

**“Atrio-Ventricular Plane Displacement: As a predictor of in
hospital outcome of Major Adverse Cardiac Events after
Acute ST-Elevation Myocardial Infarction”**

By

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Dissertation submitted to BLDE (Deemed to be University), Vijayapura



In partial fulfillment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

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Under the guidance of

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DR. SAINATH M. KUDUMBALE

LIST OF ABBREVIATIONS USED

AVPD	:	ATRIO VENTRICULAR PLANE DISPLACEMENT
HF	:	HEART FAILURE
PE	:	PULMONARY EDEMA
CS	:	CARDIOGENIC SHOCK
DD	:	DIASTOLIC DYSFUNCTION
RWMA	:	REGIONAL WALL MOTION ABNORMALITY
STEMI	:	ST ELEVATION MYOCARDIAL INFARCTION
NSTEMI	:	NON-ST ELEVATION MYOCARDIAL INFARCTION
ACS	:	ACUTE CORONARY SYNDROME
VPC	:	VENTRICULAR PREMATURE COMPLEXES
VT	:	VENTRICULAR TACHYCARDIA
LA	:	LEFT ATRIUM
LV	:	LEFT VENTRICLE
MACE	:	MAJOR ADVERSE CARDIAC EVENTS
UA	:	UNSTABLE ANGINA
MI	:	MYOCARDIAL INFARCTION

ABSTRACT

AIM:

To study Atrioventricular plane displacement as predictor of in hospital outcome of Major Adverse Cardiac Events in patients with acute ST-elevation myocardial infarction.

MATERIAL and METHODS:

Cross sectional study carried out in patients admitted in our hospital ICCU with a diagnosis of Acute Coronary Syndrome. Clinical, echocardiographic and laboratory profile and in-hospital outcome of patients with ACS were assessed as a part of work up. Atrioventricular plane displacement (AVPD) was calculated with 24 - 48 hours of admission. Patients with AVPD \geq 10 mm were compared with those AVPD \geq 10 mm. The patients during hospital stay and were observed for development of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death.

RESULTS:

Atrio Ventricular Plane Displacement <10mm was observed in 29 patients (28.43%) Atrio Ventricular Plane Displacement <10mm was associated with old age, alcohol consumption, tobacco chewing, hypertension and higher incidence of major adverse cardiac events like heart failure (44.83%, p<0.01), pulmonary edema(41.38%, p<0.01), cardiogenic shock(27.58%, p<0.01), death (31%, p<0.01), a higher incidence of reduced ejection fraction when compared with patients having Atrio Ventricular Plane Displacement \geq 10mm. The most common major adverse cardiac event were cardiogenic shock, pulmonary edema, heart failure followed by death.

CONCLUSION:

Decreased Atrioventricular plane displacement AVPD < 10 mm was found to be an important predictor of in-hospital major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death.

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INTRODUCTION

Acute coronary syndrome (ACS) has highest rate of patients getting cardiovascular morbidity and deaths in the modern world, the weight of the disease is difficult for young patients with middle or low socio-economic status, than in patients with high socio-economic status. This difference is associated with various risk factors, but also with drugs and related treatments(1).

The patients who belong to low and middle incomes, they die before arrival at the hospital, most patients not receiving medication for secondary prevention and do not change way of life after as compared with patients with high income group(2).

Acute coronary syndrome (ACS) is comprised of three things such as Unstable Angina (UA), myocardial infarction without increase of the ST segment which is NSTEMI and myocardial infarction with ST segment elevation i.e. STEMI(3).

All of these conditions above have a common pathology that is sudden coronary insufficiency due to the shift plaques with the additional formation of a blood clot. Where there is a complete blockage in coronary artery compared within the cases of a partial blockage(4).

Acute coronary syndrome which has three different types which are Unstable angina, STEMI and NSTEMI are one of the main causes of death in the Asia-Pacific region and is the half of the world's population. In these patients, cardiovascular outcomes whether be poor or good prognosis vary by age, sex, nationality and the education they have received(5).

Predisposing factors such as age, sex, diabetes mellitus, and the former history of acute myocardial infarction are seen commonly in patients with acute myocardial infarction (AMI) are. The risk of complications such as heart failure, cardiogenic shock, pulmonary edema, arrhythmias are considered as major adverse effects. And

for the evaluation of the function of the myocardium is essential for the planning of further management and the development of new approaches and algorithms to improve the results. Therefore, we need to identify the patients with acute myocardial infarction, who are at risk of being subjected to serious adverse cardiovascular events during hospitalization, in order to reduce the damage of the heart and its muscles and compromising the left ventricular dysfunction(6).

Echocardiography for the first time introduces into medical practice in the 1970's. This is very accurate in the evaluation of the operation of left ventricle and to diagnose the disease of the valves. Thanks to these features this technique quickly raised in popularity all over the world. It is a method of imaging that is commonly used in the cardiovascular system. It mainly helps in the quick and non-invasive analysis of the structure and function of the heart in various hospital environments.

In the past, many studies have shown the usefulness of echocardiographic dimensions, such as the AV-Plane displacement (AVPD) and the ejection fraction are important so that adverse cardiac events during hospital stay can be evaluated in patients who develop ST-segment myocardial infarction (STEMI)(6).

Atrioventricular plane displacement is measured at the left ventricle plane in systole and diastole where can be carried out in four different parts of the AV-plane, which is apart from chest wall and corresponds to the Septal, anterior, lateral and posterior heart walls. It's simple, fast, and very reproducible. It is especially useful, because the AVPD can be also measured in echocardiograms with a poor image quality(7).

AIMS AND OBJECTIVES

To study Atrioventricular plane displacement as predictor of in hospital outcome of Major Adverse Cardiac Events in patients with acute ST-elevation myocardial infarction.

REVIEW OF LITERATURE

In 1953 Edler and Hertz invented a non-invasive diagnostic device which later was known as echocardiology. Edler initially used this method and technique for non-invasive study of mitral stenosis and regurgitation at mitral valve. This technique was continued by other cardiologists over next few decades from rest of the world and further helped in its development by adding further modalities such as the two-dimensional doppler echocardiography and transesophageal echocardiography. These are now the norms in the research, study and diagnosis of acute myocardial infarction, heart failure, etc. Edler also influenced other neurologists and obstetricians from all over the world to use this technique in their field. For this striking invention, Edler is recognized as the "father of echocardiography"(8).

M-mode was considered as "picture of the heart" in the beginning as it was a chart that showed how the location of the structure of heart during the cardiac cycle changes. When to Edler's first machine son electronic toothbrush was added then the world's first commercially successful 2-dimensional echocardiograph was developed and for the first-time visualization of actual picture of the heart became possible. Then came a time that by adding Doppler imaging to it, the two-dimensional imaging and pulsed Doppler imaging allowed the detection of the flow signal in a number of places inside the heart. At the same time, another important discovery in the Doppler came when change of the equation of Bernoulli was used to detect the pressure gradients for the stenotic valve; it was shown that the hemodynamic information is also determined with the use of Doppler Echo. For the best display of transesophageal echocardiography has been also used by heart surgeons while operating. The echocardiography was added to their Arsenal. It was also used, where it can be connected to the coronary artery during heart surgery to evaluate the physiological

importance of the coronary arteries of patient. This, together with transesophageal echocardiography has been used for, internal monitoring of Heart Valve repair and replacement, review and correction of congenital abnormalities and monitoring for abnormal wall motion. The area of echocardiography is still developing(8).

ATRIOVENTRICULAR PLANE DISPLACEMENT

In 1988 Höglund C et al. first measured Atrioventricular plane displacement using M mode echocardiography method to know left Atrioventricular valve plane displacement. He used a method where, by measuring the displacement of the atrioventricular of the left ventricle in systole and diastole two cursors measured four sites at AV plane which corresponded to the septal, anterior, lateral and posterior myocardial walls. The difference in the values of systole and diastole was measured and a mean of it was taken by him. There was no significant difference in the recordings from the four sites. Fractional shortening, however, could not be demonstrated. This new technique of measuring AV plane displacement helped in the introduction of a new and which was a simple method in assessment of left ventricular displacement(7).

The hearts action of pumping, is the result of contraction of myocardial fibers which is carried out in different directions and at different levels, both longitudinal and spiraling muscles shortening of the ventricle. The longitudinal shortening of the atrioventricular plane can be observed, such as the movement of the base of ventricles upwards in a contraction. The stroke volume percentage explained by AVPD may vary in athletes, normal adults and patients with reduced heart function and may be important for understanding the differences in pump physiology of myocardium between these groups. It can be suggested that stroke volume due to AVPD (SVAVPD), can be obtained from the multiplication of AVPD from the epicardial LV

range is 2-3 cm apical to the base of the heart. However, this method does not take into account the change in the diameter of the ventricles at AV plane (9).

In females who aren't professional athlete's aerobic endurance training when given for a short period of time it helps in increase AVPD during workout and exercise. This increased AVPD indicates an improved contraction of the heart during the period of workouts. Hence AVPD can be considered a good parameter of left ventricular systolic function. And it can also differentiate between healthy untrained heart and trained heart. (10)

MEASUREMENT OF THE ATRIOVENTRICULAR PLANE DISPLACEMENT

The AV plane displacement by two-dimensional echocardiography is easy, non-invasive, inexpensive and has no influence on the patient's hemodynamic stability and become a standard method to evaluate left ventricular function. Use of one or two M-mode cursors in apical 4 and 2 chamber views is taken. In apical 4 chamber view the cursor is placed at AV plane at the ventricular septum. M-mode line shows the length of the displacement towards the apex during systole. Which shows displacements of lateral and septal wall. In apical 2 chamber view the cursor is placed at AV plane at the ventricular septum. M-mode line shows the length of the displacement towards the apex during systole (Figure 1). Which shows displacements of the anterior and posterior walls. This displacement of AV plane is measured. Each AV plane displacement is measured in mm which is taken at these four sites. It implies that distance travelled by the mitral annular plane.(11)

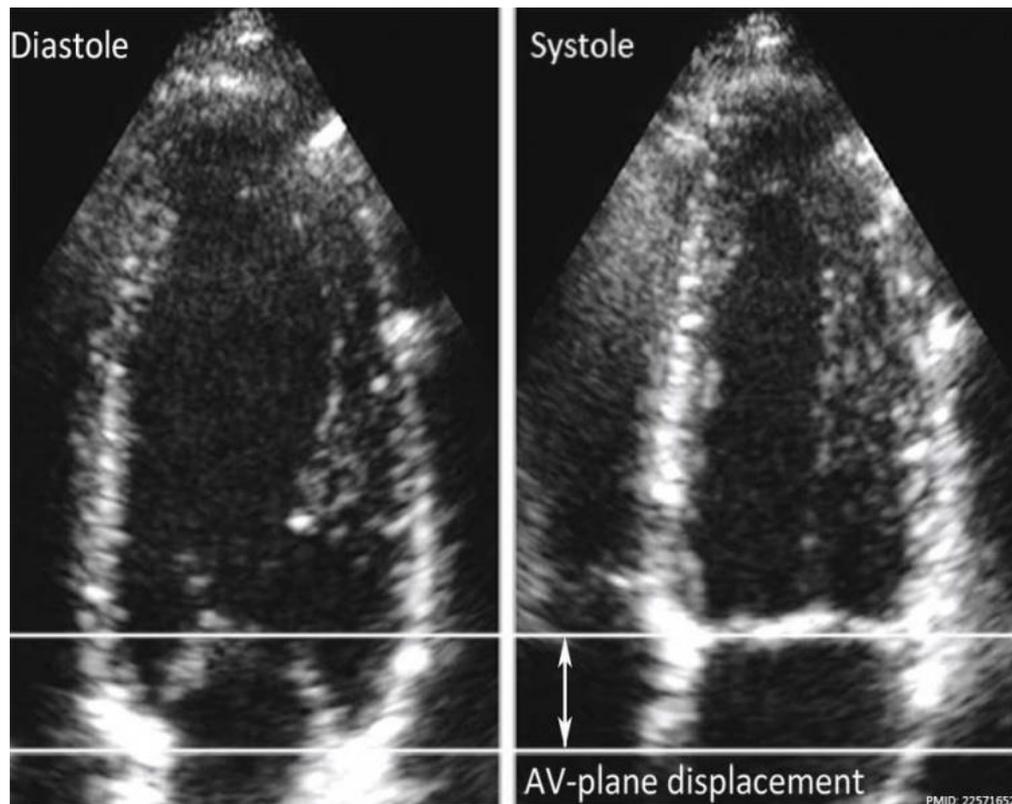


FIGURE 1: ATRIOVENTRICULAR PLANE MEASUREMENT

Normal ventricular size, ventricular volumes, systolic and diastolic functions also varies as per age and sex changes are there, several researches and studies from the past have shown that in all individuals either be young or elderly, hearts oxygen perfusion at the level of myofibrils increased due to endurance training for a long time. But it is difficult to assess the effect of lifetime endurance on the ventricular function when measured in longitudinal and radial components of stroke volume (SV). Heart's ventricular function is important so that it allows the atrium and ventricle to fill. Hence, heart beating is optimally maintained by cardiac output(12).

The overall LV function Evaluation (e. g., the ejection fraction) has a prognostic significance in patients with Acute Coronary Syndrome. There are many a correlating study of significance of ejection fractions and severe chronic congestive heart disease. In patients with myocardial infarction, the average EF as the ejection fraction was significantly reduced compared with patients with myocardial infarction. In addition

to the reduced displacement of the AV plane, especially at the infarct sites, reduced regional displacement of the abnormal wall motion of the affected left ventricular band. With the Exception of the inferior wall was in the correlation ship between a particular Wall of LV and the displacement of the AV that corresponds to the level of good and very important Position. The poor correlation between the wall motion of the pointer to the bottom wall and the displacement of the AV plane at this location is probably due to the inclusion of the apical part in the calculation of the wall motion for the inferior wall. The direction of the ultrasound beam is almost parallel with the movement of the AV plane in a ventricle, which rotate slightly along the axis, both in the healthy myocardium and infarction. In addition, the recording of the displacement of the AV levels provides an absolute value for the assessment of regional myocardial function in acute MI.

That's why measuring AV plane displacement by echocardiography is therefore determined by association in a good relationship with the displacement of the pointer of the walls. An average of 10 mm or more, has been considered as sensitive and specific in relation of normal ejection fraction.(13).

