calcium-channel blockers continue to constitute the primary medication therapy Mohlenkamp et al.(2002) and surgical myotomy is considered for refractory cases (Katznelson et al., 1996). Percutaneous coronary interventions in the form of stenting have been used, but however, major complications such as stent fracture and coronary perforation have been reported (Broderick et al., 1996).

### 2.3.1 Prevalence of Myocardial Bridging

Rate of reported myocardial bridging differ based on the mode of evaluation (Lee & Chen, 2015). Several autopsy series have been performed and rate of reporting has been from 5 to 86 %. Research by Risse and Weiler (1985) was one of the largest autopsy studies. The study which involved 1056 patients reported intramyocardial coronary artery course in 26 % of patients. This rate was much higher than angiographically reported bridging, which detects systolic compression at rates from 0.5-12 % even though deduction can go as high as 40 per cent when provocation tests are used (Mohlenkamp et al., 2002). Recent studies that used CCTA have deducted bridged coronary segments at rates as it was in the autopsy series (Wymore et al., 1989). In certain populations, like hypertrophic cardiomyopathy patients (Mohiddin et al., 2000), and heart transplant patients, MB rates are much higher than the general population.

### 2.3.2 Morphology and Histology

Myocardial bridging is frequently found at the mid-LAD (Left anterior descending) artery on coronary angiography. It has been deducted at right coronary artery (RCA) and left circumflex (LCX) in some autopsy series at rates at almost levels (Polacek & Kralove, 1961). Diagonal and marginal branches, secondary arteries are covered on histology. Depth of a typical myocardial bridge is 1-10 mm and its length is 10-30 mm (Angelini et al., 1983a). Differences in bridging owing to gender or age have not been observed.

There does not appear to be a difference in prevalence of bridging by gender or age. Myocardial bridges of the LAD artery deducted by pathology are categorized into two discrete subtypes by Ferreira et al. (1991). “Superficial” bridges which is found in 75 % cases are more common are located at LAD, in the intraventricular channel and crossed by a bundle perpendicularly or at an acute angle.

Second subset of myocardial bridge is a “deep” bridge in which the LAD artery takes a deviant course towards the right ventricle and gets into the intraventricular septum, with a epicardial longitudinal muscle bundle emerging from the right ventricular apex and moving across the tunneled segment diagonally, in a roundabout or spiral manner before ending its course in the intraventricular septum. Another subset of myocardial bridging is the myocardial loop in which an overlying vessel runs through a segment of atrial myocardium (Polacek & Kralove, 1961).

### 2.3.3 Pathophysiology of Ischemia

The rate of myocardial ischemia and its symptoms is prime facie considered to be a grave danger arising out of perceived anomalies coronary flow by myocardial bridging. However, as the most of the coronary filling occurs in diastole, systolic compression on the artery should have only weak impact on the effective myocardial perfusion (Marcus et al., 1999).

## 2.4 Review of Existing Studies

Dulk et al. (1983) reported that ischemia of the sub-nodal conduction system resulting from compression of the left anterior descending coronary artery can be the cause of paroxysmal block. The study reported the finding of (Binet et al., 1975b) that myocardial bridging can cause sudden death during exercise in people who are otherwise healthy. The study reported the case of a patient who did not suffer from angina pectoris despite an extensive septal defect was observed on the exercise thallium perfusion scan.

Lauer and Carlson (1998) reported a case of an 84-year-old woman with exertional chest pain having undergone a preoperative evaluation for a colectomy. Echocardiography test showed hyperdynamic left ventricular systolic function and moderate concentric left ventricular hypertrophy. On coronary arteriography, the patient's arteries were found to be free of atherosclerotic disease. Nevertheless, systolic bridging in the middle of the left anterior descending coronary artery was seen. After the patient's chest pain subsided, she was administered β-blocker and thereafter underwent a successful colectomy without suffering a cardiac event.

Yano et al. (2001) conducted a study to elucidate the clinical importance of myocardial bridging in patients with inferior wall myocardial infarction and shock. The study reported that myocardial bridging when in a state of increased myocardial oxygen consumption can lead to ischemia. Oxygen consumption by the anterior wall myocardium is likely to increase when there is compensatory hypercontraction of the anterior wall as a response to decreased inferior wall motion caused by myocardial infarction of the interior wall. The study noted that the major cause of shock in patients was due to the inability to meet the increased oxygen demands of the anterior wall myocardium after the inferior wall.

Bauters (2002) reported a case of 28-year-old man with heavy chest pain which had lasted for four hours. The patient had been healthy and had no risk factors for coronary heart disease. A large intraluminal filling defect was seen in the left anterior descending artery (LAD) indicative of an extensive intracoronary thrombus. The patient was given abciximab therapy for 24 hours and enoxaparin for 10 days and was later discharged on daily dose of aspirin 160 mg and clopidogrel 75 mg. After three months, the patient was asymptomatic, for systematic control angiography indicated complete disappearance of the thrombus. LAD was evident with a characteristic image of myocardial bridging.

Mohlenkamp et al. (2002) reported that motivation for clinical interest and systematic research came after an observed association of myocardial bridging with myocardial ischemia. The study reported high prevalence of myocardial bridging in heart transplant recipients and in patients with hypertrophic obstructive cardiomyopathy (HOCM). In the case of patients with HOCM, more rigorous contraction may reveal otherwise undetectable bridges. The authors noted that that primary studies in the LAD are related to association of myocardial bridging with coronary atherosclerosis. They reported that the segment near to the bridge frequently showed atherosclerotic plaque formation, but, however, the tunneled portion is typically unaffected, which was observed by Ishii et al. (1998) and Ishikawa et al. (1997). The study also reported that severe ischemia and associated symptoms cannot be sufficiently explained only by nonsignificant stenosis proximal to the bridge or systolic compression of the tunneled segment. The study concluded that myocardial bridging can rarely cause clinically significant complications although it is usually a benign condition. Thus, it should be given due attention especially in patients at low risk for coronary atherosclerosis, angina, or myocardial ischemia.

Schunkert (2003) reported a case of a 61-year-old man who had come for evaluation of retrosternal chest pain. eccentric stenosis pf the proximal left anterior descending coronary artery was observed on coronary angiography. Primary implantation of 3.5 x 12 mm stent successfully revascularized the LAD. Post-interventional angiogram showed residual stenosis. Extensive myocardial bridging involving the mid-LAD and a prominent first diagonal branch was seen distal to the lesion.

Bekkers (2006) reported a case of a 52-year-old man without any of history of cardiac disease having been admitted to the coronary care unit because of an acute anterior wall myocardial infarction. Coronary angiography showed single-vessel disease of the left anterior

descending artery. In addition to the bridging segment, there were no additional residual stenosis found. Only minimal myocardial damage was found and the patient had an uneventful recovery.

Duygu et al. (2007) conducted a research to investigate the demographic, clinical and angiographic characteristics of the patients with myocardial bridging observed on coronary angiography. The authors concluded that myocardial bridging should be considered in young patients with angina or in case the same symptoms persisted in the patients with only one risk factor for coronary artery disease. The authors reported that myocardial bridge may trigger the development of atherosclerotic lesion or may pave way for the progression of atherosclerosis in the proximal segment of the vessel. The risk of acute coronary syndrome rises when atherosclerosis overlaps myocardial bridging.

Geiringer (1951) reported that myocardial bridging can be considered as a congenital variant. Ge et al. (1995) agreed with the previous pathoanatomic studies by Giampalmo et al. (1964) and Angelini et al. (1983) in the finding that atherosclerotic plaques were found proximal to the bridged section. This was the case even in patients with normal appearing coronary angiograms, but the bridged sections were free of the disease. With coronary atherosclerosis being observed in 85 % men and 55 % of women in the general population, it continued to be uncertain if the amount of distribution of atherosclerosis in the proximal section are different from that found in a control population as myocardial bridging is a common occurrence and might coincide with another frequent disease. Ishikawa et al. (2009) stated that there is evidence for the role of myocardial bridging as a congenital anatomic risk factor for coronary atherosclerosis and myocardial infarction. They conducted a study to analyze the extent and distribution of coronary atherosclerosis in 100 consecutive autopsy hearts from patients with myocardial infarction. They found that almost half the number had myocardial bridge. Further, they analyzed 200 normal hearts, in which 100 had myocardial bridging. The observed that coronary atherosclerosis was more definite and led up to the coronary ostium augmenting natural history of the disease predisposing to myocardial infarction.

Bandyopadhyay et al. (2010) noted that although myocardial bridging is a congenital anomaly, it is detected on angiography during investigation for heart diseases and there is an association between myocardial bridging and atherosclerosis. The authors added that it is very difficult to establish a direct association between myocardial bridging and myocardial

infarction. The study also reported that the length of myocardial bridge has a definite role in producing ischemia symptoms as the longer the myocardial bridge, the more significant will be the systolic compression on the coronary arteries.

