PREVALENCE AND PREDICTORS OF RENAL ARTERY STENOSIS IN HYPERTENSIVE PATIENTS UNDERGOING CORONARY ANGIOGRAPHY

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BRANCH - II

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CERTIFICATE

This is to certify that the dissertation entitled **"PREVALENCE AND PREDICTORS OF RENAL ARTERY STENOSIS IN HYPERTENSIVE PATIENTS UNDERGOING CORONARY ANGIOGRAPHY"** is a bonafide work done by **DR.S.SATHISHKUMAR**, Department of Cardiology, Government Rajaji Hospital and Madurai Medical College, Madurai in partial fulfillment of the University rules and regulations for award of DM degree**, Branch II (Cardiology)** under my guidance and supervision during the academic year from **August 2011** to **July 2014.**

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DECLARATION

I, **Dr.S. SATHISH KUMAR** solemnly declare that the dissertation titled **"PREVALENCE AND PREDICTORS OF RENAL ARTERY STENOSIS IN HYPERTENSIVE PATIENTS UNDERGOING CORONARY ANGIOGRAPHY"** has been prepared by me. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of D.M**., Branch II (Cardiology)** to be held in **August 2014.**

Madurai **Dr. S. SATHISH KUMAR**

Date :

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CONTENTS

Prevalence and predictors of renal artery stenosis in hypertensive patients undergoing coronary angiography

ABSTRACT:

SETTING: Department of Cardiology**,** Government Rajaji Hospital, Madurai, Tamilnadu.

OBJECTIVES: (i) To study 1) The prevalence of renal artery stenosis (RAS) in hypertensive patients with suspected coronary artery disease (CAD) undergoing coronary angigraphy by renal angiography. 2) Predictors of significant RAS. 3) Association between hypertension, CAD and RAS.

DESIGN: Single centre cross section observational study**.** This study was conducted among 75 hypertensive patients who were admitted in our hospital with diagnosed CAD for coronary angiography since December 2013.After completion of CAG, renal angiography was done selectively using the same judkins right catheter which was used during cardiac catheterization.

RESULTS: The mean age of the patients in our study is 59.5yrs.The Male: Female ratio-1.8:1. The prevalence of Renal artery stenosis in our case is 20% (15/75). Unilateral RAS is 17.33% (13/15). Bilateral RAS is 2.66% (2/15).Age >55yrs,female sex, Stage II hypertension, resistant hypertension, duration of hypertension, multivessel disease and the presentation of heart failure are statistically significant predictors of RAS. The presence of angiographically

documented CAD patients were (71/75).Only 4 patients had normal coronaries and all the 4 patients did not have RAS.

Conclusion: In our study the absolute number of patients is small. However the prevalence is significantly higher if hypertensive population with CAD is screened for ARAS. Female sex, elderly age, severe HT, triple vessel disease are significant association with ARAS. We recommend screening RAS in hypertensive CAD patients with high risk predictors.

Keywords: CAD, RAS, CAG, renal angiography

INTRODUCTION

Atherosclerotic renovascular disease is a frequently overlooked and potentially correctable disease. Unsuspected renal artery stenosis of varying severity coexists with coronary artery disease (CAD) patients. It is increasingly recognized that atherosclerotic renal artery stenosis (ARAS), accounting for about 90% of cases of renal artery stenosis is an important cause of renal insufficiency, refractory hypertension, and cardiac destabilization syndromes (unstable angina and flash pulmonary edema)

The clinical entity Peripheral arterial disease (PAD) is used to define stenotic, occlusive, and aneurysmal diseases of the aorta and its branch arteries. The important point is that coronary arteries are not included in the definition.[1]

The recommendation from American Heart Association scientific committee is that to screen high risk patients for renal artery stenosis as part of cardiac catheterization. These high risk patients are identified to be potential candidates of renal vascularization.[2]

The prevalence of renal artery stenosis (RAS) ranges from 3-30% in patients undergoing coronary angiography for suspected coronary artery disease. The prevalence of atherosclerotic RAS in Hypertensive patients undergoing coronary angiography is low, but substantially higher in patients with established peripheral (50%) and/or coronary artery disease (30%), and elderly population.[3]

RAS coexists with CAD of varying severity. There is a linear correlation between RAS and severity of CAD. It independently predicts mortality in CAD patients undergoing CAG. The increased mortality is mainly attributed by cardiovascular diseases. The coexistence of Hypertension, Coronary artery Disease and renal artery stenosis forms a deadly combination.

The indications for renal artery intervention are a matter of continuing controversy. It is no longer tenable to practice renal intervention indiscriminately. The real challenge is to identify high risk patients who would really benefit from revascularization. If the patient's renal function remains stable over a period of 6-12 months and if the Hypertension is under control, then patient can be maintained with medical management.

The best recommendation is for bilateral significant RAS and cardiac destabilization syndromes.

RAS accounts for 5% of all cases of Hypertension. Males are commonly affected than females. Population based studies showed 9% for males and 5% for females. When studied for coronary artery disease the incidence is 19%.when studied for peripheral disease the incidence is 35-50%. In half of the renal artery stenosis cases, bilateral disease is seen. Significant renal artery stenosis is when > 60% stenosis is seen. Over 5yrs the atherosclerotic plaque progress to complete occlusion in 15 % of the case. Atherosclerosis shows predilection to origin of renal arteries. The age group affected is in the middle age and elderly.[4]

AIM

Prevalence and predictors of renal artery stenosis in hypertensive patients undergoing coronary angiography

To Study

- The prevalence of renal artery stenosis (RAS) in hypertensive patients with suspected coronary artery disease (CAD), by renal angiography.
- Predictors of significant RAS.
- Association between hypertension, CAD and RAS.

REVIEW OF LITERATURE

Atherosclerosis is a systemic disease. It involves multiple vascular beds. Atherosclerotic Renal Artery stenosis is a under recognized clinical entity. Its coexistence with coronary artery disease (CAD) and peripheral artery disease is increasing as the ageing population is increasing.[5] ARAS has frequent association with Hypertension whether hypertension is a cause or consequence is still unresolved and unanswerable. It is also frequently associated with decreased renal function. The cardiovascular events are high in patients with renal artery stenosis and coexisting hypertension. Systemic Hypertension is found associated in unquantifiable numbers in ARAS. Secondly ARAS could be primary causative role for hypertension. The mechanisms are activation of renal angiotensin system, heightened sympathetic activity, oxidative stress, inflammation and fibrosis. The management of ARAS has only two options medical management and percutaneous transluminal renal angioplasty (PTRA). The Meta analysis assessed the cardiovascular events in patients with Hypertension and ARAS comparing drug versus PTRA. It concluded that PTRA has no superiority over medical therapy in preventing CV events, in reducing BP and improving renal function.[6] But there are specific groups bilateral

RAS, unexplained pulmonary edema, refractory angina and resistant Hypertension where PTRA has a definite role although this subgroup is not individually studied.[7]

Definitions: Hypertension:

Hypertension is defined as values >140 mmHg SBP and/or >90mmHg DBP, based on the evidence from RCTs that in patients with these BP values treatment-induced BP reductions are beneficial.

Definitions and Classification of office blood pressure levels (mmhg)

Category	**Systolic**		**Diastolic**
Optimal	<120	and	<80
Normal	120 - 129	and /or	80- 84
High normal	130-139	and / or	85 – 89
Grade – 1 Hypertension	140- 159	and/or	90-99
Grade – 2 hypertension	160- 179	and/ or	100- 109
Grade – 3 Hypertension	<180	and /or	<110
Isolated Systolic Hypertension	< 140	and	<90

The above classification cannot be applied to children and adolescents. It has special recommendations based on special criteria. It is very well adopted in the young, middle age and elderly groups.[8 9]

It is found that renal artery stenosis coexists in all stages of hypertension. The stenosis is hemodynamically significant, but it is more commonly recognized in patients with stage 2 or resistant hypertension. As these are the individuals who are clinically suspected and carefully evaluated for additional problems. If both kidneys show evidence of bilateral renal artery stenosis then there is a greater threat to the viability of kidneys. It can lead to reduced kidney function leading to ischemic nephropathy.

Changes in blood pressure classification

JNC 6 category	SBP/ DBP	JNC 7 category
Optimal	<120 /80	Normal
Normal Borderline	120 – 129 /80 -84 130 -139 / 85 -89	Pre hypertension
Hypertension	>140 /90	Hypertension
Stage 1 Stage 2 Stage 3	140 -159 / 90- 99 160 – 179 / 100-109 >180 /110	Stage 1 Stage 2

ARAS

Atherosclerotic Renal Artery Stenosis:

The criteria established to define RAS are

1) Renal angiographic stenosis ≥ 60% and < 100%

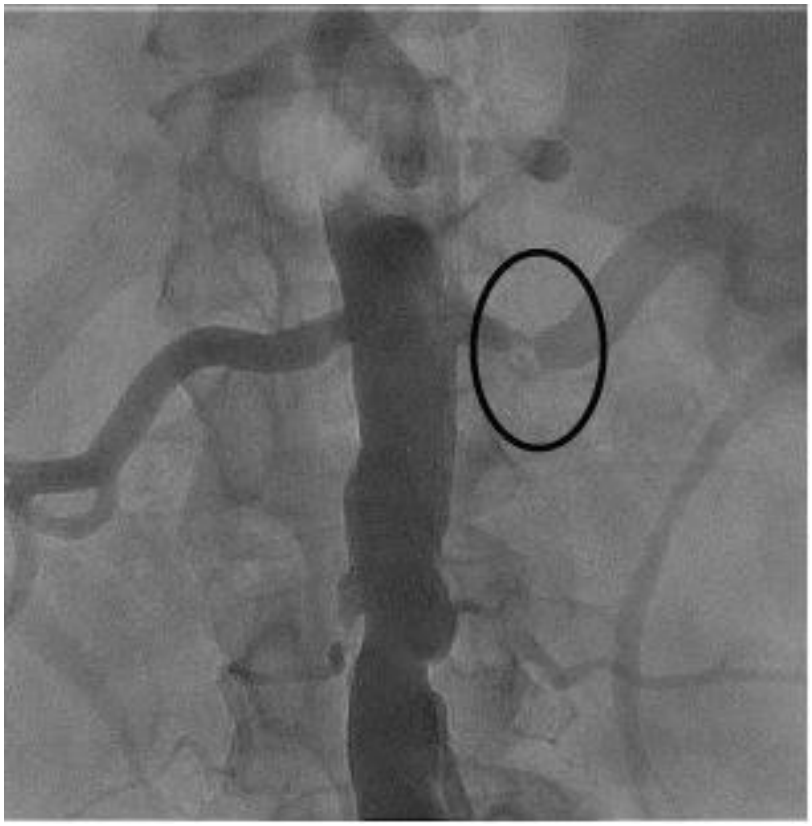
2) Systolic velocity of >300 cm/sec in duplex ultrasonography

3) Core lab approved MRA

4) Core lab approved CTA

Significant atherosclerotic RAS (ARAS), defined as a decrease of at least 60% in luminal diameter. The Quantitative angiography should show 60% diameter stenosis or a minimum luminal diameter less than 1.7mm.The Doppler criteria to define ARAS includes peak systolic velocity >320 cm/s. The renal aortic ratio of 3.8 is for diagnosis of ARAS.[10]

Renal artery disease (RAD) is defined as a stenosis of the main renal artery or its proximal branches. Atherosclerosis is the most common cause of renal artery stenosis, but may also be due to fibrous dysplasia of the renal artery. In children, most common cause of renal artery stenosis is due to fibromuscular dysplasia.