A history of the atrioventricular plane is in dispensable fibrous Region that in the heart valves that separate the heart valves from the heart wall. During the heartbeat, the AV plane moves as a piston pump during contraction and returns to the initial position during the expansion. A reduced AVPD is associated with diseases and aging, while the regular exercise in young male athletes the maintenance of the AVPD will be there even in similar level as in the young master athletes. In the clinical settings for the measurement of AVPD and reduce the long-axis function echocardiography can be used and it seems to be a healthcare prognosis effect for the future clinical adverse cardiac event. The displacement of the systolic plane, annular

mitral valve stenosis can easily be used as a good indicator as the function of the left ventricle. (14).

ACUTE MYOCARDIAL INFARCTION

The term acute coronary syndrome (ACS) is used to describe three heart diseases associated with myocardial ischemia.:

1. Unstable angina pectoris (UA)
2. St-Segment Elevation Myocardial Infarction (STEMI)
3. Myocardial Infarction Without ST Segment Elevation (NSTEMI)

Myocardial infarction can be defined based on clinical, electrocardiographic (ECG), biochemical and pathological characteristics.

“Myocardial infarction is an indication of cardiac myocyte death due to long-term myocardial ischemia.”

The myocardial infarction can be assessed with the symptoms of myocardial infarction and ecg changes as follows:

1. "Patients with ST-segment elevation, i.e. new ST-segment elevation at the J point with the cut-off points ≥ 0.2 mV from V1 to V3 and ≥ 0.1 mV in other leads," or
2. "Patients with no ST-segment elevation, i.e. ST-segment depression or T wave abnormalities."

The widely accepted definition of myocardial infarction is "any Q wave in leads V1 to V3, or Q wave ≥ 0.03 s in leads I, II, aVL, aVF, V4, V5 or V6.”

In Myocardial infarction blood levels of cardiac biomarkers such as troponin t or i, with / without ck-mb gets elevated in patients with chest pain and suspected to have of acute myocardial ischemia.(3).

Ideal st-elevation in patients with acute myocardial infarction “which is measured at point j, will be found in two contiguous leads and will be ≥ 0.25 mv in men under 40 years of age and ≥ 0.2 mv in men over 40 years of age. In women, ≥ 0.15 mv is considered significant.”

In patients with inferior wall myocardial infarction, precordial leads (v3r and v4r) should be recorded with elevated st segment. It is associated with a right ventricular infarction. Similarly, st-segment depression in v1, v2, and v3 leads suggests myocardial ischemia is of septum.

In patients with suspected myocardial ischemia and st segment elevation or new or perceived lbbb, reperfusion therapy should be initiated immediately. Ecg may be normal in the early hours of the presentation. Even after a myocardial infarction is revealed, the ecg may show st-segment elevation and new q-wave formation. If the ecg appears normal, or changes confirm the clinical suspicion of patients with myocardial infarction, the ecg should be repeated and the current of the ecg should be compared with the previous one. In addition to ecg recordings, such as lead v7, v8, v9, it will be helpful to make the diagnosis to individual with posterior wall myocardial infarction(4).

Clinical presentation

The term Myocardial Infarction refers to the death of cardiac myocytes due to the long duration of ischemia. The imbalance between oxygen supply and demand leads to ischemia of the myocardium. Ischemia in bed can be traced on the basis of the patient's history and 12-lead ECG.

Specific symptoms in patients with suspected myocardial ischemia include chest pain, abdominal pain, pain in arm, forearm, wrist, or jaw during exercise or rest.

In patients with acute myocardial infarction, the above symptoms last longer than 20 minutes.

Discomfort with shortness of breath, sweating, nausea, vomiting, and is usually very local, and never a strong character for diagnosis. The patient may come in with unusual symptoms, such as discomfort epigastric region (this symptom often is a digestive) radiating to shoulder, hand, wrist, jaw or back without any chest pain .

Localized pain in the chest does not change with the movement of the muscles. That does not change from deep inhalation. Chest pain does not change with the change of conditions.

Associated symptoms may be nausea and vomiting, shortness of breath and weakness, dizziness or syncope in various combinations. These symptoms usually occur along with chest pain, and sometimes, symptoms may occur without chest pain(3)(4).

The pathogenesis of acute myocardial infarction

Acute coronary syndrome due to a sudden decrease in cardiac output arterial blood flow rate. This is due to the lack of atherosclerosis with blood clot, with or without associated vasoconstriction. The symptoms with which the patient is in the hospital and the results of the patients depend on the location of the coronary occlusion and the severity and duration of ischemia of the myocardium.

In patients with acute myocardial infarction with increased st-segment, thrombosis is usually persistent. The sediment factor for the deadliest coronary arteries, sudden breakage of the sensitive platelets. About three-quarter of all myocardial infarction-related thrombosis will develop due to the platelets, leading to mild to moderate stenosis before and after infarction(3).

Thrombosis is composed of two different processes where it is an extension of the endothelium stripping process, so that large areas of the surface of the sub endothelium binding tissue in contact. A blood clot that adheres to the surface of the plaque, which is formed. This process has become known as erosion of the endothelium. Observations have shown a relationship between loss of endothelial cells and the distance from the macrophages. Macrophages are highly activated and induce the death of endothelial cells by apoptosis and the production of protease, which is isolated endothelial cells from entering the vascular wall.

The second mechanism for the formation of thrombus is a breakage of the plaque (synonyms, breaking, cracking). Here, the capsule of the plaque is broken to expose the lipid core to the blood. In the Central area, it is highly thrombogenic and include tissue factor, collagen fragments, and crystalline surfaces, in order to accelerate coagulation of the blood thrombus. Initially forms in the plaque itself, which is expanded and distorted from the inside, then a clot can spread (15).

Myocardial infarction with total occlusion of the coronary artery begins to occur after 15-30 minutes of ischemia. The thrombus size depends on sub endocardium which occurs gradually(4).

Of myocardial infarction without increase of the st segment due to a sudden imbalance between the oxygen demand of the myocardium thrombosis and plaque.

It is very often because of the low myocardial extracts. It is often the case, because a non-occlusive thrombus arises in a fancy atherosclerotic-plaque and eventually leads to a non-occlusive or completely occlusive thrombosis of the coronary blood vessels that supply the myocardial cell.

Various events that can lead to a ST elevation myocardial infarction:

- Plaque breakage with non-obstructive thrombus or embolism, which leads to constriction of coronary artery.
- Dynamic obstruction to coronary artery
- Inflammatory mechanisms, such as vasculitis
- External factors that lead to heart failure, coronary artery arterial hypotension, hypoxemia and hypovolemia.

The most common cause is a plaque breakage or obstructive atherosclerosis.

Breakage of the plaque occurs, usually the weakest and most thin at the point, i.e., in the shoulder of atherosclerotic capsules.

Broken cells are a large number of inflammatory cells such as T lymphocytes, monocytes, macrophages (16).

Natural course and history of acute myocardial infarction

The true Natural History of acute myocardial infarction is difficult to see, for the following reasons:

1. Two-three quarters of deaths occur outside the hospital. These deaths may be due to a heart attack or a power. It is impossible to distinguish the mechanism in most patients
2. What do you mean? What do you mean? Death certificates are unreliable, many deaths that have been proven to be due to coronary artery disease, especially in the elderly, in fact, is not classified.
3. About 25% of non-fatal heart attacks and even healing unweighted(17).

COMPLICATIONS OF ACUTE CORONARY SYNDROME

Acute coronary syndrome may result in a number of complications.

They are grouped into the following types such as: ischemic, mechanical, conduction, arrhythmic, embolic, and pericarditis (18).

" Mechanical Complications of STEMI

1. Left Ventricular (Lv) Failure and Cardiogenic Shock.
2. Right Ventricular Myocardial Infarction (RVMI)
3. Ventricular Septal Rupture (VSR)
4. Mitral Regurgitation (MR)
5. Cardiac Free Wall Rupture
6. Pseudoaneurysm
7. True Ventricular Aneurysm
8. Dynamic Left Ventricular Outflow tract (Lvot)

Ischemic Complication of STEMI

Reinfarction

Conduction Abnormalities of STEMI

Post infarction conduction abnormalities

Arrhythmic Complications of STEMI

Sudden cardiac death

Embolic complications of STEMI

Stroke."

AVPD WITH MAJOR ADVERSE CARDIAC EVENTS

About 10% to 20% of patients hospitalized with acute coronary syndrome develop heart failure during hospital stay. With ACS patients complicated by heart failure, increases in mortality.

Heart failure and a low ejection fraction is the main cause of circulatory failure and mortality in patients admitted in hospital for acute myocardial infarction. Clinical features of circulatory failure show low volume pulse, decreased blood pressure, cold and soft limbs a third heartbeat (S3 gallop), lung crackles, low body temperature and sweating. However, there is circulatory failure in patients with acute myocardial syndrome. A special approach is necessary for diagnosis and the treatment of these Patients. High degree of severity of left ventricular function, plus severity of acute myocardial injury ischemia / infarction.

A decrease in cardiac performance begins when injuries in the heart muscle in the range of 20 to 25%. Cardiogenic shock usually occurs when the heart muscle undergoes 40% or more damage. The most common clinical effects of lung crackles and S3 and S4 gallop.

Predicting the outcome in individuals becomes difficult. Heart failure syndrome begins with respiratory distress, mild fatigue, sinus tachycardia third heart disease (S3 gallop), lung crackles that can be identified in the lung bases, but with the progress of the disease, it also includes the entire lung. The development of pulmonary edema is not (19).

Myocardial systolic Dysfunction and Diastolic Dysfunction:

The main problem in non-valvular HF is perturbed by left ventricular function (LV) which causes a decrease in cardiac output. This decrease in cardiac force results in activation of the various compensatory neurohormonal mechanisms. This activation of the neurohormonal mechanism increases myocardial contractility and reduces coronary perfusion.

Activation of the sympathetic nervous system helps to maintain normal cardiac performance by increasing heart rate, myocardial contractile function of

peripheral vasoconstriction, and activation of the renin-angiotensin aldosterone (RAS) system. It causes vasoconstriction and increases blood volume with salt and water retention. The concentration of vasopressin sodium peptides such as BNP and NT Pro - BNP also gets increased. This may lead to a progressive dilatation of the heart or a change in cardiac structure, or both.(20)

Diastolic refers twice to clinical HF syndrome with a left ejection fraction, i.e. $\geq 40\%$, without major cardiovascular disease. A decrease in heart strength is evident; this type of tiredness and increase in diastolic pressure is then transmitted from the veins of the lungs to the capillaries of the lungs, resulting in short breathing.

Cardiogenic Shock:

Cardiogenic shock (CS) is a serious form of left ventricular failure, arterial pressure falls below 80 mm hg systolic and reduces heart pressure, despite an increase in left ventricular filling pressure. The reason is that the function of the heart tissue, that is more than 40% of the left ventricle. Cardiogenic shock has a very high mortality rate, more than 70%, which is independent and immediate.

Risk factors for ACS in myocardial infarction and a large area of myocardial infarction, history of acute myocardial infarction and diabetes. Acute myocardial infarction in patients with suspected Cardiogenic Shock, hemodynamic monitoring is required. The main cause of the situation is the ventricle of the lung heart, which lifts up acutely. These complications require rapid diagnosis and treatment.(21)

NT pro BNP and Heart failure and Atrioventricular Plane Displacement

The relation of heart failure and neurohormonal system is already proven. There are three types of natriuretic peptides, (atrial, brain and C-type). Atrial and brain natriuretic peptide are used to diagnose and treat cardiovascular diseases. These natriuretic peptides are synthesized from the prohormone-cardiomyocytes, which are

coded by genes at different level. Prohormone of brain natriuretic peptide is broken as N amino-terminal. The functions of these peptides are vasodilation, suppression of renin- angiotensin-aldosterone system and activation sympathetic nervous system. These hormones are released with increased heart wall stress and in patients with systolic or diastolic dysfunction of ventricles. Hence NT pro BNP can be used as biomarker for heart failure. With heart failure AVPD is decreases and the NT pro BNP increases but not in a fixed proportion as there is no evidence for the proportionate increase of NT pro BNP. Relation between AVPD and plasma concentrations of NT-pro BNP in a population with ACS and heart failure is inversely proportional.(22).

The technique calculation of AV plane displacement can be easily learnt as compared to conventional echocardiographic technique. Atrioventricular plane displacement can be related easily with major adverse cardiac events such as death and heart failure in heart failure patients. It can be confirmed with other investigation which support this. Atrioventricular plane displacement might improve given the fact that patient should take treatment properly and each individual should be compliant to it. This overall helps in assessing atrioventricular plane displacement in day to day use while doing echocardiography and for further studies. (23)

Left ventricular contractility can be corelated with Atrioventricular plane displacement with good or bad prognosis with patients who have developed heart failure after STEMI. Atrioventricular plane displacement is a different parameter as compared to ventricular ejection fraction. Atrioventricular plane displacement and ventricular function can be co related to each other and it gives further evaluation for prognosis and for major adverse cardiac events. (24)

MATERIALS AND METHODS

1. STUDY POPULATION

This study was done in Shri B M Patil Medical College Hospital and Research center, Vijayapura from December 2016 to June 2018, individuals with Acute ST elevation myocardial infarction.

3. INCLUSION CRITERIA:

Patients with Acute ST segment elevation myocardial infarction

3. EXCLUSION CRITERIA

- 1) Significant valvular lesions (mitral stenosis or greater than moderate mitral regurgitation)
- 2) Non- STEMI
- 3) Bundle branch block

4. METHODOLOGY

The study was conducted on patients who are admitted in Shri B M Patil Medical College Hospital and Research center, Vijayapura with prolonged chest discomfort typical of myocardial ischemia, ST segment elevation ≥ 1 mm in at least two contiguous leads will undergo a standardized assessment with clinical history and examination, electrocardiogram at admission, trans thoracic 2D echo cardiography and doppler examination within first 24 - 48 hours of admission, cardiac enzymes – Troponin T, CPK-MB and other necessary laboratory investigations.

