"SIGNIFICANCE OF CORONARY ARTERY CALCIUM SCORING IN PATIENTS WITH OBSTRUCTIVE AND NONOBSTRUCTIVE CORONARY ARTERY DISEASE FOLLOWING STEMI"

Dissertation submitted for

D.M. DEGREE EXAMINATION

BRANCH II – CARDIOLOGY

MADRAS MEDICAL COLLEGE

AND

GOVERNMENT GENERAL HOSPITAL

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CHENNAI – 600 032

AUGUST 2010

"Learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled **"SIGNIFICANCE OF CORONARY ARTERY CALCIUM SCORING IN PATIENTS WITH OBSTRUCTIVE AND NONOBSTRUCTIVE CORONARY ARTERY DISEASE FOLLOWING STEMI"** is the bonafide original work of **DR.P.PACHAIYAPPAN** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from July 2007 to July 2010.

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DECLARATION

I **Dr.P.PACHAIYAPPAN,** solemnly declare that this dissertation entitled, **"SIGNIFICANCE OF CORONARY ARTERY CALCIUM SCORING IN PATIENTS WITH OBSTRUCTIVE AND NONOBSTRUCTIVE CORONARY ARTERY DISEASE FOLLOWING STEMI"** is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2009 – 2010 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor **Geetha Subramanian M.D.D.M.** This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai

Date: 31.05.10

Dr. P.PACHAIYAPPAN

ACKNOWLEDGEMENTS

A great many people made this work possible. I thank my Dean for allowing me to conduct this study.

My warmest respects and sincere gratitude to our beloved **Prof.Geetha Subramanian** Professor and Head of the Department of Cardiology, Government General Hospital, Chennai. But for his constant guidance this study would not have been possible.

My respectful thanks to **Prof.R.Subraamanian** (Retd), **Prof.M.Prabhakaran** for his constructive ideas, personal guidance and involvement in this study.

I am indebted to **Prof. Geetha Subramanian, Prof. B.Ramamurthy, Prof. M.Somasundaram, Prof. P.Arunachalam and Prof. V.E.Dhandapani** without whom, much of this work would not have been possible.

I acknowledge **Dr M.A.Rajasekar** for the many useful comments he made during this project.

In addition, I am grateful to**, Dr.G.Gnanavelu, Dr.S.Venkatesan, Dr.G.Ravishankar, Dr.JustinPaul, Dr.G.Prathapkumar Dr.C.Elangovan, Dr.V.Ravi, Dr.S.Raghothaman, Dr.Rajasekar ramesh, Dr.K.Sabapathy** for tracing all those waveforms and guidance.

Last but not the least I thank all my patients for their kind cooperation.

CONTENTS

INTRODUCTION

Coronary artery calcification (CAC) occurs in small amounts in the early lesions of atherosclerosis that appear in the second and third decades of life, but it is found more frequently in advanced lesions and in older age.

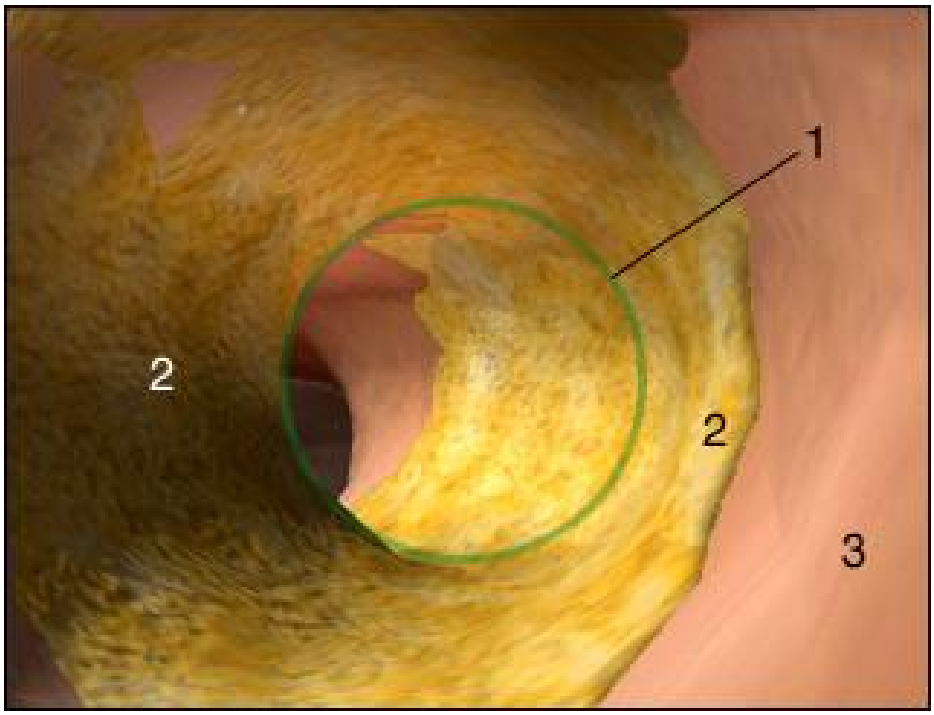
Coronary arterial calcification is part of the development of atherosclerosis, occurs almost exclusively in atherosclerotic arteries, and is absent in the normal vessel wall

A positive CT study (defined as presence of any CAC) is nearly 100% specific for atheromatous coronary plaque. Since both obstructive and non-obstructive lesions can have calcification present in the intima, CAC is not specific for obstructive coronary disease.

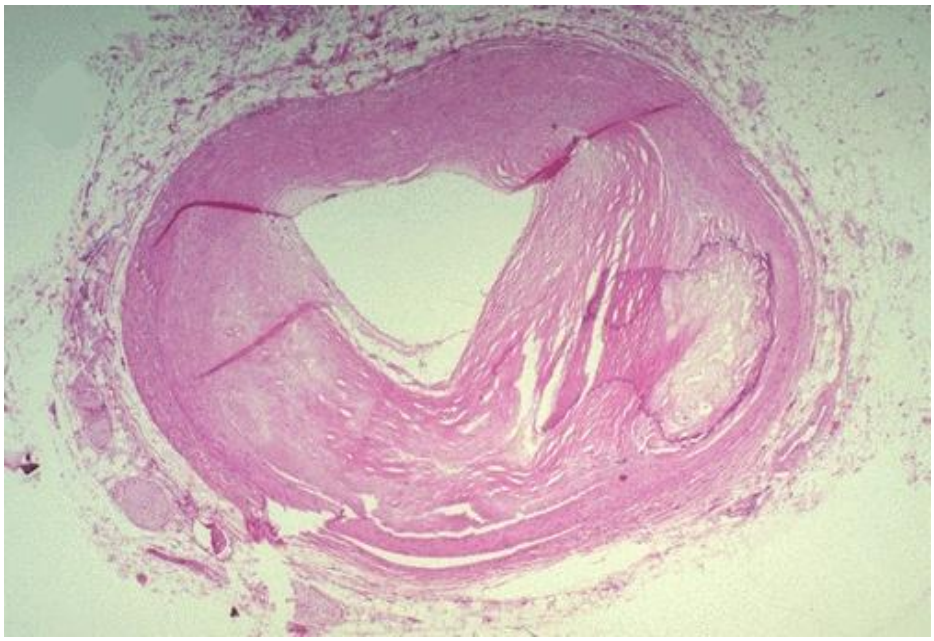
The site and the amount of coronary artery calcium and the percent of coronary luminal narrowing at the same anatomic site, the relation is nonlinear and has large confidence limits. As the occurrence of calcification reflects an advanced stage of plaque development, some researchers have proposed that the correlation between coronary calcification and acute coronary events may be suboptimal based largely on angiographic series[5].

In order to understand this apparent conflict between the stability of a calcified lesion and CHD event rates, one must recognize the association

Atherosclerotic Hardening of the Artery showing Plaque with Calcification



1) Intima 2) Media 3) Adventitia



between atherosclerotic plaque extent and more frequent calcified and non-calcified plaque[6.]

That is, patients who have calcified plaque are also more likely to have non-calcified or "soft" plaque that is prone to rupture and acute coronary thrombosis [6.]

PATHOPHYSIOLOGY OF CORONARY CALCIFICATION

Calcium is a well known component of the atherosclerotic plaque calcification of atherosclerotic plaque occurs by means of an active process resembling the bone formation under the control of complex cellular pathways .a large number of invitro studies have highlighted the importance of calcium in the process of vascular calcification of osteoblast like cells, cytokines, transcription factors, and bone morphogenic proteins found in the normal bone.

Calcification of the intima is characterized by cellular apoptosis, inflammation, lipoprotein, phospholipid accumulation, and finally hydoxyapatite deposition. Calcification is first noticed in the lipid core of the atheroma juxtaposed to the inflammatory cells that infiltrate the fibrocalcific plaque.

The basic mechanism initiating the process of calcification is unknown, but it seems to require apoptosis of intralesional cells, likely the smooth muscle cells. The apoptotic bodies would then work as nucleating foci of calcification.

a. The CAC score is age and gender specific and therefore there has to be a comparison of the individual data to a normal cohort in order to produce meaningful data, usually presented as the percentile distribution. In general, CAC develops 10 to 15years later in life in women than in men. Similarly CAC is generally 5 to 7 times lower at any given age in women than in men.

b. In patients at intermediate clinical risk for coronary events the CAC score can help to reclassify patients to a higher or lower risk group. For instance a CAC score of zero confirms low risk of events. Conversely a CAC score of greater than 400 is observed with a significant cardiac event rate in patients who appear to be intermediate risk by Framingham score.

c. Because statins have no documented effect on CAC progression, there is no value in repeating CAC in persons with a score of greater than 100 or the 75 percentile.

d. More common in men, diabetics and renal failure pts. The role of CAC scoring in determining risk in patients with CKD and/or ESRD is unclear due to a limited number of clinical studies in these populations.

Some studies suggest that patients with CKD and ESRD develop calcification in the tunica media layer of the arterial wall, unlike the typical intimal calcification that is known to be associated with plaque burden. The role of medial calcification as a marker of cardiovascular risk is not well defined. Some studies reveal an association

between coronary calcium and prevalent cardiovascular disease in patients undergoing dialysis and coronary calcium score is associated with risk for total mortality[19] .

Principles of Computed Tomography:

Computed tomography (CT) imaging was introduced in 1972. The ability to obtain cross sectional images of the body revolutionized medicine and, for the development of computer assisted tomography, Sir Geoffrey N. Hounsfield and Allan M. Cormack were awarded the Nobel Prize in Medicine in 1979.

CT is an x-ray–based technique. An x-ray source that rotates on a circular path around the patient emits a fan-shaped beam of x-rays that passes through the body. Collimators are used to confine the x-ray beam to the slice that shall be imaged; its thickness can vary from less than one to several millimeters. Opposite the x-ray source, extremely sensitive detectors record the intensity of x-rays that have passed through the body. Based on the x-ray attenuations obtained from a multitude of angles, a cross-sectional image of the body can be calculated.

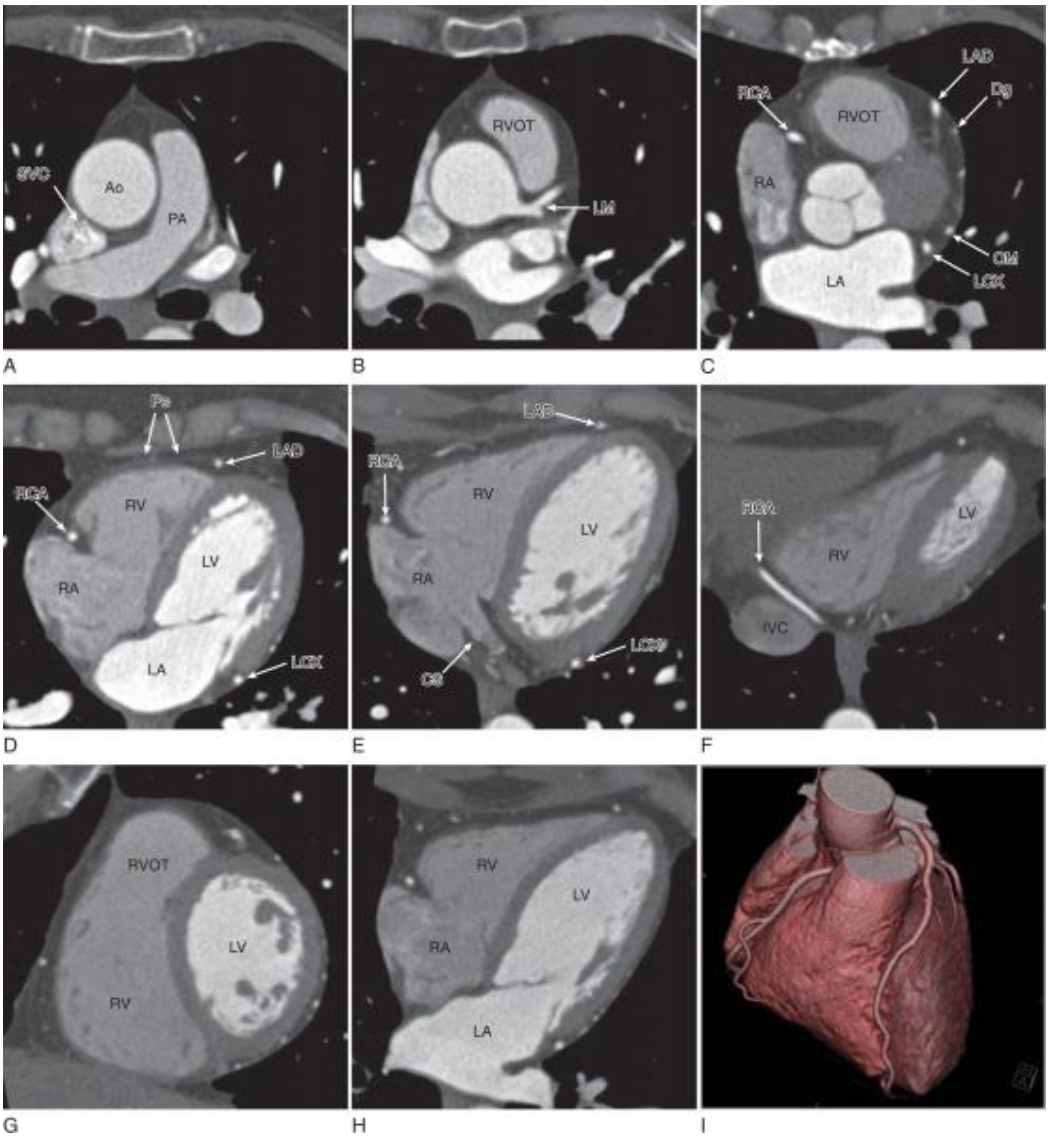


Figure 1-1 showing normal cardiac anatomy as depicted by contrast-enhanced multidetector computed tomography

A, Level of the ascending aorta (Ao) andpulmonary artery, usually the topmost level of a cardiac CT image data set.

B, Level of the left main coronary artery(LM), which can be seen originating from the aortic root and dividing into the left anterior descending coronary arteryand left circumflex coronary artery (arrow).

C, Level of the proximal right coronary artery (RCA).

D, Midventricularlevel.

E, Level of the caudal right atrium. The drainage of the coronary sinus into the right atrium (RA) can be seen.

F, Level of the distal right coronary artery.

G, Multiplanar reconstruction to create a short axis view.

H, Multiplanarreconstruction to create a four-chamber view.

I, Three-dimensional surface reconstruction, shown from an anterior view. The coronary arteries can be recognized on the surface of the heart.

CS = coronary sinus; Dg = diagonal branch; IVC = inferior vena cava; LA = left atrium; LAA = left atrial appendage; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; LV = left ventricle, OM = obtuse marginal branch; Pc =pericardium; PA = pulmonary artery; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract; SVC = superior vena cava.