NON SELECTIVE RENAL ANGIOGRAM SHOWING ATHEROSCLEROTIC RENAL ARTERY STENOSIS

The anatomical definition to label renal artery stenosis is >50% occlusion but to be hemodynamically significant the stenosis should exceed by 70%. Hemodynamically significant lesions are the major concern as they may result in renovascular hypertension (RVHT) or ischemic nephropathy.

RVHT is defined as systemic hypertension secondary to hemodynamically significant renal artery stenosis. In unilateral renal artery stenosis (RAS), affected kidney increased renin secretion and it is suppressed by the contra-lateral kidney. Angiotensin II constricts blood vessels and leads to hypertension, and enhances the aldosterone synthesis. Aldosterone causes sodium and fluid retention. With sodium retention, volume expansion, and hypertension, the contralateral kidney responds with diuresis leading to sodium and water excretion to restore plasma volume to normal. In bilateral RAS, both kidneys increase renin secretion. Impaired kidney function rapidly increases plasma volume and with plasma volume, renin secretion will ultimately decrease. Sympathetic nervous system, nitric oxide, and others are involved in the development of renovascular hypertension.[11]

RVHT can be effectively treated with ACE inhibitors or ARBs, but may be complicated by an increased risk for adverse effects,

specifically an acute decline in GFR soon after beginning therapy. Ischemic nephropathy is defined as decreased GFR due to hemodynamically significant RAD.

Etiology of Renal artery stenosis

The etiology for renal artery stenosis is mainly contributed by atherosclerosis in >90% of the population and the rest by fibromuscular dysplasia .This is from western data. The scenario is entirely different in the Indian subcontinent up to 70% RAS is mostly caused by takayasu's arteritis. The atherosclerotic process in renal arteries is long standing leading to parenchymal injury and renal impairment and hence revascularization may not be beneficial. There is a real challenge in identifying patients who will benefit from renal revascularization.[4]

The ARAS involves the ostium, proximal one third and perirenal portion of aorta. The rate of progression of ARAS is not clearly evident from studies. In the 90s the data shows 51% progression at 5yrs after diagnosis an occlusion rate of 5% per year. The DRASTIC study showed 16% in the drug cohort progressed to ARAS at 1 year.[12] In a study of 1189 patients undergoing cardiac cathetrization11% progressed and 0.3% showed total occlusion.[13]

Fibromuscular dysplasia is seen in younger age. It is most commonly seen in females. It can involve all the three layers of vessel. It shows a classic “string of beads” appearance on Digital subtraction angiography. It is heterogeneous in its presentation and long term studies not available on the rate of progression. It responds well to balloon angioplasty. The rate of restenosis is less.

Etiology of renal artery stenosis and occlusion

Disease		**Prevalence**
➢ Atherosclerotic renal artery disease	-	85 – 90%
➢ Fibromuscular disease	-	~ 10%
➢ Acute renal artery occlusion (thrombosis, embolism , trauma)	-	< 2%
➢ Aortic dissection with renal Artery involvement	-	< 1%
➢ Takayasu / Giant cell Arteries	-	< 1%
➢ Mid – Aortic syndrome	-	Rare

The other causes are takayasu arthritis is relatively common in Indian subcontinent. It involves aorta and its major branches .the renal artery involvement in seen in 26% of the cases.[10]

Vascular Injury to the kidney

The renal circulation is unique. It is loaded with the function of filtering 140-150 liters of fluid daily. There is extensive glomerular capillary network. Even a slightest insult will present with predictors that will helpful to risk satisfy both renal as well as systemic disease.

The insult to kidneys, not only threaten the viability of kidney, but in large compromise the fluid & electrolyte homeostasis.

Hypertension, atherosclerosis, embolism, inflammation and hematological vascular diseases are the common causes.

The renal endothelial injury easily present as increased urinary albumin excretion. It serves as a marker of athero vascular disease (AVD).

Patho physiology

The genesis of atherosclerosis usually progress from clinically silent vasculopathy to adverse Cardiovascular (CV) events. The non modifiable risk factors include age, sex, birth weight and genetic factors.

The modifiable risk factors include hypertension, smoking, diabetes, dyslipidemia and obesity. All factors result in endothelial dysfunction.

The renal manifestations are HT and decreased GFR. The renin and sympathetic nervous system gets activated resulting in frequent flushing, nocturnal BP non dippers, and rapid BP swingers. Flash pulmonary edema is a common clinical manifestation of renal artery stenosis. RAS cases are more likely to suffer from CV events than ischemic nephropathy.

Renal artery stenosis, Hypertension and cardiovascular risk

The concept that many hypertensive populations do not have elevated blood pressure at the time of presentation and the CV risk should be taken into account of the global risk. The risk factors are additive and complement each other. The combination of ARAS, HT is a high risk category and its management includes aggressive management of risk factors and hypertension. The CV risk increases linearly from a blood pressure level as low as 115mm HG. There is a doubling of mortality for every 20mm Hg increase in systolic and every 10mm hg increase in diastolic BP.[8]

The cardiovascular mortality is on the decrease due to advances in the management particularly intervention is on the peak. Still ARAS is

silent killer of heart. It is an independent cardiovascular risk factor. It is turning out to be leading cause of death 16%year.[10]

The increased cardiovascular risk in ARAS is due to hypertension, decreased GFR and concomitant atherosclerosis in other CV beds. The mechanisms are due to RAAS and sympathetic activation. The RAAS system activation is a transient phenomenon and the blood pressure rise is sustained by alternative pathways such as inflammation, endothelial dysfunction and oxidatory pathways.[14] The levels of renin starts to decline as the perfusion pressures decreases that corresponds to a trans lesion gradient 10-20%.however the lateralized plasma renin assays are helpful in suggesting a significant kidney disease. The captopril renin assays has lost its place as it does not give a visual estimation of RAS. The test is no longer recommended as ARAS hypertension is not renin dependent.

Risk factors of RAS

The concept of risk factor is that it should detect future event risks of the particular system in the body. The major risk factors for the atherosclerotic disease are listed below.

Conventional risk factors	**other risk factors**
1. Hypertension	1. Obesity
2. Diabetes	2.Familyhistory
3. Male sex	3.Dyslipidemia
4. Smoking	
5. Advancing age	

Risk factor for ischemic renal disease

1. Hypertension: It can be cause as well as consequence. 35% of the patients with renal diseases have normal blood pressure.
2. Renal insufficiency
3. Diabetes
4. Smoking

The conventional risk factors are not useful in screening RAS. Screening RAS has been important as it helps in early intervention that helps ameliorate BP rise, prevent renal insufficiency and protect heart from cardio destabilization syndromes. These established risk factors for atherosclerotic diseases cannot be exactly extrapolated to renal artery disease.

There are emerging predictors of renal artery stenosis

1. Homocysteine
2. C –Reactive protein
3. Fibrinogen
4. Lipoprotein-A

The role for this emerging risk factor is helpful in detecting ARAS at an earlier stage .Modification of this risk factor with drugs like Statins may prevent progression of ARAS.[15]

Hypertension is known and well established risk factor. It damages the intimal layer and causes endothelial dysfunction. For every 20/10 mmHg blood pressure increase there is doubling of mortality for ischemic heart disease and stroke. The mortality risk starts with the blood pressure of 115/75mmHG. CAD, Hyperlipidemia and Hypertension are the most important predictors of renal artery disease.[16] ARAS has higher incidence of HT approximately 80% .Of the HT patients 60% approximately, are resistant HT. Bilateral renal disease presents with refractory HT. An interesting finding is that hypokalemia is associated with severe HT. Age greater or equal to 65 years, resistant

hypertension, type2 diabetes mellitus, multi vessel CAD are considered independent risk factors for ARAS.

The Chinese study tried to create a logistical model including risk factors for patients undergoing CAG. The risk factor included age >65yrs, gfr <60ml/min/m^2, multivessel CAD and resistant hypertension.

The equation is"P/ (1−*P*) = exp (−2.618+1.112 [age≥65 years] + 1.891 [resistant hypertension] + 0.453 [type 2 diabetes] + 0.587 [Ccr≤60 ml/min] +2.254 (multivessel coronary disease)"

This regression model has been used to screen ARAS with a sensitivity of 81.2%, specificity of 88.9%, and positive and negative predictive accuracies of 53.8% and 96.7%, respectively.[17]

Early recognition is extremely important in detecting this deadly disease. It is a progressive and the rate of occlusion between 10-20%.the clinical clues as given by guidelines should be picked up and investigated further. When the clinical suspicion is high and the other noninvasive tests inconsistent invasive renal angiography has been recommended. It can also be apart of coronary angiography if patients are found at increased risk for RAS.[18]

RAS and CAD

The incidence of RAS in CAD is higher compared to normal coronaries .The incidence tends to be higher in multivessel disease particularly in triple vessel disease. The coronary anatomy has higher predictive role in identifying ARAS. The screening for RAS has been recommended in those who have greater than two vessel disease. The other risk factors that have even higher risk are elevated systolic BP, Diabetes, and reduced eGFR.[18] The natural history for ARAS in asymptomatic cases are favorable, in fact intervention should be deferred.

Clinical features: Clues to suspect RAS

Clinical clues to renovascular disease include (1)onset of hypertension before age 30 (especially without a family history) or recent onset of significant hypertension after age 55; (2) an abdominal bruit, particularly if it continues into diastole and is lateralized; (3) accelerated or resistant hypertension;(4) recurrent (flash) pulmonary edema; (5)renal failure of uncertain etiology, especially with a normal urinary sediment; (6) coexisting diffuse atherosclerotic vascular disease, especially in heavy smokers; or (7) acute renal failure precipitated by antihypertensive therapy, particularly ACEIs or ARBs.

Clinical clues to the diagnosis of Atherosclerotic Renal Artery Stenosis :

1. Hypertension onset before the age of 30y or severe hypertension after the age 55yrs (class I . level of evidence (LOE) B)
2. Resistant, malignant and accelerated hypertension (class I, LOE C)
3. New azotemia or the development of worsening renal function after treatment with an angiotensin – converting enzyme inhibitor or angiotensin receptor blocker (class I, LOE B)
4. When there is no cause for atrophic kidney or discrepancy in size between the two kidneys > 1.5 cm between kidneys (class I , LOE B)
5. Development of sudden unexplained pulmonary edema (class I)
6. Unexplained deterioration of kidney function, including patients starting renal replacement treatment (Class II a, LOE B)
7. The presence of 3VD or Multi vessel coronary artery disease or peripheral arterial disease (class II b, LOE B)
8. The presence of Unexplained congestive heart failure or refractory angina (class II, LOE C).[1]

Investigations of RAS

It should begin with non invasive modalities. The most common screening tool is doppler USG. The sensitivity is 70%.