Abhilash et al. (2011) studied the clinical and angiographical profile of myocardial bridging form consecutive coronary angiograms performed over five years at Thiruvanthapuram Medical college. The aim of the study was to assess the risk of cardiovascular events and the risks of accelerated atherosclerosis in isolated myocardial bridging. The study reported that use of inotropes heightened the severity of myocardial bridges by adding to the contractility of heart. The study added incidence of myocardial bridging was higher in patients with lower systematic blood pressure and rheumatic heart disease. The study concluded that myocardial bridging should be considered mainly in patients at low risk for coronary atherosclerosis, with angina- like chest pain or well diagnosed myocardial ischemia.

Xu et al. (2011) reported a case of 57-year-old man with a long history of frequent chest pain. The patient had undergone coronary artery bypass grafting with the left internal mammary artery to the segment of the left anterior descending coronary artery grafting for myocardial bridging in LAD even after having been administered β-blockers and calcium channel blockers. Nevertheless, within three months after the surgery, the patient suffered repeated angina. Coronary narrowing caused by myocardial bridging not only occurs in systole but also in a lagged and partial vessel diameter gain during mid-to late diastole, thus affecting the principal phase of the coronary perfusion (Schwarz et al., 1997). Because of the risk of stent thrombosis and restenosis, stenting should not be recommended in myocardial bridging (Tandar et al., 2008). For patients with persistent symptoms even with intensive medication, surgical myotomy will serve as a radical correction procedure. Xu et al. (2011) recommended supra-arterial myotomy should be the first-preferred treatment in symptomatic patients with myocardial bridging in the case of they do not respond to medical therapy.

Corban et al. (2014) observed that patient with myocardial bridging are often asymptomatic, but there is a probability of this anomaly being associated with angina, cardiac arrhythmias, acute coronary syndrome, syncope or even sudden cardiac death. They emphasized the need for advocating aggressive risk factor modification and considering antiplatelet therapy in patients with myocardial bridging since they are at increased risk for developing atherosclerosis. To evaluate symptomatic patients, they recommended that a

series of noninvasive and invasive diagnostic modalities that reveal the pathophysiology of myocardial bridging can be deployed. They reported that medical therapy with beta-blockers and calcium channel blockers, continued the predominant form of treatment, but, however, for patients who do not respond to intensified medical therapy, surgical intervention, or less preferably PCI with DES can be considered. In the backdrop of limitations of the literature on myocardial bridging, Corban et al. (2014) stated that to establish the prevalence of myocardial bridging, clinical identification should require two angiographic views obtained after nitroglycerin administration instead of using computed tomography, which should be used only for measuring the length and depth of the bridge.

Angelini (2014) also reported that chest pain, myocardial infarction and sudden death are not systematically associated with myocardial bridging of any anatomic severity and most myocardial bridges are benign. Corban et al. (2014) noted that myocardial bridges, in fact, prevent coronary artery disease inside the affected segments. Still there is a misconception about the location of plaque in relation to the myocardial bridging. It is not at the entrance of the bridge there is maximal plaque burden, but on average 20 mm to 30 mm near the entrance of the bridge (Ishikawa et al., 2013; Lin et al., 2013).

Meena et al. (2014) conducted a study to determine the frequency, angiographic characteristics, anatomical aspects, clinical presentations and possible associations of myocardial bridges in large urban Indian population of adults who undergo coronary angiography. The retrospective study analyzed the angiographic data of 3275 adult patients. The study found that myocardial bridge was present in 42 of the 3275 patients. Myocardial bridge was found in LAD in 40 patients and in 2 patients it was found in left circumflex coronary artery. The study concluded that chest pain was the common reason for angiography in patients with myocardia bridge. The study noted that prevalence of myocardial bridge may vary based on the population and myocardial bridging can aggravate atherosclerosis and quicken the pace of myocardial infarction.

Nakaura et al. (2014) conducted a study to investigate if myocardial bridging is an independent risk factor for coronary atherosclerosis. The study concluded that the portion of coronary artery near to the segment with myocardial bridging has increased risk of atherosclerosis and therefore adequate attention should be paid to the development of coronary atherosclerosis in the segment near myocardial bridging especially in the case of elderly patients with diabetes mellitus.

Bergmark et al. (2015) reported that myocardium overlying the epicardial coronary arteries is much more common at autopsy as it is being found in 30 % to 85 % of adults. The study reported that prevalence is high among heart transplant recipients and patients with hypertrophic cardiomyopathy with de novo myocardial bridging reported in the heart transplant patients. Like many previous studies, the authors reported that although myocardial bridging is typically benign, several clinical presentations have been attributed to it such as ischemia, infarction, ventricular tachycardia and even sudden cardiac death. The authors further noted that although many studies have shown the encroachment of compression into mid- or even late diastole with decreased diastolic epicardial, and coronary sinus blood flow, vessel compression is naturally considered to occur primarily during systole.

Lee and Chen (2015) reported that the bridged segment itself is unaffected from atherosclerosis possibly due to favourbale shear forces that lead to increased expression of vasoactive agents and morphological changes in endothelial and smooth muscle cells in the region. The authors added that hemodynamic effects of bridging are systolic coronary flow reversal near the bridge and decrease in coronary flow reserve and their clinical consequences range from angina to acute coronary syndrome to sudden cardiac death.

Yuan (2016) reported pathophysiology of myocardial bridging is inadequately understood and added that hemodynamic and structural changes such as disturbance in blood flow, malperfusion, deposits of lipids and mucopolysaccharides and elastic damages can be observed in the coronary artery portion proximal to myocardial bridge. These changes facilitate the formation of atherosclerotic plaques in the intima of the coronary artery segment. The study concluded that since myocardial bridging is associated with a series of severe cardiovascular events such as myocardial infarction, arrhythmia and sudden death, symptomatic patients must be treated conservatively, interventionally or surgically based on the patient's condition.

Poloński (2015) reported that plaque distribution is not consistent. Atherosclerosis more commonly occurs in the LCA (left anterior descending artery) in the RA (right artery) but the LCx (left circumflex branch) is less frequently affected. Septal perforators emerging from the LAD and RCA trigger higher prevalence of atherosclerosis in the two arteries, whose effect gets aggravated by multiple branching points of the RCA and LAD in comparison to the LCx. The structure of coronary anatomy branching demonstrates

the un-organized distribution of atherosclerotic plaques and prevalence of atherosclerosis in the LAD and RCA in comparison to the LCx.

Jukić et al. (2017) reported that myocardial bridging can be common cause of chest pain especially in patients with more atypical presentation. The study in its conclusion noted that considering the low sensitivity of invasive coronary angiography (ICA) for deducting myocardial bridging, ICA should not be the complete diagnostic option and CCTA can be coronary computed tomography angiography better suited for young women with a typical chest pain with low intermediate risk for coronary artery disease.

## 2.5 Research Gap

Review of the existing studies indicate that most of the authors agree that though MB is thought to be a benign condition, it might be an independent risk factor for complications as angina, acute coronary syndrome left ventricular dysfunction and stunning, arrhythmias and even sudden cardiac death. So, they see the need for a comprehensive multicenter clinical database to identify the parameters that justify the role of myocardial bridges in adverse cardiac events. Therefore, the aim of this study is to determine the frequency, angiographic characteristics, anatomical aspects, clinical manifestations, and possible associations of myocardial bridges.

# CHAPTER III: RESEARCH METHODOLOGY

The aim of this retrospective study was to evaluate the frequency, angiographic features, anatomical characteristics, clinical presentations and factors that are associated with myocardial bridges in urban Indian adults who were going through coronary angiography.

Observational studies are categorized under analytical study design. They are further sub-divided into observational or experimental study designs. The objective of analytical studies is to deduct and evaluate causes or risk factors of diseases or events that are related to health (Song & Chung, 2010). Retrospective cohort studies, which are otherwise called historical cohort studies, looks at the past to investigate certain medical events or outcomes (Song & Chung, 2010). The factor that differentiates observational and experimental study is that in experimental study design the study group comprises of subjects are either subjected to an intervention or not, whereas in the observational study design the investigator does not have any control over the study subjects. The investigator only observes and evaluate the strength of the association between the exposure and disease variable.

Retrospective study selects its subjects based on the exposure status and outcome data which were measured in the past are reconstructed for analysis. Advantage of retrospective study design are investigator's limited control over data collection and, since the data is immediately available, this study design is less expensive and time consuming. In addition, retrospective studies like prospective studies provide specific advantages of measuring disease occurrence and its association with an exposure. The most important factor in cohort studies such as retrospective study is categorizing the selected set of subjects based on the status at the start of the investigation. A prominent characteristic od subject selection is to have the exposed and unexposed groups from the same source population.