Analysis of Coronary Artery Calcium

Electron-beam computed tomography (EBCT) and multi-detector computed tomography (MDCT) are the primary fast CT methods for CAC measurement at this time. Both technologies employ thin slice CT imaging, using fast scan speeds to reduce motion artifact.

Thirty to 40 adjacent axial scans usually are obtained. A calcium scoring system has been devised based on the X-ray attenuation coefficient or CT number measured in Hounsfield units and the area of calcium deposits. A fast CT study for coronary artery calcium measurement is completed within 10 to 15 min, requiring only a few seconds of scanning time.

Cardiac computed tomography has been used with increasing frequency in the United States and other countries during the past 15 years, initially with the goal of identifying patients at risk of having obstructive coronary artery disease based on the amount of coronary calcium present.

However, in the past 5 to 10 years, fast CT methods have been used primarily for 2 purposes: 1) To assist in coronary heart disease (CHD) risk assessment in asymptomatic patients, and 2) To assess the likelihood of the presence of CHD in patients who present with atypical symptoms which could be consistent with myocardial ischemia.

Table 1 -1 showing significance of age and CAC score.

AGE IN YEARS	CAC SCORE	SENSITIVITY (%)	SPECIFICITY (%)	NEGATIVE PREDICTIVE VALUE FOR ZERO SCORE(%)
40 to 49	50	71	91	98
50 to 59	50	74	70	94
50 to 59	300	74	81	100

Agatston Coronary Artery Calcium Scoring system:

The agatston coronary calcium volume score is the most frequently used scoring system. It is derived by measuring the area of each calcified coronary lesion and multiplying it by a coefficient of 1 to 4, depending on the maximum CT attenuation within that lesion. It is important to realize the reproducibility of the agatston score before applying the recommended guidelines for cut points. Importantly the variability in score has very little meaning at the very high and very low scores. Inter-reader variability can be as high as 3%[17].

a. The CAC score can be classified in to five groups.

1) 0 - No coronary calcification

2) upto 100 - Mild coronary calcification

3) 100 to 399 - Moderate calcification

4) 400 to 999 - Severe calcification

5) >1000 - Extensive calcification.

Detection of Coronary Artery Calcification

The standard EBCT imaging protocol is to acquire 40 consecutive 3-mm-thick images at a rate of 100 ms per image from the base of the heart to just below the carina. Images are obtained at end-inspiration, with ECG triggering typically at 80 percent of the R-R interval (end-diastole). Image pixel size using a 512 x 512 reconstruction matrix is 0.26 or 0.34 mm^2 based on a 26- or 30-cm field of vision, respectively.

A calcified lesion is generally defined as either two or three adjacent pixels (0.68 to 1.02 mm^2 for a 512^2 reconstruction matrix and camera field size of 30 cm) of >130 Hounsfield units (HUs). Using the traditional Agatston method, each calcified lesion is multiplied by a density factor as follows: 1 for lesions with a maximal density between 130 and 199 HU; 2 for lesions between 200 and 299 HU; 3 for lesions between 300 and 399 HU; and 4 for lesions >400 HU.

The total coronary artery calcium score (CACS) is calculated as the sum of each calcified lesion in the four main coronary arteries over all the consecutive tomographic slices. The EBCT-derived CACS correlates well with calcified areas

found in individual coronary arteries as determined by histomorphometric measurements ($r = 0.96$, $p < 0.0001$)[16].

MULTIROW DETECTOR COMPUTED TOMOGRAPHY:

Advancements in CT technology have improved image acquisition speed and patient throughput. Multidetector computed tomography (MDCT) scanners produce images by rotating an x-ray tube around a circular gantry through which the patient advances on a moving couch. Increased numbers of detectors have allowed much faster throughput, essentially reducing the time to image the entire cardiac anatomy to <10 seconds.

The introduction of multirow spiral CT detector systems (i.e., multislice CT) currently allow acquisition of 4 to 64 simultaneous images, with slice thickness reduced to 0.5 to 0.625 mm. Improvements in gantry rotation speeds and the development of partial reconstruction algorithms have reduced effective single-image acquisition time to <200 msec.

However, image acquisition within 50 ms is required to completely avoid cardiac motion artifacts. The coronary arteries also move independently throughout the cardiac cycle and even at slow heart rates (i.e., <70 beats/min) exhibit significant translational motion of up to 60 mm/sec for the right coronary artery (RCA) and 20 to 40 mm/sec for the left anterior descending (LAD) and circumflex coronary arteries[44].

Retrospective gating with MDCT employs acquisition of multiple images throughout each cardiac cycle. With multirow detector CT systems, temporal resolution may be further improved by selecting specific partial image sector data from different heartbeats and detector rings to reconstruct a complete 240-degree image data set. With retrospective gating, several hundred images can be acquired during a single cardiac study, allowing one to *pick and choose* images with the least amount of motion-related distortion prior to final image reconstruction. However, this oversampling leads to significant excess radiation exposure to the patient.

The typical radiation exposure from an electron-beam computed tomography (EBCT) study is <1.0 rad, whereas MDCT scanners using retrospective gating can increase exposure approximately 13-fold.[44]

Prospective gating during either spiral or nonspiral acquisitions employs image triggering only at a specific temporal location of the cardiac cycle, thereby significantly reducing radiation exposure. Gating works relatively well at slow heart rates (i.e., <60 beats/min), where the R-R interval is >1000 ms and the fastest imaging protocols are used. However, at faster heart rates, a 200-msec acquisition effectively covers most of the cardiac cycle, thus obviating any potential benefit from gating the image acquisition

MDCT imaging protocols vary among different camera systems and manufacturers. Generally 40 consecutive 2.5- to 3-mm-thick images are acquired per cardiac study. Calcified lesions are defined as two or three adjacent pixels with a

tomographic density of either >90 or >130 HU. Effective pixel size for a reconstruction matrix of 512 x 512 pixels with a common field of view of 26 cm is 0.26 mm^2. Calcium scoring is usually based on the traditional Agatston method (i.e., initial density of >130 HU). As with EBCT scoring, the total CACS is calculated as the sum of each calcified plaque over all the tomographic slices.

Multirow Detector Computed Tomography compared to Electron Beam Computed Tomography:

The comparability of MDCT- and EBCT-derived coronary artery calcium scores has been explored in separate studies involving approximately 400 patients.[17–19] The MDCT protocols vary considerably in these studies, ranging from conventional CT to single-slice CT (with either retrospective or prospective gating) to multislice CT .

EBCT imaging was performed using the standard protocol conventionally used in routine clinical practice. Coronary calcification was defined as >130 HU for EBCT but varied from 90 to 130 HU for MDCT. Although high correlation coefficients were reported between EBCT and MDCT CACS, there was significant variability in individual CACS results (range 17 to 84 percent).

Table 1-2 EBCT versus Mechanical CT[a]

Author	Year	Number of Patients	Age	Average Ca^{2+} Score	Mechanical CT Technique	Gating	Number of Detectors	Correlation Coefficient	Mean % Difference
Becker[20]	1999	50	61	983	Nonspiral	No	Single	0.98	42%
Budoff[17]	2001	33	54	52	Nonspiral	No	Single	0.68	84%
Knez[19b]	2002	99	60	722	Spiral	Prosp	4	0.99	17%

Ca^{2+}, calcium; CT, computed tomography; EBCT, electron-beam computed tomography; prosp, prospective; retrosp, retrospective.

[a]Agatston score except as indicated. [b]Volumetric score.

A more recent study by Knez and coworkers compared MDCT to EBCT using prospective ECG gating for both techniques.[19] The CACS was calculated using the volumetric (rather than the Agatston) calcium scoring method.

Variability in CACS between the two techniques ranged from 20 percent (CACS <100) to 15 percent (CACS >100), with a mean variability of 17 percent. Further research is still needed to determine which MDCT technique, imaging protocol, calcium criterion, and scoring system best approximates the values determined by EBCT, especially with the new 64-detector systems. No calcium data is yet available from these state of the art scanners.

Coronary Artery Calcification and Atherosclerotic Plaque burden:

The presence of CAC is clearly indicative of coronary atherosclerosis.[25,26] Furthermore, the CACS severity, as assessed by EBCT, is directly related to the total atherosclerotic plaque burden present in the epicardial coronary arteries.[25,26] Coronary calcification is thought to begin early in life, but it progresses more rapidly in older individuals who have further advanced atherosclerotic lesions.[27]

Calcification is an active, organized, and regulated process occurring during atherosclerotic plaque development where calcium phosphate in the form of hydroxyapatite precipitates in atherosclerotic coronary arteries in a similar fashion as observed in bone mineralization.[28–30] Although lack of calcification does not categorically exclude the presence of atherosclerotic plaque, calcification occurs exclusively in atherosclerotic arteries and is not found in normal coronary arteries.

The presence and extent of histologically determined plaque area has been compared to the total calcium area as assessed by EBCT in individual coronary arteries derived from autopsied hearts.[25] A strong linear correlation exists between total coronary artery plaque area and the extent of CAC as found in individual hearts ($r = 0.93$, $p < 0.001$) and in individual coronary arteries ($r = 0.90$, $p < 0.001$). However, the total calcium area underestimates total plaque area, with approximately five times as many noncalcified as calcified plaques.[25]

Table 1-3 showing accuracy of EBCT Coronary Artery Calcification in Detecting Significant (>50%) Coronary Artery Stenosis as Defined by Angiography

Investigator	Year	Number of Subjects	Sensitivity (%)	Specificity (%)	Positive Predictive Accuracy	Negative Predictive Accuracy
Agatston[17]	1990	584	96	51	31	98
Budoff[43]	1996	710	95	44	72	84
Detrano[1]	1996	491	95	31	51	89
Baumgart[36]	1997	57	97	21	56	86
Schmermund[3]	1997	118	95	88	99	58

EBCT, electron-beam computed tomography.

Coronary Artery Calcification and Stenosis severity:

Significant (>50 percent) coronary artery stenosis by angiography is almost universally associated with the presence of coronary artery calcium as assessed by EBCT. However, the severity of angiographic coronary artery stenosis is not directly related to the total CACS. A recent study compared calcium extent to coronary artery luminal diameter stenosis determined by morphologic examination of 723 coronary artery segments.[26]

Although coronary stenosis severity increased with increasing CAC, this relationship was poor and could not be used to estimate angiographic stenosis severity on a segment-by-segment basis. One explanation is that coronary artery remodeling occurs with increasing plaque burden so as to maintain luminal diameter and arterial patency.[31] Although the extent of coronary calcification does not precisely predict stenosis severity, noncalcified plaques are almost universally associated with <50 percent diameter stenosis and typically <20 percent stenosis.[26] These data indicate that lack of coronary calcification predicts a very low likelihood of obstructive CAD.

Clinical angiographic trials confirm the relationship between CACS severity and the presence of significant (50 percent) CAD.[32] Although the diagnostic accuracy of EBCT improves with age, most patients younger than 50 years with obstructive CAD also have coronary calcification (85 percent).[33,] To date there are 15 studies evaluating EBCT with coronary angiography in which obstructive CAD was defined as >50 percent luminal diameter stenosis[32].

In these studies, the overall sensitivity and specificity for detecting obstructive CAD were 97 and 39 percent, respectively. In the largest series, Haberl and colleagues performed EBCT within 30 days of coronary angiography in 1764 patients who had suspected CAD.[41] Only 5 of 940 patients (0.5 percent) with significant (50 percent) coronary artery stenosis had a normal EBCT, and four of these were younger than 45 years of age. Although differences in CACS were noted among men and women, EBCT predicted CAD equally well in both genders, based on

age-specific CACS thresholds.[41] Coronary artery calcification (CAC) assessment may also be useful for detecting CAD in heart transplant recipients.[4]

The poor specificity of coronary calcium scanning can be reconciled by the fact that the coronary calcification confirms the presence of atherosclerotic plaque but it may not necessarily be obstructive.. The CACS severity may be a better barometer of obstructive CAD than the mere presence of calcium.

Budoff and coworkers observed that specificity increased with the number of calcified coronary arteries (i.e., high calcium scores).[33] Two separate reports in patients referred for coronary angiography found that a CACS >100 best predicted obstructive CAD with an equally high sensitivity and specificity of 80 percent.

There appears to be a threshold CACS above which most patients will have significant coronary artery stenosis. The accuracy for identifying significant CAD based on CACS may be further improved by incorporating age, gender,[39] and traditional risk-factor information. However, despite the relationship between obstructive CAD and CACS severity, the latter is still too imprecise in itself to be used as a definitive criterion for proceeding directly to coronary angiography.

The current American College of Cardiology/American Heart Association (ACC/AHA) guidelines on coronary angiography do not recommend coronary angiography on the basis of a positive EBCT but do suggest angiography may be avoided with the finding of a negative (zero score) study

Prognosis by Coronary Artery Calcium Measurements:

In the prior ACC/AHA expert consensus document published in 2000, only 3 reports on the prognostic capability of CAC scoring were available to develop risk assessment indications in asymptomatic individuals. At the time, the ACC/AHA document concluded that the body of evidence using CAC measurement to predict CHD events was insufficient[7].

A critical component to that recommendation was that the independent prognostic value of CAC had not been established. In a separate but similar evaluation using data published through 2002, the U.S. Preventive Services Task Force (USPSTF) concluded that limited clinical outcomes data were available and recommended against routine screening for the detection of silent but severe CAD or for the prediction of CHD events in low risk, asymptomatic adults.

In the past several years, however, a number of publications have reported on the incremental prognostic value of CAC in large series of patients including asymptomatic self-referred and population cohort.

A major rationale for the current document is the need for an update including recent publications regarding CAC as it relates to the estimation of CHD death or nonfatal myocardial infarction (MI).

Although earlier evidence included the use of "soft" endpoints including coronary revascularization as a primary outcome, more recent data are available on the

estimation of CHD death or MI. Models predicting "hard" cardiac events (i.e., CHD death or MI) are less subjective and less likely to overestimate the predictive accuracy of CAC scoring.

Other Uses:

1. To differentiate between ischemic and non ischemic cardiomyopathy. One large study in 120 patients with heart failure of unknown etiology demonstrated the presence of CAC was associated with 99% sensitivity for ischemic cardiomyopathy. Another study also demonstrated similarly high sensitivity using fast CT to differentiate ischemic from non-ischemic cardiomyopathy [11]

2. To triage chest pain patients in Emergency Department.

3. Presence of any calcium – There is a fourfold risk of coronary events in the next 3 to 5 years.

4. To reclassify intermediate risk group to either low or high risk group based on Framingham risk score. The accumulating evidence suggests that asymptomatic individuals with an intermediate FRS may be reasonable candidates for CHD testing using CAC as a potential means of modifying risk prediction and altering therapy. On the other hand, there is little to be gained

by testing with CAC in patients with a low FRS. Furthermore, patients with a high FRS should be treated aggressively consistent with secondary prevention goals based upon the current NCEP III guidelines and thus should not require additional testing, including CAC scoring, to establish this risk evaluation [13].

5. Statins has no effect on CAC progression if score is more than 100.

AIM OF THE STUDY

- To compare CAC (coronary artery calcium) score in patients with Obstructive and Non obstructive CAD.
- To compare CAC score in patients with single and multivessel disease.
- To compare CAC score in males and females.
- To compare CAC score in those with and without HT, Smoking and Diabetes.
- To compare CAC score between IRA and other vessels in multivessel disease.

REVIEW OF LITERATURE

Theoretical relationship between Coronary Calcification and CHD events:

There are conflicting results regarding the site, extent of coronary artery calcification and the angiographic grading based on various available data. Atherosclerotic plaque proceeds through progressive stages where instability and rupture can be followed by calcification, perhaps to provide stability to an unstable lesion.

As the occurrence of calcification reflects an advanced stage of plaque development, some researchers have proposed that the correlation between coronary calcification and acute coronary events may be suboptimal based largely on angiographic series.

There is no known relationship between vulnerable plaque and coronary artery calcification[3]. The relation of arterial calcification, like that of angiographic coronary artery stenosis, to the probability of plaque rupture is unknown[4].