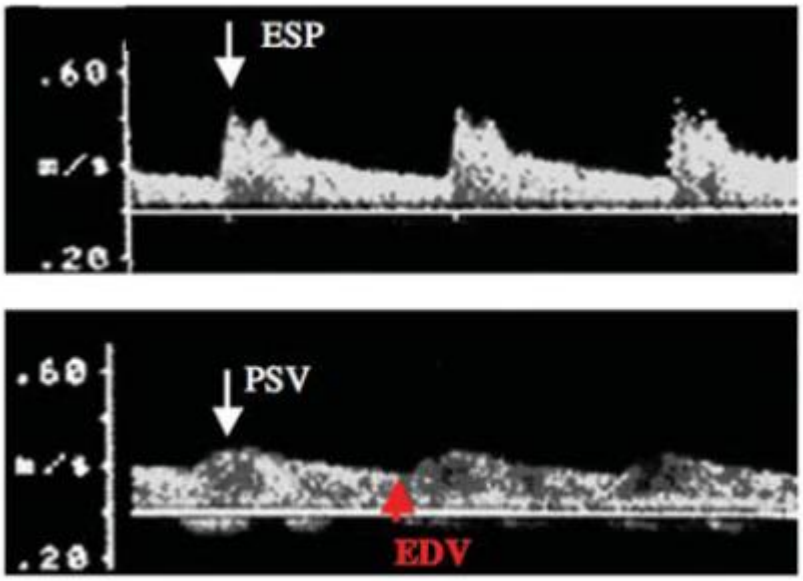
Doppler USG

The Doppler is a valuable tool in assessing significant RAS. This is a initial screening tool in evaluation of suspected RAS. The pulse Doppler spectrum of renal parenchyma shows early peak systolic velocity and the end diastolic velocity. Resistive index difference is defined as a ratio of end diastolic velocity to peak systolic velocity. A value of > 0.05 indicates significant RAS of >70%. The other indices helpful in suspecting RAS are Peak systolic velocity between 200 - 320cm/s or Renal Aortic ratio >3.5-3.8.

Measurement of intra renal resistive index is more valuable in assessing parenchymal disease. A RI < 0.8 is a prerequisite for revascularization in renal artery stenosis. It is a prognostic tool in assessing recovery following RAS stenting.

The contralateral kidney usually shows compensatory Hypertrophy. The absence of hypertrophy should arouse suspicion of bilateral disease. Hypertensive nephropathy and diabetic nephropathy are other causes since they result in parenchymal disease. [10]

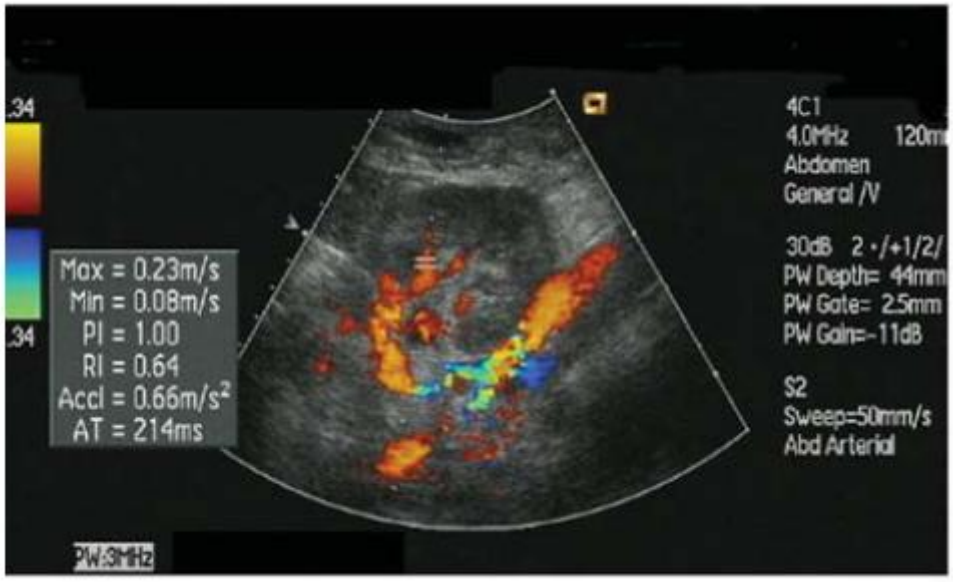
Normal intra-parenchymal Doppler signal
ESP: early systolic peak



Poststenotic Doppler signal
PSV: peak systolic velocity; EDV: end-diastolic velocity
RI difference > 0.05

In 2001, Radermacher et al evaluated 5950 patients with hypertension for renal artery stenosis using color doppler ultrasonography to predict the outcome of therapy for RAS. Resistance index can be used to select appropriate patients for treatment. Among138 patients with >50% of the luminal diameter underwent renal angioplasty or surgery and the procedure was successful in 131 (95%) patients. In 35 patients who had resistance index if least 80 before revascularization, mean arterial pressure did not decrease by 10mm Hg or more after revascularization in 34(97%). In 96 patients (73%) with a resistance index of <80, mean arterial pressure reduced at least 10% in all expect 6 patients. Study concluded that resistance index value of at

least 80 reliably identify patients with renal artery stenosis in whom surgical intervention will not help improving BP, renal function or kidney survival.[20]

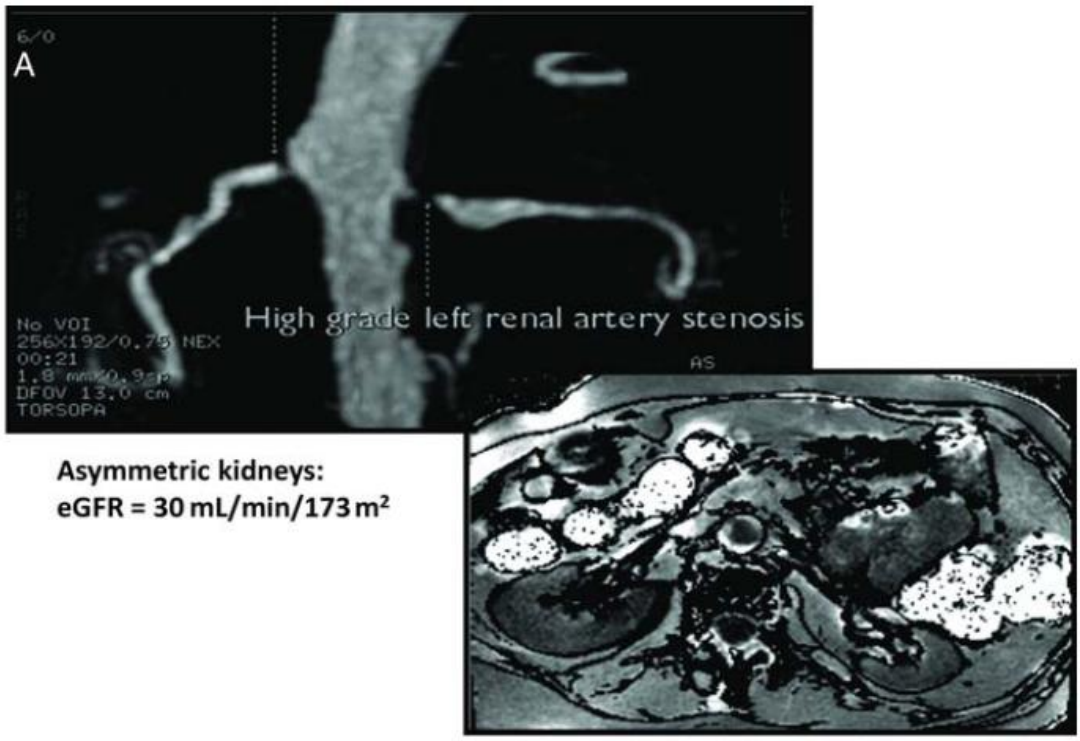


CT Angiography

CT angio has advantage of faster acquisition of images but the ionizing risk and nephrotoxicity is high. It delineates accurately smaller sized vessels. The sensitivity and specificity compared to renal angiography is 94% & 93% respectively.[21]It is a less invasive procedure. It has its own limitations; the presence of calcium obscures the luminal stenosis. Further it is not a physiological assessment of renal artery stenosis.

MR Angio

This topographic imaging gives anatomic and physiologic assessment of kidneys accurately. It validates glomerular filtration as well as renal blood flow. The cross sectional area of renal arteries is well assessed. A new emerging MR technique called BLOOD OXYGEN LEVEL DEPENDENT (BOLD) helps in greater functional assessment of ARAS. It assess the renal levels of deoxyhemoglobin thereby functional status of kidneys after the stenosis .The image below shows bilateral renal artery stenosis and asymmetric kidneys.[10]



Echocardiography

The echo is useful in assessing cardio vascular and renal risk.The ASE has recommendations that diastolic assessment is an important paramater in assessment of heart failure with preserved ejection fraction.Relative wall thickness is 2 x posterior wall thickness divided by end diastolic left ventricle dimension. It gives whether it is concentric or eccentric Left ventricular hypertrophy. The Left ventricular mass divided to body surface area has been more accurate in assessing organ damage.the Left atria volume has grater prognostic implications.

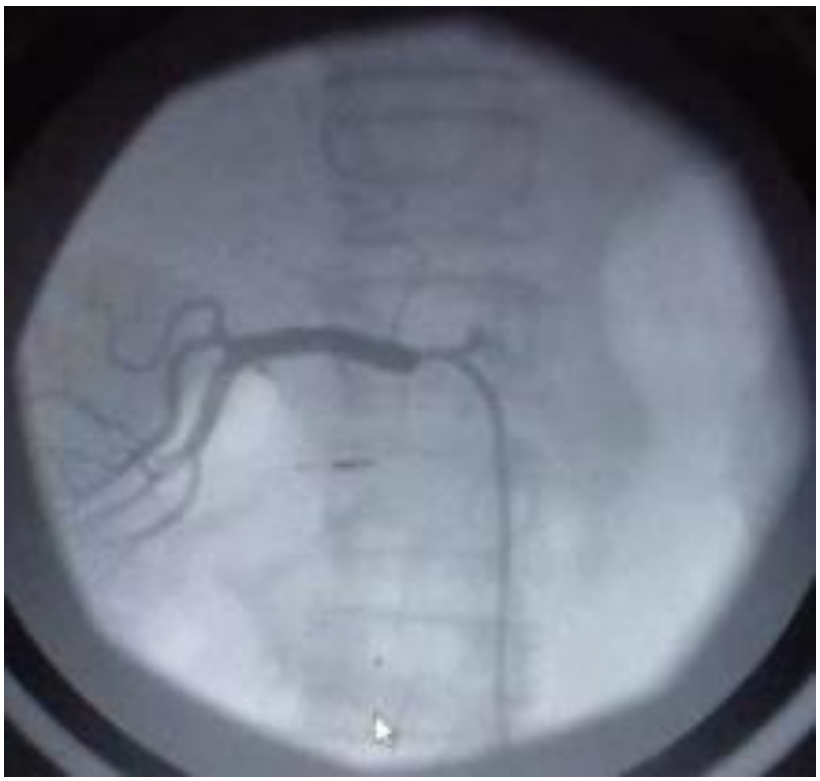
Renal Angiography

Anatomical Landmark L1 –L2 Vertebrae

Accessory renal artery is seen in 25% of the patients .I t usually supplies lower pole of kidney.

Renal angiography is the gold standard technique for imaging renal arteries. It is mostly required in ambiguous non-invasive imaging diagnosis of RAS or as apart of cardiac catheterization.

Renal Arteries originates from the Abdominal Aorta laterally .The access to the renal artery is done through, trans-femoral, trans-radial, trans-brachial routes. The take off arteries decides the route. Mostly after completion of cardiac catheterization, renal arteries are accessed



RIGHT RENAL ANGIOGRAM SHOWING
NORMAL RENAL ARTERY

commonly through the trans-femoral route. Downward angulation of renal arteries necessitates trans–renal or trans–brachial routes.

The renal angiography is initially done non-selectively by injecting contrast in a multi hold catheter like pig tail .After accessing renal arteries, through catheters like judkins right, internal mammary and hockey stick catheters selective renal angiography is done.

As the origin of the renal arteries is variable, the views should be placed such that the origin of renal arteries displayed in face. Usually AP view with slight LAO/RAO tilt is used. Caution should be exercised. No touch technique should be used, such that the atheroma in the aorta is not dislodged in to the renal artery during catheter manipulation.