INVESTIGATIONS

1. Blood

- Complete blood count
- Blood glucose
- Renal function test

-Serum electrolytes

-Troponin I

-CPK MB

2. Urine Examination

3. Electrocardiogram.

4. Chest X ray

5. 2D Echocardiography and color doppler study.

-AVPD measurements

Echocardiographic evaluation was done by Esaote MyLab 25 Gold Ultrasound Machine_two-dimensional- guided M-mode with A 2.5 MHz phased array probe as the distance covered by the atrioventricular plane delineated at the mitral valve annulus from the position most remote from the apex, to the position closest to the apex. AVPD will be measured from the septal and lateral segments in apical 4 chamber view, and from the anterior and inferior segments in apical 2-chamber view. The mean value of AVPD is calculated for each patient. LVEF is measured from the apical 4- and 2-chamber views. Patients were grouped according to the AVPD into Group A with a mean AVPD < 10 mm, and Group B with a mean AVPD \geq 10 mm.

Patients were followed up during their in-hospital course for the occurrence of major adverse cardiac events (MACE) namely: death, heart failure, complex ventricular arrhythmias (sustained ventricular tachycardia or ventricular fibrillation), early post-infarction angina, or mechanical complications. Heart failure was diagnosed clinically according to the standard criteria. Complex ventricular arrhythmia by monitoring ECG strip or by 12 lead ECG recording. Early post-infarction angina- recurrent typical chest discomfort during hospital admission

following relief of that of the index myocardial infarction. Mechanical complications including: acute mitral regurgitation, rupture of the interventricular septum, LV pseudo-aneurysm formation, and rupture of the LV free wall.

5. TYPE OF STUDY:

Cross sectional study.

6. SAMPLE SIZE:

By using the formula

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Z= z statistic at 5% level of significance (1.96)

d is margin of error

p is anticipated prevalence rate (1-10%)

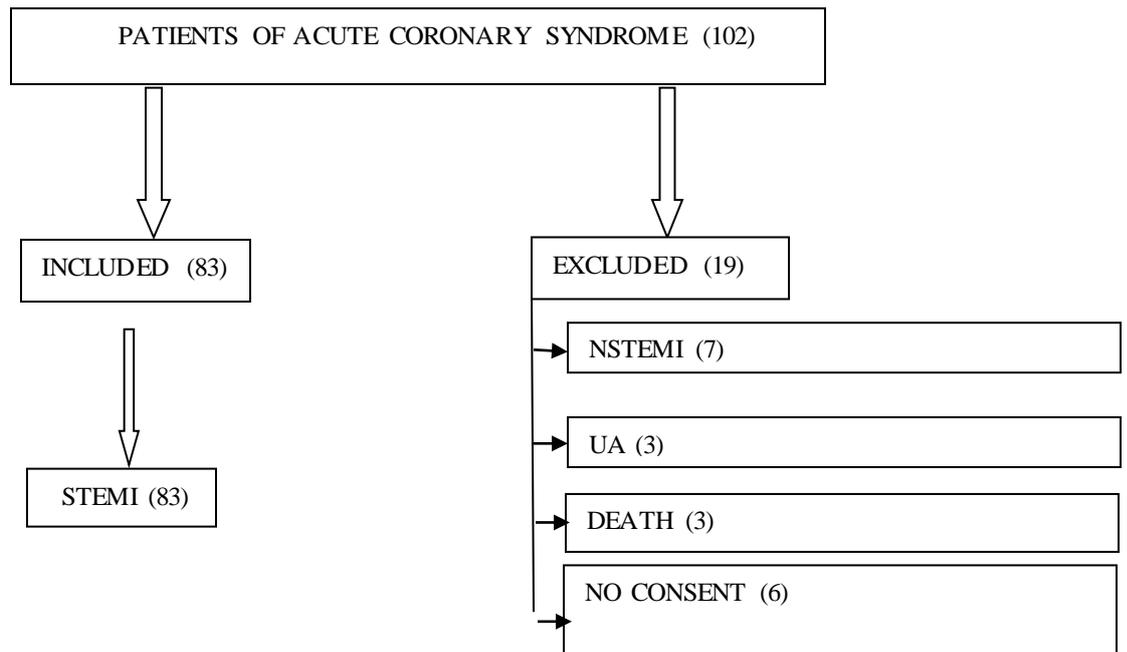
A sample size of 81 subjects were allowed in the study to assess the variation in parameters by AVPD cut-off among AMI patients with 95% confidence level and margin of error of $\pm 10\%$ with finite population correction.

7. STATISTICAL ANALYSIS:

Data was represented using Mean \pm SD, and analyzed by Chi square test for association, comparison of means using t test, ANOVA for comparison and diagrammatic presentation.

RESULTS

The study of Atrio Ventricular Plane Displacement as a predictor of early in-hospital major cardiovascular events in acute coronary syndrome was done in patients admitted to ICCU of Shri B M Patil Medical College and Research Centre from December 2016 to June 2018. Total of 102 patients were admitted with acute coronary syndrome. Total of 83 patients were included in the study, who fulfilled the inclusion criteria and 19 patients were excluded from the study as per exclusion criteria. Out of which Seven patients had NSTEMI, three patients had UA, three patients died before taking study values and six patients did not give consent for study.



AGE DISTRIBUTION

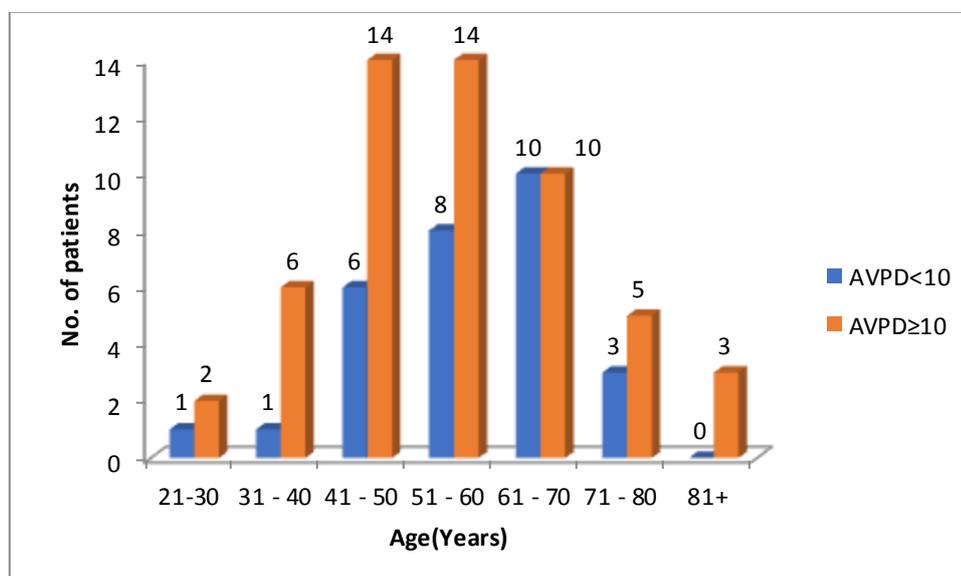
The mean age of study group was 56.81 ± 5 years. Out of 83 patients included in the study, patients aged 21-30 years were 3 (2.9), patients between the age 31-40 years were 8(7.8%), patients between the age 41-50 years were 22 (21.6%), patients between the age 51-60 were 28(27.5%), patients between the age 61-70 years were

28(27.5%), patients between the age 71-80 years were 9(8.8%), patients aged more than 81 years were 4(3.9%) The most common age group were two groups of 51-60 and 61- 70 years (28 patients). Table 1, Figure 2.

TABLE 1: DISTRIBUTION OF CASES ACCORDING TO AGE AND AVPD

Age(Years)	(Group A) AVPD<10		(Group B) AVPD≥10		Chi square test
	N	%	N	%	
21- 30	1	3.4	2	3.7	P=0.521 NS
31 - 40	1	3.4	6	11.1	
41 - 50	6	20.7	14	25.9	
51 - 60	8	27.6	14	25.9	
61 - 70	10	34.5	10	18.5	
71 - 80	3	10.3	5	9.3	
81+	0	0	3	5.6	
Total	29(100)	100.0	54	100	

FIGURE 2: DISTRIBUTION OF CASES ACCORDING TO AGE AND AVPD



SEX DISTRIBUTION

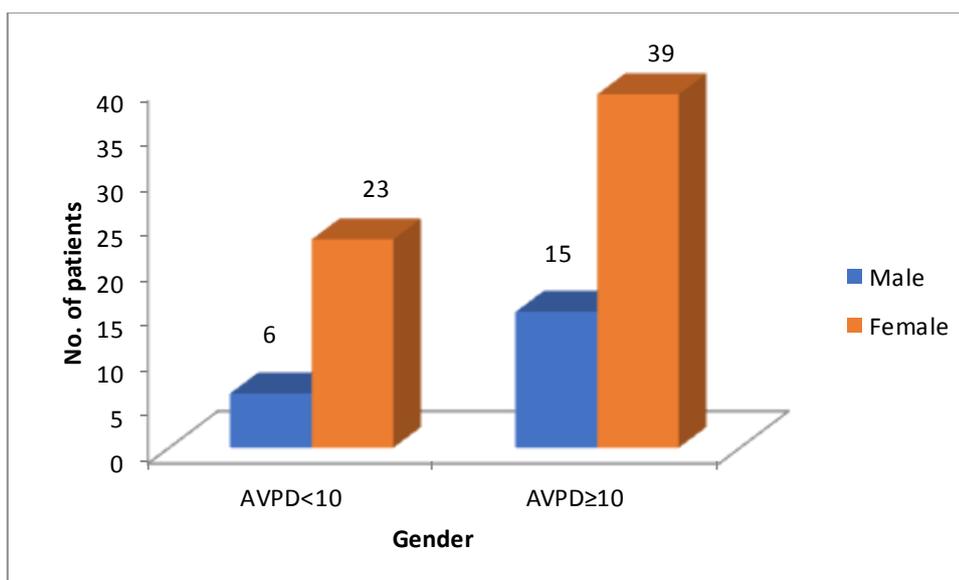
Out of 102 patients in the study, 74 patients (72.5%) were male and 28 patients (27.5%) were female. In this study male patients were more than females.

Table 2, Figure 3.

TABLE 2: DISTRIBUTION OF CASES ACCORDING TO SEX

Gender	(Group A) AVPD<10		(Group B) AVPD≥10		Total		Chi square test
	N	%	N	%	N	%	
Male	6	20.7	15	27.8	21	25.3	P=0.479 NS
Female	23	79.3	39	72.2	62	74.7	
Total	29	100.0	54	100	83	100	

FIGURE 3: DISTRIBUTION OF CASES ACCORDING TO SEX



DISTRIBUTION ACCORDING TO AGE AND SEX:

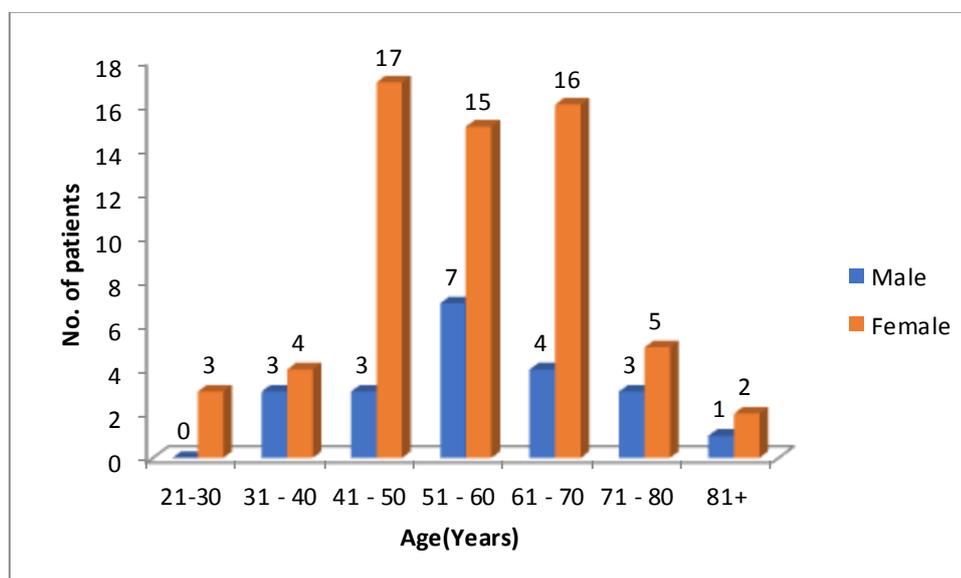
Among male patients the most common age groups were 61-70 years (22 patients). Among female patients common age group was 51-60 years (11 patients)

Table 3, Figure 4.

TABLE 3: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

Age(Years)	Male		Female		Total		Chi square test
	N	%	N	%	N	%	
21-30	0	0	3	4.1	3	3.6	P=0569 NS
31 - 40	3	14.3	4	6.5	7	8.4	
41 - 50	3	14.3	17	5.4	20	24.1	
51 - 60	7	33.3	15	24.2	22	26.5	
61 - 70	4	19.0	16	25.8	20	24.1	
71 - 80	3	14.3	5	8.1	8	9.6	
81+	1	4.8	2	3.2	3	3.6	
Total	21	100.0	62	100	83	100	

FIGURE 4: DISTRIBUTION OF CASES ACCORDING TO AGE and SEX



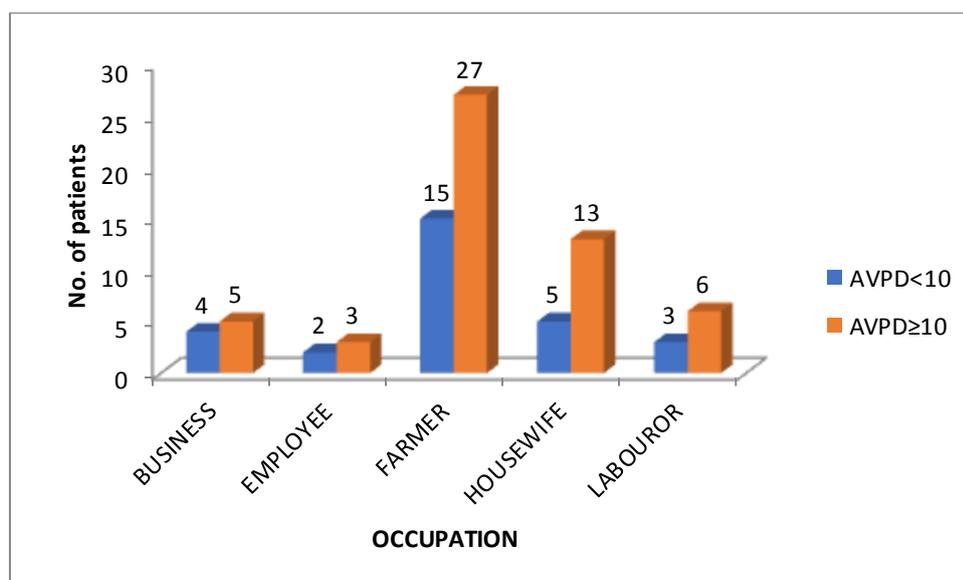
DISTRIBUTION ACCORDING TO OCCUPATION :

Out of 102 patients in the study, 49 patients(48%) were farmers, 24 patients(23.5%) were housewife, 13 patients(12.8%) were laborer's, 9 patients(8.8%) were business, 5 patients(4.9). The most common occupation associated with ACS in this study was Farming followed by home makers, laborer, business, employee. Table 4, Figure 5.