This study was conducted at……. The study retrospectively analyzed the angiographic data of 100 patients who were undergoing coronary angiography for deducting myocardial bridging. There were ……men and ……. women; mean age, years; age range,……. The patients were admitted at ……. between ……and ……with the diagnosis of likelihood for coronary artery disease and thus required diagnostic angiography. Each patient had one or more risk factors of coronary artery disease such as smoking, diabetes mellitus, family history, hyperlipidemia and hypertension. Patient characteristics such as age, gender, vascular risk factors and clinical presentation were recorded.

Myocardial bridging was described angiographically by Portmann and Iwig in 1960 (Ripa et al., 2007). The prevalence of this anomaly was more frequent than it was thought to be. Pathophysiological mechanisms of clinical presentations of myocardial bridge are supposed to be related to tendencies in the patients to develop atherosclerosis. Coronary angiography first began with the human heart catheterization in 1929. The method has evolved with a series of progressive technical and cultural developments. It has emerged into as a selective coronary angiography, direct needle vascular puncture, pre-shaped dedicated coronary catheters and refinement of radial access and thus has begun to make indispensable contribution to endovascular intervention (La Vecchia, 2013). Characteristic angiographic feature of a myocardial bridge is systolic narrowing of an epicardial artery. This is most of the time completely resolved during the diastolic phase of the cardiac cycle. The diagnosis depends on the change in diameter between the systole and diastole within the bridged coronary segment. Coronary angiography presents a milking effect when there is 70 % and above reduction in minimal luminal diameter during systole and more than 35 % stable minimal luminal reduction during mid-to-late diastole. Intracoronary nitroglycerin injection is used to accentuate the systolic narrowing at the bridge, by dilatating the blood vessels proximal to non-bridged coronary segments.

In this study, before coronary angiography was administered on the patients, they went through echocardiogram and routine pre-procedure care by using standard procedures. Two qualified cardiologists who determined the diameters of coronary lumen reviewed each cineangiogram retrospectively. Programmable digital caliper was used for angiographic quantification of systolic lumen compression. Systolic lumen diameter reduction and the length were measured. Left anterior oblique position as well as systolic lumen diameter reduction and length were measured. Only after the disparities between the measurements of the two investigators was less than 20 % they were accepted.

Patients were divided into three groups corresponding to the degree of systolic compression. Patients in group 1 were those less than 50 per cent of systolic compression of the epicardial vessel imposed by myocardial bridge. It was considered as mild level. Patients in group were those with 50-70 % systolic compression, which was of a moderate range. Patients in whom there was more than 70 % systolic compression, which was considered of a significant range, comprised the third group. Two cardiologists who reviewed the cineagiographs were not given any information about patient's medical history. Cineangiographic projection that showed the highest level of bridging was used as to evaluate

maximal systolic compression and the total length of the bridged segment. Every artery was observed closely for the presence of maximal percentage of systolic compression, myocardial bridging, total length of the bridging, and diameter of the bridged segment. Calibration with the given diameter of the coronary artery catheter was used to measure the length and diameter of arterial segment. In the case of patients with multiple sites of bridging, the lengths of each bridged segment were added for the total length. The area impacted by bridging was defined using standard nomenclature of coronary artery disease.

The study was ratified by the institutional clinical review board as a retrospective research. All the patients gave their consent in writing for enrolling them in the study. All the procedures were performed in adherence with the ethical standards of the ……. Ethics Committee.

## 3.1 Statistical Analysis

SPSS was used for data analysis. All data were verified for normal distribution prior to analysis and suitable test was used. All values were written as mean ± standard deviation unless they have not been suggested in the study. For determining the difference in the thickness of MB, diameter and systolic lumen and length between the end-systolic phase and end-diastolic phase independent sample test was used. Value less than 0.05 was considered statistically significant value.

# CHAPTER IV: RESULTS

## 4.1 Introduction

This study is a retrospective study. In this study evaluated patients seen in our medical centre between January 2013 and September 2013 admitted with a diagnosis of possible coronary artery disease (CAD) requiring diagnostic coronary arteriography.The angiographic data of adult patients undergoing coronary angiography were retrospectively analysed for the diagnosis of myocardial bridge. Quantitative coronary angiography was used for analysis. The sample size taken for the study n=142. Thus, using SPSS software the present study results analyzed. The analysis carried out was percentage analysis to find out the demographical information of respondents. Descriptive statistical measures were carried out in each variable. Analysis of Variance (ANOVA) is to compare the mean between more than two categorical variables. Chi-square analysis was used to find the association between categorical variables. P< 0.05 was considered statistically significant.

**Table 1: Prevalence of Myocardial bridging patients with Angiograms in 5 years**

| | Count (n) | Prevalence (%) |
|---|---|---|
| Myocardial Bridging | 142 | 1.5 |
| Angiograms in 5 years | 9464 | |

Table 1 reveals the prevalence of myocardial patients. Of total 9464 angiograms patients in 5 years in which 142 (1.5%) patients were affected by myocardial bridging.

**Table 2: Frequency of sex**

| Gender | Frequency (n) | Percentage (%) |
|---|---|---|
| Male | 104 | 73.2 |
| Female | 38 | 26.8 |
| **Total** | **142** | **100.0** |

Table 2 shows sex of the patients. Of total 142 patients, majority 104 (73.2%) of the patients were male while 38 (26.8%) were female.

**Figure 1: Percentage for sex**

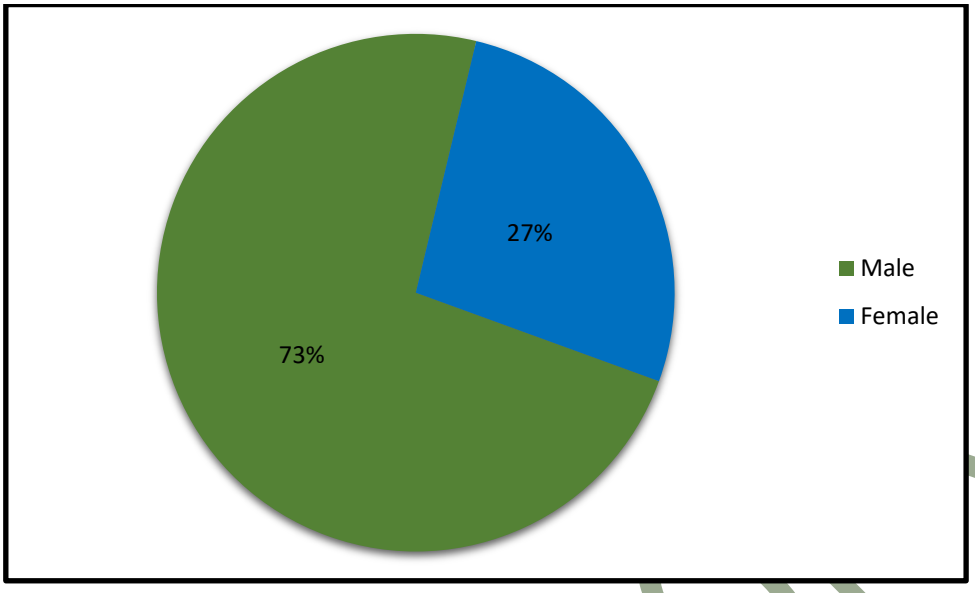


**Table 3: Frequency of age group**

| Age group | Frequency (n) | Percentage (%) |
|---|---|---|
| <=45 years | 32 | 22.5 |
| 46-60 years | 77 | 54.2 |
| >=60 years | 33 | 23.2 |
| **Total** | **142** | **100.0** |

Table 3 shows the frequency of age group of the patients. Majority 54.2% of the patienss were under 46-60 years age group followed by, 23.2% of the patients were >=60 years age group while 22.5% of the patients were <=45 years age group respectively.

**Figure 2: Percentage for age**

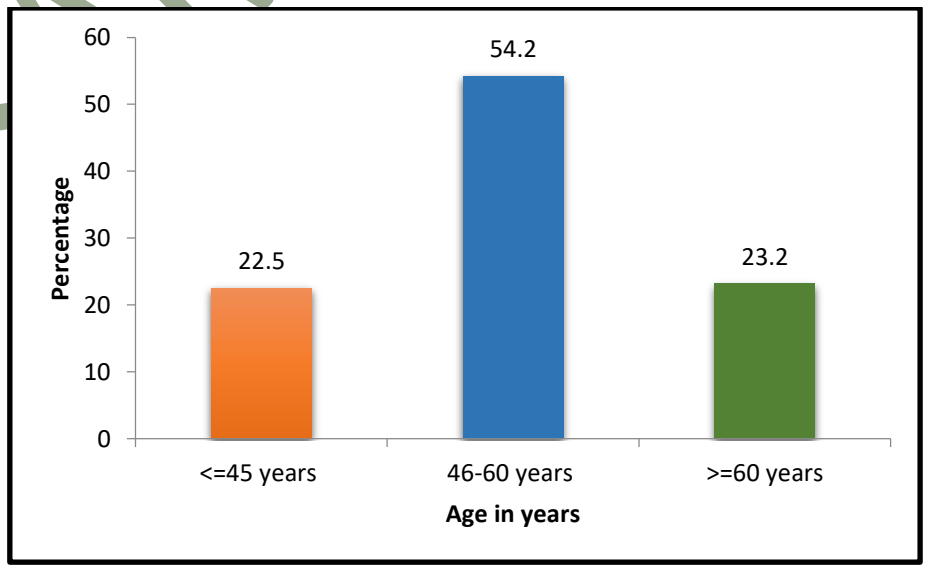

**Table 4: Frequency of Diabetes Mellitus**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| Yes | 75 | 52.8 |
| No | 67 | 47.2 |
| **Total** | **142** | **100.0** |

Table 4 shows the frequency of diabetes mellitus. Majority 52.8% of the patients are have diabetes mellitus while 47.2% of the patients do not have diabetes mellitus respectively.