In indeterminate renal angiography, the hemodynamic significance of the lesion is accessed by Trans stenotic gradient. It is achieved by using .014 inch FFR wire. The presence of systolic gradient more than 20mm Hg or a mean gradient of 10mm Hg is considered significant. The role of IVUS is very helpful, when per-cutaneous renal vascularization is planned. The IVUS gives greater image resolution of all renal artery sizes.

Once the severity of renal artery is accessed, the stenting can be performed according to AHA guidelines. The rate of complication of using catheter based angiography is very low.

The complications of renal angiography are

1. Athero-embolization
2. Contrast induced nephropathy
3. Access site complication/damage
4. Bleeding
5. Contrast allergy

Procedural Angiographic complications

Dissection

Branch vessel occlusion

Angiographic distal embolization

Wire perforation

Vessel rupture

Pseudo aneurysm

Pre-procedure preparation

1. Hydration with intravenous fluids before injection of contrast
2. Tablet N-Acetyl cysteine 600mg BD before and after the procedure
3. Iso–osmolar / non-ionic contrast
4. Maximal information obtained through non-invasive imaging studies
5. IV sodium bicarbonate may be considered

It is highly sensitive and specific test, but it is invasive. To reduce the radiation exposure, digital subtraction angiography should be considered.

Alternative angiographic techniques like carbon dioxide/ gadolinium contrast agents can be used.

The catheter size should be 5F or 6F of renal angiography.[22 23]

Percutaneous renal intervention

After the RAS is measured and quantified by radial angiography, the angioplasty balloon is advanced over the guide wire and positioned at the lesion. The balloon dilation should be at the ratio of 1:1.While removing the balloon after dilation the catheter should be pushed for correct deployment of stent. Distal protection devices can be used to protect the cholesterol embolization.

"SZABO technique is another safe and reproducible technique to exactly deploy stents at the aortic renal artery junction. This technique was initially used in coronary artery ostial lesions and can also be used in guiding and deploying renal stents into position at the aortic renal junction. Here, for the exact deployment, two 0.014 inch wire (with the second wire inserted through the last cell of a stent) are used for stent deployment. This stent tail wire or anchor technique facilitates precise aorta-ostial stent deployment in case of atherosclerotic RAS. It also

helps to eliminate errors of improper stent positioning at this aorto renal junction, and may possibly minimize patient exposure to ionizing radiation and contrast dye".

Screening ARAS: should renal angiogram (RAG) be a part of Coronary angiogram (CAG)

The AHA science advisory recommends screening renal angiography as part of cardiac catheterization on patients at high risk for ARAS who are potential candidates for renal intervention/surgical revascularization.[24] Is routine screening for ARAS worthful, useful? Although the procedure is safe and convenient whether it has a high diagnostic yield? Even if it yields does it leads to a successful outcome?. The committee advises against routine screening for RAS during cardiac catheterization except in patients at high risk for RAS.

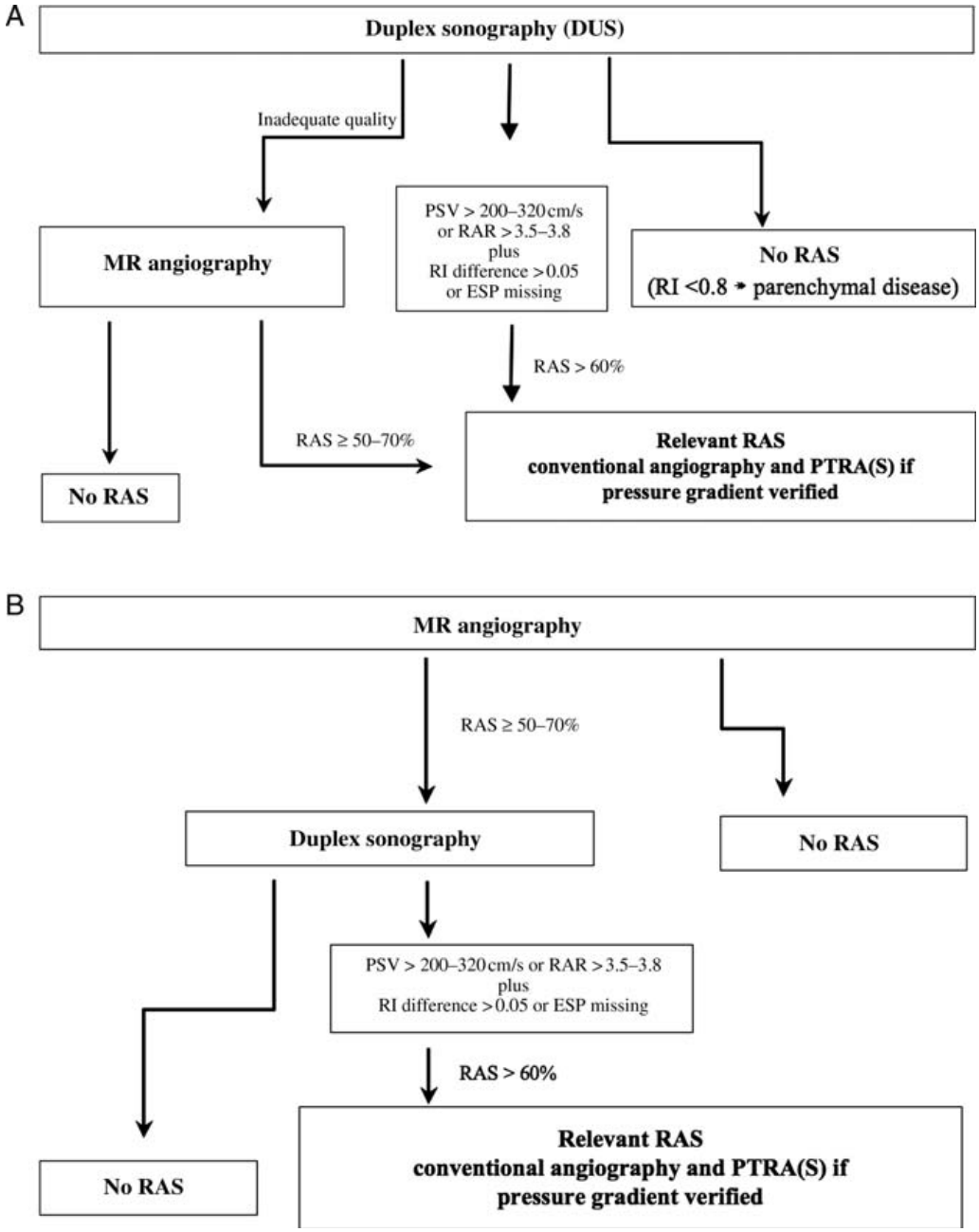
There are dangers of early intervention, the natural history of disease unclear. The editorial is against routine screening that may lead to over diagnosis and over treatment. The screening for RAS does not lead to increased survival.

Any screening program's purpose is to serve by improving survival. The patients on cardiac disease are already on maximal treatment. The additional diagnosis of ARAS had great enthusiasm among interventional cardiologists. They literally jumped in doing renal

intervention .But till today all the trials has failed to show renal stenting s' superiority over medical management.

But there are patients to be identified who would really benefit from revascularization. It is still a difficult question that is still unanswered.

Flow chart of primary renal assessment [10]



Management of RAS

All renal artery stenosis need not be stented and should not be. So far all the randomized control trials did not favor routine stenting .it is a controversy continuing with lot of hot debates? Interventional cardiologists show a trend towards percutaneous renal artery stenting. The arguments put forward favoring intervention is that the major trials are seriously flawed. The inclusion of RAS included lesions greater than 50% most clinicians recommend lesions >70% are significant; stenting should be preferred over balloon angioplasty, lot of crossover between groups. The current indications of stenting ARAS should follow AHA guidelines till large RCTs modify the current scenario.

ASTRAL

The Angioplasty and Stenting for Renal Artery Lesions (ASTRAL)

This trial was designed on the background that if renal artery patency is maintained then it should derive clinical benefit. But contrary to the expectations it did not show improvement in renal function which is a primary endpoint. This 34 yr median follow up study showed higher complication rates included death and limb loss. It did not find upper hand over medical management.

Authors' conclusions

"We found substantial risks but no evidence of a worthwhile clinical benefit from revascularization in patients with atherosclerotic renovascular disease". [25]

Star

"Stent Placement in Patients with Atherosclerotic Renal Artery Stenosis and Impaired Renal Function"

The aggressive medical management of RAS offers greater benefit compared to stenting. The exact mechanism how it acts on the intra renal compartment is not addressed. This study compared the two strategies. They concluded that renal artery stenting did not score over medical therapy. *"Stent placement with medical treatment had no clear effect on progression of impaired renal function but led to a small number of significant procedure-related complications*".[26]

Drastic trial

The experiments conducted by gold blatt showed renal constriction resulted in high blood pressure. Initially surgical revascularization was the only treatment option available for RAS. But the great enthusiasm that was shown towards renal angioplasties in treating RAS formed the background of the study. In the treatment of

patients with hypertension and renal-artery stenosis, angioplasty has little advantage over antihypertensive-drug therapy." The limitations in the study are

- The sample size was insufficient
- Balloon angioplasty without stenting was Used
- Renal artery stenosis was defined as greater than 50% stenosis
- Twenty-two of the 50 patients randomized to medical therapy crossed over to the angioplasty group

There was great success in fibromuscular dysplasia. But majority of RAS is due to atherosclerosis. The primary endpoint is control of BP. The study results shows no greater reduction of BP compared to medical management.[12]

Coral

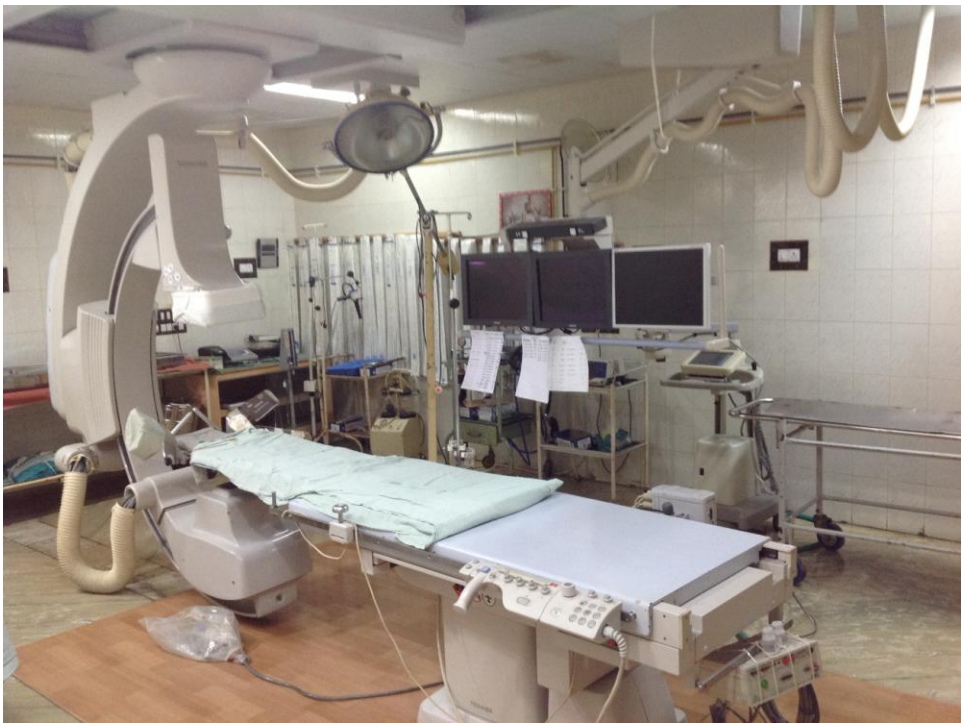
"A Randomized Multicenter Clinical Trial of Renal Artery Stenting in Preventing Cardiovascular and Renal Events": Results of the CORAL Study. It concluded "Renal artery stenting did not confer a benefit to the prevention of clinical events when added to comprehensive, multi-factorial medical therapy in people with atherosclerotic renal artery stenosis and hypertension or chronic kidney disease".