TABLE 4: DISTRIBUTION OF CASES ACCORDING TO OCCUPATION

Occupation	(Group A) AVPD<10		(Group B) AVPD≥10		Total		Chi square test
	N	%	N	%	N	%	
BUSINESS	4	13.8	5	9.3	9	10.8	P=0.933 NS
EMPLOYEE	2	6.9	3	5.6	5	6	
FARMER	15	15.7	27	50	42	50.6	
HOUSEWIFE	5	17.2	13	24.1	18	21.7	
LABOUROR	3	10.3	6	11.1	9	10.8	
	29	100.0	54	100	83	100	

FIGURE 5: DISTRIBUTION OF CASES ACCORDING TO OCCUPATION



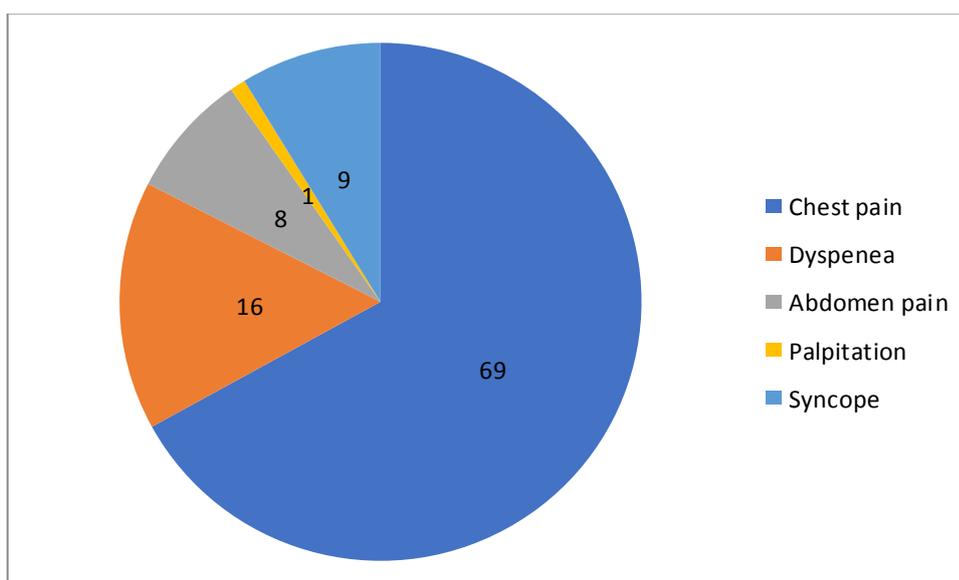
DISTRIBUTION ACCORDING TO SYMPTOMS:

Out of 102 patients in this study, 69 patients (67.6%) had chest pain, 16 patients(15.7%) had dyspnea, 8 patients(7.8%) had abdominal pain, 9 patient(8.8%) had syncope and one patient had palpitations.(1%). The common presenting symptom was chest pain (69 patients). Table 5, Figure 6.

TABLE 5: DISTRIBUTION OF CASES ACCORDING TO SYMPTOMS

CHIEF COMPLAINTS	No of patients	%
Chest pain	69	67.6
Dyspnea	16	15.7
Pain in Abdomen	8	7.8
Palpitation	1	1.0
Syncope	9	8.8

FIGURE 6: DISTRIBUTION OF CASES ACCORDING TO SYMPTOMS



DISTRIBUTION ACCORDING TO RISK FACTORS:

Among risk factors, out of 102 patients in the study, age more than 50 years was present in 32 patients with $AVPD \geq 10$ compared to 21 patients with $AVPD < 10$. Male sex was seen in 62 patients (74.7%) of which 39 patients had $AVPD \geq 10$ and 23 patients had $AVPD < 10$. Diabetes mellitus was seen in 22 patients of which 7 patients had $AVPD \geq 10$ and 15 patient had $AVPD < 10$ Hypertension was seen in 21 patients of which 11 patients had $AVPD \geq 10$ and 10 patients had $AVPD < 10$. Smoking habit was seen in 10 patients of which 7 patients had $AVPD \geq 10$ and 3 patients had $AVPD < 10$. Alcohol consumption was present in 8 patients of which 5 patients had $AVPD \geq 10$ and 3 patient had $AVPD < 10$ Tobacco chewing was seen in 11 patients, of which 7 patients had $AVPD \geq 10$ and 4 patients had $AVPD < 10$. Table 6.

TABLE 6: DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS

Risk Factors			(Group A) AVPD<10		(Group B) AVPD≥10		TOTAL		Chi square test
			N	%	N	%	N	%	
Non- Modifiable	AGE	<50 Years	8	27.60	22	40.74	30	36.14	P=0.234 NS
		≥50 Years	21	72.41	32		53	63.86	
	Gender	MALE	23	79.32	39	72.22	62	74.7	P=0.479 NS
		FEMALE	6	20.68	15	27.78	21	25.3	
Modifiable	Smoking		3	10.34	7	12.9	10	12.5	P=0.727 NS
	Alcohol		3	10.34	5	9.26	8	9.6	P=0.873 NS
	Tobacco Chewing		4	13.79	7	12.96	11	13.25	P=0.915 NS
	Diabetes Mellitus		7		15		22		P=0.048*
	Hypertension		10		11		21		P=0.043*
	Chest pain		15		44		59		P=0.004*
	Dyspnea		3		7		10		P=0.727 NS
	Abdominal Pain		4		2		6		P=0.091 NS
	Palpitation		0		1		1		P=0.461 NS
	Syncope		4		4		8		P=0.347 NS

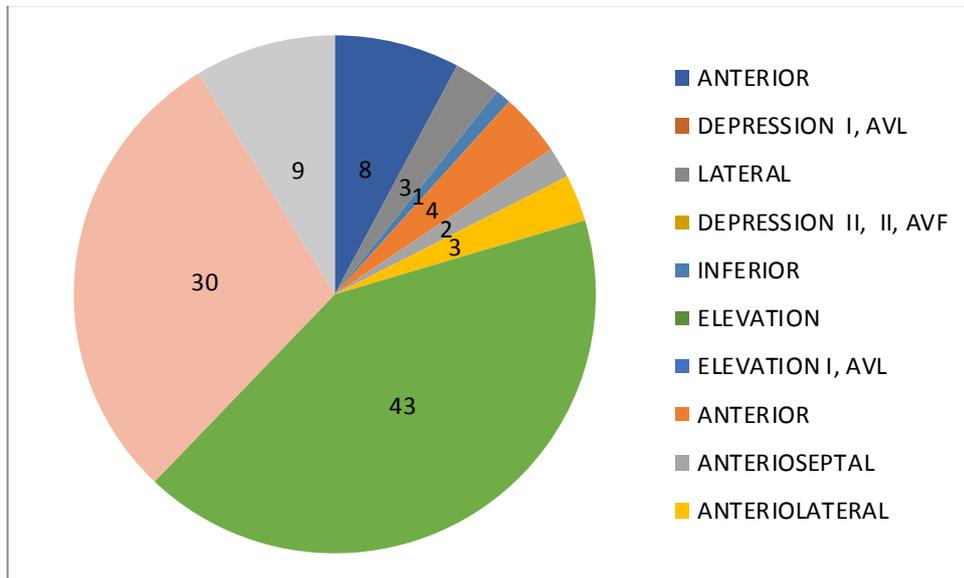
DISTRIBUTION ACCORDING TO ELECTROCARDIOGRAPHIC VARIABLES:

Out of 102 patients in the study, ST segment was elevated in 91 patients(88%), depressed in 12 patients(12%). Out of 91 patients with ST segment elevation in anterior leads (such as anterior, anteroseptal and anterolateral leads ST elevation) was seen in 48 patients, inferior leads ST elevation was seen in 43 patients. The most common ST segment elevation was seen in anterior leads. Table 7, Figure 8.

**TABLE 7: DISTRIBUTION OF CASES ACCORDING TO
ELECTROCARDIOGRAPHIC VARIABLES**

ST SEGMENT	No of patients	%
DEPRESSION V2,V3		
ANTERIOR	8	7.843137
DEPRESSION I, AVL		
LATERAL	3	2.941176
DEPRESSION II, III, AVF		
INFERIOR	1	0.980392
ELEVATION I, AVL		
ANTERIOR	4	3.921569
ELEVATION I, AVL		
ANTERIOSEPTAL	2	1.960784
ELEVATION I, AVL		
ANTERIOSEPTAL	3	2.941176
ELEVATION II,III, AVF		
INFERIOR	43	41.17647
ELEVATION V2,V3		
ANTERIOR	30	29.41176
ELEVATION V2,V3		
ANTERIOSEPTAL	9	8.823529

FIGURE 7: DISTRIBUTION OF CASES ACCORDING TO ELECTROCARDIOGRAPHIC VARIABLES



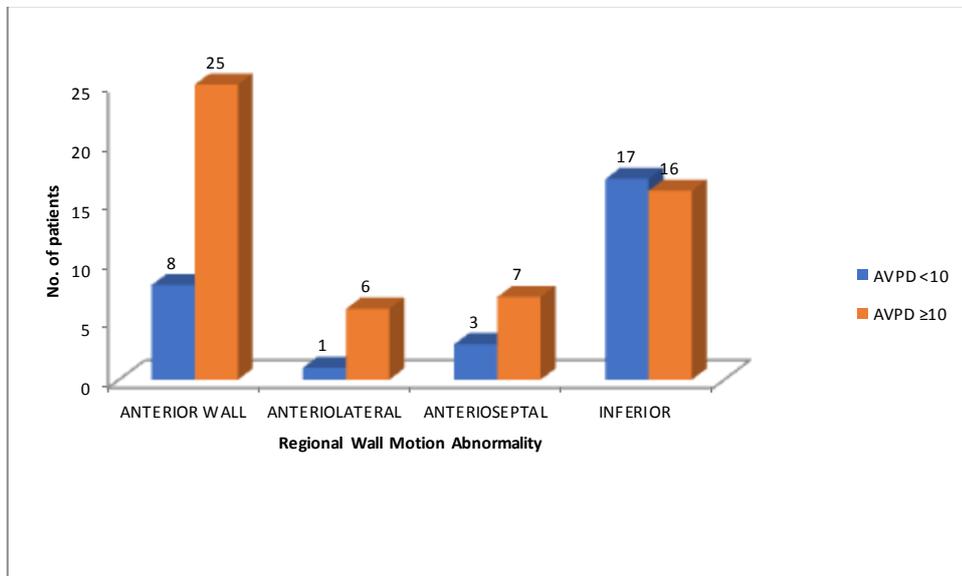
DISTRIBUTION ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In this study of 83 patients, echocardiographic parameters like regional wall motion abnormality, ejection fraction, Atrio Ventricular Plane Displacement had been studied. Out of 83 patients, 33 patients had anterior wall hypokinesia, 7 patients had anteriolateral wall hypokinesia, 10 patients had anterioseptal wall hypokinesia, 33 patients had inferior wall hypokinesia. Here 54 patients had AVPD \geq 10 where 29 patients had AVPD $<$ 10 . Ejection fraction calculated mean was 46.76% in patients with AVPD \geq 10 and 43.27% in patients with AVPD $<$ 10 with p value 0.0448 which is statistically significant. Table 8.

**TABLE 8: DISTRIBUTION OF CASES ACCORDING TO
ECHOCARDIOGRAPHIC VARIABLES**

ECHOCARDIOGRAPHIC VARIABLES		AVPD <10		AVPD ≥10		Chi square test	
		N		N			N
Regional Wall Motion Abnormality	Anterior wall	8	27.58	25	46.29	33(39.76)	P=0.070 NS
	Anteriolateral	1	3.45	6	11.11	7(8.4)	
	Anterioseptal	3	10.34	7	12.96	10(12)	
	INFERIOR	17	58.62	16	29.63	33(39.6)	
			Mean	Standard Deviation	Mean	Standard Deviation	
LVEF		43.27	7.4	46.76	7.84		P=0.0448*

**FIGURE 8: DISTRIBUTION OF CASES ACCORDING TO
ECHOCARDIOGRAPHIC VARIABLES**



DISTRIBUTION ACCORDING TO AVPD AND MAJOR ADVERSE CARDIAC EVENTS:

Patients are grouped as AVPD more than 10 and AVPD less than 10. Out of 83 patients in study, 54 patients had $AVPD \geq 10$ whereas rest 29 patients had $AVPD < 10$ (Table 9, Figure 9). Among these groups Heart failure was found in 1 patients with $AVPD \geq 10$ as compared to 13 patients with $AVPD < 10$. Pulmonary edema was found in 2 patients with $AVPD \geq 10$ as compared to 12 patients with $AVPD < 10$. Cardiogenic shock was present in 6 patients with $AVPD \geq 10$ as compared to 8 patients with $AVPD < 10$. In $AVPD \geq 10$ group, one patient had died. In $AVPD < 10$ group 9 patients had died (Table 10, Figure 10). In this study, major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, death were higher in patients with $AVPD < 10$ with p value < 0.001 , which is statistically very significant.