Although radiographically detected coronary artery calcium can provide an estimate of total coronary plaque burden, due to arterial remodeling, calcium does not concentrate exclusively at sites with severe coronary artery stenosis [5].

It is the co-occurrence of calcified and non-calcified plaque that provides the means for estimating acute coronary events. Furthermore, although CAC detection cannot localize a stenotic lesion or one that is prone to rupture, CAC scoring may be able to globally define a patient's CHD event risk by virtue of its strong association with total coronary atherosclerotic disease burden, as shown by correlation with pathologic lesions.

The Committee judged that it may be reasonable to consider use of CAC measurement in such patients based on available evidence that demonstrates incremental risk prediction information in this selected (intermediate risk) patient group. This conclusion is based on the possibility that such patients might be reclassified to a higher risk status based on high CAC score, and subsequent patient management may be modified[9].

In order to understand this apparent conflict between the stability of a calcified lesion and CHD event rates, one must recognize the association between atherosclerotic plaque extent and more frequent calcified and non-calcified plaque. That is, patients who have calcified plaque are also more likely to have non-calcified or "soft" plaque that is prone to rupture and acute coronary thrombosis.

A subset analysis of the predictive accuracy of CAC in patients with an intermediate FRS reveals that for a score greater than or equal to 400, the patient's 10-year CHD risk would achieve risk equivalent status similar to that noted with diabetes

or peripheral arterial disease . Thus, clinical decision-making could potentially be altered by CAC measurement in patients initially judged to be at intermediate risk (10% to 20% in 10 years)[12.]

Most unexpected cardiovascular events occur in persons at intermediate risk of coronary artery disease (10%–20% 10-year risk). The absence of CAC by cardiac CT is associated with a low adverse event risk and therefore could be used as a tool to counsel patients about their risk of such events[13].

Coronary artery scanning using electron beam computed tomography is a diagnostic tool with application to high-risk and symptomatic subjects that can assist in diagnosing or excluding coronary artery disease. Although there is ample evidence for the utility of this and related technologies for diagnosis in symptomatic subjects, this remains an unproven technology for screening healthy asymptomatic subjects[1]

Multiple logistic regression analysis determined male sex, presence of diabetes and left anterior descending (LAD) and circumflex (LCX) coronary calcium scores, independent from more distal calcium localization, as independent predictors for identification of three-vessel and/or left main CAD[2].

On the basis of a simple algorithm ("noninvasive index"), EBCT calcium scanning in conjunction with risk factor analysis can rule in or rule out angiographically severe disease, i.e., three-vessel and or left main CAD, in symptomatic patients[2].

On average, significant coronary disease (greater than 50% or greater than 70% stenosis by coronary angiography) was reported in 57.2% of the patients. Presence of CAC was reported on average in 65.8% of patients (defined as a score greater than 0 in all but one report)

Higher coronary calcium scores increased the likelihood of detecting significant coronary disease (greater than 50% or greater than 70% luminal stenosis). A threshold of detectable calcium or a score greater than 5 was associated with an odds of significant disease of 25.6-fold (95% CI 9.6 to 68.4)[1].

Because of its potential in this regard, further research should be encouraged to determine its place in the armamentarium of diagnostic tools. In contrast to its unproven utility for screening asymptomatic populations, electron beam computed tomographic coronary calcium has shown fairly accurate association with coronary angiographic findings in symptomatic patients referred for angiography for chest pain syndromes[1].

MESA STUDY - The Multi Ethnic Study of Atherosclerosis (6800 subjects) has reported that all modern Multi Detector Row CT systems are at least as reliable as EBCT for performing and reproducing coronary calcium measurements [1].

From the ST. FRANCIS HEART STUDY, measured risk factor data were available in 1293 of the total enrolled cohort of 4903 asymptomatic individuals. In univariable (p less than 0.0001) and multivariable ($p = 0.01$) models estimating CHD

events at 4.3 years of follow-up, CAC scores were independently predictive of CHD outcome above and beyond both historical and measured risk factors[37.]

RECALL STUDY- The Heinz-Nixdorf Risk factors Evaluation of Coronary Calcium, and Lifestyle study (4200 subjects) provides unbiased information on the extent of coronary calcium in the general German population from a suburban community[3.]

In this study again the coronary calcium score was superior to conventional risk factors for predicting coronary heart disease. This was true even for all four major racial and ethnic groups in the United States [1.]

Coronary atherosclerotic changes may appear calcified, noncalcified or mixed plaque lesion. Noncalcified lesions are found predominantly in patients who have AMI, whereas calcified lesions are found more often in patients who have chronic stable angina [14].

The CT density of noncalcified plaques is significantly lower in the culprit coronary segment of patients studied at the time of acute coronary syndromes as compared with those who have chronic stable disease[15]. In patients who have an acute coronary syndrome, a noncalcified lesion in the coronary artery may correspond to an intracoronary thrombus[16].

The current gold standard to detect coronary atherosclerosis in vivo is intravascular ultrasound (IVUS).studies comparing IVUS with multidetector row

CT (MDCT) have shown a good correlation between the echogenicity by IVUS and the CT density of coronary atherosclerotic lesions.

The sensitivity and specificity for CT to detect calcified and non calcified coronary atherosclerosis are 78% and 94%, respectively the sensitivity to comparison between CTA and IVUS is only 52%. However, probably because of the lower spatial resolution if CTA [18].

Coronary Artery Calcification: Prognostic Implications

The likelihood of plaque rupture and the development of acute cardiovascular events is related to the total atherosclerotic plaque burden.[27] Although controversy exists as to whether calcified or noncalcified plaques are more prone to rupture[28] extensive calcification indicates the presence of both plaque morphologies.[7]

There is a direct relationship between the CACS severity, the extent of atherosclerotic plaque, and the presence of silent myocardial ischemia. Many studies have now demonstrated an increased risk for cardiac events in asymptomatic patients who have extensive silent myocardial ischemia[29.] Therefore, the CACS could be useful for risk assessment of asymptomatic individuals and potentially guide therapeutics.

Several recent trials in both symptomatic[1] and asymptomatic[13] patients have studied whether the extent of CAC as assessed by EBCT can predict subsequent patient outcome. In 422 symptomatic patients followed for 30 ± 12 months[29] cardiac

events were 10-fold higher in patients with a CACS above the 75th percentile for age (9.5 percent) versus those below the 25th percentile (0.9 percent). Another study of 288 symptomatic patients referred for coronary angiography[30] showed that patients with a CACS >100 had a 3.2-fold higher relative risk of death or MI than those with a lower CACS (95 percent confidence limit: 1.17–8.71).

In the longest study of EBCT scanning of the coronary arteries, the SOUTH BAY HEART WATCH STUDY, 1196 asymptomatic patients were followed (median = 7.0 years) and it was demonstrated that the CACS score added predictive power beyond that of standard coronary risk factors and C reactive protein.[31]

Among 1173 asymptomatic patients followed for 3.6 years after an initial screening EBCT,[32] no events occurred in patients with a normal study and the negative predictive value was 99.8 percent in patients with a CACS <100. These results show a 5, 7, and 13 percent hard cardiac event rate in individuals with a CACS 80, 160, and 600, respectively.[32] The CACS remained the best single predictor of risk after adjustment. Wong and colleagues also showed that the CACS severity predicted subsequent events independent of age, gender, and patient risk-factor profile[33].

Table 3-1 Multivariate Analyses of the Association of Coronary Artery Calcium Scores and Self-Reported Traditional Coronary Disease Risk Factors with All Events[a]		
	Variable	**Odds Ratio (95% CI)**
Independent of EBCT	Elevated cholesterol	3.9 (1.3–11.7)
	Hypertension	2.8 (1.2–6.5)
	Diabetes	5.4 (2.0–14.9)
With EBCT CACS 80	CACS >80	14.3 (4.9–42.3)
	Age >55 y	3.3 (1.3–8.4)
	Elevated cholesterol	4.0 (1.3–12.2)
	Hypertension	2.6 (1.1–6.1)
	Diabetes	4.8 (1.6–13.9)
With EBCT CACS 160	CACS >160	19.7 (6.9–56.4)
	Age >55 y	4.5 (1.6–12.2)
	Elevated cholesterol	3.7 (1.2–11.5)
	Hypertension	3.0 (1.2–7.4)
	Diabetes	5.8 (2.1–19.7)
With EBCT CACS 600	CACS >600	20.2 (7.3–55.8)
	Age >55 y	2.9 (1.1–7.9)
	Elevated cholesterol	3.5 (1.1–10.8)
	Hypertension	2.9 (1.2–7.3)
	Diabetes	4.4 (1.4–13.7)

CI, confidence interval; EBCT, electron-beam computed tomography.

[a]Analysis were performed with and without the coronary artery calcium scores (CACS)[32].

Raggi and coworkers reported on 172 patients who had EBCT within 60 days of an unheralded MI and on 632 asymptomatic patients who were referred for a screening EBCT and then followed for 32 ± 7 months.[34] Ninety-six percent of all patients with infarction were abnormal by EBCT, and the CACS was 100 in 62 percent and 400 in 47 percent of patients.

Both the absolute CACS and the relative CACS percentiles adjusted for age and gender predicted subsequent death and nonfatal MI. Hard cardiac events occurred in only 0.3 percent of subjects with a normal EBCT, but this increased to 13 percent in those with a CACS >400. A very high CACS 1000 may portend a particularly high risk of death or MI (i.e., 25 percent per year).[35]

Larger trials have been reported, demonstrating approximately 10-fold increased risk with the presence of CAC. [36] in one of the largest observational trials to date,

Shaw and colleagues reported all-cause mortality among 10,377 asymptomatic patients (4191 women and 6186 men) who had a baseline EBCT and were then followed for 5.0 ± 3.5 years.[38] Most subjects had cardiac risk factors including a family history of CAD (69 percent), hyperlipidemia (62 percent), hypertension (44 percent), and current cigarette smoking (40 percent). The CACS was a strong independent predictor of mortality ($x^2 = 36.6$, $p < 0.00001$) with 43 percent additional predictive value contained within the CACS beyond risk factors alone. Mortality significantly increased with increasing CACS.

Similarly, in a younger cohort of asymptomatic persons[35] the 3 year mean follow up in 2000 participants (mean age 43 years) showed that coronary calcium was associated with an 11.8-fold increased risk for incident coronary heart disease (CHD) ($p < 0.002$) in a Cox model controlling for the Framingham risk score.

The ROTTERDAM HEART STUDY[39] investigated 1795 asymptomatic participants (mean age 71 years) who had CAC and measured risk factors. During a mean follow up of 3.3 years, the multivariate-adjusted relative risk of coronary events was 3.1 (95 percent CI, 1.2–7.9) for calcium scores of 101 to 400, 4.6 (95 percent CI, 1.8–11.8) for calcium scores of 401 to 1000, and 8.3 (95 percent CI, 3.3–21.1) for calcium scores >1000 compared with calcium scores of 0 to 100.

The COOPER CLINIC STUDY[40] included 10,746 adults who were 22 to 96 years of age and free of known CHD. During a mean follow up of 3.5 years, 81 hard events (CHD death, nonfatal MI) occurred. Age-adjusted rates (per 1000 person-years) of hard events were computed according to four CAC categories: no detectable CAC and incremental sex-specific thirds of detectable CAC; these rates were, respectively, 0.4, 1.5, 4.8, and 8.7 (trend $p < 0.0001$) for men and 0.7, 2.3, 3.1, and 6.3 (trend $p < 0.02$) for women.

The association between CAC and CHD events remained significant after adjustment for CHD risk factors. A Munich Study determined the extent of CAC by MDCT in 924 patients (443 men, 481 women, aged 59.4 ± 18.7 years).

During the 3-year follow-up period, the event rates for coronary revascularization (5.4 %/y vs. 2.9 %/y), MI (3.8 %/y vs. 1.8 %/y), and cardiac death (2.1 %/y vs. 1.0 %/y) in patients with volume scores above the 75th percentile were significantly higher compared to the total study group and no cardiovascular events occurred in patients with scores of zero. Receiver operating characteristic (ROC) analysis demonstrated it outperformed both PROCAM and Framingham models ($p < 0.0001$), where 36 percent and 34 percent of MIs occurred in the high risk cohorts, respectively.

Coronary artery calcium score in diabetes population.

A study demonstrated the risk stratification in uncomplicated type 2 diabetes in a prospective evaluation of CAC and MPS.[42] Risk factors and CAC scores were prospectively measured in 510 asymptomatic type 2 diabetic subjects (mean age 53 ± 8 years, 61 percent males) without prior cardiovascular disease with a median follow up of 2.2 years. In the multivariable model, the CAC score and extent of myocardial ischemia were the only independent predictors of outcome ($p < 0.0001$).

ROC analysis demonstrated that CAC predicted cardiovascular events with the best area under the curve (0.92), significantly better than the United Kingdom Prospective Diabetes Study Risk Score (0.74) and Framingham Score (0.60, $p < 0.0001$). The relative risk to predict a cardiovascular event for a CAC score of 101 to 400 was 10.13, and increased to 58.05 for scores >1000 ($p < 0.0001$). No cardiac

events or perfusion abnormalities occurred in subjects with CAC 10 Agatston units up until 2 years of follow up.

The CAC score appears to provide complementary prognostic information to that obtained by the Framingham risk model. Combining EBCT results with biochemical markers, such as C-reactive protein, may more precisely define risk than either test alone. . More data is needed in different ethnic groups prior to widespread application.[41]

Calcium Score and Ethinicity

Finally data from the MESA study[1] and other series demonstrated that whites have a higher prevalence of CAC and CAC scores than the other races, and this raised the question of the validity of CAC in non- whites .Two recent publications addressed the value of CAC as a marker of risk in four different races (White, African American, Chinese and Hispanic)in the united states .

Nasir and his colleagues evaluated the use of CAC to predict all cause mortality (505 deaths during the 10 years follow up) in 14,812 patients .the prevalence of CAC was higher in whites, although blacks and Hispanics had a greater clustering of risk factors for CAD.

Despite a low prevalence of CAC and lower scores compared with other races , black patients demonstrated the highest mortality rates even after multivariable adjustment for clinical risk factors and baselines CAC scores (p,<.0001).compared

with whites the relative risk for death was 2.97 (95%cl:1.87-4.72) in blacks ,1.58 (95%cl:0. 92-2.71) in Hispanics and 0.85 (95% cl:0.47-1.54) in Chinese.

Detrano and his colleagues[1] showed that CAC is a strong predictor of CVD, non fatal myocardial infraction ,angina and revascularization independent of race in 6722 MESA patients (the risk increased 7.7 fold in patients with a CAC score between 101 and 300 compared with 0 and 9.7 fold in patients with a score >300). Furthermore CAC added incremental prognostic value beyond traditional risk factors for prediction of all events in all races.

Hence, CAC seems to be an excellent marker of risk in all races so far investigated although the prognostic significance of score categories may vary among the racial groups.

The evidence surrounding the CAC was recently reviewed into two statements of the American Heart Association[12] and the American College Of Cardiology, which recognized the potential utility of CAC screening for refinement of risk assessment in intermediate risk peoples.

MATERIALS AND METHODS.

- This study was conducted in the Department of Cardiology, Government General Hospital, Chennai, during the year 2009 – 2010.The study is a prospective observational non interventional study involving 100 patients.
- Ethical committee clearance was obtained to conduct the study in our hospital.
- All subjects provided written informed consent to participate in the study before inclusion.