**Figure 3: Percentage for Diabetes Mellitus**

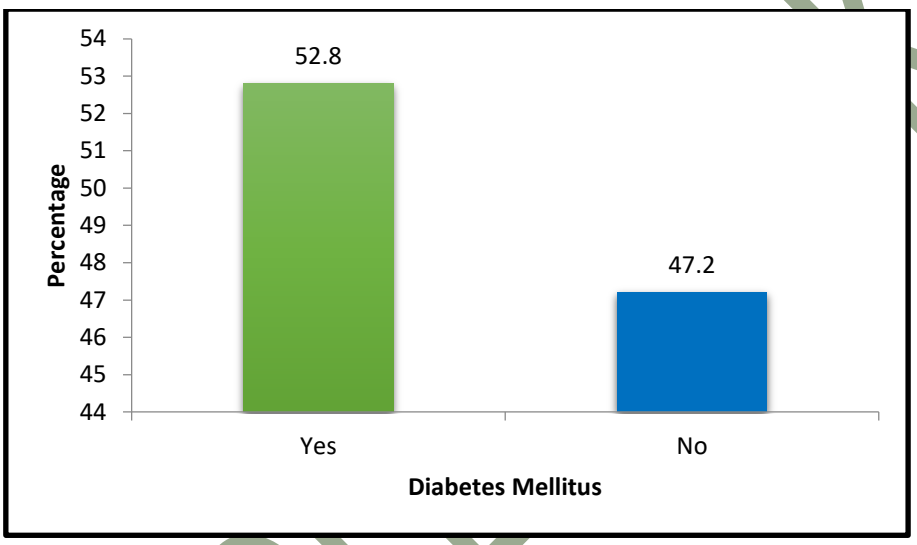


**Table 5: Frequency of Hypertension**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| Yes | 69 | 48.6 |
| No | 73 | 51.4 |
| **Total** | **142** | **100.0** |

Table 5 shows the frequency of hypertension. Majority 51.4% of the patients are have hypertension while 48.6% of the patients do not have hypertension respectively.

**Figure 4: Percentage for Hypertension**

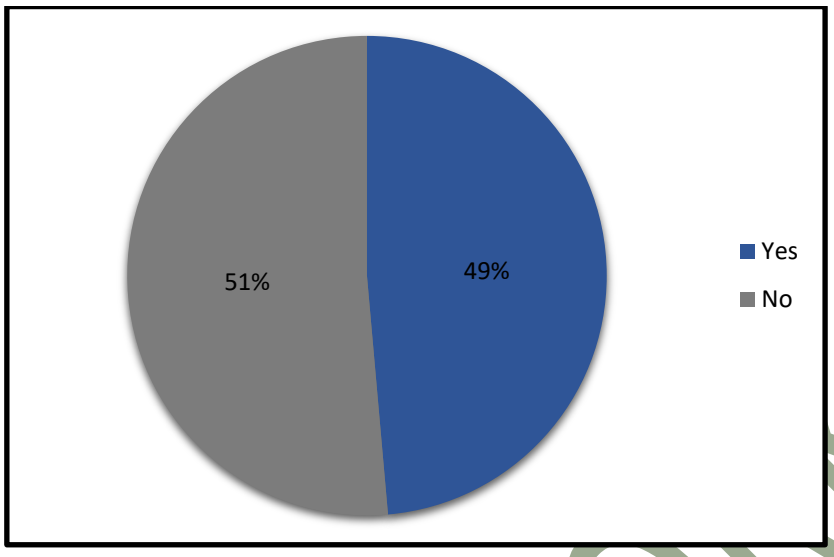


**Table 6: Frequency of Treadmill test**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| Nil | 78 | 54.9 |
| Positive | 64 | 45.1 |
| **Total** | **142** | **100.0** |

Table 6 shows the frequency of treadmill test of the patients. Out of 142 patients, majority 54.9% of the patients are nil while 45.1% of the patients are positive respectively.

**Figure 5: Percentage for Treatmill test**

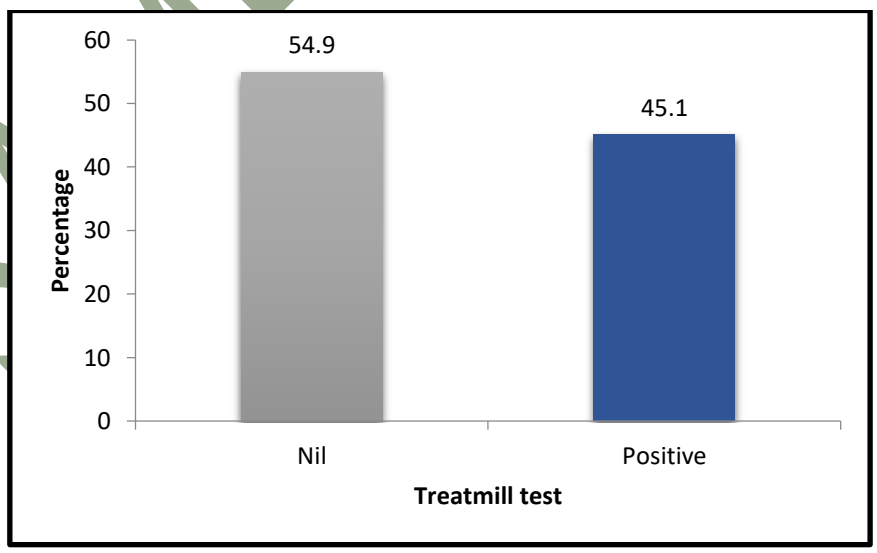

**Table 7: Frequency of angina**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| Yes | 101 | 71.1 |
| No | 41 | 28.9 |
| **Total** | **142** | **100.0** |

Table 7 shows the frequency of angina of the patients. Out of 142 patients, majority 71.1% of the patients are have angina while 28.9% of the patients don't have angina respectively.

**Figure 6: Percentage for angina**

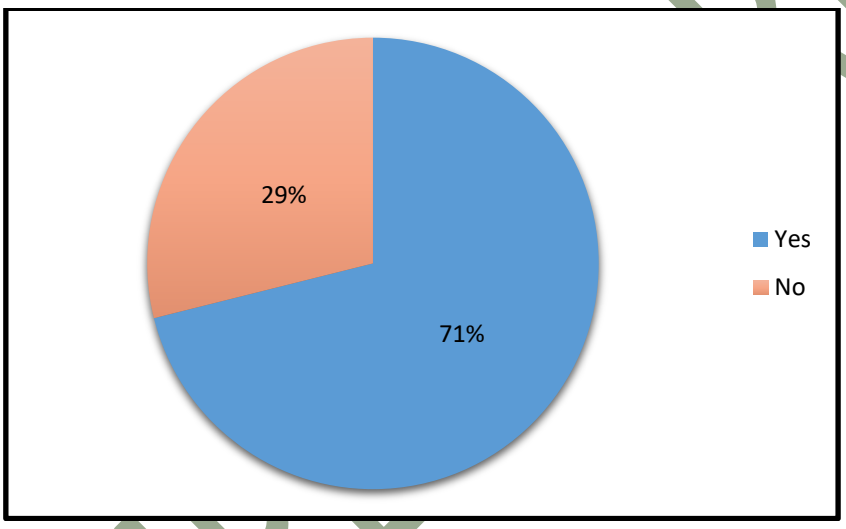


**Table 8: Frequency of artery**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| AN. Left Circumflex | 1 | 0.7 |
| Distal. Left anterior descending artery | 25 | 17.6 |
| Distal. Right Coronary Artery | 1 | 0.7 |
| Mid-portion Left anterior descending | 115 | 81.0 |
| **Total** | **142** | **100.0** |

Table 8 shows the frequency of artery. Out of 142 patients, majority 81% of the patients are have Mid-portion Left anterior descending followed by, 17.6% of the patients have Distal. Left anterior descending artery while 0.7% of the patients have Distal. Right Coronary Artery and AN. Left Circumflex respectively.