TABLE 9: DISTRIBUTION OF PATIENTS ACCORDING TO ATRIOVENTRICULAR PLANE DISPLACEMENT (AVPD)

AVPD	No of patients	%
AVPD <10	29	28.43
AVPD ≥10	54	52.94
TOTAL	83	100.0

FIGURE 9: DISTRIBUTION OF CASES ACCORDING TO AVPD

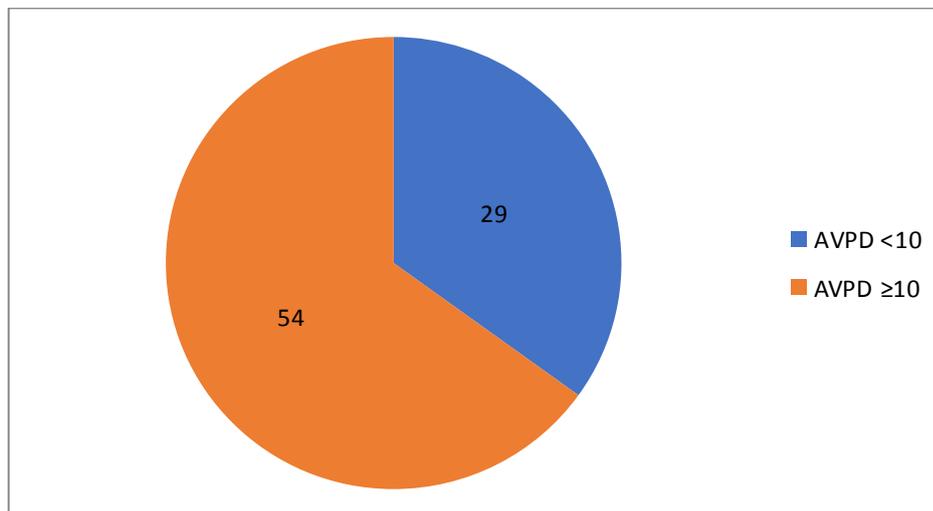
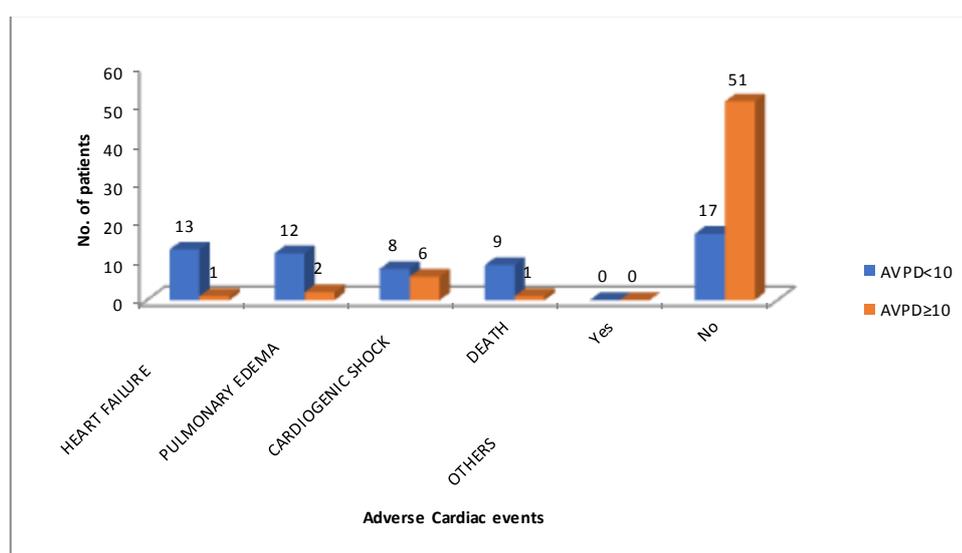


TABLE 10: DISTRIBUTION OF MAJOR PREVIOUS ADVERSE CARDIAC EVENTS ACCORDING TO AVPD

MACE	AVPD<10		AVPD≥10		TOTAL	p value
	N		N	%	N	
HEART FAILURE	13	44.83	1	1.85	14	0.0001*
PULMONARY EDEMA	12	41.38	2	3.70	14	<0.001*
CARDIOGENIC SHOCK	8	27.58	6	11.1	14	<0.001*
DEATH	9	31.03448	1	1.9	10	
OTHERS	3	10.34483	2	3.6	5	0.001*

Note: * significant at 5% level of significance (p<0.05)

FIGURE 10: DISTRIBUTION OF MAJOR ADVERSE CARDIAC EVENTS ACCORDING TO AVPD



DISCUSSION

This study is a Cross sectional study conducted over a period of two years from December 2016 to June 2018 to analyze Atrio Ventricular Plane Displacement as a predictor of in-hospital major cardiovascular events in acute coronary syndrome. 83 patients included in this study were analyzed to predict in-hospital major adverse cardiac events.

Patients were observed in-hospital for development of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death.

AGE:

In this study most, The most common age group was 51-60 (22 patients) with mean age of 56.81 ± 5 years. Similarly, in a study done by Filho N et al (25) in 520 patients, they found that patients mean age was 62.0 (± 12.2) years and found that patients ageing more than 55 had more common occurrence of having ACS.

Another study done by Mesas C et al (26) in 50 patients hospitalized with acute coronary syndrome in Southern Brazil, they observed that, mean age group was 59 years (standard deviation SD = 10.5 years) which was significantly similar as in this study. In a study done by Ajay VS et al (27) it was found than mean age group has changed in last 50 years, where it was equal in both Indian and western countries, now it is found that acute coronary syndrome occurs a decade earlier in Indian population compared to western countries. The reason could be lack of education about disease and risk factors, evidence-based treatment, lack of compliance of medications.

SEX:

In this study there was male predominance i.e., 74.7% of patients and female patients were 25.3%, which was similar to study done by Scholz K et al (28) where

male patients were 74% and female patients were 26%. In a study done by Cenko E et al (29) out of 8834 subjects studied, male patients were 69.9% and female were 30.1%.

OCCUPATION:

In this study the most common occupation associated with acute coronary syndrome 42 (50.6%) patients were farmers, 9 (10.8%) patients were businessmen , 5 (6%) patients were employee, 18 (21.7%) patients were housewife, 9 (10.8%) patients were laborer. Most of these patients belong to low and middle socioeconomic status. The reason could be lack of education about disease, risk factors, inability to afford for treatment, lack of compliance to medication, inability to modify risk factors and lack of regular follow up.

SYMPTOMS:

In this study, 69 patients (67.6%) had chest pain, 16 patients(15.7%) had dyspnea, 8 patients(7.8%) had abdominal pain, 9 patient(8.8%) had syncope and one patient had palpitations.(1%). The common presenting symptom was chest pain (69 patients). Similarly in a study done by Goel PK et al (30) in 609 patients admitted with ACS for 1 year in 2008, they found that the most common symptom in patients with acute coronary syndrome was chest pain (84%), followed by dyspnea (8.7%) and epigastric pain(2.6%).

In other study done by Conto J G et al (31) in 434877 patients admitted with acute myocardial infarction, they found that chest pain was present in 67% of patients which is same as that observed in this study.

RISK FACTORS:

In this study Diabetes mellitus was seen in 22 patients, hypertension was seen in 21 patients , smoking habit was seen in 10 patients, alcohol consumption was

present in 8 patients , tobacco chewing was seen in 11 patients. There is significant variation in various risk factors and their association with acute coronary syndrome in different studies. In this study diabetes mellitus was observed in 22.4% which is higher than that is reported by Yusuf S et al (32) study done in 2004, where patients in 52 other countries were included and comparable to an Indian study done by Xavier et al (1) In a study done by Krishnan M N et al (33) in 2016, in patients with acute coronary syndrome, diabetes mellitus was present in 15% patients, hypertension in 28% patients and smoking 28% of patients. Another study done by Rao V et al (34) in 2017, in 100 patients with acute coronary syndrome, it was observed that diabetes was present in 67% of patients, hypertension in 52% of patients, smoking was present in 61% of patients, alcohol consumption in 21% of patients. This study has high incidence of risk factors like diabetes mellitus, hypertension, smoking and alcohol consumption for acute coronary syndrome compared to this study. In study done by Alashetty AS et al (35) in 2014, in 61 patients with STEMI, diabetes mellitus was most common risk factor associated with ACS. These risk factors if modified can lead to increased life expectancy in patients with ACS. In a study done by Unal et al (36) between 1981 to 2000, they concluded that, life expectancy of patients with ACS can be increased four times than that is increased by modern cardiological treatment by modest reduction in major risk factors like smoking, hypertension, diabetes mellitus. Hence there is need for policies to control tobacco use, promote healthy diet and educate patients regarding control of diabetes mellitus which help in improving life expectancy of patients with ACS.

ELECTROCARDIOGRAM:

In this study of out of 102 patients, ST segment was elevated in 91 patients (88%), depressed in 12 patients(12%). Out of 91 patients with ST segment elevation in anterior leads (such as anterior, anteroseptal and anterolateral leads ST elevation) was seen in 48 patients, inferior leads ST elevation was seen in 43 patients. The most common ST segment elevation was seen in anterior leads. In a study done by Alashetty AS et al (35) in 2014, only 48.2% of patients had ST segment elevation which was significantly lower than this study. In another study done by Saczynski J et al (37) out of 7215 patients with acute myocardial infarction, STEMI was found in 61.56% and 38.04% were NSTEMI which is significantly lower than this study. The most common ST segment elevation was seen in anterior leads. In a study done by Reddy C S T et al (38)in 2013, they concluded that electrocardiogram identification of culprit artery helps in not only localization of proximal or distal occlusion but also to predict severity of myocardial infarction and plan emergency management.

ECHOCARDIOGRAPHY:

In this study of 83 patients, echocardiographic parameters like regional wall motion abnormality, ejection fraction, Atrio Ventricular Plane Displacement had been studied. Out of 83 patients, 33 patients had anterior wall hypokinesia, 7 patients had anterolateral wall hypokinesia, 10 patients had anterioseptal wall hypokinesia, 33 patients had inferior wall hypokinesia. In a study done by Savith.A et al (39) patients had higher incidence of inferior wall myocardial infarction and was present in 48% of patients, followed by anterolateral wall MI in 26% patients and anteroseptal wall MI was in 22% of patients.

MAJOR ADVERSE CARDIAC EVENTS AND AVPD:

In this study it was observed that increased AVPD, measured at admission in patients with STEMI, is an important predictor of major cardiovascular events. Out of 83 patients in study heart failure was found in 1 patients with $AVPD \geq 10$ as compared to 13 patients with $AVPD < 10$. Pulmonary edema was found in 2 patients with $AVPD \geq 10$ as compared to 12 patients with $AVPD < 10$. Cardiogenic shock was present in 6 patients with $AVPD \geq 10$ as compared to 8 patients with $AVPD < 10$. In $AVPD \geq 10$ group, one patient had died. In $AVPD < 10$ group 9 patients had died. In this study, major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, death were higher in patients with $AVPD < 10$ with p value < 0.001 , which is statistically very significant. Patients with $AVPD < 10$ had worse prognosis in terms of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death. So, in general, patients with $AVPD \geq 10$ has good outcome.

The observations in this present study are similar to study done by Nammas W. et al (6) in 2012, where they studied 93 patients and found that in 22 patients with $AVPD < 10$, here patients who developed heart failure was 17 and 1 patients died, the incidence of major adverse cardiac events like heart failure and death were significantly higher compared to patients with $AVPD \geq 10$ predict prognosis in patients with acute coronary syndrome. In study done by Brand B. et al (40) in 2002 studied 271 patients and found that 43.8% patients died and heart failure developed in 14.6% of patients where in this study death was seen in 33% of patients and heart failure was seen in 47% of patients.

In another study done by Willenheimer R. et al (23) in 1997 and 181 patients were studied death occurred in 41 patients (22.7%) where $AVPD < 10$ and 19

(10.5%) died where AVPD was ≥ 10 . Hence AVPD is useful in prediction of morbidity and mortality in patients with ST Segment Elevation Myocardial Infarction.

SUMMARY

One hundred and two patients with acute coronary syndrome were studied at B.L.D.E. (DEEMED TO BE UNIVERSITY) Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura between December 2016 to June 2018.

This study was conducted to know Atrio Ventricular Plane Displacement as a predictor of early in-hospital major cardio vascular events in acute coronary syndrome.

1. The common age group was 51- 60 years. Age more than 50 years was associated with increased Atrio Ventricular Plane Displacement and occurrence of in-hospital major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death.
2. In the present study male patients (74.7%) were more common than female (25.3%).
3. Risk factors like diabetes and hypertension was most common occurring in 22 patients had diabetes and 21 had hypertension, and there is significant association between AVPD and risk factors indicated by p value < 0.05 .
4. Out of 102 patients, the common wall motion abnormality was anterior wall occurring in 50 patients. There was no significant association between AVPD and wall motion abnormality as p value was ≥ 0.05 .
5. Mean ejection fraction was 46.76% in patients with AVPD ≥ 10 and 43.27% in patients with AVPD < 10 which was significant, p value < 0.001 .
6. Of the 83 patients with STEMI, the AVPD was less than 10 in 29 patients and more than 54 in patients

7. Mean Atrio Ventricular Plane Displacement was \pm 11mm with 7.8mm being lower limit and 16mm being the highest limit.
8. Atrio Ventricular Plane Displacement less than 10 was associated with occurrence of in-hospital major adverse cardiac events like heart failure (47%), pulmonary edema (45%), cardiogenic shock (39%) and death (33%) with p value<0.001.
9. In general patients with Atrio Ventricular Plane Displacement more than 10 had good prognosis.
10. Atrio Ventricular Plane Displacement should be routinely measured in patients with acute coronary syndrome.

CONCLUSION

In the present study of 102 patients with ACS only 83 patients were of STEMI and there was increased incidence of in-hospital major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death in patients aged more than 50 years. Echocardiographic parameters like ejection fraction and diastolic dysfunction were significantly associated with decreased Atrio Ventricular Plane Displacement. AVPD < 10 was found to be an important predictor of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death.