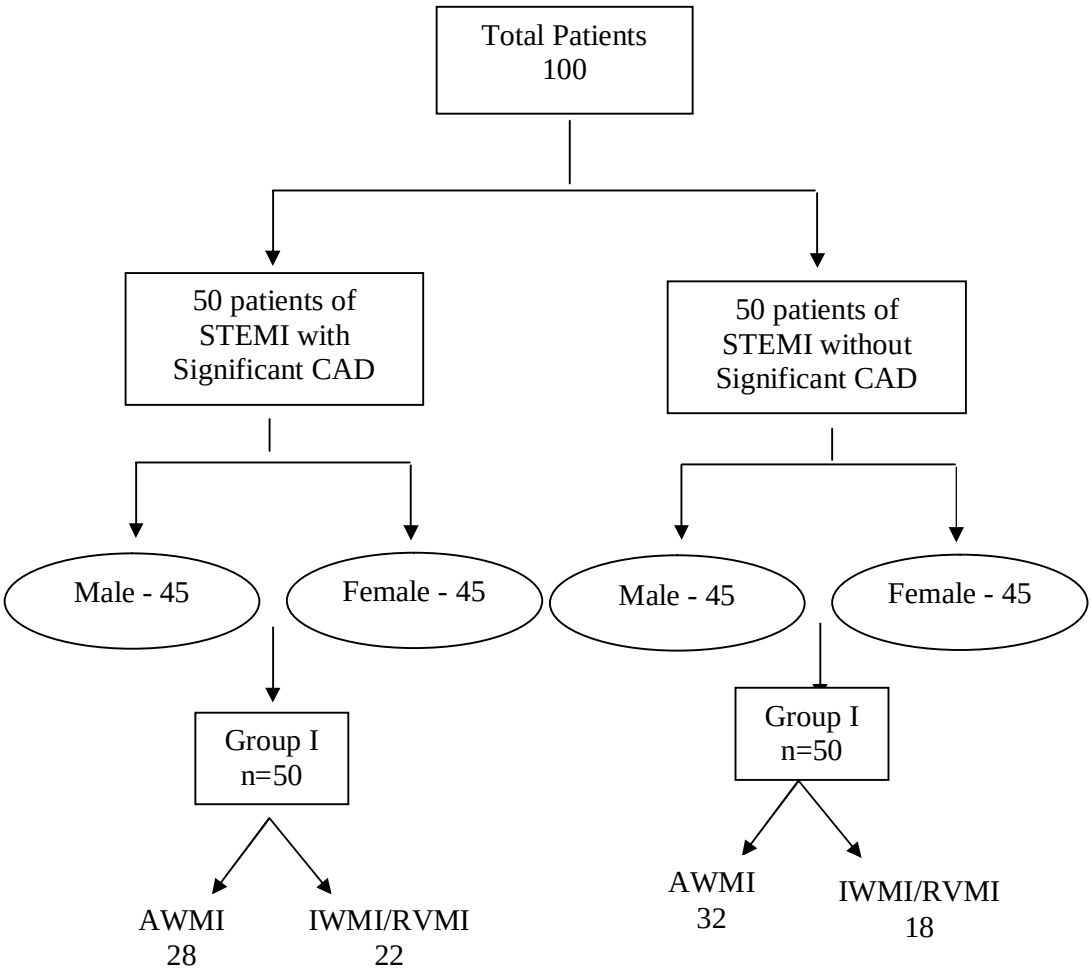
Inclusion Criteria:

- All patients following STEMI (MI diagnosed by History, ECG. ECHO & Enzymes) including both recent and old myocardial infarction irrespective of age and sex.
- Both thrombolysed & not thrombolysed patients.
- Patients with or without LV dysfunction.

Exclusion Criteria

- All acute coronary syndrome patients.
- All chronic stable angina patients.
- Chronic kidney disease.
- Uncontrolled tachycardia.
- Technically inadequate CT.

Flow chart showing patient characteristics of the study population.



Study Centre

- Department of Cardiology and Department of Radiology- Government General Hospital, chennai – 3.

Single centre, Prospective Observational, Non-interventional study.

Detailed history was obtained from all the patients, including the presence of risk factors like

- Diabetes mellitus,

- Hypertension,

- Smoking and

- Family history of ischemic heart disease.

Baseline investigations were done in all patients including complete blood count, blood sugar, renal function tests, lipid profile, chest X-ray. ECG, ECHO, Cardiac enzymes, namely, Creatinine kinase and CK-MB were done in all patients.

Patient characteristics:

The study population included 100 patients (91 male and 9 female) admitted to the department of cardiology Govt. General Hospital, Chennai – 3, for coronary angiography evaluation following STEMI.

Table 4-1 Showing Patient Characteristics:

	Group 1 (n=50)	**Group 2 (n=50)**	**TOTAL**
Age < 40 years	7	10	17 (17%)
40 to 60 years	11	5	16 (16%)
>60 years	32	35	67 (67%)
Male	46	45	91 (91%)
Female	4	5	9 (9%)
Hypertension	15	10	25 (25%)
Diabetes	8	13	31 (31%)
Smoking	16	5	21 (21%)
F/H of CAD	4	1	5 (5%)

Our study population contains predominantly male (90%). One fourth of the population had hypertension and one third of the study group are diabetics.

One fifth are smokers. Only few patients gave history of smoking.

Coronary Angiography:

- All patients following STEMI admitted to undergo coronary angiography

  (After an average period of 4 to 6 weeks following STEMI) in the department of cardiology underwent CAG using Philips Integri 3000 machine. Government General Hospital,Chennai-3.

- CAG was done through both the femoral and radial route, using properly sized sheath, Judkin's catheter, Amplatz catheter and Tiger catheters if necessary. Multiple angulations and views were used. The CAG was analyzed and the lesions are quantified in detail. Lumen diameter narrowing was graded as 0, 25,50,75,90 and 100%. A detail report with pictures are prepared and tabulated.

Complications:

Five patients of the study group developed minor complications in the form of minor hematoma, transient benign arrhythmias. There is no death, MI or CVA in the study group following the procedure.

Based on CAG findings the study population is categorized into study

Group I– With obstructive CAD (defined as ≥50% luminal obstruction irrespective of the infarct related artery) and

Group II – With non obstructive CAD (<50% luminal obstruction in any of the epicardial coronaries as well as normal coronaries).

CAC score measurements:

Then the patients were referred to Barnard Institute of Radiology, Government General Hospital, Chennai-3 for assessment of CAC score. (Average waiting period for CAC measurement following CAG is 2 weeks). It was done using Philips Brilliance 64 slice MDCT machine based on Agatston scoring system by a expert radiologist who has no knowledge about the CAG lesions of the patients concerned. Any score greater zero is considered as positive score based on agatston scoring system. And the results of both groups who underwent CAC scores were tabulated, compared and analyzed in detail.

RESULTS

Statistical analysis:

The results were analyzed by the following statistical methods.

1) Chi-square test

2) Mann Whitney U Wilcox on Rank Sum test

3) Correlation coefficient methods

4) Multiple regression analysis.

The p values are categorized as follows.

a) 0 to 0.01 - Significant at 1% level.

b) 0.01 to 0.05- Significant at 5% level.

c) > 0.05 - No statistical significance.

Table 5-1 showing patient's CAG baseline profile in Group I :

VESSEL INVOLVEMENT	LAD	LCX	RCA	LAD & LCX	LAD & RCA	LCX & RCA	LAD, LCX & RCA
NO OF CASES	12	3	5	7	14	2	7

Table 5-2 showing Types of MI and the vessels involved.

VESSELS INVOLVED	AWMI	IWMI / RVMI
LAD	13	1
LCX	0	3
RCA	2	3
LAD & LCX	4	1
LCX & RCA	3	4
LAD & RCA	6	2
LAD,LCX & RCA	6	1

About 90% of AWMI patients showed LAD involvement whereas LCX & RCA are the predominant culprit vessels among patients with IWMI/RVMI.

Table 5-3 showing the significance of risk factor and CAC scores in patients in Group I:

S.No	**Variable**	Positive CAC	Negative CAC	p value	Significance
1	**DIABETES**	9	3	0.067	SIGNIFICANT
2	**HYPERTENSION**	8	7	0.901	NOT SIGNIFICANT
3	**SMOKING**	11	5	0.103	NOT SIGNIFICANT
4	**FAMILY HISTORY**	4	0	0.045	SIGNIFICANT
5	**SEX**	M-24,F-2	M-21,F-3	0.571	NOT SIGNIFICANT
6	**AGE**	MEAN 50.82	MEAN 48.84	0.025	SIGNIFICANT

The above analysis are done using chi-square test regarding the significance of the above variable and total CAC score among GROUP 1 patients.

It showed that though the conventional risk factors like hypertension, smoking and male sex are associated with increased CAC scores they are not statistically significant whereas the diabetes, age and positive family history is predictive of increased CAC scores in patients with obstructive CAD and it is statistically significant.

Figure 1 showing risk factor and CAC correlation in GROUP I patients.

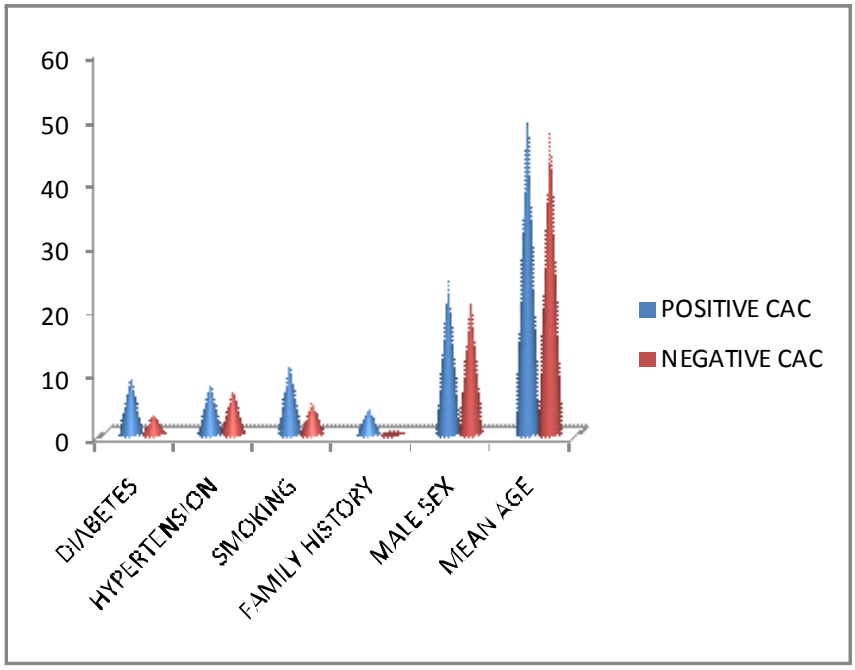


Figure 2 showing risk factor and CAC correlation in GROUP I patients.

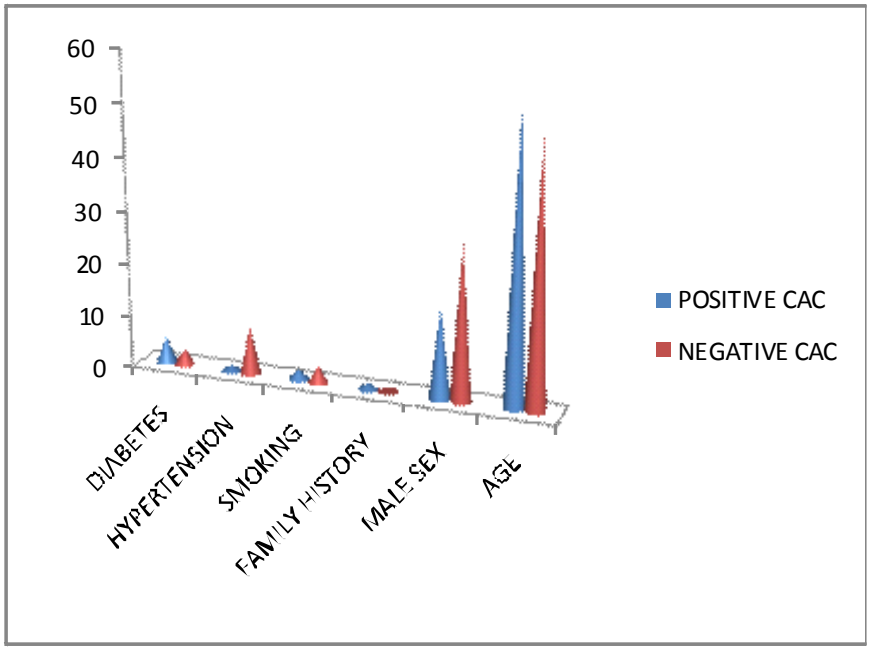


Table 5-4 showing the significance of risk factor and CAC scores in patients in Group II:

S. No	Variable	Positive CAC	Negative CAC	P value	Significance
1	**DIABETES**	5	3	0.119	NOT SIGNIFICANT
2	**HYPERTENSION**	1	9	0.041	SIGNIFICANT
3	**SMOKING**	2	3	0.922	NOT SIGNIFICANT
4	**FAMILY HISTORY**	1	0	0.196	NOT SIGNIFICANT
5	**SEX**	M - 17, F- 2	M – 29, F - 2	0.606	NOT SIGNIFICANT
6	**AGE**	MEAN 52.84	MEAN 49.12	0.072	SIGNIFICANT

The above analysis is done using chi- square test regarding the significance of the above variable and total CAC score among GROUP II patients.

It showed that though the conventional risk factors like diabetes, smoking, family history and male sex are associated with increased CAC scores they are not statistically significant except age which has good correlation with statistical significance. It was also found that history of hypertension shows negative predictive value for CAC scores in patients with non obstructive CAG and it is statistically significant (p- 0.041).

Table 5-5 showing the significance of individual vessel involvement and CAC scores in patients in Group II.

S.NO		**LAD CAC**	**LCX CAC**	**RCA CAC**	**TOTALCAC**	**P VALUE**
1	**LAD CAG**	0.0598 P=0.68	0.0599 P=0.68	0.761 P=0.6	0.0739 P=0.61	NOT SIGINIFICANT
2	**LCX CAG**	0.0038 P= 0.979	0.1617 P=0.289	0.1529 P=0.289	0.0439 P=0.762	NOT SIGINIFICANT
3	**RCA CAG**	0.0893 P= 0.538	0.075 P = 0.605	0.1718 P = 0.233	0.1204 P =0.405	NOT SIGNIFICANT

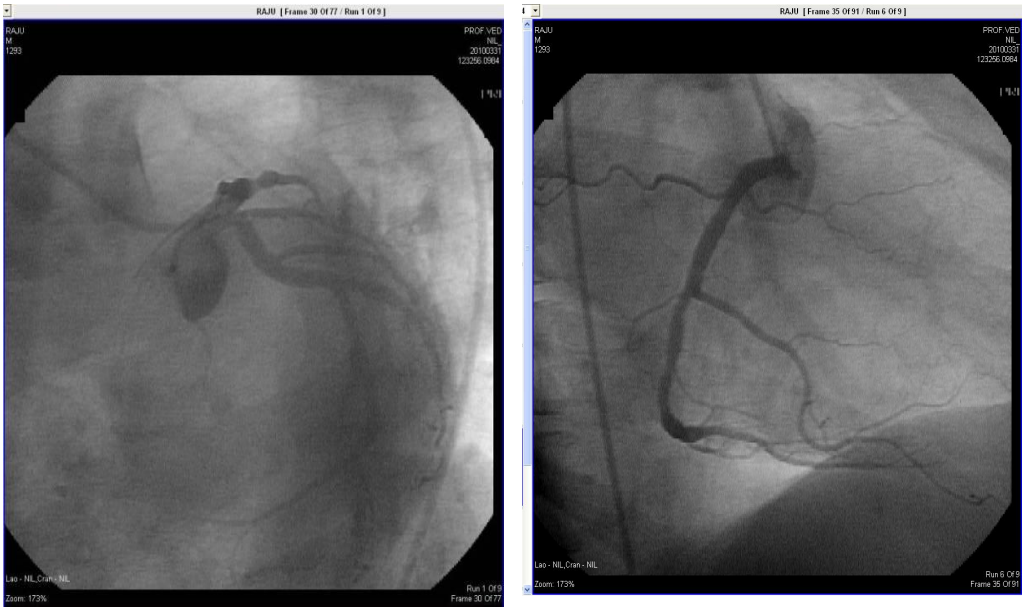
A detailed analysis of CAG lesions of individual vessel was correlated with the CAC scores of corresponding vessel in patients among Non obstructive CAD. The above details of Group II comparing CAG with CAC score were analyzed using the correlation coefficient method and the details revealed no statistical significance. Thus it shows that there is no correlation between the CAG stenosis and the CAC score of the vessel involved among patients with Non obstructive CAD.