**Figure 7: Percentage for artery**

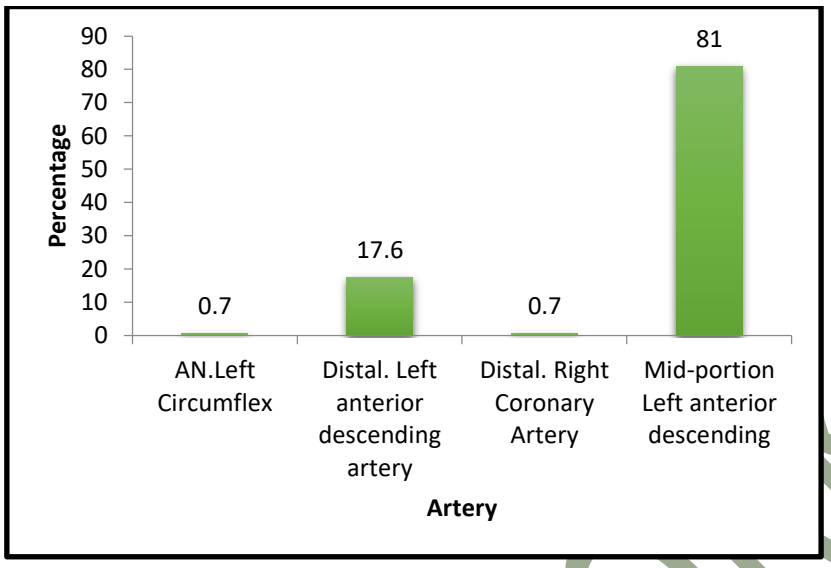


**Table 9: Frequency of systolic length**

| | Frequency (n) | Percentage (%) |
|---|---|---|
| Below 10 | 2 | 1.4 |
| 10 to 20 | 44 | 31.0 |
| 20 to 30 | 57 | 40.1 |
| 30 to 40 | 36 | 25.4 |
| Above 40 | 3 | 2.1 |
| **Total** | **142** | **100.0** |

Table 9 shows the systolic length of the patients. Majority 40.1% of the patients were 20 to 30 systolic length followed by, 31% of the patients were 10 to 20 systolic length, 25.4% of the patients are were 30 to 40 systolic length while least 1.4% of the patients were below 10 systolic length respectively.

**Figure 8: Percentage for systolic length**

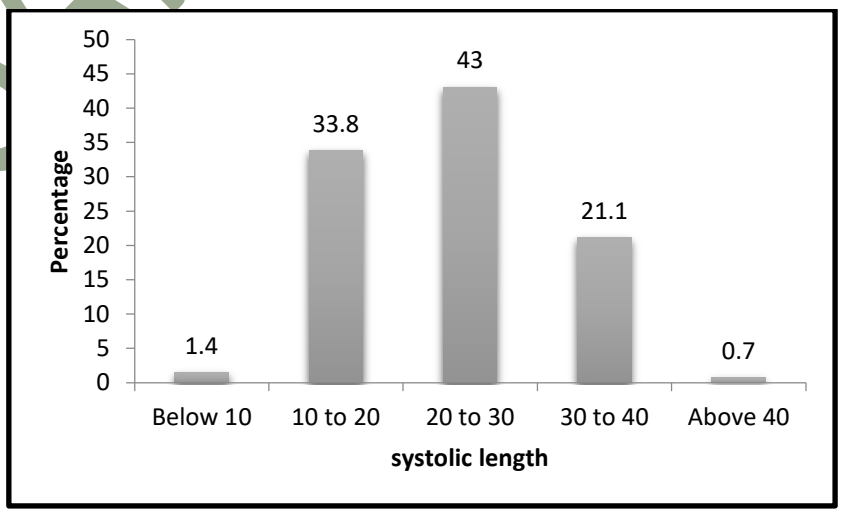

**Table 10: Descriptive statistics of diastolic and systolic**

| | Mean | SD | Max | Min |
|---|---|---|---|---|
| DIASTOLIC-ST.LENGTH | 25.37 | 7.33 | 40.40 | 8.46 |
| MLD% | 15.21 | 6.38 | 36 | 0 |
| SYSTOLIC-ST.LENGTH | 23.99 | 7.29 | 41.72 | 8.44 |
| MLD% | 34.55 | 9.5 | 62 | 16 |

Table 10 shows the descriptive statistics of diastolic and systolic length of the patients. The average diastolic length is 25.37 SD 7.33 with maximum 40.4 and minimum 8.46, Minimal Lumen Diameter has average 15.21, SD 6.38 maximum 36 and minimum 0. While average length of systolic is 23.99, SD 7.29 with maximum 41.72 and minimum 8.44 respectively.

**Table 11: Relationship between systolic length and diastolic length**

| | DIASTOLIC-ST.LENGTH | SYSTOLIC-ST.LENGTH |
|---|---|---|
| DIASTOLIC-ST.LENGTH | 1 | .898** |
| SYSTOLIC-ST.LENGTH | | 1 |

**p<0.01

Table 11 presents the Pearson correlation analysis. The correlation analysis shows the linearity between the variables not the strength of association between dependent and independent variables represented by r and p value, while r is a degree of correlation and p signifies significance level. It is evident from the table that diastolic length does showed a significant positive linear relationship with systolic length (r=0.898, p< 0.01).

**Table 12: Relationship between systolic mld% and diastolic mld%**

| | Systolic MLD% | Diastolic MLD% |
|---|---|---|
| Systolic MLD% | 1 | .268** |
| Diastolic MLD% | | 1 |

**p<0.01

Table 12 presents the Pearson correlation analysis. The correlation analysis shows the linearity between the variables not the strength of association between dependent and independent variables represented by r and p value, while r is a degree of correlation and p signifies significance level. It is evident from the table that Systolic MLD% does showed a significant positive linear relationship with Diastolic MLD% (r=0.268, p< 0.01).

**Table 13: Difference in mean diastolic and systolic between male and female**

| | Sex | | | |
|---|---|---|---|---|
| | **Male (n=104)** | **Female (n=38)** | **t value** | **p value** |
| | **Mean±SD** | | | |
| DIASTOLIC st length | 25.73±7.65 | 24.37±6.34 | 0.951 | 0.331 |
| MLD% | 15.91±6.54 | 13.29±5.57 | 4.822 | **0.030*** |
| SYSTOLIC st length | 24.25±7.40 | 23.28±7.02 | 0.488 | 0.486 |
| MLD% | 35.88±9.46 | 30.92±8.70 | 7.842 | **0.006**** |

*p<0.05, **p<0.01

Table 13 reveals difference in mean difference in mean diastolic and systolic between male and female. Since p-value for diastolic length of MLD% (0.030<0.05) systolic length of hence MLD% (0.006<0.05), hence there is a significant difference in mean difference in mean diastolic and systolic between male and female. In male patients, diastolic length of MLD% has high mean (15.91±6.54) while compared to female patients diastolic length of MLD% has low mean (13.29±5.57). In male patients. In male patients, systolic length of MLD% has high mean (35.88±9.46) while compared to female patients systolic length of MLD% has low mean (30.92±8.70).

**Table 14: Difference in mean diastolic and systolic between with and without diabetes mellitus patients**

| | Diabetes Mellitus | | | |
|---|---|---|---|---|
| | **Yes (n=75)** | **No (n=67)** | **t value** | **p value** |
| | **Mean±SD** | | | |
| DIASTOLIC st length | 25.30±7.12 | 25.44±7.61 | 0.014 | 0.907 |
| MLD% | 14.77±5.98 | 15.70±6.28 | 0.75 | 0.389 |
| SYSTOLIC st length | 24.05±7.65 | 23.92±6.92 | 0.012 | 0.914 |
| MLD% | 34.31±9.20 | 34.82±9.99 | 0.102 | 0.750 |

Table 14 reveals difference in mean diastolic and systolic length between with and without diabetes mellitus patients. Since p>0.05, hence there is no significant difference in mean diastolic and systolic between with and without diabetes mellitus patients.

**Table 15: Difference in mean diastolic and systolic between Hypertension**

| | Hypertension | | f value | p value |
|---|---|---|---|---|
| | Yes (n=69) | No (n=73) | | |
| | Mean±SD | | | |
| Diastolic st length | 25.40±7.50 | 26.18±7.11 | 1.871 | 0.174 |
| MLD% | 15.13±6.99 | 15.29±5.81 | 0.021 | 0.884 |
| Systolic st length | 23.40±7.81 | 24.89±6.69 | 2.286 | 0.133 |
| MLD% | 33.30±7.68 | 36.01±10.94 | 3.592 | 0.060 |

Table 15 reveals difference in mean diastolic and systolic length between hypertension. Since p>0.05, hence there is no significant difference in mean diastolic and systolic length between hypertension.