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ETHICAL CLEARANCE CERTIFICATE



B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

OUTWARD
No. 61/2016
06/10/2016
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College, BIJAPUR-586103

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04-10-2016 at 3-pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Atrio-ventricular Plane Displacement : As a predictor of in hospital outcome of Major Adverse Cardiac Events after Acute ST-Elevation Myocardial Infarction

Name of P.G. student Dr. Sainath M. Kudumbale
Dept. of Medicine

Name of Guide/Co-investigator Dr. Badiger Sharanabasappa
Professor of Medicine

DR. TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College, BIJAPUR-586103.

Following documents were placed before E.C. for Scrutination

- 1) Copy of Synopsis/Research project.
- 2) Copy of informed consent form
- 3) Any other relevant documents.

CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL
COLLEGE HOSPITAL AND RESEARCH CENTER, BIJAPUR-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned, _____, S/O D/O W/O
_____, aged _____ years, ordinarily resident of
_____ do hereby state/declare that **DR. SAINATH M. KUDUMBALE**
of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me
thoroughly on _____ at _____ (place) and it has been explained
to me in my own language that I am suffering from _____ disease
(condition) and this disease/condition mimic following diseases. Further Doctor Dr.
SAINATH informed me that he/she is conducting dissertation/research titled
“Atrioventricular plane displacement As a predictor of in-hospital outcome of Major
Adverse Cardiac Events after acute ST-elevation myocardial infarction” under the
guidance of Dr. Badiger Sharanabasawappa requesting my participation in the study.

Apart from routine treatment procedure, the pre-operative, operative, post-
operative and follow-up observations will be utilized for the study as reference data.
Doctor has also informed me that during conduct of this procedure like adverse results
may be encountered. Among the above complications most of them are treatable but
are not anticipated hence there is chance of aggravation of my condition and in rare
circumstances it may prove fatal in spite of anticipated diagnosis and best treatment
made available.

Further Doctor has informed me that my participation in this study help in evaluation of the results of the study which is useful reference to treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made photographs video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes. The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during the course of treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis.

At the same time I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged. After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Shri/Smt _____ under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Witness:

Date:

Place:

**B.L.D.E (DEEMED TO BE UNIVERSITY) SHRI B.M.PATIL MEDICAL
COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR**

“Atrioventricular plane displacement as a predictor of in-hospital outcome of Major
Adverse Cardiac Events after acute ST-elevation myocardial infarction”

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Residence:

Presenting complaints:

History of present illness:

Past History:

Family History:

Personal History:

Diet/appetite

Sleep

Bladder and bowel habits:

Smoking/Tobacco chewing/Alcohol

General Physical Examination:

Vitals

PR :

BP :

RR :

Temp:

Icterus:

Clubbing:

Generalized lymphadenopathy:

Pallor:

Cyanosis:

Pedal edema

SYSTEMIC EXAMINATION**Cardiovascular System**

Arterial system:

Pulse

Rate

Rhythm

Volume

Character

Condition of vessel wall

Radio radial

Radio femoral delay

Other peripheral pulses

Venous system:

Engorged veins in neck

Blood Pressure

Precordial examination

Inspection

Palpation

Auscultation

Respiratory System:

Per abdomen:

Central Nervous System:

INVESTIGATIONS

HAEMATOLOGY –

Hemoglobin	gm %
Total WBC counts	Cells/mm ³
Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour

BIOCHEMISTRY–

Random blood sugar	
Blood urea	
Serum creatinine	
Serum sodium	
Serum potassium	

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

TROPONIN I:

CPK MB:

ECG-

	ECG
Standardization	
Rate	
Rhythm	
P wave	
PR interval	
QRS complex	
QRS configuration	
QRS duration	
QRS Axis	
ST Segment	
T wave	
QT	
QTc	
Arrhythmias	
Ectopics	
Heart blocks	

Ecg diagnosis

Echocardiographic and colour doppler findings:

MEASUREMENTS:

LVIVSd:

LVIDd:

RVIDd:

LVPWd:

LVISd:

Aorta:

LAV:

EF%:

Pulmonary Artery:

VALVES:

Mitral Valve:

Aortic Valve:

Tricuspid Valve :

Pulmonary Valve:

CHAMBERS:

Left Ventricle

Right Ventricle

Left Atrium

Right Atrium

SEPTAE

GREAT ARTERY

Aorta

Pulmonary Artery

DOPPLER STUDY

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

REGIONAL WALL MOTION ABNORMALITY

PERICARDIAL EFFUSION

CLOT/VEGETATION

AVPD Measurements:

Major adverse cardiac events:

- 1) Heart Failure
- 2) Pulmonary Edema
- 3) Hypotension
- 4) others

KEY TO MASTER CHART

P	-	PRESENT
A	-	ABSCENT
M	-	MALE
F	-	FEMALE
ST	-	SINUS TACHYCARDIA
VPC	-	VENTRICULAR PREMATURE COMPLEX
VT	-	VENTRICULAR TACHYCARDIA
LVEF	-	LEFT VENTRICULAR EJECTION FRACTION
GR	-	GRADE
MR	-	MITRAL REGURITATION
TR	-	TRICUSPID REGURITATION
HB	-	HEART BLOCK

MASTER CHART

SL NO.	PATIENT NAME	AGE	SEX	OCCUPATION	IP NO.	D.O.A	D.O.D	SYMPTOMS					DIABETES	HYPERTENSION	FAMILY HISTORY	HABITS			VITALS				HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	S-CREATININE	S-SODIUM	S-POTASSIUM	TROPONIN T	CPK MB	ECG		2D-ECHO		LVEF	ATRIOVENTRICULAR PLANE DISPLACEMENT (AVPD)	NT pro BNP (pg/ml)	PREVIOUS ADVERSE CARDIAC EVENTS				INCLUSION/ EXCLUSION
								CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PAPITATION	SYNCOPE				SMOKING	ALCOHOL	TOBACCO CHEWING	PR	BP	RR	TEMPERATURE											ST-SEGMENT	RHYTHM	REGIONAL WALL MOTION ABNORMALITY	HEART FAILURE				PULMONARY EMBOLISM	CARDIOGENIC SHOCK	OTHERS		
1	BHASHASAB	50	M	WORKER	36325	11/2/2016	11/8/2016	A	P	A	A	A	A	A	A	A	A	78	120/80	19	37	15.6	9080	-	-	-	-	-	-	N	-	ELVATION V2,V3	NILL	ANTERIOR	40%	-	-	NO	NO	NO	NO	E		
2	HEMALATHA	70	F	HOUSEWIFE	36451	11/2/2016	11/6/2016	P	A	A	A	A	A	P	A	A	A	80	110/80	16	37.4	-	-	-	168	-	0.8	132	3.3	N	40	DEPRESSION I,AVL	NILL	ANTERIOR	55%	-	-	NO	NO	NO	NO	E		
3	MALLAPA	60	M	FARMER	36764	11/5/2016	11/10/2016	A	P	A	A	A	-	-	-	-	-	-	-	-	10	12160	-	98	-	0.9	137	4	-	ELEVATION I,AVL	NILL	INFERIOR	35%	-	-	NO	NO	NO	NO	E				
4	MEHBOOSAB	70	M	FARMER	37924	11/15/2016	11/19/2016	A	P	A	A	A	A	A	A	A	A	80	110/70	14	37.5	13	22500	-	181	-	1	134	3.6	-	ELEVATION V2,V3	NILL	ANTERIOR	40%	12.4MM	-	NO	NO	NO	NO	I			
5	GOURAWWA	76	F	HOUSEWIFE	38034	11/16/2016	11/21/2016	A	P	A	A	A	A	A	A	A	A	64	130/80	16	37.1	12.4	12910	-	-	-	-	-	N	20	ELEVATION V3,V4	NILL	ANTERIOR	45%	11.4MM	-	NO	NO	NO	NO	I			
6	GURUBAI BIRADR	75	F	HOUSEWIFE	38564	11/30/2016	12/4/2016	P	A	A	A	A	A	A	A	A	A	74	140/70	14	37.6	11	15120	-	157	20	0.9	137	4	N	40	DEPRESS V2,V3	NILL	ANTERIOR	50%	-	-	NO	NO	NO	NO	E		
7	RAMESH RATHOD	34	M	FARMER	40855	12/11/2016	12/17/2016	P	A	A	A	A	A	A	A	A	A	84	136/84	12	37.2	12.9	18520	-	115	-	0.9	132	5.6	P	223	ELEVATION V2,V3	NILL	ANTERIOR	45%	16MM	-	NO	NO	NO	NO	I		
8	BHARAT	52	M	FARMER	41571	12/17/2016	12/19/2016	P	A	A	A	A	A	A	A	A	A	84	140/90	14	37.1	14.4	11960	-	200	-	0.8	131	4.2	P	62	ELEVATION V2,V3	RBBB	ANTERIOR	-	-	-	NO	NO	NO	NO	E		
9	BASAPPA	75	M	FARMER	50/17	1/2/2017	1/10/2017	A	P	A	P	A	A	A	A	A	A	90	124/84	15	37.1	17.3	27010	-	231	-	1	136	4.5	N	55	ELEVATION V2,V3	NILL	ANTERIOR	55%	11.2MM	-	NO	NO	NO	NO	I		
10	SHIVAPPA	55	M	LABOUROR	1837	1/17/2017	1/19/2017	P	A	A	A	A	A	A	A	A	A	88	110/70	16	37.6	16.1	11850	-	100	32	0.8	-	-	-	-	ELEVATION V3,V4	NILL	ANTRIOSEPTAL	55%	12.2MM	-	NO	NO	NO	NO	I		
11	RACHAYYA	56	M	BUSINESS	2267/17	1/20/2017	1/26/2017	P	A	A	A	A	A	A	A	A	A	104	180/100	15	37.1	9.2	36950	20	125	20	0.6	142	4	P	52	ELEVATION II, III, AVF	NILL	INFERIOR	55%	13.3MM	-	NO	NO	NO	NO	I		
12	NEELAMMA	50	F	HOUSEWIFE	2388/17	1/22/2017	1/29/2017	P	A	A	A	A	A	P	A	A	A	96	150/100	14	37	11.2	18460	85	257	36	1	141	4.4	P	50	ELEVATION III,AVF	NILL	INFERIOR	45%	10.75MM	-	NO	NO	NO	NO	I		
13	TOTAPPA	48	M	FARMER	3082/17	1/28/2017	2/4/2017	P	A	A	A	A	A	P	A	A	A	90	180/120	14	37	16	8900	10	180	25	0.8	138	4	N	29	ELEVATION V2-3	NILL	ANTERIOR	40%	12.2MM	-	NO	NO	NO	NO	I		
14	PARVATHI	65	F	HOUSEWIFE	3085/17	1/28/2017	2/3/2017	P	A	A	A	A	A	P	A	A	A	104	170/100	14	37	13.4	12500	30	129	30	0.6	141	4.3	P	133	ELEVATION V2-4	NILL	ANTERIOR	45%	11.8MM	-	NO	YES	NO	NO	I		
15	SATTAPPA	60	M	FARMER	6619/17	2/28/2017	3/4/2017	P	A	A	A	A	P	P	A	A	P	74	116/70	16	37.1	13.5	19760	10	314	24	0.6	138	4.2	P	85	ELEVATION V2& II	NILL	ANTERIOATERAL	40%	11.8MM	-	NO	NO	NO	NO	I		
16	ALISAB	65	M	LABOUR	7989/17	3/11/2017	3/16/2017	P	A	A	A	A	A	P	A	P	A	88	140/90	16	37	13.4	11530	40	120	33	1.4	140	4	P	95	ELEVATION V1-3	NILL	ANTERIOSEPTAL	45%	10.9MM	7032pg	NO	NO	NO	NO	I		
17	BALA SAHEB	60	M	FARMER	8854/17	3/19/2017	3/16/2017	P	A	A	A	A	A	A	P	A	A	64	120/80	16	36.9	13.7	11840	20	153	20	2.6	137	3.8	P	112	ELEVATION II, III, AVF	NILL	ANTERIOATERAL	50%	13.3MM	1652pg	NO	NO	NO	NO	I		
18	GANGA	60	F	FARMER	9056/17	3/21/2017	3/29/2017	P	A	A	A	A	P	A	A	A	A	102	190/100	16	36.8	10.8	13600	20	422	26	0.9	141	4.7	P	53	ELEVATION II, III, AVF	NILL	ANTERIOATERAL	55%	13.2MM	3292pg	NO	NO	NO	NO	I		
19	BAHUBALI	30	M	EMPLOYEE	10063/17	3/30/2017	4/6/2017	A	P	A	A	A	A	A	A	A	A	120	96/50	32	37	10	17110	40	85	18	0.8	140	4.5	P	72	ELEVATION II, III, AVF	NILL	ANTERIOATERAL	55%	13MM	5250pg	NO	NO	YES	NO	I		
20	SAIBANNA	65	M	FARMER	10311/17	4/1/2017	4/7/2017	A	P	A	A	A	A	A	P	A	A	100	170/90	14	37.2	12	11400	20	210	84	2	138	6.9	N	78	DEPRESSION V3-5, I,AVL	NILL	INFERIOR	55%	-	-	NO	NO	NO	NO	E		
21	KAMALA BAI	60	F	FARMER	10754/17	4/5/2017	4/6/2017	A	A	P	A	A	P	A	A	A	A	80	130/80	14	37	11.5	12100	70	269	34	1.5	142	3.2	P	35	ELEVATION V2-5	NILL	ANTERIOR	40%	8.87MM	>2500pg	NO	YES	NO	DEATH	I		
22	YELLAPPA	50	M	FARMER	11078/17	4/7/2017	4/12/2017	P	A	A	A	A	A	A	P	P	A	70	120/70	16	37.1	12.1	3830	65	122	22	0.7	133	4	P	69	ELEVATION II,III,AVF	NILL	INFERIOR	45%	10.75MM	2232pg	NO	NO	NO	NO	I		
23	GOLLAPPA	87	M	FARMER	11399/17	4/10/2017	D 11-4/17	P	A	A	A	A	P	A	A	A	P	86	90/50	26	37	10.5	12200	45	104	31	1.3	137	4.5	P	114	ELEVATION V2-6	NILL	ANTERIOSEPTAL	45%	10.6MM	1692pg	YES	YES	YES	DEATH	I		
24	GANGARAM	52	M	PHARMACIST	11400/17	10/1/2017	4/16/2017	P	A	A	A	A	P	A	A	A	A	88	130/80	14	37	13.6	21150	5	93	30	0.8	141	4.3	P	206	ELEVATION V1-4	NILL	ANTERIOSEPTAL	45%	13.75MM	466pg	NO	NO	NO	NO	I		
25	MACHINDRA	50	M	FARMER	11608/17	4/12/2017	4/18/2017	P	A	A	A	A	A	A	A	A	P	60	90/60	14	37.4	14	11620	15	112	24	1	137	4.8	P	225	ELEVATION V1-4	NILL	ANTERIOSEPTAL	45%	13.22MM	1112pg	NO	NO	YES	NO	I		
26	MALLIKARJUNA	65	M	EX EMPLOYEE	13708/17	4/30/2017	5/5/2017	P	A	A	A	A	P	P	A	A	A	80	120/80	14	37.1	13.1	10140	40	312	22	1.2	132	5.2	P	55	ELEVATION V2-5	NILL	ANTERIOSEPTAL	50%	12.65MM	2261pg	NO	NO	NO	BRADY	I		