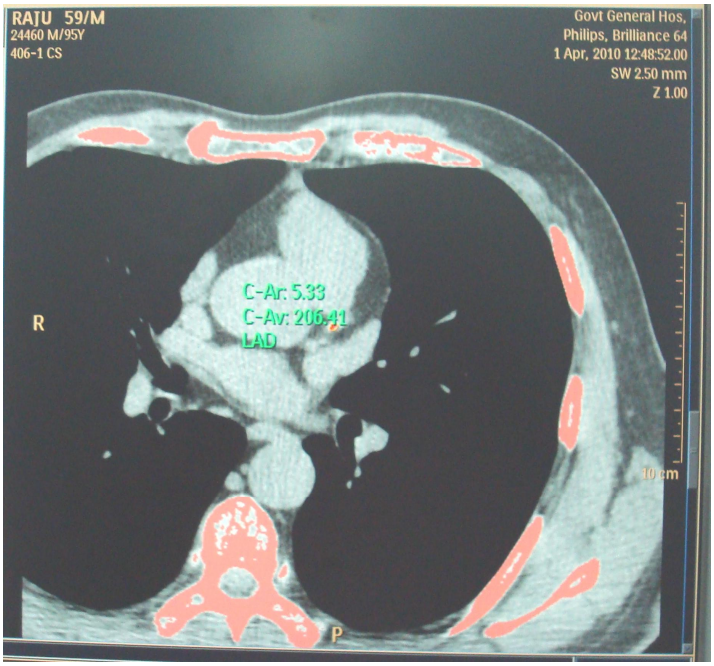
Table 5-6 showing the significance of individual vessel involvement and CAC scores in patients in Group I

CAC		LAD	LCX	RCA	TOTAL	P VALUE
CAG	**LAD**	0.1545 P=0.284	0.0258 P=0.859	0.0161 P=0.912	0.1128 P=0.436	NOT SIGINIFICANT
	LCX	0.0038 P= 0.979	0.1617 P=0.289	0.1529 P=0.289	0.0298 P=0.837	NOT SIGINIFICANT
	RCA	0.0893 P= 0.538	0.075 P = 0.605	0.1718 P = 0.233	0.1663 P =0.248	NOT SIGNIFICANT

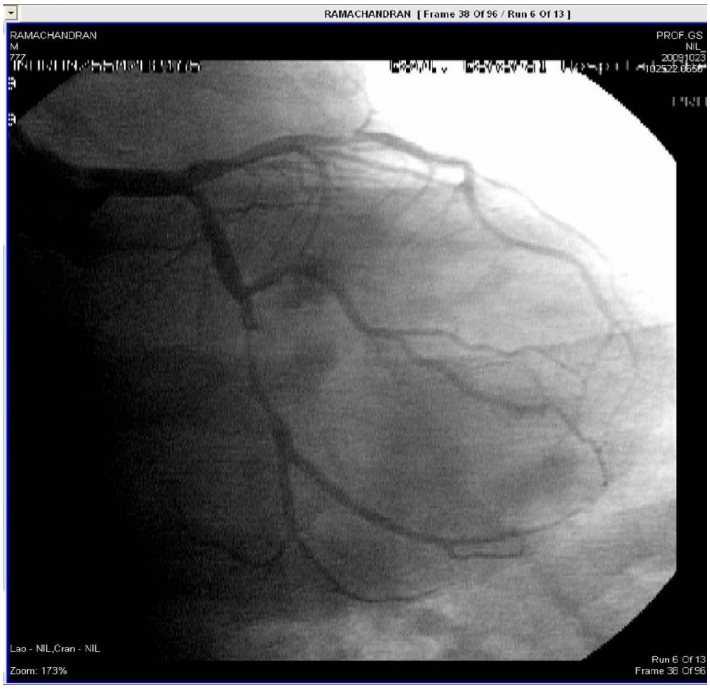
A detailed analysis of CAG lesions of individual vessel was correlated with the CAC score of the corresponding vessel of patients among obstructive CAD. The above details of Group 1 comparing CAG with CAC score were analyzed using the correlation coefficient method and the details revealed no statistical significance. Thus it shows that there is no correlation between the stenosis and the CAC score of the vessel involved.

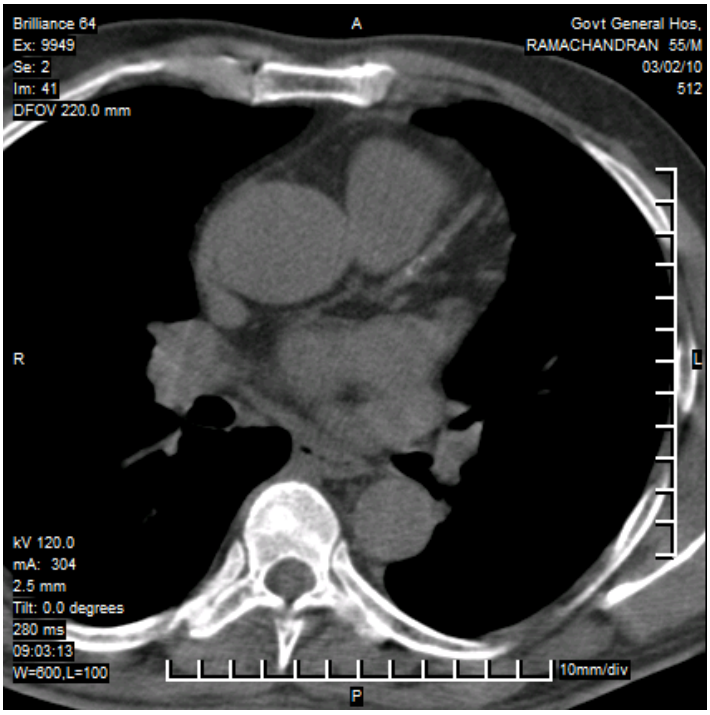
Picture -8. Raju 59/M – Old AWMI:
a) CAG – LAD 80% lesion
b) Mid RCA- 70% lesion c) CAC – score 448



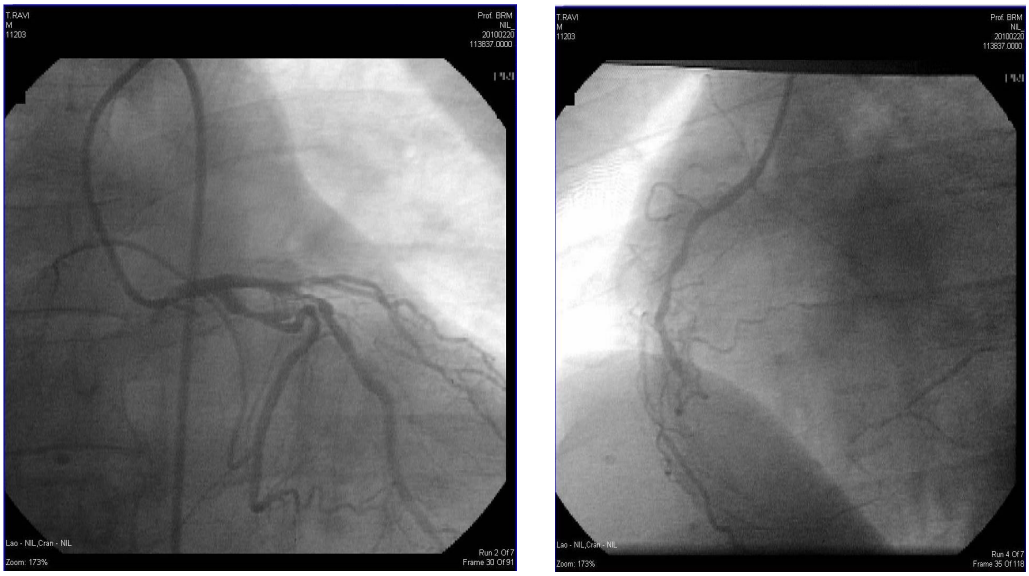


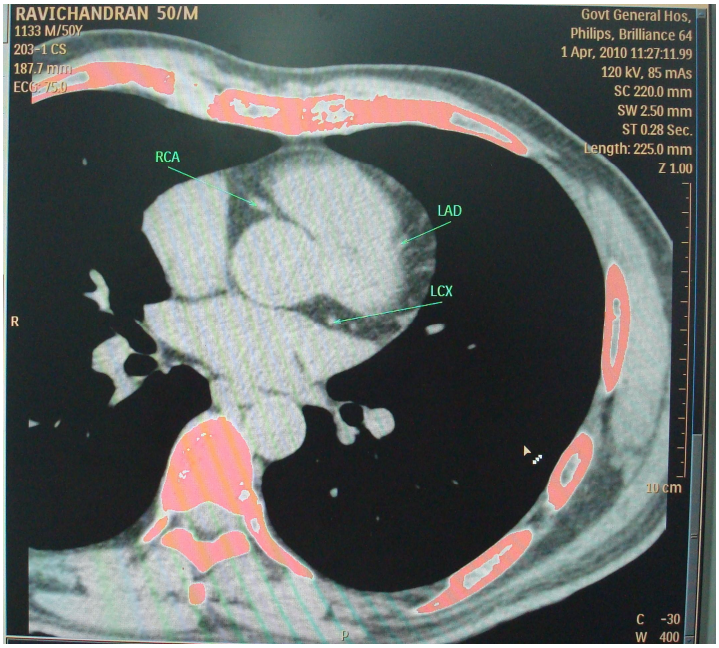
Picture -2. Ramachandran 55/M – Old AWMI:
a) CAG – Mid LAD 70% lesion & Mid RCA- 70% lesion
b) CAC – LAD score is 2.



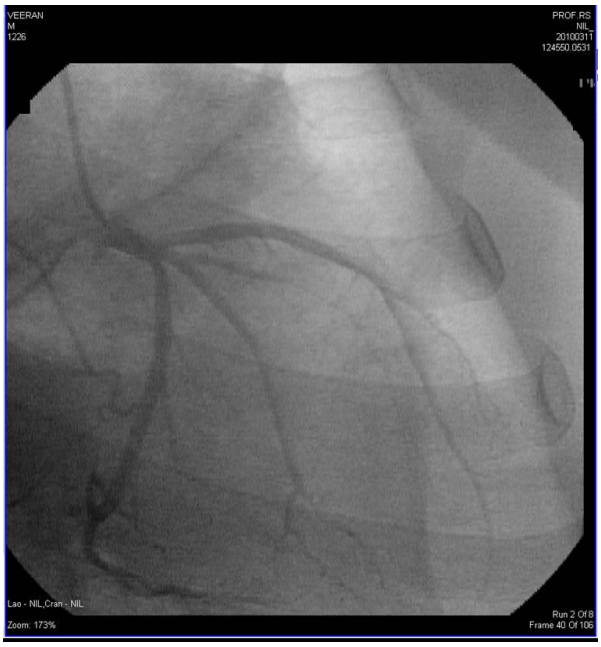


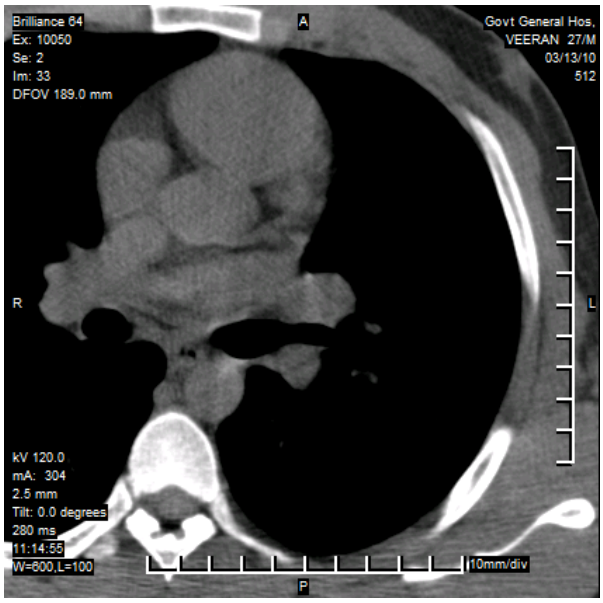
Picture -3. Ravichandran 50/M – Old AWMI: a) CAG – LAD 80% lesion b) Mid RCA- 100% lesion c) CAC – score is zero.



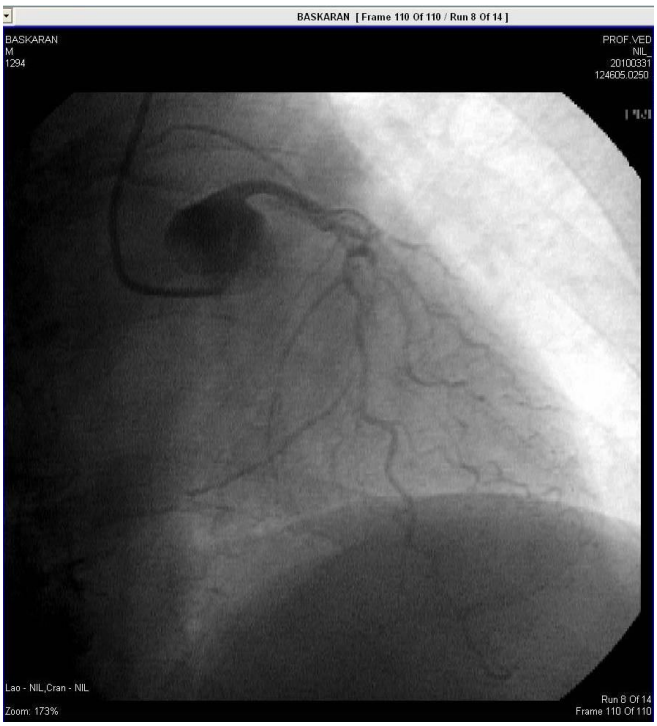


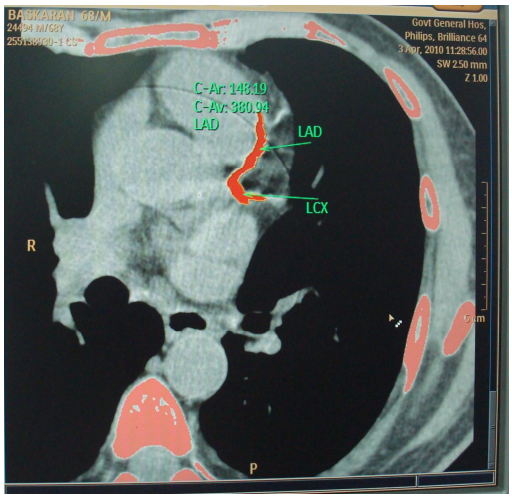
Picture -4. Veeran 27/M – Old AWMI: a) CAG – Normal LCA
b) CAC – score is zero.