**Table 16: Difference in mean diastolic and systolic between Treadmill tests**

| | Treadmill tests | | f value | p value |
|---|---|---|---|---|
| | Yes (n=78) | No (n=64) | | |
| | Mean±SD | | | |
| Diastolic st length | 25.74±7.85 | 24.92±6.67 | 0.441 | 0.508 |
| MLD% | 14.83±6.43 | 15.67±6.34 | 0.604 | 0.438 |
| Systolic st length | 24.30±7.49 | 23.62±7.07 | 0.302 | 0.583 |
| MLD% | 34.18±10.32 | 35.00±8.85 | 0.258 | 0.612 |

Table 16 reveals difference in mean diastolic and systolic length between treadmill test. Since p>0.05, hence there is no significant difference in mean diastolic and systolic length between treadmill test.

**Table 17: Difference in mean diastolic and systolic between angina**

| | Angina | | f value | p value |
|---|---|---|---|---|
| | Yes (n=41) | No (n=101) | | |
| | Mean±SD | | | |
| Diastolic st length | 25.54±6.52 | 25.30±7.66 | 0.032 | 0.857 |
| MLD% | 15.78±5.39 | 14.98±6.72 | 0.456 | 0.501 |
| Systolic st length | 24.60±6.93 | 23.74±7.45 | 0.404 | 0.529 |
| MLD% | 37.41±9.95 | 33.39±9.48 | 5.342 | **0.022*** |

*p<0.05

Table 17 reveals difference in mean diastolic and systolic length between angina. Since p-value for systolic length of MLD% (0.022<0.05), hence there is a significant difference in mean diastolic and systolic between angina. In systolic length of MLD% has high mean (37.41±9.95) while compared to systolic length of MLD% has low mean (33.39±9.48).

**Table 18: Association between Diabetes Mellitus and sex**

| | Diabetes Mellitus | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| Male | 48 (64.0) | 56 (83.6) | 104 (73.2) | **0.009**** |
| Female | 27 (36..0) | 11 (16.4) | 38 (26.8) | |
| **Total** | **75 (100.0)** | **67 (100.0)** | **142 (100.0)** | |

Chi-Square: 6.923, **p<0.01

Table 18 reveal the association between diabetes mellitus and sex. It is observed that 73.2% of the diabetes patients are belongs to male. Further 64% of the male patients are have diabetes while 83.6% of the patients do not have diabetes. From the observed chi-square value of 6.923 and p value of 0.009 which is less than 0.01, hence there is an association between diabetes mellitus and sex.

**Table 19: Association between hypertension and sex**

| | Hypertension | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| Male | 43 (62.3) | 61 (83.6) | 104 (73.2) | **0.004**** |
| Female | 26 (37.7) | 12 (16.4) | 38 (26.8) | |
| **Total** | **69 (100.0)** | **73 (100.0)** | **142 (100.0)** | |

Chi-Square: 8.167, **p<0.01

Table 19 reveal the association between hypertension and sex. It is observed that 73.2% of the hypertension patients are belongs to male. Further 62.3% of the male patients are have hypertension while 83.6% of the patients do not have hypertension. From the observed chi-square value of 8.167 and p value of 0.004 which is less than 0.01, hence there is an association between hypertension and sex.

**Table 20: Association between treadmill test and sex**

| Sex | Treadmill test | | Total | p value |
|---|---|---|---|---|
| | Nil | Positive | | |
| | n(%) | | | |
| Male | 55 (70.5) | 49 (76.6) | 104 (73.2) | 0.418 (N.S) |
| Female | 23 (29.5) | 15 (23.4) | 38 (26.8) | |
| **Total** | **78 (100.0)** | **64 (100.0)** | **142 (100.0)** | |

Chi-Square: 0.656, N.S: Not Significant

Table 20 reveal the association between treadmill test and sex. It is observed that 73.2% of the patients are belongs to male. Further 70.5% of the male patients have not done treadmill test while 76.6% of the male patients have positive treadmill test. From the observed chi-square value of 0.656 and p value of 0.418 which is greater than 0.05, hence there is no association between treadmill test and sex.

**Table 21: Association between angina and sex**

| | angina | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| Male | 70 (69.3) | 34 (82.9) | 104 (73.2) | 0.097 (N.S) |
| Female | 31 (30.7) | 7 (17.1) | 38 (26.8) | |
| **Total** | **101 (100.0)** | **41 (100.0)** | **142 (100.0)** | |

Chi-Square: 2.760, N.S: Not Significant

Table 21 reveal the association between angina and sex. It is observed that 73.2% of the angina patients are belongs to male. Further 69.3% of the male patients have angina while 82.9% of the male patients have not angina. From the observed chi-square value of 2.760 and p value of 0.097 which is greater than 0.05, hence there is no association between angina and sex.

**Table 22: Association between artery and sex**

| | Artery | | | | Total | p value |
|---|---|---|---|---|---|---|
| | AN.Left Circumflex | Distal. Left anterior descending artery | Distal. Right Coronary Artery | Mid-portion Left anterior descending | | |
| | n(%) | | | | | |
| Male | 1 (100.0) | 16 (64.0) | 0 (0.0) | 87 (75.7) | 104 (73.2) | 0.209(N.S) |
| Female | 0 (0.0) | 9 (36.0) | 1 (100.0) | 28 (24.3) | 38 (26.8) | |
| **Total** | **1 (100.0)** | **25 (100.0)** | **1 (100.0)** | **115 (100.0)** | **142 (100.0)** | |

Chi-Square: 4.533, N.S: Not Significant

Table 22 reveal the association between artery and sex. It is observed that 73.2% of the artery patients are belongs to male. Further 64% of the male patients have Distal. Left anterior descending artery while 75.7% of the male patients have Mid-portion Left anterior descending. From the observed chi-square value of 4.533 and p value of 0.209 which is greater than 0.05, hence there is no association between artery and sex.

**Table 23: Association between diabetes mellitus and age group**

| Age group | Diabetes Mellitus | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| <=45 years | 9 (12.0) | 23 (34.3) | 32 (22.5) | **0.004**** |
| 46-60 years | 44 (58.7) | 33 (49.3) | 77 (54.2) | |
| >=60 years | 22 (29.3) | 11 (16.4) | 33 (23.2) | |
| **Total** | **75 (100.0)** | **67 (100.0)** | **142 (100.0)** | |

Chi Square: 10.947, **p<0.01

Table reveals 23 the association between diabetes millitus and age group. It is observed that 54.2% of the diabetes patients are belongs to 46-60 years age group. Further 58.7% of the 46-60 years age group patients have diabetes while 49.3% of the 46-60 years age group patients don't have diabetes. From the observed chi-square value of 10.947 and p value of 0.004 which is less than 0.01, hence there is an association between diabetes millitus and age group.

**Table 24: Association between hypertension and age group**

| Age group | Hypertension | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| <=45 years | 9 (13.0) | 23 (31.5) | 32 (22.5) | **0.029*** |
| 46-60 years | 41 (59.4) | 36 (49.3) | 77 (54.2) | |
| >=60 years | 19 (27.5) | 14 (19.2) | 33 (23.2) | |
| **Total** | **69 (100.0)** | **73 (100.0)** | **142 (100.0)** | |

Chi Square: 7.100, *p<0.05

Table 24 reveals the association between hypertension and age group. It is observed that 54.2% of the hypertension patients are belongs to 46-60 years age group. Further 59.4% of the 64-60 years age group patients have hypertension while 49.3% of the 46-60 years patients don't have hypertension. From the observed chi-square value of 7.100 and p value of 0.029 which is less than 0.05, hence there is an association between hypertension and age group.

**Table 25: Association between treadmill test and age group**

| Age group | Treadmill test | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| <=45 years | 21 (26.9) | 11 (17.2) | 32 (22.5) | 0.346 |
| 46-60 years | 41 (52.6) | 36 (56.2) | 77 (54.2) | |
| >=60 years | 16 (20.5) | 17 (26.6) | 33 (23.2) | |
| **Total** | **78 (100.0)** | **64 (100.0)** | **142 (100.0)** | |

Chi Square: 2.120

Table 25 reveals the association between treadmill test and age group. It is observed that 54.2% of the patients are belongs to 46-60 years age group. Further 52.6% of the 46-60 years age group patients have done treadmill test while 56.2% of the 46-60 years age group patients not done treadmill test. From the observed chi-square value of 2.120 and p value of 0.346 which is greater than 0.05, hence there is no association between treadmill test and age group.

**Table 26: Association between angina and age group**

| Age group | Angina | | Total | p value |
|---|---|---|---|---|
| | Yes | No | | |
| | n(%) | | | |
| <=45 years | 10 (24.4) | 22 (21.8) | 32 (22.5) | 0.936 |
| 46-60 years | 22 (53.7) | 55 (54.5) | 77 (54.2) | |
| >=60 years | 9 (22.0) | 24 (23.8) | 33 (23.2) | |
| **Total** | **41 (100.0)** | **101 (100.0)** | **142 (100.0)** | |

Chi Square: 0.133

Table 26 reveals the association between angina and age group. It is observed that 54.2% of the angina patients are belongs to 46-60 years age group. Further 53.7% of the 46-60 years age group patients have angina while 54.5% of the 46-60 years age group patients done have angina. From the observed chi-square value of 0.133 and p value of 0.936 which is greater than 0.05, hence there is no association between angina and age group.