27	TARABAI	64	F	HOUSEWIFE	1374317	5/1/2017	5/6/2017	A	A	P	A	A	P	P	A	A	A	A	98	140/80	14	37	10.7	10530	80	169	20	0.7	140	3.5	N	61	DEPRESSION V2-5	NILL	ANTERIOPLATERAL	55%	-	-	NO	NO	NO	NO	E
28	BHEERAPPA	65	M	FARMER	1401217	5/3/2017	5/9/2017	P	A	A	A	A	A	A	P	A	A	80	1224/80	14	37.1	14.1	10200	10	176	22	0.9	130	4	N	34	ELEVATION IILIAVAF	NILL	ANTERIOPLATERAL	40%	9.5MM	680pg	NO	NO	NO	NO	I	
29	HEMA	40	F	HOUSEWIFE	1385117	5/1/2017	5/10/2017	P	A	A	A	A	A	A	A	A	A	80	120/70	16	37.2	10.4	9030	15	159	60	0.8	140	4.5	N	42	ELEVATION IILIAVAF	NILL	INFERIOR	40%	11.2MM	-	NO	NO	NO	NO	I	
30	NINGAPPA	95	M	NONE	1562017	5/16/2017	5/22/2017	A	A	A	A	A	A	P	A	P	A	A	80	140/90	16	37	13.4	11960	35	241	50	1.2	141	4.3	P	39	DEPRESSION V3-6	NILL	INFERIOR	30%	-	-	NO	NO	YES	NO	E
31	MAHABOOB	48	M	LABOURER	1614717	5/21/2017	5/26/2017	P	A	A	A	A	P	A	A	A	A	40	90/60	14	37.1	11.3	16430	5	458	79	2.9	130	5	P	129	ELEVATION III AVF	31 AV BLOC K	INFERIOR	60%	10.15MM	4031pg	NO	NO	YES	NO	I	
32	PARVATHI	77	F	HOUSEWIFE	1619817	5/22/2017	5/28/2017	P	A	A	A	A	P	A	A	A	A	80	140/80	15	37	10.8	7620	15	204	20	0.7	135	4.1	N	16	ELEVATION V5&I AVL	NILL	ANTERIOPLATERAL	45%	11.3MM	2920pg	NO	NO	NO	NO	I	
33	YEMKAWWA	80	F	HOUSEWIFE	1783117	6/3/2017	6/7/2017	P	A	A	A	A	A	A	A	A	A	76	90/60	14	37	13.3	17650	20	121	34	0.7	134	3.4	P	234	ELEVATION II, III, AVF	NILL	INFERIOR	45%	10.6MM	6829pg	NO	NO	YES	NO	I	
34	RADHA	38	F	HOUSEWIFE	1825317	6/7/2017	6/14/2017	P	A	A	A	A	A	A	A	A	A	90	110/80	15	37.1	11.8	28030	10	185	26	0.6	133	4.2	N	15	ELEVATION V2-4	NILL	ANTERIOSEPTAL	60%	11.8MM	482pg	NO	NO	NO	NO	I	
35	PARVATHI	60	F	HOUSEWIFE	1852317	6/9/2017	6/15/2017	P	P	A	A	A	A	A	A	A	A	120	190/110	16	37	8.8	15490	70	520	32	0.7	142	3.1	P	57	DEPRESSION V3-6	NILL	ANTERIOPLATERAL	40%	-	-	NO	NO	NO	NO	E	
36	DEVAPPANNAGOU DA	56	M	BUSINESS	1853417	6/9/2017	6/15/2017	P	A	A	A	A	A	A	A	P	P	A	80	120/90	16	37.1	15.1	8950	10	199	14	0.6	131	4	N	28	ELEVATION V2&I AVL	NILL	ANTERIOSEPTAL	35%	11.9MM	248pg	NO	NO	NO	NO	I
37	TULASABAI	54	F	HOUSEWIFE	1864717	6/10/2017	6/17/2017	P	A	A	A	A	P	A	A	A	A	98	140/80	14	37	10.5	15330	55	280	28	0.8	131	5.1	N	34	DEPRESSION IILIAVAF	NILL	INFERIOR	50%	-	-	NO	NO	NO	NO	E	
38	NANAGOUDA	55	M	FARMER	1944317	6/16/2017	6/22/2017	P	A	A	A	A	A	P	A	A	A	130	160/106	15	37	15.9	16190	5	166	31	0.7	144	4.1	P	85	ELEVATION V2&I AVL	NILL	ANTERIOR	40%	9.85MM	90pg	NO	NO	NO	NO	I	
39	SANGANNA GOUDA	41	M	FARMER	1979617	6/19/2017	6/26/2017	P	A	A	A	A	A	A	A	P	P	A	80	136/90	16	36.8	19.8	18270	5	77	20	0.8	140+AB A 41: AD41	3.1	P	20	ELEVATION II, III, AVF	NILL	INFERIOR	60%	15.5MM	841pg	NO	NO	NO	NO	I
40	CHENNAMMA	60	F	HOUSEWIFE	1738117	6/19/2017	8/26/2017	P	A	A	A	A	A	A	A	A	A	96	130/80	14	36.8	12.3	10850	80	190	20	0.6	129	2.9	P	45	DEPRESSION V2-5	NILL	ANTERIOSEPTAL	40%	-	-	NO	NO	NO	NO	E	
41	KAMALA BAI	55	F	HOUSEWIFE	2055817	6/24/2017	30-60-17	P	P	A	A	A	A	A	A	A	A	84	130/70	16	36.9	11.6	20970	10	180	12	0.6	136	3.2	N	33	ELEVATION II, III, AVF	NILL	INFERIOR	50%	11.2MM	358pg	NO	NO	YES	C-HB	I	
42	KEMPAYYA	65	M	FARMER	2065717	6/24/2017	7/1/2017	P	A	A	A	A	A	A	A	A	P	88	120/80	16	37	13.6	9640	35	447	20	0.8	135	4.5	N	64	DEPRESSION V2-6	VPC	ANTERIOPLATERAL	45%	-	-	NO	NO	NO	NO	E	
43	MAHAVEER	45	M	LABOURER	2066817	6/25/2017	7/2/2017	P	A	A	A	A	A	A	A	A	A	98	150/80	14	37	13.7	14700	20	124	20	0.7	140	3.9	P	78	ELEVATION V2.6, II, III, AVF	NILL	ANTERIOR	50%	13.2MM	1197pg	NO	NO	NO	NO	I	
44	SHARAWWA	60	F	HOUSEWIFE	2073117	6/16/2017	7/4/2017	P	A	A	A	A	P	P	A	A	A	80	120/70	14	37	12.9	9460	15	180	21	0.7	136	4	P	79	ELEVATION V2,V3,AVL	NILL	ANTERIOPLATERAL	40%	11.6MM	-	NO	NO	NO	NO	I	
45	RAYAWWA	65	F	HOUSEWIFE	2500517	7/30/2017	8/4/2017	P	A	A	A	A	A	A	A	A	A	60	130/100	20	37.1	11	11810	20	159	21	0.6	132	3.8	P	72	ELEVATION II, III, AVF	HB	INFERIOR	50%	9MM	372pg	NO	NO	NO	NO	I	
46	PRAKASH	58	M	LABOURER	2500117	7/31/2017	8/5/2017	P	A	A	A	A	A	A	A	P	A	90	140/90	16	36.8	7.4	12650	60	127	22	0.8	132	4.2	N	82	ELEVATION V2-5	NILL	ANTERIOR	45%	13.7MM	2786pg	NO	NO	NO	NO	I	
47	HANAMANTH	65	M	FARMER	2751917	8/19/2017	8/25/2017	P	A	A	A	A	A	A	A	A	P	84	130/80	16	37	11.8	11400	40	122	22	1.2	136	4.7	P	45	ELEVATION V1.4, I AVL	NILL	ANTERIOSEPTAL	40%	9MM	25000pg	NO	NO	NO	VT	I	
48	NEELAKANTAYYA	78	M	FARMER	2751617	8/19/2017	8/28/2017	A	P	A	A	A	A	P	A	A	A	130	150/110	16	37	10.1	1000	90	332	30	1.4	139	4.8	P	30	ELEVATION V1-3	VT	ANTERIOR	40%	9MM	25000pg	NO	YES	NO	VT	I	
49	MALAKAWWA	50	F	HOUSEWIFE	2777417	8/21/2017	8/28/2017	P	A	A	A	A	P	A	A	A	A	72	130/80	14	37.1	12.3	17710	20	169	27	0.6	136	0.2	P	300	ELEVATION V2.4, I AVL	NILL	ANTERIOR	40%	11MM	820pg	NO	NO	NO	NO	I	
50	LAXMIBAI	60	F	LABOURER	4110	2/2/2018	2/7/2018	P	A	A	A	A	A	A	A	A	P	80	110/70	14	37.2	11.3	4310	10	169	20	0.8	135	4.3	P	24	ELEVATION II,III	NILL	INFERIOR	55%	-	-	NO	NO	NO	NO	E	
51	SATISH	35	M	UNEMPLOYE D	5011	2/9/2018	2/10/2018	A	A	P	A	A	P	P	A	A	P	80	170/100	18	37.4	17.7	20080	5	333	36	2.1	132	3.5	P	-	DEPRESSION V2,V3,V4	NILL	ANERIOSEPTAL	35%	-	-	NO	NO	NO	DEATH	E	
52	IRRAPA	70	M	FARMER	6327	2/20/2018	2/20/2018	A	A	A	A	P	A	A	A	A	A	FEEBLE	NOT RECORDABLE	20	38	-	-	-	93	35	1.4	144	4.4	-	42	ELEVATION IILIAVAF	NILL	INFERIOR	60%	-	-	NO	NO	YES	DEATH	E	
53	SABEWWA	60	F	HOUSEWIFE	6385	2/21/2018	2/26/2018	P	A	A	A	A	P	A	A	A	A	80	120/80	18	36.5	15	13340	5	320	-	1.5	134	5.6	P	53	ELEVATION IILIAVAF	NILL	ANTERIOSEPTAL	30%	8.1MM	-	NO	NO	NO	DEATH	I	
54	BAGEWWA	70	F	HOUSEWIFE	6365	2/20/2018	2/26/2018	A	A	A	A	P	A	P	A	A	A	84	150/90	18	37	7.2	10620	50	126	-	0.8	138	4.7	P	50	ELEVATION IILIAVAF	NILL	INFERIOR	40%	9.7MM	-	NO	NO	NO	NO	I	
55	NARAYAN	63	M	FARMER	6761	2/23/2018	3/1/2018	A	P	A	A	A	P	P	A	A	A	114	110/70	16	37	8.1	6510	-	106	95	7.4	136	4.5	P	22	DEPRESSIONV5V6	NILL	LATERAL	40%	-	-	NO	NO	NO	NO	E	
56	HARICHANDRA	65	M	FARMER	7218	2/27/2018	3/5/2018	A	P	A	A	A	A	A	A	P	A	110	80/60	22	37	12.1	12700	15	61	42	3.1	127	5.3	P	109	ELEVATION IILIAVAF	NILL	INFERIOR	45%	-	-	NO	NO	NO	NO	E	
57	AMBAJI	43	M	LABOURER	7269	2/28/2018	2/28/2018	P	A	A	A	A	A	A	P	P	A	70	130/80	16	37.4	12	23440	-	-	28	0.8	-	-	-	-	ELEVATION V1-V4	NILL	ANTRIOSEPTAL	-	-	-	YES	YES	YES	DEATH	E	
58	SIDRAM	45	M	FARMER	8302	3/8/2018	3/13/2018	P	A	A	A	A	A	A	A	A	A	70	110/60	14	37.1	13.6	13490	-	121	30	1.1	145	4.4	P	145	ELEVATION IILIAVAF	NILL	INFERIOR	50%	10.9MM	-	NO	NO	NO	NO	I	
59	SAVALAGAPPA	55	M	WATCHMAN	8910	3/15/2018	3/22/2018	P	A	A	A	A	A	A	A	A	A	108	130/80	12	38.3	12.6	2090	25	126	-	0.7	141	4.5	P	47	ELEVATION IILIAVAF	NILL	INFERIOR	60%	10.05MM	-	NO	NO	NO	NO	I	
60	SUNANDA	48	F	HOUSEWIFE	9249	3/16/2018	3/28/2018	A	A	A	A	A	A	A	A	A	A	110	140/80	16	37	12.7	9240	67	143	24	0.8	141	4	P	27	ELEVATION V1-V4	NILL	ANTERIOR	30%	11MM	-	NO	NO	NO	NO	I	
61	SUKADEV	45	M	NURSE	9477	3/18/2018	3/27/2018	P	A	A	A	A	A	A	A	A	A	100	150/90	16	37.1	13.6	14640	15	180	-	0.6	145	4	P	13	ELEVATION V1-V4	NILL	ANTERIOSEPTAL	35%	9MM	-	NO	NO	YES	Y	I	
62	MALAPPA	63	M	FARMER	11022	3/30/2018	4/4/2018	P	A	A	A	A	P	P	A	A	A	90	150/80	14	37.5	16.8	14470	15	380	-	0.9	141	5.2	-	50	ELEVATION IILIAVAF	NILL	INFERIOR	40%	12MM	-	NO	NO	NO	NO	I	
63	NINGAWWA	35	F	LABOURER	12310	4/11/2018	4/18/2018	A	A	P	A	A	A	A	A	A	A	80	110/60	14	37	11.3	11260	10	301	-	0.6	136	3.8	P	42	ELEVATION IILIAVAF	NILL	INFERIOR	40%	13.2MM	-	NO	NO	NO	NO	I	
64	YALLAPA	64	M	FARMER	12746	4/11/2018	4/18/2018	P	A	A	A	P	A	A	A	A	A	82	112/80	11	37.2	13.8	14398	5	189	27	0.9	131	4.4	P	74	ELEVATION V1-V4	NILL	ANTERIOR	45%	11MM	-	NO	NO	NO	NO	I	