CORAL confirms the current ACC/AHA guidelines. “Medical therapy should be the initial therapy for renal artery stenosis and difficult to control blood pressure. In patients in whom blood pressure cannot be controlled on 3 medications, one of which is a diuretic or for patients who cannot tolerate medications and who have severe renal stenosis, renal artery stenting remains a reasonable option”.[27]

In 2000, Long term effects of balloon angioplasty on hypertension in patients with atherosclerotic renal artery stenosis were studied in 106 patients by Jaarsveld and his team. At three months, the blood pressures were similar in the two groups. Patients in the angioplasty group were taking 2.1 & 1.3 defined daily doses of medication and those in the drug- therapy group were taking 3.2 & 1.5 daily doses. 22 patients in the drug treatment group underwent angioplasty after 3 months because of persistent blood pressure despite of taking more than 3 drugs. At 12 months, there was no difference between both drug treatment and balloon angioplasty group in SBP, DBP, daily drug dose or renal function.

Dorres G et al in 2002 conducted multicenter trial in1058 patients with renal artery stenosis. Patients underwent revascularization procedure with palmaz stent and followed up for four years. At 4 years, SBP and DBP had significantly decreased with concomitant decrease in

number of drugs used. Cumulative probability of survival was 74% at 4 years. Survival was good for patients with normal (85% +/- 3%) baseline renal function, fair (78% +/- 5%) with mildly impaired renal function, and poor (49% +/- 5%) with severely impaired renal function (baseline creatinine > or =2.0 mg/dl). The combination of impaired renal function and bilateral disease adversely effected survival. Survival was adversely effected by renal dysfunction despite adequate revascularization.[28]



CARDIAC CATHETERIZATION LABORATORY

MATERIALS AND METHODS

This study was done with the aim of screening renal artery stenosis in patients with hypertension who are admitted in our hospital for coronary angiography and predicting risk factors association with ARAS.

Place of study	Department of cardiology, Government Rajaji hospital and Madurai medical college, Madurai.
Type of study	Observational analytical study
Institutional ethics committee	Approval obtained
Collaborating departments	Department of Biochemistry
Period of Study	6 months
Financial Support	Nil
Conflict of Interest	Nil

STUDY POPULATION:

This study was conducted among 75 Hypertensive Patients admitted for coronary angiogram / intervention in cardiology ward at the Government Rajaji Hospital, Madurai, after getting the institutional ethics committee approval. Informed and written consent was obtained from each patient before being included in the study.

Inclusion Criteria

Patients admitted with a history of Hypertension and planned CAG / intervention for suspected/diagnosed CAD in cardiology ward.

Exclusion criteria

1. Doesn't opt for inclusion in the study
2. Pregnancy
3. Age less than 30yrs
4. Chronic renal insufficiency GFR <60ml/min/m2
5. Patients with EF less < 30%
6. History of allergy

Data Collection and Methods

CLINICAL analysis includes presence of signs of hypertension, CAD and RAD. etc. LABORATORY PROFILE study includes blood counts, urea, creatinine, lipid profile, ECG, echocardiogram for all patients, treatment details to be collected including drugs for HT, Heart and kidney. CAG and Renal angiogram

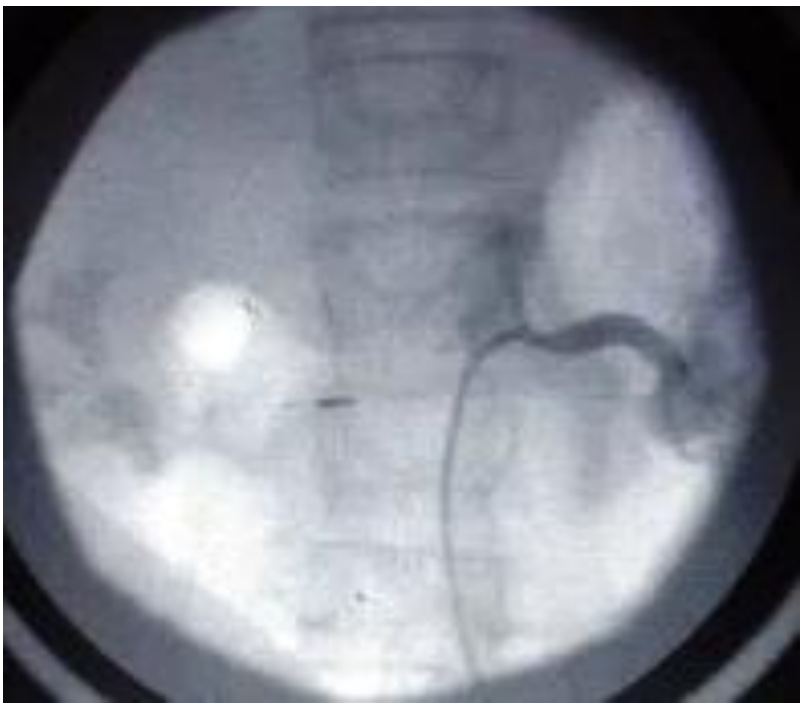
OUTCOME includes identify patients with high risk clinical predictors and prevalence of significant RAS thereby recommend Screening in that subgroup and planned revascularization.

PROCEDURE:

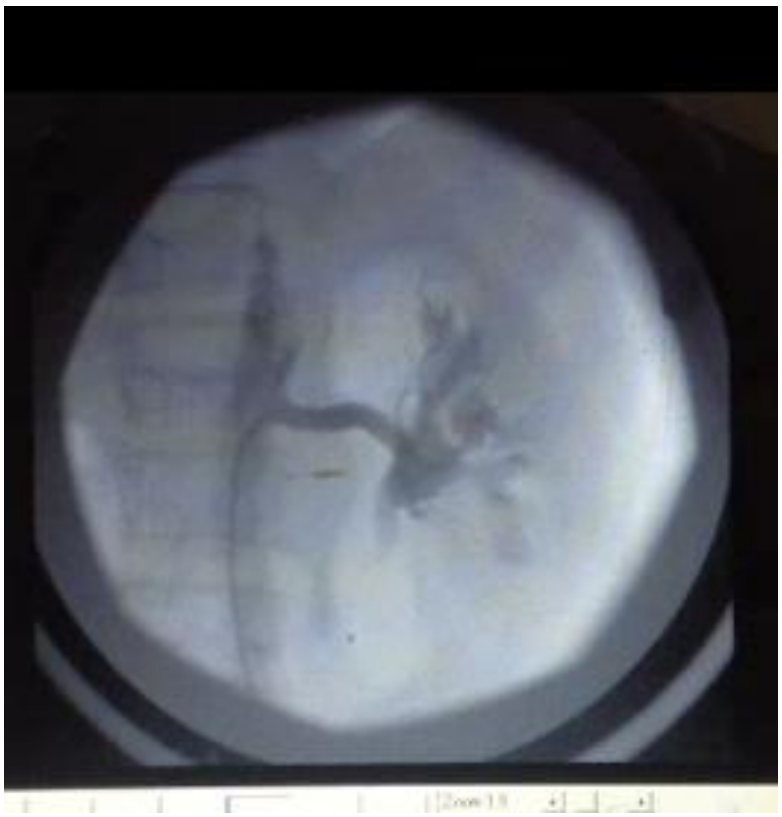
The patients undergoing cardiac coronary angiography, renal angiography is planned after completion of the CAG. Immediately after injecting contrast in right coronary artery the catheter (Judkins right) is slowly brought down in the aorta to the anatomical land mark of renal arteries(L1_L2 vertebrae).The renal arteries are selectively cannulated and renal angiogram is completed. In difficult cases non-selective renal angiogram is done using pig tail catheter.

Statistical Analysis:

The information collected regarding all the selected cases were recorded in a master chart. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012). Using this software, range, frequencies, percentage, mean, standard deviation and ‘p’ value were calculated through One way ANOVA, Chi square, Pearson and Spearman Correlation test and P value of < 0.05 was taken as significant.



SELECTIVE LEFT RENAL ANGIOGRAM USING JUDKINS RIGHT CATHETOR SHOWING RENAL ARTERY STENOSIS IN THE PROXIMAL ONE THIRD



RENAL ANGIOGRAM AFTER

RENAL ARTERY STENTING

RESULTS

Table – 1

Age Distribution

Age in Years	Total no. of cases	RAS
< 40 years	4	0
41 – 55	22	1
56 – 70	43	11
> 70 years	6	3
Total	75	15

Mean Age - 59.5 years

Range - 34-80 years

< 55 years - 1 / 26

> 55 years - 14 / 49

'p' value - 0.033 Significant

In this study, Out of 75 cases, 26 cases in less than 55 years age group and 49 cases in more than 55 years age group.

When compared with age group, most of the RAS cases (93%) is in > 55 years age group. It is also statistically significant i.e. 'p' value is 0.033.

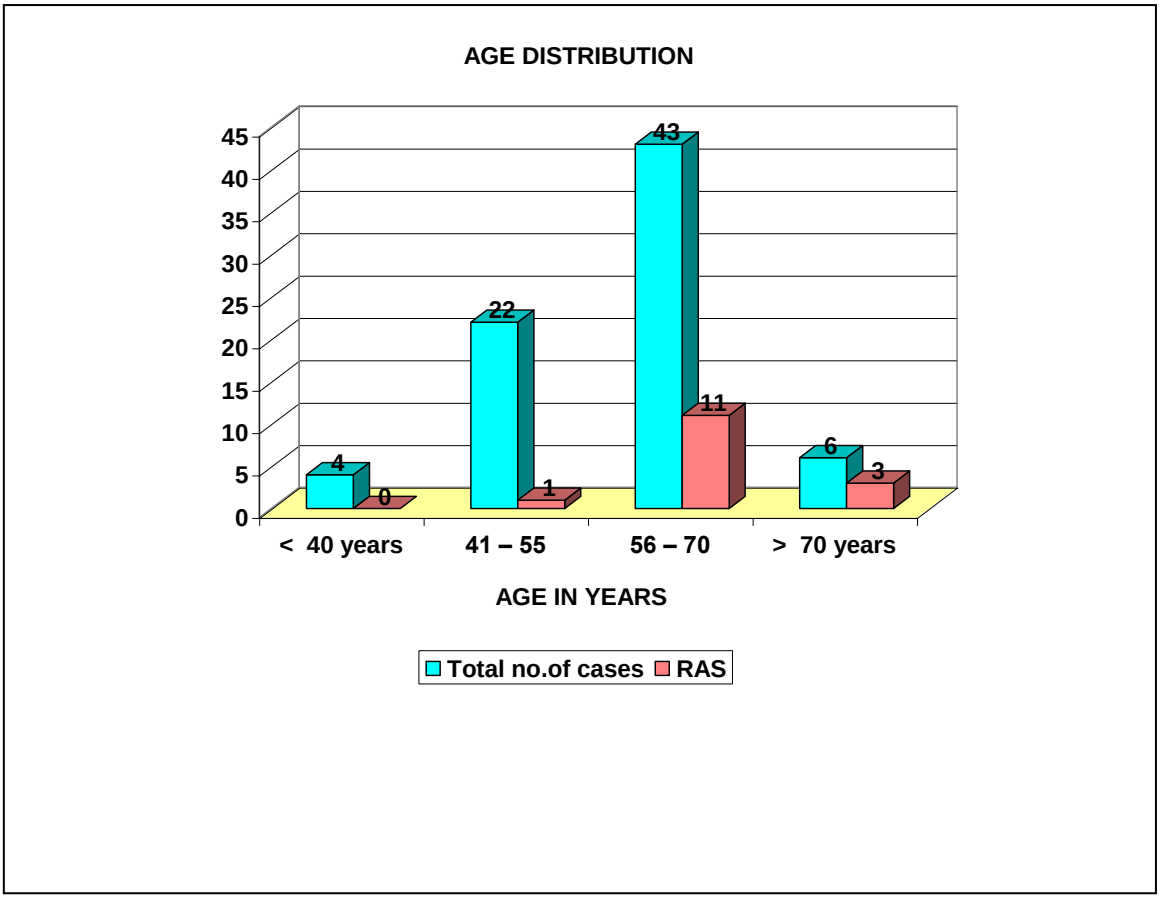

AGE DISTRIBUTION
45
40
35
30
25
20
15
10
5
0
4
0
22
1
43
11
6
3
< 40 years
41 – 55
56 – 70
> 70 years
AGE IN YEARS
Total no.of cases
RAS