**Table 27: Association between angina and age group**

| Age group | Artery | | | | Total | p value |
|---|---|---|---|---|---|---|
| | AN. Left Circumflex | Distal. Left anterior descending artery | Distal. Right Coronary Artery | Mid-portion Left anterior descending | | |
| | n(%) | | | | | |
| <=45 years | 0 (0.0) | 6 (24.0) | 0 (0.0) | 26 (22.6) | 32 (22.5) | 0.941 |
| 46-60 years | 1 (100.0) | 13 (52.0) | 1 (100.0) | 62 (53.9) | 77 (54.2) | |
| >=60 years | 0 (0.0) | 6 (24.0) | 0 (0.0) | 27 (23.5) | 33 (23.2) | |
| **Total** | **1 (100.0)** | **25 (100.0)** | **1 (100.0)** | **115 (100.0)** | **142 (100.0)** | |

Chi Square: 1.746

Table 27 reveals the association between artery and age group. It is observed that 54.2% of the artery patients are belongs to 46-60 years age group. Further 52% of the 46-60 years age group patients have Distal. Left anterior descending artery while 53.9% of the 46-60 years age group patients have Mid-portion Left anterior descending. From the observed chi-square value of 1.746 and p value of 0.941 which is greater than 0.05, hence there is no association between artery and age group.

**Table 28:Descriptive statistics for degree of flow reduction**

| | Mean | SD | Max | Min |
|---|---|---|---|---|
| Degree of flow reduction (Systolic-Diastolic) | 19.338 | 9.971 | 45.00 | 1.000 |

Table 28 shows the descriptive statistics of degree of flow reduction. Mean of degree of flow reduction is 19.338, standard deviation 9.971 with maximum45.00 and minimum 1.000 respectively.

**Table 29: Frequency of degree of flow reduction**

| | Frequency(n) | Percentage (%) |
|---|---|---|
| Mild | 80 | 56.3 |
| Moderate | 36 | 25.4 |
| Severe | 26 | 18.3 |
| **Total** | **142** | **100.0** |

Table 29 shows the frequency of degree of flow reduction. Majority 56.3% of the patients are mild followed by, 25.4% of the patients are moderate and 18.3% of the patients are severe respectively.

**Table 30: Association between Sex and Degree of flow reduction**

| Sex | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| Male | 55 (68.8) | 28 (77.8) | 21 (80.8) | 104 (73.2) | 0.377 (N.S) |
| Female | 25 (31.2) | 8 (22.2) | 5 (19.2) | 38 (26.8) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:1.953

Table 30 reveal the association between sex and degree of flow reduction. It is observed that 73.2% of the patients are belongs to male. Further 68.8% of the male patients have mild, 77.8% of the male patients have moderate and 80.8% of the male patients have severe. From the observed chi-square value of 1.953 and p value of 0.377 which is greater than 0.05, hence there is no association between sex and degree of flow reduction.

**Table 31: Association between diabetes mellitus and Degree of flow reduction**

| Diabetes Mellitus | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| Yes | 41 (51.2) | 19 (52.8) | 15 (57.7) | 75 (52.8) | 0.849 (N.S) |
| No | 39 (48.8) | 17 (47.2) | 11 (42.3) | 67 (47.2) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:0.327

Table 31 reveal the association between diabetes mellitus and degree of flow reduction. It is observed that 52.8% of the patients are belongs to diabetes mellitus. Further 51.2% of the diabetes patients have mild, 52.8% of the patients have moderate and 57.7% of the patients have severe. From the observed chi-square value of 0.327 and p value of 0.849 which is greater than 0.05, hence there is no association between diabetes mellitus and degree of flow reduction.

**Table 32: Association between hypertension and Degree of flow reduction**

| Hypertension | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| Yes | 42 (52.5) | 19 (52.8) | 8 (30.8) | 69 (48.6) | 0.132 (N.S) |
| No | 38 (47.5) | 17 (47.2) | 18 (69.2) | 73 (51.4) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:4.048

Table 32 reveal the association between hypertension and degree of flow reduction. It is observed that 51.4% of the patients are not belongs to hypertension. Further 52.5% of the hypertension patients have mild, 52.8% of the patients have moderate and 69.2% of the patients have not severe. From the observed chi-square value of 4.048 and p value of 0.132 which is greater than 0.05, hence there is no association between hypertension and degree of flow reduction.

**Table 33: Association between treadmill test and Degree of flow reduction**

| Treadmill test | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| Yes | 44 (55.0) | 18 (50.0) | 16 (61.5) | 78 (54.9) | 0.666 (N.S) |
| No | 36 (45.0) | 18 (50.0) | 10 (38.5) | 64 (45.1) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:0.812

Table 33 reveal the association between treadmill test and degree of flow reduction. It is observed that 54.9% of the patients are not belongs to treadmill test. Further 55% of the patients have mild, 50% of the patients have and have not moderate and 61.5% of the patients have severe. From the observed chi-square value of 0.812 and p value of 0.666 which is greater than 0.05, hence there is no association between treadmill test and degree of flow reduction.

**Table 34: Association between angina and Degree of flow reduction**

| Angina | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| Yes | 18 (22.5) | 12 (33.3) | 11 (42.3) | 41 (28.9) | 0.121 (N.S) |
| No | 62 (77.5) | 24 (66.7) | 15 (57.7) | 101 (71.1) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:4.216

Table 34 reveal the association between angina and degree of flow reduction. It is observed that 71.1% of the patients are not belongs to angina. Further 77.5% of the patients have not mild, 66.7% of the patients have not moderate and 57.7% of the patients have not severe. From the observed chi-square value of 4.216 and p value of 0.121 which is greater than 0.05, hence there is no association between angina and degree of flow reduction.

**Table 35: Association between artery and Degree of flow reduction**

| Artery | Degree of flow reduction | | | Total | p-value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| AN. Left Circumflex | 0 (0.0) | 1 (2.8) | 0 (0.0) | 1 (0.7) | 0.395 (N.S) |
| Distal. Left anterior descending artery | 14 (17.5) | 7 (19.4) | 4 (15.4) | 25 (17.6) | |
| Distal. Right Coronary Artery | 0 (0.0) | 1 (2.8) | 0 (0.0) | 1 (0.7) | |
| Mid-portion Left anterior descending | 66 (82.5) | 27 (75.0) | 22 (84.6) | 115 (81.0) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:6.256

Table 35 reveal the association between artery and degree of flow reduction. It is observed that 81% of the artery patients are belongs to Mid-portion Left anterior descending. Further 82.5% of the patients have mild, 75% of the patients have moderate and 84.6% of the patients have severe. From the observed chi-square value of 6.256 and p value of 0.395 which is greater than 0.05, hence there is no association between artery and degree of flow reduction.

**Table 36: Association between age group and degree of flow reduction**

| Age group | Degree of flow reduction | | | Total | p value |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | | |
| | n(%) | | | | |
| <=45 years | 19 (23.8) | 4 (11.1) | 9 (34.6) | 32 (22.5) | **0.023*** |
| 46-60 years | 37 (46.2) | 27 (75.0) | 13 (50.0) | 77 (54.2) | |
| >=60 years | 24 (30.0) | 5 (13.9) | 4 (15.4) | 33 (23.2) | |
| **Total** | **80 (100.0)** | **36 (100.0)** | **26 (100.0)** | **142 (100.0)** | |

Chi-Square:11.328, *p<0.01

Table 36 reveal the association between age group and degree of flow reduction. It is observed that 54.2% of the patients are belongs to 46-60 age group. Further 46.2% of 46-60 years age group patients have mild followed by, 75% of 46-60 years age group patients have moderate and 50% of 46-60 years age group patients have severe. From the observed chi-square value of 11.328 and p value of 0.023 which is less than 0.05, hence there is an association between age group and degree of flow reduction.

# CHAPTER V: DISCUSSION AND CONSLUSION

The present chapter discusses the findings of the present research wherein the following are the objectives to recap-

To study the relationship between the systolic length and the diastolic length

To study the relationship between the systolic MLD% and diastolic MLD%

To study the difference in mean diastolic and systolic between male and female

To study the difference in mean diastolic and systolic between angina

To study the association between diabetes mellitus and gender

To study the association between hypertension and gender

To study the association between diabetes mellitus and age group

In this regard, the results of the present research are discussed as follows:

A total of 9464 angiograms patients in 5 years are considered in which 142 patients are affected by the myocardial bridging, which comes around 1.5%. In these 142 patients, the number of male and female patients is found to be 104 and 38 respectively and the percentage distribution is found to be 73.2% and 26.8%. The increased number of male patients with myocardial bridging is evident in another research done by Sujatha et al. (2015). In this study, 64 myocardial bridge positive cases were considered. Among them about 62.5% had been male and about 37.5% had been female patients.