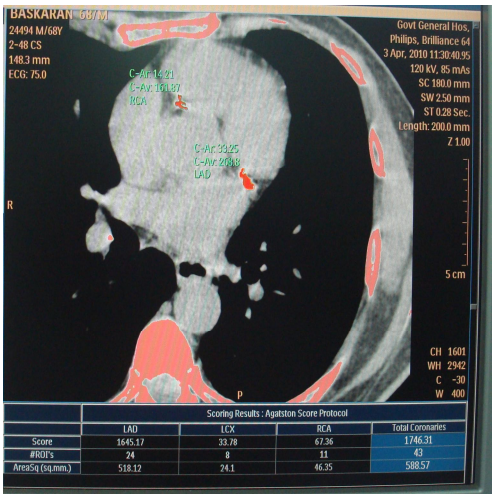




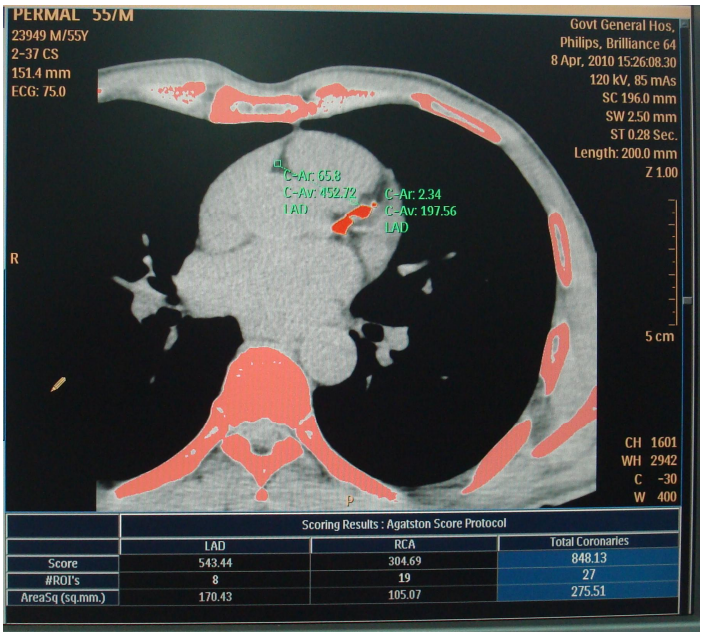
Picture -5. Baskar 68/M – Old AWMI: a) CAG – Normal LAD & LCX.
b) CAC – dense calcium in LAD 1573, ,RCA 37.







Picture -6. Perumal 55/M – Old IWMI/RVMI: showing Mid LAD irregularities with dence calcium in LCA.



Picture -7. Nayanan 62/M –Correction between normal CAG with normal CAC despite advanced age

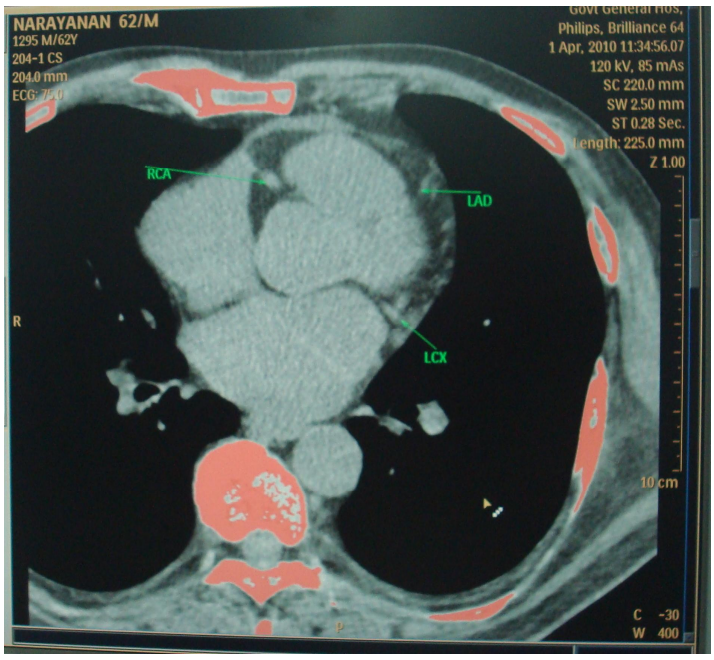


Table 5-8 showing the significance of multivessel involvement and CAC scores in patients in both groups.

S.NO	**VESSELS INVOLVED**	**MULTIPLE R**	**SIGNIFICANCE**
1	**LAD & LCX**	0.112	NO
2	**LAD & RCA**	0.245	NO
3	**LCX & RCA**	0.166	NO
4	**LAD, LCX & RCA**	0.250	NO

The significance of correlation of multivessel involvement and total calcium scoring was analyzed by multiple regression analysis. The CAC score was analyzed between double and triple vessel involvement with single vessel disease. It was found that there is no increase in either the positivity or the degree of CAC score with multivessel involvement when compared to single vessel disease.

DISCUSSION

Our study showed that though the conventional risk factors like hypertension, smoking and male sex are associated with increased CAC scores, they are not statistically significant whereas the diabetes, age and positive family history is predictive of increased CAC scores in patients with obstructive CAD and it is statistically significant.

Further it was also found that though the conventional risk factors like diabetes, smoking, family history and male sex are associated with increased CAC scores they are not statistically significant except age which has good correlation and history of hypertension shows negative predictive value for CAC scores in patients with non obstructive CAG and it is statistically significant.

Smoking:

A strong dose–response relationship between cigarette smoking and CHD has been observed in both sexes, in the young, in the elderly, and in all racial groups.[11] Cigarette smoking increases risk two- to threefold and interacts with other risk factors to multiply risk. There is no evidence that filters or other modifications of the cigarette reduce risk. Pipe smoking and cigar smoking increase the risk of CHD. More than 1 in every 10 cardiovascular deaths in the world in the year 2000 were attributable to smoking.[25]

Figure 3 – Sex distribution in Group I with CAC score.

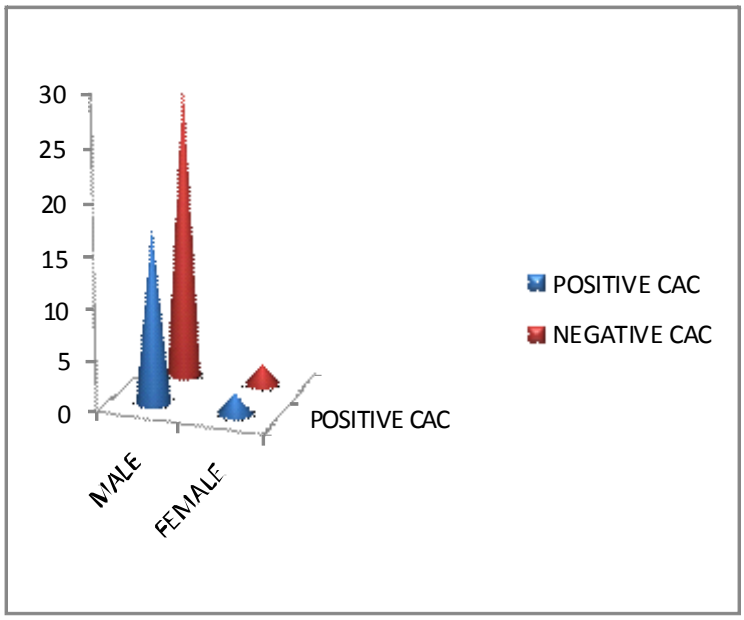
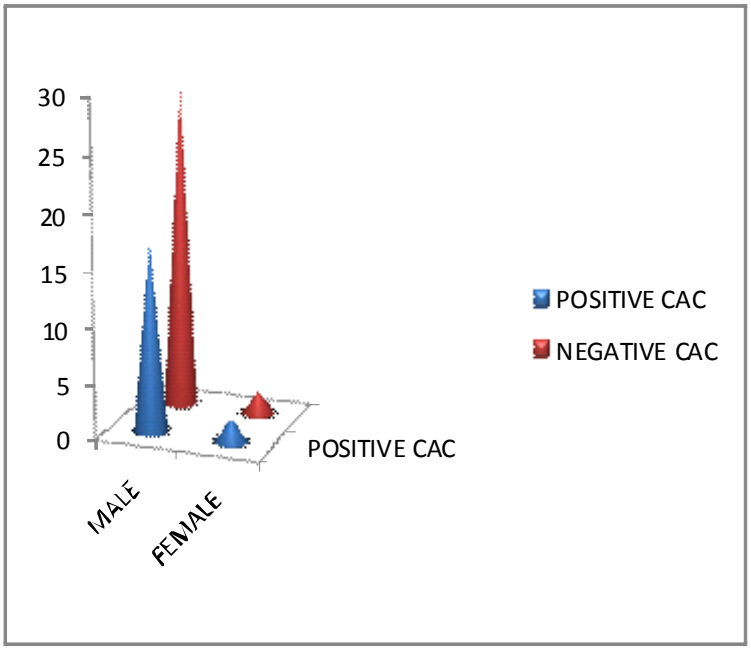


Figure 4 – Sex distribution in Group II with CAC score.



Smoking does not carry a significant risk for coronary calcification as compared to international studies .There were 16 patients (32%) in group I and 5 (10%) patients in group II with smoking history. But in both groups smoking does not show a statistically significant correlation of increased CAC score.(p – 0.103 in Group I: P-0.922 in Group II)

CAC Scores and Gender:

Gender differences in utility and accuracy of imaging tests are typically related to differences in the epidemiology of coronary heart disease, with women having later onset of clinical CHD than men. Gender differences in incidence and prevalence of CAD are most marked in middle-aged populations, the typical target age group for CHD screening. In addition, emerging data suggest that there may be actual gender differences in the anatomy of atherosclerosis.

Thus, it is important to consider gender-specific data when evaluating the potential uses of any new cardiac test. There are limited data broadly specific to women on the relationship between CHD outcomes and CAC. Existing data confirm an association between CAC scores and all-cause mortality and CHD events in elderly women.

The Prospective Army Coronary Calcium (PACC) Project[19] found a higher prevalence of coronary calcium in white (19.2%) than black (10.3%) active-duty military personnel with a mean age of 42 years; the difference persisted after adjusting for cardiovascular disease risk factors.

Budoff et al. described similar findings in white men referred for CAC testing compared with black men; however, in this study, black women had a higher prevalence of coronary calcium than white women. In addition, Asian men and women had a lower prevalence of coronary calcium, and the prevalence in Hispanics was similar to the whites[20]

The utility of CAC screening has also been investigated in special subsets of populations such as women, diabetic patients and elderly .Two original investigations and one meta analysis supported the utility of CAC for risk stratification in women. The authors' group[11] compared the occurrence of all-death in approximately 4000 women and 6000 men referred for CAC screening by primary vary physicians.

CAC scores were lower in women than in the men ($p<.001$), but death rates were higher among the older, diabetic, hypertensive and smoking patients of both the gender. .In risk adjusted models; women had a greater probability of death than the men for the CAC score importantly. CAC score added incremental prognostic value to the FRS ($p<.0001$) in both the genders.

Lakoski and colleagues conducted gender analyses of the Multi Ethnic Study of Atherosclerosis (MESA) data and noted that a CAC score greater than 0 was strong predictor of coronary heart and CVD events in 2684 women considered to be at low risk by Framingham categories compared with patients without CAC (hazard ratios 6.5and 5.2 respectively) finally in a meta-analysis of three prospective and two observational registries. Bellasi and his colleagues concluded that CAC screening is

Figure - 9 showing types of AMI and vessels involved in GROUP I.

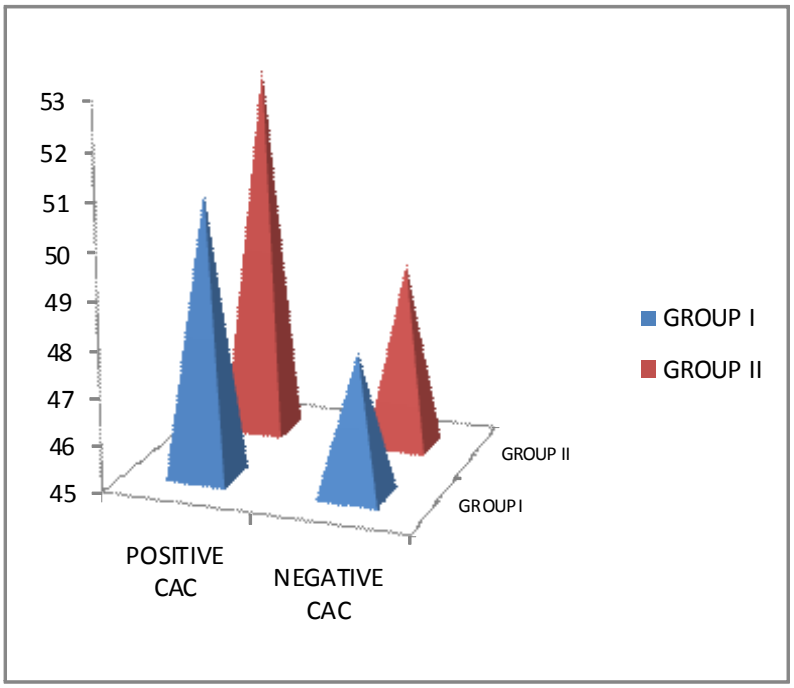
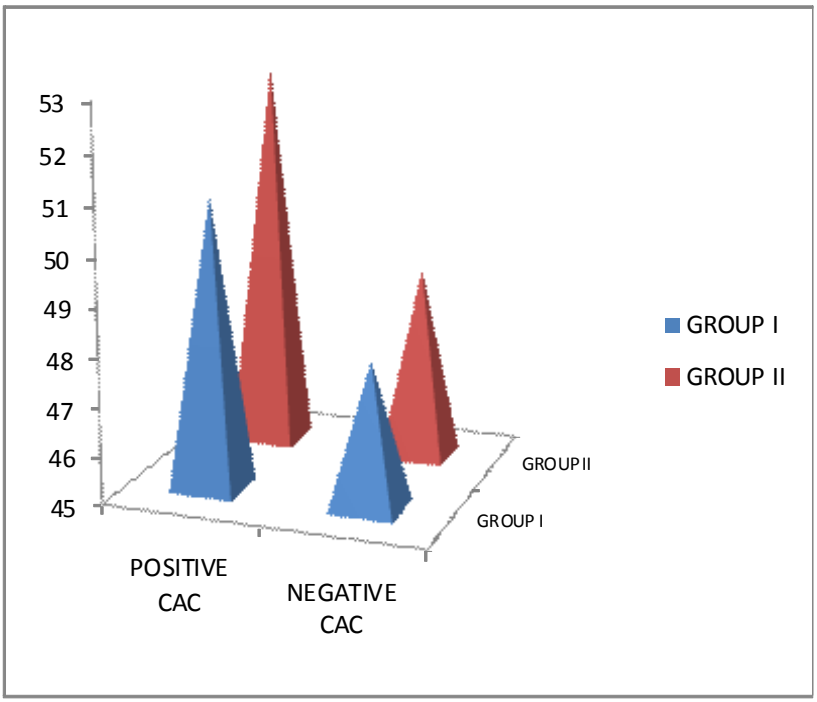


Figure - 10 showing correlation of Mean Age and CACS in both groups.



equally accurate in stratifying risk for all-cause death and CVD events in women and in men.

There are only limited no of female patients involved in our study because of social reasons as many of our female patients are not willing to enroll in our study. In our study there are only 4 female in Group I and 5 female in Group II. There is no positive correlation of CAC score in males compared to females in both groups.

Calcium Score in the Elderly:

Age is an important predictive factor for coronary artery calcification in our study independent of CAG lesions. Several recent cohorts have been published including prospective observational registries in predominantly male, younger and middle-aged , unselected and older-aged, higher risk asymptomatic cohorts[8] .

For age group 40 to 49 and 50 to 59 years, a total score of 50 resulted in a sensitivity of 71% and 74% and a specificity of 91% and 70%, respectively. For age group 50 to 59 years, a total score of 300 gave a sensitivity of 74% and a specificity of 81%.

CAC maintains its utility for risk stratification in the elderly. In the prospective Rotterdam study, 2013 participants (mean age: 71+_5.7 years) received CAC screening and measurement of traditional cardiovascular risk factors[23].

Men and women in the highest CAC score category showed an adjusted odd ratio for myocardial infarction of 7.7 (95%cl:4.1-14.5) and 6.7(95%cl:2.4-19.1),

respectively, compared with the lowest score category (0-100). The predictive power of CAC was independent of FRS category (low, intermediate or high).