Table – 2

Sex Distribution

Sex	Total no. of cases	RAS
Male	49	5
Female	26	10

Male - 5 / 49

Female - 10 / 26

'p' value - 0.043 Significant

Male: Female ratio - 1.8

In this study, males are participated more when compared with females (65% / 35%)

When compared with RAS, out of 26 females, 10 cases in RAS but only 5 cases (RAS) in males out of 49.

It is also statistically significant 'p' = 0.043

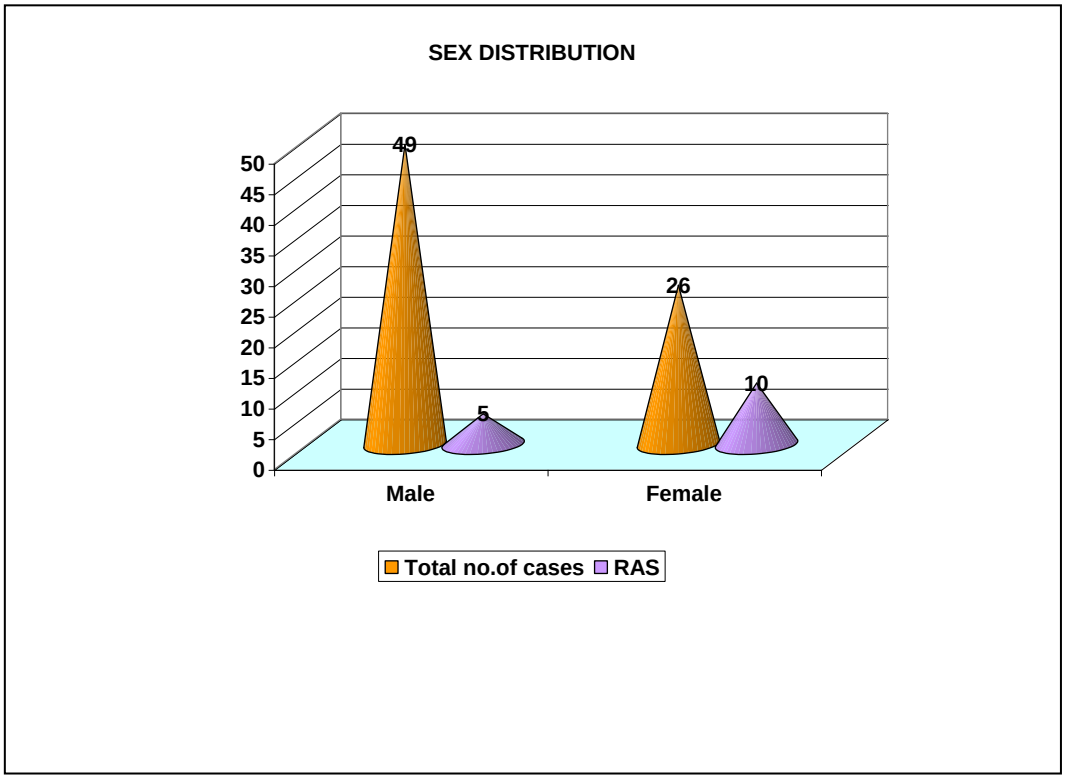

SEX DISTRIBUTION
50
45
40
35
30
25
20
15
10
5
0
49
5
26
10
Male
Female
Total no.of cases
RAS

Table – 3

Prevalence of Renal artery stenosis (N = 75 cases)

	No. of cases	Percentage
Unilateral	13	17.33 %
Bilateral	2	2.66 %
Total	15	20 %

Table – 4

Smoking, Diabetes and Hyperlipidemia

Parameters	Total no. of cases	RAS	'p' value
Smoking	20	4	0.749 Not Significant
Diabetes	19	6	0.400 Not Significant
Hyperlipidemia	13	5	0.289 Not significant

Smoking, Diabetes and Hyperlipidemia are not significantly affecting RAS factor.

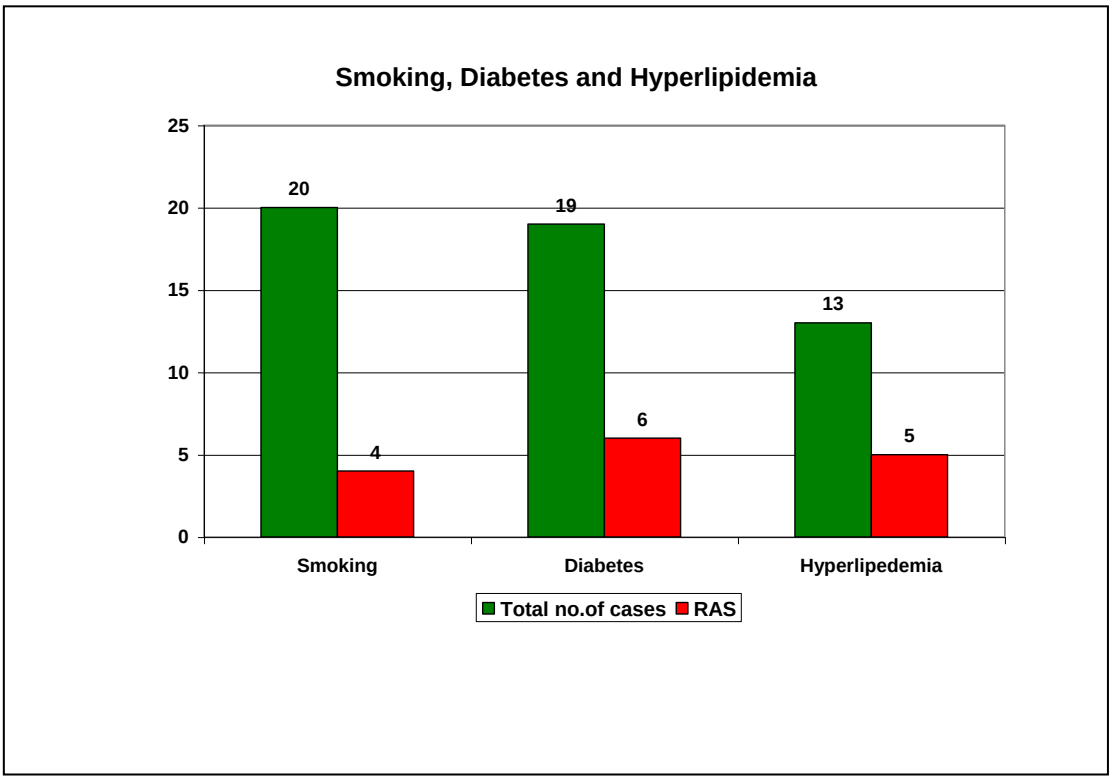

Smoking, Diabetes and Hyperlipidemia
25
20
15
10
5
0
20
4
19
6
13
5
Smoking
Diabetes
Hyperlipedemia
Total no.of cases
RAS

Table – 5

Hypertension Stages

Stages	Total no. of cases	RAS
Stage I	39	3
Stage II	36	12
Total	75	15

Stage I - 3 / 39

Stage II - 12 / 36

'P' value - 0.047 Significant

Association of RAS and Resistant HT:

There was (39/75) cases of stage I HT and the presence of RAS (3/39) were not significantly increased. There was (36/75) cases of stage II HT and the presence of RAS (12/36) were significantly increased.

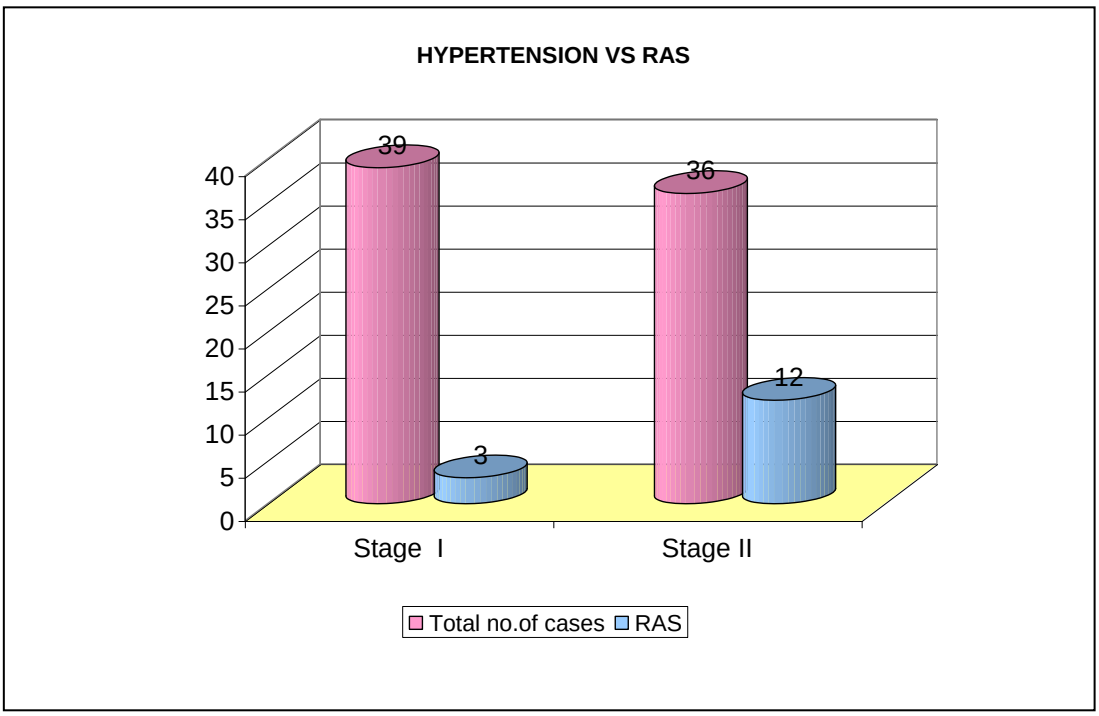

HYPERTENSION VS RAS
40
35
30
25
20
15
10
5
0
39
3
36
12
Stage I
Stage II
Total no.of cases
RAS

Table – 6

Resistant HT

	Total no. of cases	RAS
Resistant	5	4
Non Resistant	70	11
Total	75	15

Resistant - 4 / 5

Non Resistant - 11 / 70

'p' value - 0.039 Significant

Association of RAS and Resistant HT:

There was (5/75) cases of resistant HT and the presence of RAS (4/5) was significantly increased. The resistant HT is a powerful predictor of RAS in our study. The resistant HT cases had a dramatic course of adverse CV events.