Of 142 patients having myocardial bridging, majority of the patients are under less than or equal to 45 age, which constitutes about 22.2%. This is in line with the study done by Kantarci et al. (2006)

From the results of the study, it is evident from table 11 that there is a positive linear relationship between the diastolic length and systolic length. This is shown by the value of degree of correlation, which is 0.898. This is proved by the study by Gavish et al. (2008). In this study, symmetrical regression was applied to get a more valid estimate of the systolic-on-

diastolic slope. In this study, about 140 patients were considered with mean age of 56 years. Of these patients, 45% of them were men.

Relationship between the systolic MLD% and diastolic MLD can be elicited from the table 12. From this table, it is clear that there is a positive linear relationship between the systolic MLD% and diastolic MLD%. This relationship is shown by the value of degree of correlation (r) which is 0.268. From table 13, the p values of 0.030 and 0.028 for minimum lumen diameter percentage and minimum lumen area percentage respectively show that there is a significant difference in mean between the male and the female for the diastolic length. For the systolic length, the p values are 0.006 and 0.003 for MLD and MLA respectively.

From table 17, the p values for systolic length are 0.022 and 0.024 for MLD and MLA respectively. This shows a significant difference in mean diastolic and systolic between angina. According to a study done by Ferro et al. (1984), the five patients with spontaneous angina were studied during the procedure of cardiac catheterization. The electrocardiographic lead (V) and aortic pressure were registered at rest and at the onset of angina pain 5 and 10 minutes after 0.6 mg of nitroglycerin. Systemic arterial pressure, heart rate and systolic and diastolic time intervals were all recorded. After the onset of angina, the heart rate and systemic arterial pressure considerably increased. These decreased after the administration of nitroglycerin after 10 minutes. During the angina, the preejection period did not change whereas the left ventricular ejection time and electromechanical systole increased. Because of this, diastolic time which can be expressed as percentage of cardiac cycle reduced. After the administration of NTG, all values dropped back to base values and thereby resulting in the decrease in the anginal pain.

From the table 18, it is evident that, of 104 males, the percentage of male affected by diabetes is 64%. Of the remaining 38 females, the percentage of the female affected by the diabetes is 36%. The p value of 0.009 shows that there is an association between diabetes mellitus and gender. Hence it is revealed that males are affected more by diabetes mellitus than females.

The incidence of diabetes in males is two and a half times more than female. But when the females are diagnosed to have the diabetes, it seems to be very aggressive, which can predispose to cardiovascular complexities (Lutgers et al., 2009; Hilding et al., 2007). The incidence of diabetes is due to the prevalence of obesity in males. Thus, this may be one

of the causes of the males to have higher incidence of diabetes with men being diagnosed with diabetes at lower Body Mass Indices (BMI) than women (Logue et al., 2011). Hence, this explains why the middle-aged males have type 2 diabetes more commonly. Males may be less sensitive to insulin than women for any given body mass index. The males tend to accumulate more visceral fat readily than females, which in turn lead to increased abdominal girth whereas the females accumulate the fat subcutaneously around hips and thighs. According to the recent researches, the visceral fat is diabetogenic and metabolically active (Miyazaki & DeFronzo, 2009). The issue of engagement of healthcare professionals is the biggest problems in both diagnosing diabetes in at-risk groups and maintaining good diabetes control. The men care about their health and because of occupational commitments, they find it difficult to attend the traditional clinical events (White et al., 2008).

From the table 19, of 142 people, 83.6% of males are not having the hypertension, but 37.7% of the females are having the hypertension. The p value of 0.004 shows that there is an association between hypertension and sex. From table 23, of 142 people, both males and females, the high incidence of diabetes occurs in the age group of 57 to 62 years, which constitutes 28%. The next high incidence of diabetes is in the age above 63 years, which amounts to 22.7%.

## 5.1 Conclusion

Myocardial bridging which is found to be in 25% of patients based on autopsy and CT but is detectable only in 10% of the patients by angiographically detectable systolic compression. A congenital anomaly where the epicardial artery takes an intramyocardial course is called myocardial bridging. The result is the accelerated atherosclerosis caused by the flow alteration in the coronary segment which is in close proximity to the bridged segment. The smooth muscles in the area and the morphological changes in endothelial cells and the favorable shear forces resulting in the increased vasoactive agents spares the bridged portion from atherosclerosis. The decrease in coronary flow reserve and systolic coronary flow reversal close to the bridge is the result of hemodynamic effects of bridging. The consequence of this bridging results in angina to acute coronary syndrome to sudden cardiac death. The treatment involves beta blockers with medical treatment and non-dihydropyridine calcium channel blockers. Reflex sympathetic activation results in hypercontractility and nitrates are contra indicated because of secondary tachycardia. Surgical myotomy ,stenting and coronary artery bypass surgery is suugested for refractive symptoms. For the patients

with myocardial bridging a prospective randomized trial is required to identify the best treatment strategy.

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## Appendix

**Pro-Forma**

Name: ……..

Age: ……years

Sex: ❑ Male ❑ Female

Socioeconomic status: …….

IP number: …….

DOA: …. /…. / …… DOD: …. /…. / ……

Complaints & present history:

Complaints with which patient approaching MGMC OPD…….

Have to check whether anything to do with inclusion and exclusion criteria like:

| | Yes | No |
|---|---|---|
| H/o nausea / vomiting | ❑ | ❑ |
| H/o anorexia | ❑ | ❑ |
| H/o dry cough | ❑ | ❑ |
| H/o chest pain | ❑ | ❑ |
| H/o regurgitation | ❑ | ❑ |
| H/o hemetemesis/malena/ hematochezia | ❑ | ❑ |
| H/o bowel/ bladder disturbances | ❑ | ❑ |

| | | |
|---|---|---|
| H/o dyspnea/orthopnea/pnd | ❑ | ❑ |
| H/o sweating/vomiting/syncope | ❑ | ❑ |
| Higher function abnormality | ❑ | ❑ |
| H/o motor and sensory defecit | ❑ | ❑ |
| H/o renal impairment symptoms: (puffiness of face/pedal edema/ Decreased urine output) | ❑ | ❑ |

**Past History:**

| | **Yes** | **No** |
|---|---|---|
| H/o diabetes/hypertension/bronchial asthma/copd/tuberculosis | ❑ | ❑ |
| H/o thyroid symptoms/abnormalities | ❑ | ❑ |
| H/o angina/ transient ischemic attacks/ myocardial infarction | ❑ | ❑ |
| H/o peripheral vascular disease | ❑ | ❑ |
| H/o previous stroke | ❑ | ❑ |
| H/o renal function abnormalities | ❑ | ❑ |
| H/o prolonged drug intake | ❑ | ❑ |

| | | |
|---|---|---|
| H/o previous treatment | ❑ | ❑ |
| H/o ocp/hrt | ❑ | ❑ |

**Menstural History:**

| | **Yes** | **No** |
|---|---|---|
| Irregularities in menstrual cycles | ❑ | ❑ |

**PERSONAL HISTORY:**

**Life Style:** ❑ 1. Sedentary ❑ 2. Moderately Active ❑ 3.Very Active

**Diet:** ❑ 1. Vegetarian ❑ 2. Non Vegetarian

**H/O Smoking:** ❑ 1. Number of bedi ❑ 2. Cigarettes age at which started smoking

**H/O Alcohol Consumption**

| | Yes | No |
|---|---|---|
| Sleep disturbances | ❑ | ❑ |
| Bowel/bladder disturbances | ❑ | ❑ |

**Family History:**

| | **Yes** | **No** |
|---|---|---|
| H/o diabetes/ hypertension/tension/ hypercholesterolemia/ hypertriglyceridemia / obesity | ❑ | ❑ |

H/o myocardial infarction/stroke/ckd ❑ ❑

**General Examination**

Build : ❑ Thin ❑ Normal ❑ Obese

Height: ……cm

Weight: ……kg

Waist circumference:

❑Male……..

❑ Female ……

Vitals: …….

PR: ……

BP: ……. RR: ……

Anaemia/cyanosis/clubbing/icterus/

Lymphadenopathy

Thyromegaly : ❑ Yes ❑ No

Breasts : ❑ Normal ❑ Abnorma

Skin : ❑ xanthomas ❑ acanthosis nigricans

Tendon xanthomas: ❑ Yes ❑ No

CVS: ……

RS: ……

ABDOMEN: ……

CNS: ……

**Investigations**

Complete blood count …….

Blood urea …….

Serum creatinine …….

Liver function tests ……..

Fasting lipid profile …….

Urine routine …….

Fundus examination ……..

Chest x-ray ……

Ecg ……

Diagnosis: ……..

Thank you for your response



End of the Sample Work