Raggi and colleagues followed 35,388 patients ,with 3570 subjects being 70 years of age or older at screening , for an average period of 5.8+_3 years .The author 's group[11] reported an expected increase in all cause mortality rate with increasing age.

(relative hazard per age decline increase =1.09,95% cl;1.08-1.10 ;p<.000).

With higher death rates among men than women nonetheless, increasing CAC score were associated with decreasing survival rates across all age declines (p<.0001) suggesting that CAC is evident even in the elderly. Finally using CAC score categories, more than 40% of elderly patients were reclassified into lower or higher risk categories compared with their original FRS group.

In our study the mean age is 50.82 in Group I and showed a significant association with increased CAC score with statistical significance. (P- 0.025)

In Group II the mean age of the population 50.82 and showed a significant association with increased CAC score with statistical significance (P- 0.006). And this matches with the above mentioned various international studies.

Calcium Score in Diabetic patients:

Several clinical studies have shown that glucose intolerance and insulin resistance are associated with increased prevalence of CAC. Similarly frank diabetes is associated with a greater risk of CAC compared with those in non-diabetic population .Wong and colleagues and Anand and colleagues [22]demonstrated an increasing incidence of inducible ischemia on stress myocardial perfusion imaging in diabetic patients with a greater amount of CAC.

Type 2 diabetic patients with a CAC score of 10 or less,11 to100,101 to 400,401 to 1000,and greater than 1000 had an incidence of myocardial ischemia of 0%,18%,23%,48%,71%respectively,and morbidity and mortality increased proportionally with CAC score and ischemic burden In an observational registry.

Raggi and his colleagues showed a higher all- cause mortality rate for any extent of CAC for diabetic subjects than the non-diabetic patients ($p>.0001$). Of interest the 5- year mortality rate of diabetic patients with little or no CAC (approximately 30% cohort of 903 diabetic patients)was as low as that of non-diabetic subjects without CAC (approximately 1% at the end of the follow up).[13]

It is not an important predictive factor for coronary artery calcification in our study independent of CAG lesions. However, Raggi et al. found that coronary calcium predicted all-cause mortality in diabetics referred for fast coronary CT scanning.

Figure- 7 showing relationship of HT with CACS among both groups.

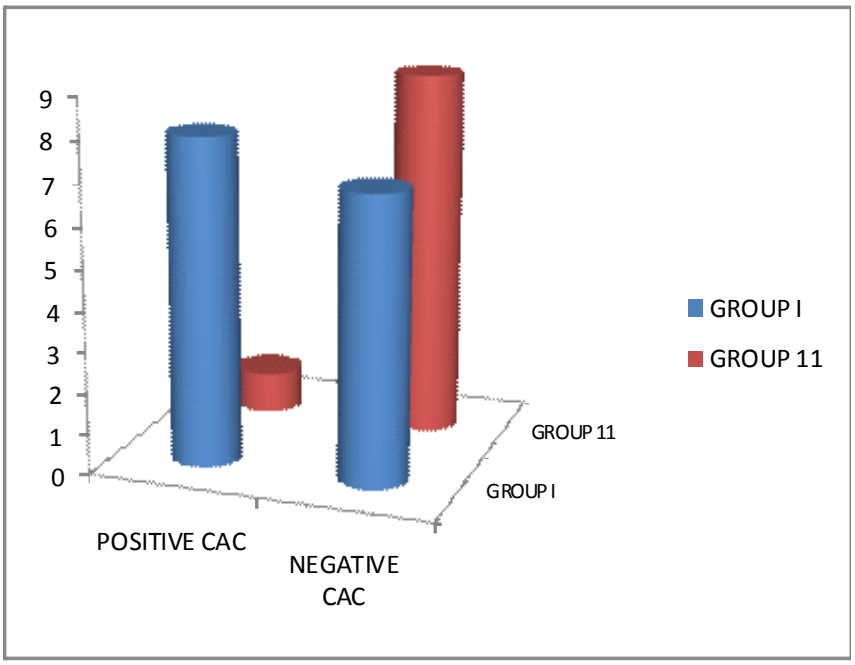


Figure-8 showing significance of CACS and Family history among both the groups.

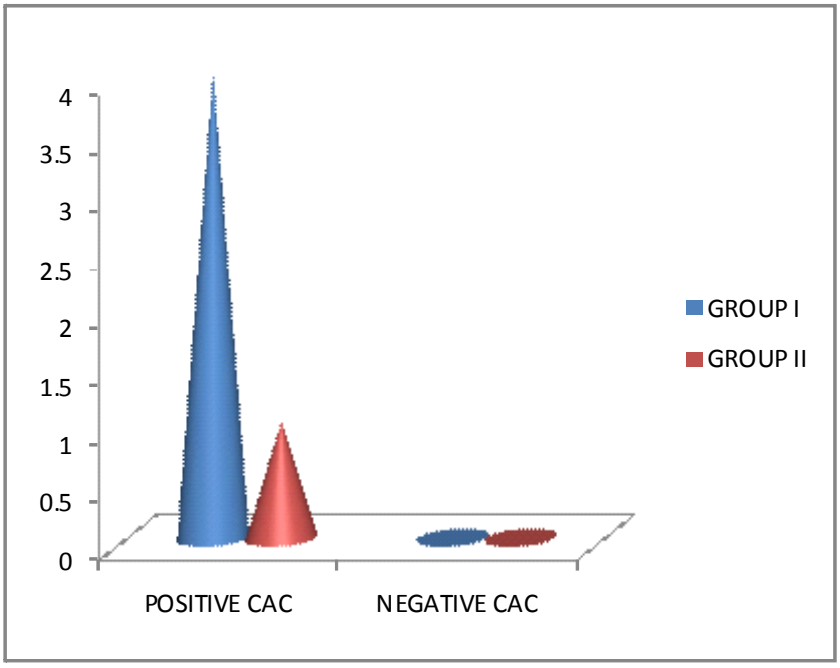


Figure-5 showing significance of Smoking and CACS among both groups.

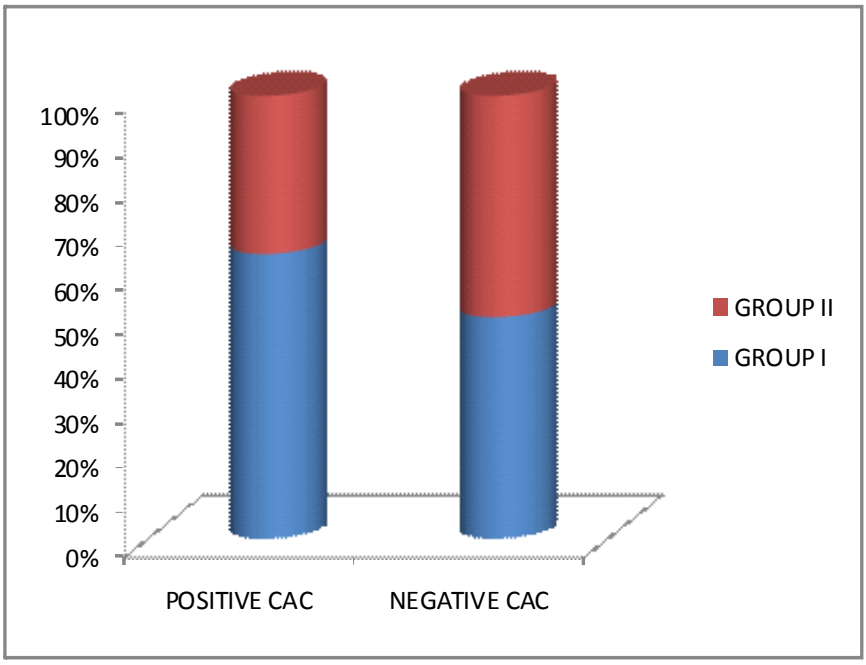
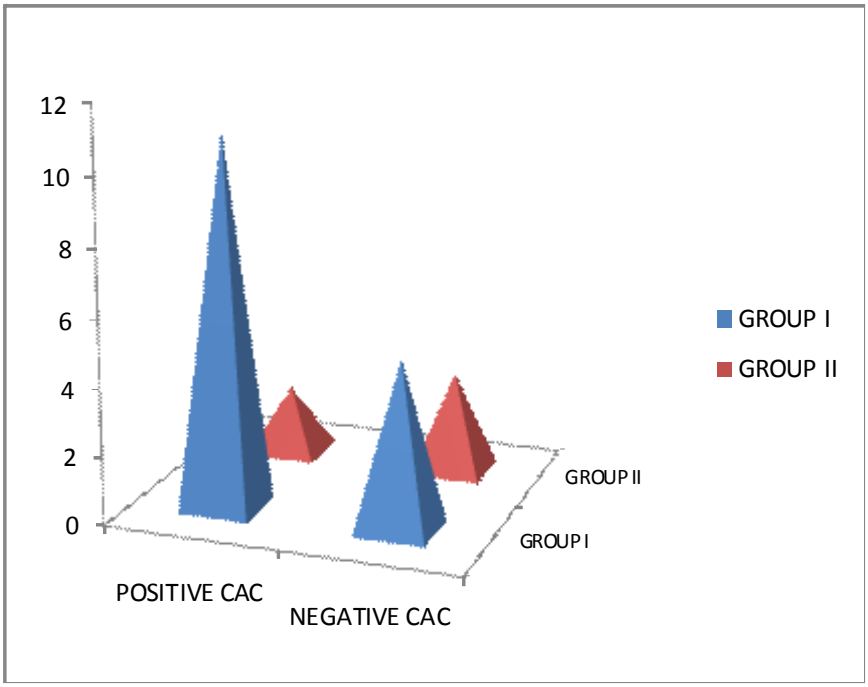


Figure- 6 showing relationship of Diabetes with CACS in both groups.



Raggi et al. also found that patients with diabetes have a greater increase in risk for mortality associated with a given degree of calcium than the non-diabetic patients Diabetic patients without any evidence of coronary calcification have a survival rate similar to non-diabetic patients with a zero calcium score during 5 years of follow-up.

These results suggest that coronary calcium might be useful to further stratify short-term risk in diabetic patients. However, until studies from non-referral populations with longer follow-up, including fatal and non-fatal cardiovascular events are completed, CAC scores should not be used to modify treatment goals in diabetic patients.

In Group I there are 12 diabetics (24%) with 9 cases (18%) showing positive CAC and 3 cases (6%) showing negative CAC with a good statistical significance. Hence diabetes showed a significant association with CAC in patients with obstructive CAD with statistical significance (P- 0.06)

In Group II there are 10 diabetics (20%) with 7 (14%) cases showing positive CAC and 3 (6%) cases showing negative CAC with no statistical association. Hence diabetes showed no significant association with CAC in patients with non obstructive CAD (P- 0.11). And this matches in certain aspects with the above mentioned various international studies

CAC Score and Multivessel involvement:

The CAC score was analyzed by multiple regression analysis between double and triple vessel involvement with that of single vessel disease. It was found that there is no increase in either the positivity or the degree of CAC score with multivessel involvement when compared to single vessel disease.

Incidental findings in patients undergoing CAC Testing:

Coronary calcium measurement by fast CT scanning of the heart includes imaging of a portion of the lungs, mediastinum, bones and upper abdomen, in addition to the aorta.

The identification of potential pathology other than coronary calcium must be considered when evaluating the benefits and costs of cardiac CT scanning. The most common incidental finding is pulmonary nodules[9] but in our study we found few aortic, pulmonary artery and pulmonary vein calcification

CONCLUSION

1) Sixty four slice MDCT derived CAC score is a useful tool to assess angiographic severity in Post MI population.

2) CAC scores showed good correlation in patients with obstructive CAD especially in Elderly, Diabetics and in those with a family history of CAD.

3) There is less correlation of CAC score with regards to other conventional risk factors like Gender, Hypertension and Smoking in both obstructive and non obstructive CAD.

4) CAC score was not useful to identity infarct related artery.

5) There was no linear correlation between CAC score and the number of vessel involvement.

6) There was a significant negative correlation in hypertensive patients among non obstructive CAD population.

LIMITATIONS OF THE STUDY

1) There are only limited no of female patients involved in our study as many of our female patients are not willing to undergo coronary angiogram.

2) IVUS which is the gold standard is not performed to study the extent of accurate plaque burden for comparison.

3) CAC scores in patients with renal disease could not be studied as there is risk in CAG regarding contrast usage.

4) We have not followed the patients for analysis regarding the prognostic implications of CAC scores.

5) CAC score is not analyzed in patients with acute coronary syndrome and chronic stable angina.

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PROFORMA

NAME: AGE/SEX: DOA:

ADDRESS:

OCCUPATION:

DO AMI:

DO CAG:

DO CAC:

CAG NO:

DIAGNOSIS:

RISK FACTORS:

DM: HT: SMOKING:

DYSLIPIDEMIA:

TC: LDL: TGL:

HDL:

FAMILY H/O CAD: CKD:

CAC SCORE:

CAG FINDINGS:

LM:

LAD:

LCX:

RCA:

GLOSSARY AND ACRONYMS

AMI- Acute Myocardial Infarction.

AWMI- Anterior Wall Myocardial Infarction.

IWMI- Inferior Wall Myocardial Infarction.

RVMI- Right Ventricular Myocardial Infarction.

LWMI- Lateral Wall Myocardial Infarction.

CAD- Coronary Artery Disease.

CAC-coronary artery calcium.

LAD- left anterior descending artery.

LCX- left circumflex artery.

RCA- right coronary artery.

OM-obtuse marginal, D-diagonal artery.

STEMI- ST segment Elevation Myocardial Infarction.

LVEF- Left Ventricular Ejection Fraction.

EBCT- Electron Beam Computed Tomography.

MDCT- Multirow Detector Computed Tomography.

MRI-Magnetic Resonance Imaging.

ECG- Electrocardiogram.

ECHO- Echocardiogram.