65	RAMESH	40	M	LABOURER	12853	4/16/2018	4/20/2018	P	A	A	A	A	A	A	A	A	P	78	110/70	14	37	15.6	5530	10	89	31	0.8	137	4.4	P	28	ELEVATION IILIAVAF	NILL	INFERIOR	40%	9.2MM	-	YES	YES	NO	NO	I
66	RAMACHANDRA	40	M	FARMER	13772	4/23/2018	4/28/2018	P	A	A	A	A	A	A	A	A	P	56	110/70	14	37.2	12.7	24770	5	50	-	0.8	128	4.2	P	113	ELEVATION IILIAVAF	NILL	INFERIOR	45%	11MM	-	NO	NO	NO	NO	I
67	SHIVANGOUDA	64	M	FARMER	11901	4/8/2018	4/11/2018	P	A	A	A	A	A	A	A	A	P	114	110/70	16	37	12.3	10850	80	19	20	0.6	129	2.9	P	45	ELEVATION V1-V4	NILL	ANTERIOR	50%	8.8MM	841pg	YES	NO	NO	NO	I
68	RAYAPPA	52	M	FARMER	14773	5/2/2018	5/8/2018	A	A	P	A	A	P	P	A	A	P	110	80/60	22	37	11.6	20970	10	18	12	0.6	136	3.2	N	33	ELEVATION IILIAVAF	NILL	INFERIOR	60%	9.2MM	1452pg	YES	NO	NO	NO	I
69	MALLAPPA	58	M	FARMER	16000	5/12/2018	5/18/2018	A	A	A	A	P	A	A	A	A	A	70	130/80	16	37.4	13.6	9640	35	44	20	0.8	135	4.5	N	64	ELEVATION V1-V4	NILL	ANTERIOR	30%	13MM	358pg	NO	NO	NO	NO	I
70	SHARANAPPA	32	M	SHOPKEEPER	16635	5/14/2018	5/17/2018	P	A	A	A	A	P	A	A	A	A	70	110/60	14	37.1	13.7	14700	20	12	20	0.7	140	3.9	P	78	ELEVATION IILIAVAF	NILL	INFERIOR	35%	14MM	-	NO	NO	NO	NO	I
71	MALLIKARJUNA	53	M	LABOURER	17016	5/20/2018	5/28/2018	A	A	A	A	P	A	P	A	A	A	108	130/80	12	38.3	12.9	9460	15	18	21	0.7	136	4	P	79	ELEVATION IILIAVAF	NILL	INFERIOR	40%	8.4MM	1197pg	YES	NO	YES	DEATH	I
72	YALLAPA	50	M	FARMER	17976	5/28/2018	6/1/2018	A	P	A	A	A	P	P	A	A	A	82	112/80	11	37.2	13.8	14398	5	18	27	0.9	131	4.4	P	74	ELEVATION V1-V4	NILL	ANTERIOR	60%	13.2MM	882pg	NO	NO	NO	NO	I
72	G M ROJARI	50	M	FARMER	18776	6/3/2018	6/7/2018	P	A	A	A	A	A	A	A	A	P	78	110/70	14	37	15.6	5530	10	89	31	0.8	137	4.4	P	28	ELEVATION V1-V4	NILL	ANTERIOR	40%	12.4MM	-	NO	NO	NO	NO	I
74	HANAMANTH	72	M	LABOURER	18795	6/3/2018	6/9/2018	P	A	A	A	A	A	A	A	A	A	56	110/70	14	37.2	12.7	24770	5	50	-	0.8	128	4.2	P	113	ELEVATION IILIAVAF	NILL	INFERIOR	50%	9MM	5436pg	NO	YES	YES	NO	I
75	RAJASAEV	45	M	FARMER	18986	6/5/2018	6/5/2018	P	A	A	A	A	A	A	A	A	A	114	110/70	16	37	12.3	10850	80	19	20	0.6	129	2.9	P	45	ELEVATION V1-V4	NILL	ANTERIOR	45%	8.8MM	2560pg	YES	NO	NO	DEATH	I
76	MAHADEVI	60	F	HOUSEWIFE	19040	6/5/2018	6/11/2018	A	A	A	A	A	A	A	A	A	A	110	80/60	22	37	11.6	20970	10	18	12	0.6	136	3.2	N	33	ELEVATION IILIAVAF	NILL	INFERIOR	50%	9.2MM	552pg	NO	NO	YES	NO	I
77	MALLIKARJUNA	68	M	BUSINESS	19415	6/8/2018	6/13/2018	P	A	A	A	A	A	A	A	A	A	56	110/70	14	37.2	11.6	20970	80	19	20	0.8	137	4.4	P	28	ELEVATION V1-V4	NILL	ANTERIOR	40%	8.6MM	5436pg	NO	YES	NO	NO	I
78	GANGADHAR	78	M	FARMER	19674	6/10/2018	6/11/2018	P	A	A	A	A	P	P	A	A	A	114	110/70	16	37	13.6	9640	10	18	12	0.8	128	4.2	P	113	ELEVATION IILIAVAF	NILL	INFERIOR	50%	9MM	882pg	YES	NO	NO	DEATH	I
79	SAHEBGOUDA	62	M	FARMER	19845	6/12/2018	6/14/2018	A	A	P	A	A	A	A	A	A	A	110	80/60	22	37	13.7	14700	35	44	20	0.6	129	2.9	P	45	ELEVATION V1-V4	NILL	ANTERIOR	45%	10MM	-	NO	NO	NO	NO	I
80	MAHADEVI	65	F	HOUSEWIFE	21669	6/27/2018	7/9/2018	P	A	A	A	P	A	A	A	A	A	60	90/60	14	37.4	12.9	9460	20	12	20	0.6	136	3.2	N	33	ELEVATION V1-V4	NILL	ANTERIOR	40%	11MM	-	NO	NO	NO	NO	I
81	RUKAMAWWA	85	F	HOUSEWIFE	22858	7/6/2018	7/14/2018	P	A	A	A	A	P	A	A	A	A	80	120/80	14	37.1	13.8	14398	15	18	21	0.8	135	4.5	N	64	ELEVATION IILIAVAF	NILL	INFERIOR	60%	13.4MM	870pg	NO	NO	NO	NO	I
82	NAGRAJ	22	M	SHOPKEEPER	23519	7/11/2018	7/17/2018	A	A	A	A	P	A	P	A	A	A	98	140/80	14	37	15.6	5530	5	50	-	0.7	140	3.9	P	78	ELEVATION IILIAVAF	NILL	INFERIOR	30%	8.6MM	552pg	YES	NO	NO	NO	I
83	BHMGOUDA	47	M	TRUK DRIVER	24054	7/16/2018	7/19/2018	A	P	A	A	A	P	P	A	A	A	80	1224/80	14	37.1	19.8	18270	80	19	20	0.7	136	4	P	79	ELEVATION V2-5	NILL	ANTERIOR	35%	9MM	4568pg	NO	YES	YES	NO	I
84	SUTAN	50	M	FARMER	24130	7/17/2018	7/22/2018	P	A	A	A	A	A	A	A	A	P	80	120/70	16	37.2	12.3	10850	10	18	12	0.9	131	4.4	P	74	ELEVATION IILIAVAF	NILL	INFERIOR	40%	8.8MM	258pg	NO	YES	YES	NO	I
85	CHIDANAND	65	M	FARMER	24481	7/20/2018	7/26/2018	P	A	A	A	A	A	A	A	A	A	80	140/90	16	37	11.6	20970	80	19	20	0.8	137	4.4	P	28	ELEVATION V2-5	NILL	ANTERIOR	60%	14MM	568pg	NO	NO	NO	NO	I
86	TUKARAM	65	M	BUSINESS	24676	7/21/2018	7/24/2018	P	A	A	A	A	A	A	A	A	A	40	90/60	14	37.1	13.6	9640	10	18	12	0.8	128	4.2	P	113	ELEVATION V1-V4	NILL	ANTERIOR	40%	7.9MM	654pg	YES	YES	NO	DEATH	I
87	TULJABAI	60	F	HOUSEWIFE	26565	8/6/2018	8/10/2018	A	A	A	A	A	A	A	A	A	A	80	140/80	15	37	13.7	14700	35	44	20	1.5	134	5.6	P	53	ELEVATION IILIAVAF	NILL	INFERIOR	50%	9.2MM	2245pg	YES	NO	NO	NO	I
88	RUDRAGOUDA	47	M	FARMER	28835	8/24/2018	8/29/2018	P	A	A	A	A	A	A	A	A	A	100	170/90	14	37.2	12.9	9460	20	12	20	0.8	138	4.7	P	50	ELEVATION V2-5	NILL	ANTERIOR	45%	13MM	-	NO	NO	NO	NO	I
89	UMESHGOUDA	51	M	FARMER	29188	8/24/2018	8/29/2018	P	A	A	A	A	P	P	A	A	A	80	130/80	14	37	11	11810	15	18	21	7.4	136	4.5	P	22	ELEVATION V2-5	NILL	ANTERIOR	40%	11MM	-	NO	NO	NO	NO	I
90	SHIVSHANKEPPA	70	M	FARMER	29189	8/27/2018	9/2/2018	A	A	P	A	A	A	A	A	A	A	70	120/70	16	37.1	7.4	12650	5	50	-	3.1	127	5.3	P	109	ELEVATION IILIAVAF	NILL	INFERIOR	60%	8.9MM	660pg	YES	NO	NO	NO	I
91	LAXMAN	45	M	FARMER	29276	8/28/2018	8/30/2018	A	A	A	A	P	A	P	A	A	A	86	90/50	26	37	11.8	11400	80	19	20	0.9	131	4.4	P	74	ELEVATION IILIAVAF	NILL	INFERIOR	40%	9MM	8987pg	NO	NO	YES	NO	I
92	SUBAS	70	M	FARMER	29454	8/29/2018	9/3/2018	A	P	A	A	A	P	P	A	A	A	88	130/80	14	37	10.1	1000	10	18	12	0.8	137	4.4	P	28	ELEVATION IILIAVAF	NILL	INFERIOR	50%	9.5MM	664pg	NO	YES	NO	NO	I
93	SHANKAR	80	M	FARMER	29790	8/31/2018	9/7/2018	A	P	A	A	A	A	A	A	P	A	60	130/100	20	37.1	12.3	17710	20	15	21	0.8	128	4.2	P	113	ELEVATION V1-V4	NILL	ANTERIOR	45%	14MM	3452pg	NO	NO	NO	NO	I
94	AMBADAS	63	M	FARMER	29923	9/2/2018	9/3/2018	P	A	A	A	A	A	A	A	P	P	90	140/90	16	36.8	11.6	20970	60	12	22	1.5	134	5.6	P	53	ELEVATION IILIAVAF	NILL	INFERIOR	40%	9.2MM	6745pg	YES	YES	NO	DEATH	I
95	PUNDALIK	56	M	FARMER	29958	9/2/2018	9/7/2018	P	A	A	A	A	A	A	A	A	A	84	130/80	16	37	13.6	9640	40	12	22	0.8	138	4.7	P	50	ELEVATION V2&L AVL	NILL	ANTERIOR	60%	12.1MM	-	NO	NO	NO	NO	I
96	SAGAR	24	M	BUSINESS	29996	9/3/2018	9/7/2018	P	A	A	A	A	A	A	A	A	A	130	150/110	16	37	13.7	14700	90	33	30	1.5	134	5.6	P	53	ELEVATION V1-V4	NILL	ANTERIOR	40%	11MM	675pg	NO	NO	NO	NO	I
97	BABU	60	M	BUSINESS	30058	9/3/2018	9/8/2018	A	A	A	A	A	A	A	A	A	A	100	170/90	14	37.2	12.9	9460	20	16	27	0.8	138	4.7	P	50	ELEVATION IILIAVAF	NILL	INFERIOR	50%	8MM	1420pg	YES	YES	A	DEATH	I
98	KHUBA	68	M	FARMER	30315	9/5/2018	9/9/2018	P	A	A	A	A	A	A	A	A	A	80	130/80	14	37	13.8	14398	10	16	20	7.4	136	4.5	P	22	ELEVATION V2-5	NILL	ANTERIOR	45%	11MM	3556pg	NO	NO	NO	NO	I
99	MALLINATH	65	M	FARMER	31569	9/14/2018	9/19/2018	P	A	A	A	A	P	P	A	A	A	70	120/70	16	37.1	15.6	5530	5	33	36	3.1	127	5.3	P	109	ELEVATION V1-V4	NILL	ANTERIOR	50%	12MM	7700pg	NO	NO	NO	NO	I
100	TULSIRAM	42	M	FARMER	31741	9/17/2018	9/18/2018	A	A	P	A	A	A	A	A	A	A	86	90/50	26	37	19.8	18270	15	18	21	0.9	131	4.4	P	74	ELEVATION IILIAVAF	NILL	INFERIOR	40%	7.8MM	1256pg	YES	YES	YES	DEATH	I
101	MALLAPPA	82	M	FARMER	32297	9/20/2018	9/26/2018	P	A	A	A	P	A	A	A	A	A	88	130/80	14	37	12.3	10850	5	50	-	0.8	137	4.4	P	28	ELEVATION V2&L AVL	NILL	ANTERIOR	50%	14MM	-	NO	NO	NO	NO	I
102	MD RAFFEQ	45	M	LABOURER	32671	9/23/2018	9/26/2018	P	A	A	A	A	P	A	A	A	A	60	130/100	20	37.1	11.6	20970	80	19	20	7.4	136	4.5	P	53	ELEVATION IILIAVAF	NILL	INFERIOR	45%	12MM	-	NO	NO	NO	NO	I