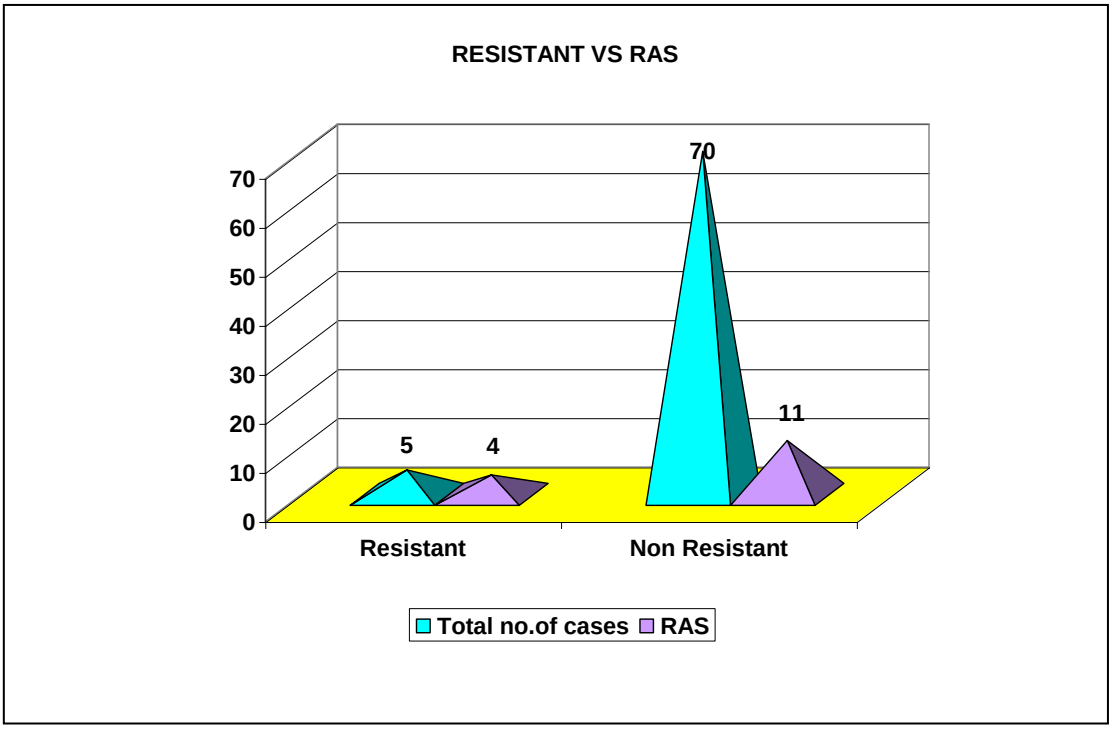

RESISTANT VS RAS
70
60
50
40
30
20
10
0
5
4
70
11
Resistant
Non Resistant
Total no.of cases
RAS

Table – 7

Duration of HT

Duration	Total no. of cases	RAS
0 – 6 months	15	1
7 months - 2 years	28	2
2 years - 5 years	20	5
5 years – 10 years	11	5
> 10 years	1	1
Total	75	5

'p' value - 0.027 Significant

Association of RAS and duration of HT:

The duration of hypertension has linear correlation. As duration of hypertension increases above 5yrs there is increased prevalence of RAS. There were (11/75) cases of HT with duration >5yrs and the presence of RAS (5/11) was significantly increased. There were (1/75) cases of HT with duration>10 yrs, and the presence of RAS (1/1) was significantly increased.

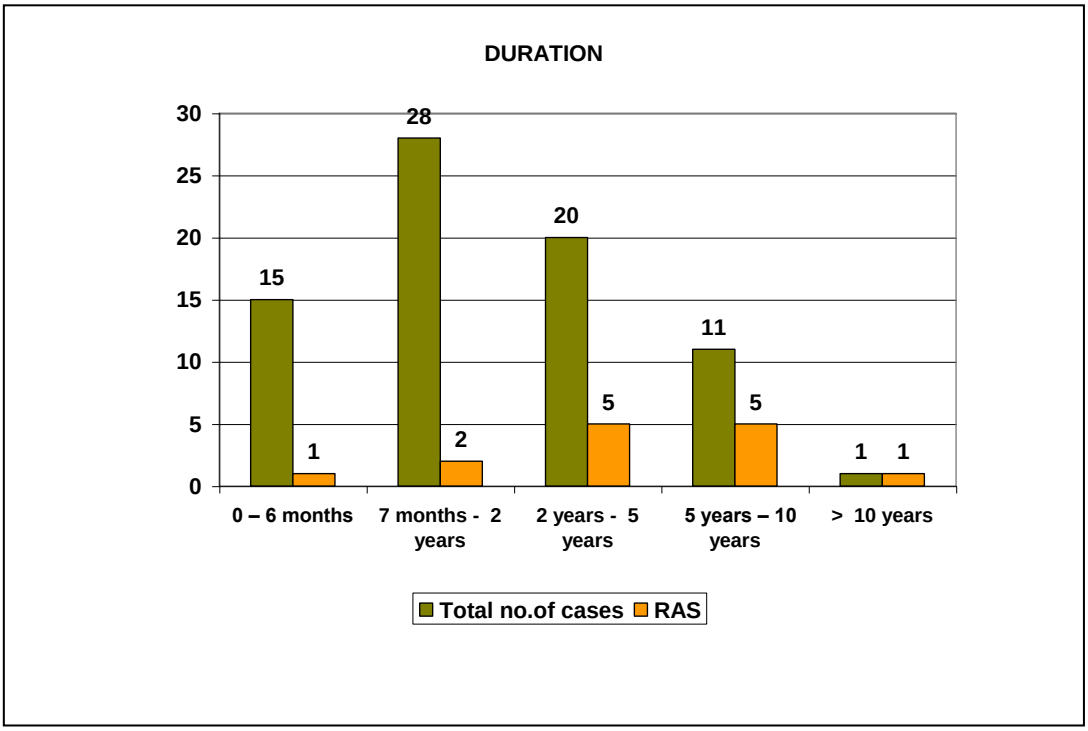

DURATION
30
25
20
15
10
5
0
15
1
28
2
20
5
11
5
1
1
0 – 6 months
7 months - 2 years
2 years - 5 years
5 years – 10 years
> 10 years
Total no.of cases
RAS

Table – 8

CAD vs RAS

	Total no. of cases	RAS
CSA	11	2
UA / NSTEMI	22	5
STEMI	42	9

CSA - 2 / 11

UA/NSTEMI - 5 / 22

STEMI - 9 / 42

'p' value - 0.952 Not significant

Association of RAS and clinical presentation of Coronary artery disease:

Most of the cases presented as STEMI (42/75). There was no significant association between clinical presentation and presence of RAS. There was (22/75) cases of unstable angina and the presence of RAS were not significantly increased. There was (2/11) cases of stable angina and the presence of RAS were not significantly increased

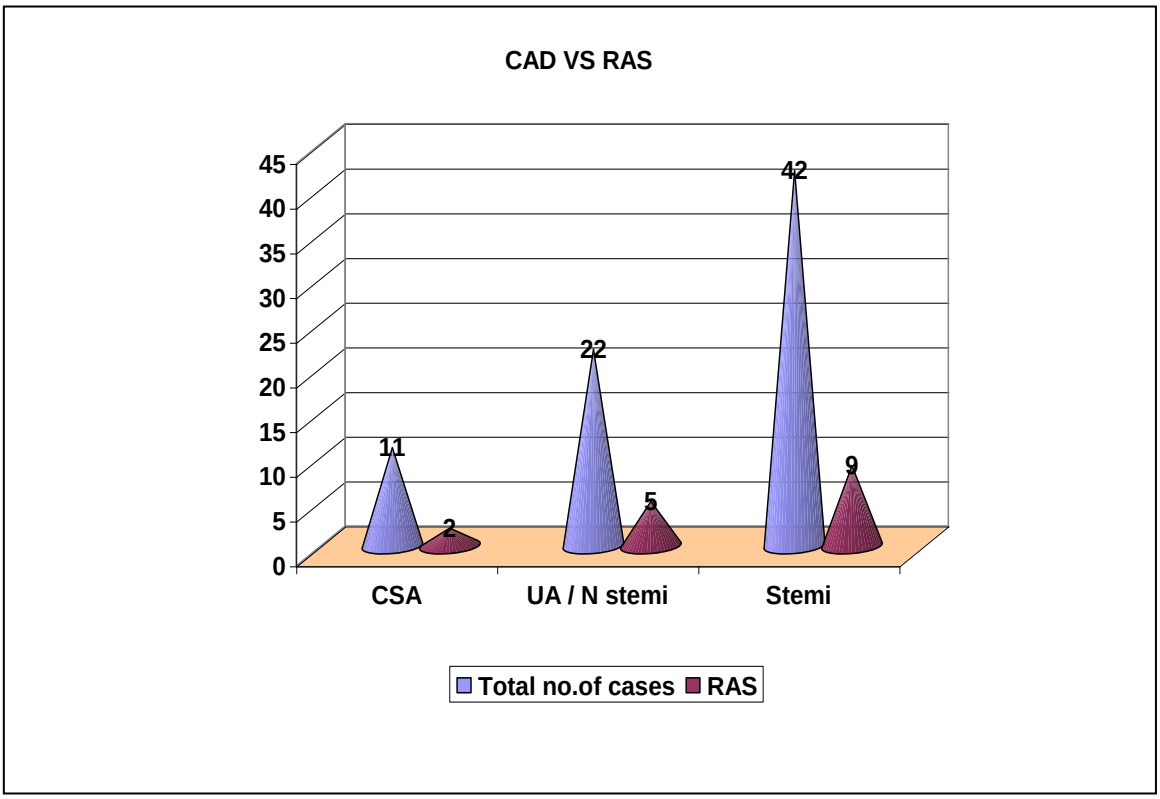

CAD VS RAS
45
40
35
30
25
20
15
10
5
0
11
2
22
5
42
9
CSA
UA / N stemi
Stemi
Total no.of cases
RAS

Table – 9

Heart Failure / pulmonary edema vs RAS

HF / PE	Total no. of cases	RAS
Yes	15	10
No	60	5
Total	75	15

HE/PE Yes - 10 / 15

No - 5 / 60

'p' value - < 0.001 Significant

Association of RAS and clinical presentation of Coronary artery disease as heart failure/pulmonary edema:

The cases presented as heart failure are (15/75). There was significant association between clinical presentation as heart failure and presence of RAS.