MASTER CHART OF GROUP I POPULATION

S.NO	NAME	AGE/ SEX	IP NO	DIAGNOSIS	CAG NO	D.O.MI	D.O.CAG	D.O.CAC	LAD CAG	LCX CAG	RCA CAG	LAD CAC	LCX CAC	RCA CAC	TOTAL CAC	DM	HT	SMOKING	FAMILY H/O CAD
1	PARAMASIVAM	M/58	24180	OLD AWMI	1280	10/2/10	29/02/10	2/5/10	70	0	0	87	34	0	122	NIL	NIL	NIL	NIL
2	RAJENDRAN	48/M	474	OLD IWMI	1041	17/1/10	22/1/10	15/2/10	0	90	0	8.87	0	0	8.87	NIL	NIL	YES	NIL
3	ABUBEKAR	50/M	3147	OLD IWMI	1046	12/1/10	22/1/10	28/4/10	0	0	100	0	0	0	0	NIL	NIL	NIL	NIL
4	SRINIVASAN	50/M	70238	OLD AWMI	599	31/7/09	12/9/09	12/4/10	70	0	90	108.32	0	0	108.32	YES	YES	YES	NIL
5	VELMURUGAN	42/M	9237	OLD IWMI	1088	16/5/09	6/2/10	11/3/10	0	0	100	0	0	0	0	NIL	NIL	YES	NIL
6	SELVAPANDIAN	49/M	64559	OLD AWMI	521	8/5/09	24/8/09	1/4/10	50	0	70	0	0	0	0	YES	NIL	NIL	NIL
7	RADHAKRISHNAN	57/M	24502	OLD AWMI	1350	10/2/10	31/3/10	5/4/10	60	0	30	21	10	1	33	NIL	NIL	YES	NIL
8	MALLIGA	63/F	6210	OLD IWMI	1205	24/1/10	6/3/10	10/4/10	70	0	70	95	0	314	409	YES	NIL	NIL	NIL
9	NARAYANAN	62/M	21555	OLD AWMI	1295	10/1/10	20/3/10	1/4/10	90	0	0	0	0	0	0	NIL	NIL	NIL	NIL
10	KUMAR	40/M	8400	OLD IWMI	1021	11/9/09	18/1/10	22/2/10	60	0	150	0	0	0	0	YES	YES	NIL	NIL
11	NAGARAJAN	47/M	27158	OLD IWMI	1410	28/1/10	12/4/10	16/4/10	0	50	70	0	0	0	0	NIL	NIL	YES	NIL
12	ARUMUGAM	37/M	70115	OLD AWMI	719	3/8/08	6/10/09	23/4/10	50	0	70	0	0	0	0	NIL	YES	NIL	NIL
13	RADHAKRISHNAN	53/M	18125	OLD AWMI	1351	6/3/10	31/3/10	1/4/10	60	0	30	21	26	0	47	NIL	YES	NIL	NIL
14	LOGANATHAN	45/M	15777	OLD IWMI	444	20/4/09	3/8/09	2/2/10	0	40	100	47	73	148	269	YES	YES	YES	NIL
15	RAJENDRAN	48/M	474	OLD IWMI	1041	24/1/05	22/1/10	25/3/10	0	90	0	0	0	0	0	NIL	YES	YES	NIL
16	NIRMALA	39/F	98983	OLD AWMI	923	16/1/09	19/12/09	7/2/10	70	70	0	27	0	0	27	NIL	NIL	NIL	NIL
17	KRISHNAN	61/M	35469	OLD AWMI	1124	14/1/09	13/2/10	6/3/10	40	0	40	0	O	0	0	YES	YES	YES	NIL
18	KANNIYAPPAN	M/63	363	OLD IWMI	389	12/5/09	14/7/09	14/3/10	0	99	0	156	7	15	178	NIL	YES	NIL	NIL
19	ISAAC	57/M	3779	OLD AWMI	1031	17/01/10	20/01/10	20/03/10	90	0	0	0	0	0	0	NIL	YES	NIL	NIL
20	JAYASHANKER	37/M	6614	OLD AWMI	1071	18/12/09	27/01/10	2/4/10	70	0	0	0	0	0	0	NIL	NIL	NIL	NIL
21	MANI	69/M	82592	OLD AWMI	924	25/5/09	19/12/09	8/3/10	40	80	70	0	0	0	0	NIL	NIL	NIL	NIL
22	MOHAMMED IBRAIM	57/M	14008	OLD AWMI	1179	12/12/09	24/02/10	13/4/10	70	0	90	0	0	0	0	NIL	NIL	NIL	NIL
23	ELLANGOVAN	49/M	18611	OLD AWMI	1255	22/1/10	12/3/10	7/4/10	70	0	0	0	0	0	0	NIL	NIL	NIL	NIL
24	RUKRUDEEN	50/M	23234	OLD AWMI	1283	16/1/10	29/02/10	3/4/10	90	0	0	26	0	0	26	YES	YES	NIL	NIL

25	DEVARAJAN	69/M	19054	OLD AWMI	1260	15/12/09	13/03/10	25/4/10	40	70	0	0	0	0	0	NIL	NIL	NIL	NIL
26	RAMALINGAM	68/M	17060	OLD IWMI	1261	1/1/10	13/03/10	22/4/10	0	0	80	166	18	5	189	NIL	NIL	NIL	NIL
27	KANTHASWAMI	60/M	17051	OLD AWMI	1262	6/1/10	13/03/10	17/4/10	90	0	0	137	0	0	137	NIL	NIL	NIL	YES
28	VASANTHA	49/F	19049	OLD AWMI	1263	28/12/09	13/03/10	19/4/10	70	30	0	0	0	0	0	NIL	YES	NIL	NIL
29	NALLANKILLI	42/F	19302	OLD IWMI	1280	3/1/10	15/03/10	3/4/10	90	0	0	0	0	0	0	NIL	NIL	NIL	NIL
30	MOHMAD IBRAHIM	M/57	14008	OLD AWMI	1180	21/2/10	24/2/10	2/4/10	70	0	90	37	60	98	195	YES	NIL	YES	NIL
31	ARUMUGAN	40/M	18053	OLD AWMI	1290	26/1/10	17/03/10	29/4/10	100	0	0	0	0	0	0	NIL	NIL	NIL	NIL
32	GOKULAKANNAN	35/M	19079	RESENT AWMI	1296	14/1/10	20/03/10	12/4/10	30	100	40	0	0	0	0	NIL	NIL	NIL	NIL
33	ANANDHI	26/F	19182	OLD AWMI	1500	10/3/10	3/5/10	13/5/10	90	50	0	0	0	0	0	NIL	YES	NIL	NIL
34	JAYARAMAN	52/M	23962	OLD AWMI	1285	4/1/10	30/3/10	14/9/10	100	100	70	237	60	148	445	NIL	NIL	NIL	NIL
35	BABU	55/M	23191	OLD AWMI	1040	19/11/09	22/1/10	27/3/10	0	0	80	16	1	1	18	NIL	NIL	YES	YES
36	KUMAR	40/M	3400	0LD IWMI	1025	2/1/10	18/1/10	23/3/10	80	0	100	221	152	19	393	NIL	YES	NIL	NIL
37	PRABAKAR	46/M	10192	OLD AWMI	1181	12/12/09	20/03/10	12/5/10	100	0	0	20	6	85	111	NIL	NIL	NIL	NIL
38	RAJU	59/M	24460	OLD AWMI	1352	3/1/10	31/3/10	1/4/10	0	0	70	237	60	148	445	NIL	NIL	YES	NIL
39	KRISHNAN	61/M	35469	REC AWMI	273	14/1/09	12/5/09	17/5/10	40	100	40	17	1	1	20	Y	Y	YES	NIL
40	KANNIYAPPAN	63/M	51219	OLD IWMI	390	10/10/08	14/7/09	14/5/10	70	99	0	156	7.5	15.1	178.6	NIL	NIL	YES	NIL
41	ARJUN	32/M	4697	OLD AWMI	1042	24/1/08	22/1/10	27/3/10	70	0	0	0	0	0	0	NIL	NIL	YES	NIL
42	ARUMUGA37M	37/M	70115	OLD IWMI	692	3/8/08	6/10/09	23/4/10	50	0	70	0	0	0	0	NIL	NIL	NIL	NN
43	JAYACHANDRAN	46/M	10100	OLD AWMI	270	12/12/09	11/5/09	12/5/10	100	0	0	0	0	0	0	NIL	NIL	NIL	NIL
44	KUMAR	25/M	10188	OLD AWMI	1305	13/1/10	22/3/10	26/4/10	70	0	0	0	0	0	0	NIL	NIL	NIL	NIL
45	MOHAMMED SHERIF	48/M	23192	OLD AWMI	1383	13/2/10	7/4/10	8/4/10	70	70	60	0	3	0	3	YES	NIL	YES	YES
46	ANANDHAN	M/66	4573	RECENT IWMI	1445	23/3/10	19/4/10	23/4/10	80	75	90	17	1	1	20	YES	NIL	NIL	NIL
47	RAMACHANDRAN	M/55	42165	OLD AWMI	777	5/8/09	23/10/09	2/4/10	70	70	60	2	0	0	2	NIL	NIL	NIL	NIL
48	SOMAN	59/M	25598	OLD AWMI	1385	10/2/10	7/4/10	8/4/10	80	50	0	254.64	9.39	139	403.43	NIL	NIL	NIL	NIL
49	VELVENDRAN	M/63	23190	OLD AWMI	1384	1/2/10	7/4/10	8/4/10	90	0	0	422	0	15	438	NIL	NIL	NIL	NIL
50	GOPAL	M/57	24453	OLD AWMI	1425	4/1/10	15/4/2010	17/4/10	90	0	70	488	0	5	493	YES	YES	YES	YES

MASTER CHART OF GROUP II POPULATION

S.NO	NAME	AGE/ SEX	IP NO	DIAGNOSIS	CAG NO	D.O.MI	D.O.CAG	D.O.CAC	LAD CAG	LCX CAG	RCA CAG	LAD CAC	LCX CAC	RCA CAC	TOTAL CAC	DM	HT	SMOKIN	FAMILY H/O CAD
1	VASUDEVAN	44/M	19111	OLD AWMI	1291	11/2/10	17/03/10	3/4/10	0	0	0	12	0	0	12	YES	NIL	NIL	NIL
2	VEERiAH	49/M	15328	OLD AWMI	768	14/10/09	21/10/09	3/4/10	0	0	0	0	0	0	0	NIL	NIL	YES	NIL
3	RAVICHANDRAN	50/M	12002	OLD AWMI	1144	9/11/09	17/2/10	13/4/10	0	0	0	0	0	0	0	YES	YES	NIL	NIL
4	KRISHNAMOORT	48/M	781578	OLD AWMI	1082	14/10/09	29/1/10	2/2/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
5	SUBRAMANI	41/M	23208	OLD AWMI	1085	20/1/10	6/2/10	5/510	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
	BASKAR	68/M	54679	0LD AWMI	1294	20/3/10	31/3/10	3/4/10	20	0	0	1573	0	37	1610	NIL	NIL	NIL	NIL
7	THANGARAJ	M/39	98890	0LD AWMI	919	30/9/09	19/12/09	12/5/10	0	0	40	14.94	0	0	14.94	NIL	NIL	YES	NIL
8	RAJU	59/M	24460	OLD IWMI	1353	17/12/09	31/3/10	1/4/10	0	30	0	160.6	25.5	206.3	392.5	NIL	NIL	NIL	NIL
9	RATHNAM	60/M	100964	OLD AWMI	1088	23/1/10	6/2/10	5/510	0	0	0	0	0	0	0	NIL	NIL	YES	NIL
10	ARJUN	34/M	4196	OLD AWMI	1031	25/2/08	19/1/10	26/4/10	0	0	0	252	40	130	423	NIL	NIL	NIL	NIL
11	JAYACHANDRAN	50/M	10152	OLD IWMI	1145	26/12/09	17/2/10	5/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
12	KUPPAN	55/M	29709	OLD IWMI	63	26/12/09	21/4/09	25/4/10	0	0	0	0	0	0	0	NIL	NIL	YES	NIL
13	ANSARI	M/32	4697	OLD AWMI	1043	20/1/10	22/1/10	16/3/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
14	RAMESH	20/M	25346	OLD AWMI	1379	19/12/09	6/4/10	26/4/10	0	0	0	542	0	304	846	NIL	NIL	NIL	NIL
15	PERUMAL	53/M	25344	OLD AWMI	1378	3/1/10	6/4/10	24/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
16	DANAPAL	61/M	26051	OLD AWMI	1377	19/12/09	7/4/10	12/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
17	JOSEPH	62/M	24829	OLD IWMI	1376	6/1/10	6/4/10	20/4/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
18	MOHMEED	45/M	26331	OLD AWMI	1391	7/12/09	9/4/10	13/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
19	GANESH	48/M	26337	OLD AWMI	1392	7/1/10	9/4/10	26/4/10	0	0	0	12	0	59	71	YES	NIL	NIL	NIL
20	SHANKAR	55/M	26345	RECENT	1393	22/12/09	9/4/10	17/4/10	0	0	0	0	0	2	2	NIL	YES	NIL	NIL
21	NATARAJAN	38/M	24751	OLD AWMI	1396	4/11/09	10/4/10	21/4/110	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
22	MURUGASEN	61/M	26778	RECENT	1397	17/8/09	10/4/10	18/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
23	RAJI	50/F	26806	OLD AWMI	1398	20/12/09	10/4/10	17/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
24	LALITHA	36/F	27160	OLD IWMI	1405	18/1/10	12/4/10	19/4/10	0	40	0	0	0	0	0	NIL	NIL	NIL	NIL

25	NAGARAJAN	47/M	27158	OLD IWMI	1406	28/1/10	12/4/10	16/4/10	0	0	0	12	14	0	26	YES	NIL	NIL	NIL
26	KALAISELVI	55/F	27159	OLD IWMI	1407	13/2/10	12/4/10	23/4/10	0	0	0	12	2	0	14	NIL	NIL	NIL	NIL
27	MANOGARAN	45/M	25141	OLD AWMI	1408	11/1/10	12/4/10	24/4/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
28	ANBU	33/M	18189	OLD AWMI	1224	26/1/10	11/3/10	27/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
29	SUNTHARAM	45/M	18410	OLD IWMI	1225	11/1/10	11/3/10	21/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
30	VEERAN	28/M	18786	OLD IWMI	1226	12/1/10	11/3/10	2/4/10	0	0	0	2	0	0	0	NIL	YES	NIL	NIL
31	ARJUNAN	45/M	18188	OLD AWMI	1227	27/12/09	11/3/10	18/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
32	ARJUN	34/M	4196	OLD AWMI	1032	25/2/08	19/1/10	26/4/10	0	0	0	0	0	0	0	YES	NIL	NIL	NIL
33	IRUTHYARAJ	51/M	20830	OLD IWMI	1263	12/2/10	20/03/10	2/4/10	30	0	0	0	0	0	0	NIL	NIL	NIL	NIL
34	SELVARAJ	55/M	21237	OLD AWMI	1267	13/1/10	22/03/10	9/4/10	0	0	0	0	0	0	0	YES	YES	NIL	NIL
35	DOSS	40/M	21433	OLD IWMI	1270	11/1/10	23/03/10	29/4/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
36	PRABAGARAN	46/M	21434	OLD IWMI	1271	23/12/09	23/03/10	28/3/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
37	SARAVANAN	45/M	20983	OLD AWMI	1274	10/2/10	23/03/10	28/3/10	0	0	40	21	0	0	21	YES	NIL	NIL	NIL
38	KUMAR	56/M	21679	OLD AWMI	1275	13/12/09	24/03/10	28/3/10	0	0	0	0	0	0	0	NIL	NIL	NIL	NIL
39	SRINIVASAN	56/M	21665	OLD IWMI	1276	1/2/10	24/03/10	28/3/10	0	0	0	31	0	12	43	YES	NIL	NIL	NIL
39	SRINIVASAN	56/M	21665	OLD IWMI	1276	14/2/2010	24/03/10	28/3/10	0	0	0	342	0	34	376	NIL	NIL	YES	NIL
40	SULTHAN	63/M	21431	OLD IWMI	1272	9/1/10	24/03/10	1/4/10	0	0	30	542	0	304	846	NIL	NIL	NIL	YES
41	BABU	58/M	23191	OLD AWMI	1193	19/11/09	29/02/10	2/3/10	O	0	0	57.25	52.4	11.27	120.92	NIL	NIL	NIL	NIL
42	SOWKATH ALI	48/M	25641	OLD AWMI	1382	13/2/10	7/4/10	8/4/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
43	KUMAR	35/M	22907	RECENT AWM	1194	5/1/10	29/02/10	14/4/10	0	0	0	83	1	304	388	NIL	NIL	NIL	NIL
44	RATHAKRISHNAN	53/M	24502	OLD AWMI	1302	13/11/09	31/02/10	5/4/10	O	O	O	0	0	50	50	NIL	NIL	NIL	NIL
45	PERUMAL	55/M	23949	OLD IWMI	1354	4/1/10	31/03/10	2/4/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
46	RAJENDRAN	50/M	13956	OLD IWMI	1305	19/12/09	31/02/10	14/3/10	0	0	0	0	0	25	25	YES	NIL	NIL	NIL
47	VENI	56/F	24768	CAD AWMI	1364	9/1/10	3/4/10	15/4/10	0	0	0	12	14	0	26	NIL	NIL	NIL	NIL
48	MUNISWAMI	60/M	25041	CAD AWMI	1365	24/1/10	3/4/10	23/4/10	0	0	0	0	0	0	0	NIL	YES	NIL	NIL
49	ARUMUGAM	50/M	25156	OLD AWMI	1372	12/2/10	5/4/10	12/4/10	0	0	0	21	0	12	33	NIL	NIL	NIL	NIL
50	NARAYANAN	62/M	21555	OLD IWMI	1264	14/1/10	20/3/10	1/4/10	0	0	0	0	0	0	0	YES	NIL	NIL	NIL