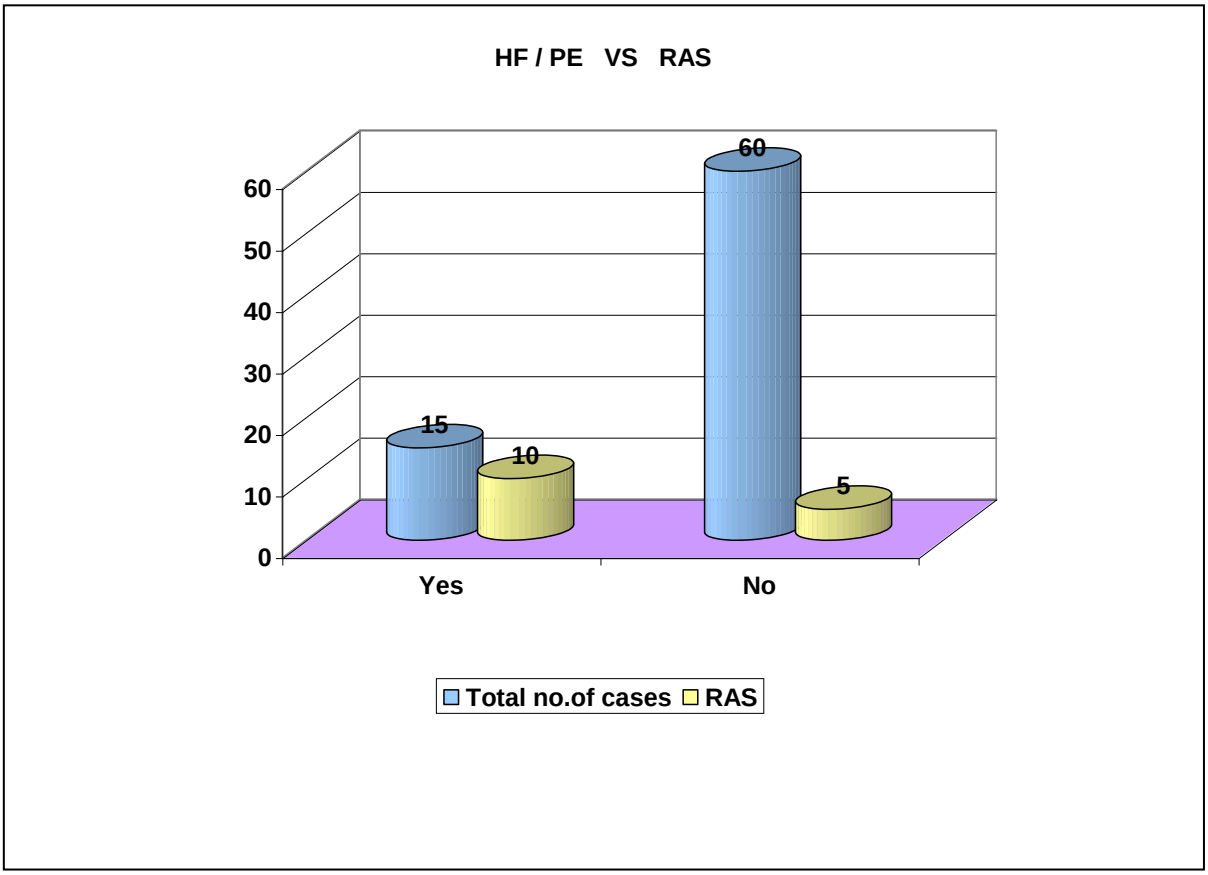

HF / PE VS RAS
60
50
40
30
20
10
0
15
10
60
5
Yes
No
Total no.of cases
RAS

Table – 10

CAD/Angio vs RAS

CAD/Angio	Total no. of cases	RAS
SVD	34	3
2VD	18	3
3VD	17	9
LMCA	2	0
Normal	4	0
Total	75	15

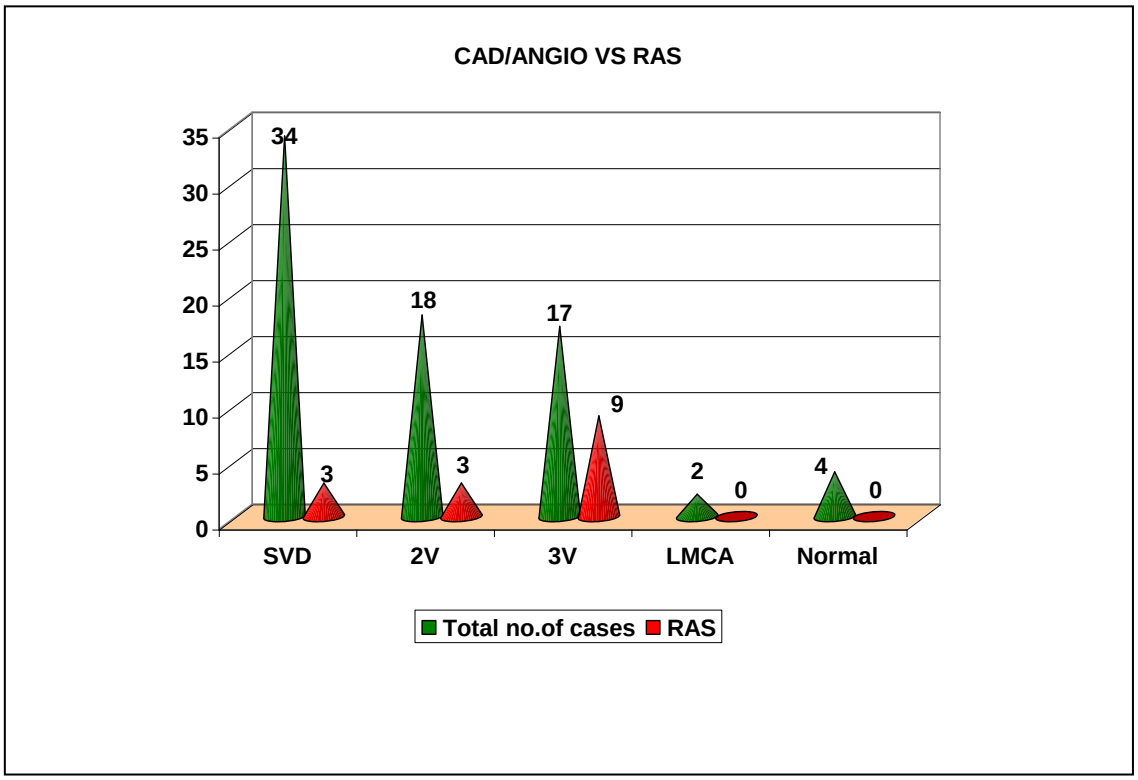
3VD - 9 / 17

Others - 6 / 58

'p' value - 0.009 significant

Association of RAS and angiographic presentation of Coronary artery disease:

Most of the cases presented as single vessel disease (34/75). There was no significant association between SVD presentation and presence of RAS (3/34). There was (18/75) cases of 2Vd and the presence of RAS (3/18) were not significantly increased. There was (17/75) cases of triple vessel disease and the presence of RAS (9/17) were statistically significantly increased.


CAD/ANGIO VS RAS
35
30
25
20
15
10
5
0
34
3
18
3
17
9
2
0
4
0
SVD
2V
3V
LMCA
Normal
Total no.of cases
RAS

DISCUSSION

Prevalence and predictors of RAS

RAS is a major comorbid and independent risk factor for cardiovascular disease. The western data shows a prevalence of 13.5-18% in patients undergoing Coronary angiography for suspected coronary artery disease. Less data is available from the Indian subcontinent.

The prevalence of RAS in our study conducted over 75 patients is 20% (15/75). The mean age is 59.5 yrs. Most of the patients are males. STEMI is the common clinical presentation. The conventional risk factors like smoking, diabetes and dyslipidemia did not find significant association. The stage II Hypertension predicted RAS. The coexistence of resistant hypertension and heart failure increases the predictability of RAS in the study group.

The prevalence of RAS in Japanese population undergoing CAG is 7%.In the sub group analysis the prevalence of RAS was 5%, 10%, 9% and 19% for 0, SVD, 2VD, 3VD respectively. The prevalence in hypertension population was 13% and 2% in non hypertensives.[29]

In the sub group analysis of our study, the prevalence of RAS was 4%, 4%, and 12% for SVD, 2VD, 3VD respectively. We therefore find that screening for RAS should be a part in triple vessel disease and Hypertensive population.

RAS has deleterious effect on heart and portends poor prognosis. It is high time that cardiologist should perceive atherosclerosis as a global risk involving multiple beds. The aggressive nature of interventional cardiologist is to be justified. Atherosclerotic RAS is the most common cause for secondary hypertension.

The Indian study conducted over 469 patients underwent coronary angiogram were screened for RAS by conventional renal angiography. The prevalence of RAS was 66/469 and 36 had significant ARAS. Of the 36 RAS patients 33 had significant CAD and had no CAD patients.[30] There is always a debate on which patients renal angio should be used for screening RAS. The renal angiography has been recommended on patients with atherosclerosis on coronary and peripheral beds.

It is also recommended on patients who undergo coronary/peripheral angiogram to screen for ARAS if they have severe atherosclerosis, resistant hypertension, flash pulmonary edema and triple vessel disease.

Another Indian study by PC Rath et al concluded that the incidence of RAS was statistically not significant but showed increased incidence among multiple risk factors and severe coronary artery disease. This study showed hypertension, diabetes smoking, females, elderly age and triple vessel disease as significant risk factors. The prevalence for significant RAS over 1000 patients was 2.5%.[31]

The patients underwent selective renal angiography. It was performed in all the patients after CAG. 84.9% of the patients had significant coronary artery disease. Of the total patient cohort, 103 (10.3%) patients had renal artery disease. But significant RAS over 1000 patients was 2.5% only.

This study made us to screen RAS in a subset of patients such that unnecessary renal angio could be avoided in a vast majority of patients. Though renal artery stenosis can be present in non hypertensives and normal coronary patients the number is too low. Hence we excluded this population from our study. We have also excluded PAD in our study as to reduce access site complications.

Our study found age >55yrs, female sex, duration of HT, 3VD, and resistant HT as predictors of RAS.

Though renal artery stenosis is not recognized at its first presentation, it is subsequently recognized at follow ups. Renal artery stenosis is increasingly recognized to be an independent predictor of survival in coronary artery disease. The prevalence compared to western data is relatively low.

Therefore long term follow up of patients with CAD is needed. Our study is only a cross sectional observational study. Since routine renal angiography is not warranted as screening purpose, we propose screening as part of CAG in selected subset like, 3VD, resistant HT heart failure/pulmonary edema

Rimoldi SF et al, in 2010, retrospectively studied 1403 patients who underwent drive-by renal arteriography. The hypertensive population is the subset. It was conducted to determine the prevalence and predictors in unselected hypertensives. The prevalence of significant RAS was 8%. Independent predictors were coronary (OR 5.3%), peripheral (OR 3.3), and cerebral artery (OR 2.8) impaired renal function (OR 2.9). It was concluded that unselected patients undergoing coronary angiography was a low.[32]

In our study we selected a Hypertensive population since the prevalence is very low in unselected patients. We excluded normal

coronary patients from screening.

In 2001, Shurrab and colleagues have reviewed 249 patients undergoing angiography over the period of 18 months to identify the patients having the greatest likelihood of atherosclerotic renovascular disease. 166 (66.7%) patients had no evidence of ARVD, while only 83 (33.3%) patients showed some degree of ARVD, 29 (35%) of which had bilateral renal artery disease. There was no significant difference between the ARVD group and the non-ARVD group for mean age (69.0 years vs 63.3 years), male to female ratio, history of smoking (68.7% vs 55.4%), severe hypertension (10.8% vs 9.0%), hypercholesterolemia (61.4% vs 47.0%), diabetes mellitus (28.6% vs 25.3%) or angiotensin converting enzyme inhibitor-related renal dysfunction (9.6% vs 6.1%).

The other risk factors diabetes, smoking and dyslipidemia has no significant association with RAS.[33]

Conlon PL et al investigated the effect of the severity of RAS on all-cause mortality with total of 3987 patients undergoing abdominal aortography immediately after coronary angiography. Significant RAS was found to be present in 4.8% of patients was bilateral in 0.8%. Four year unadjusted survival for patients with and without significant RAS was 57% and 89% respectively. Presence of RAS, increased age, the severity of coronary artery disease, and presence of comorbid disease,

reduced ejection fraction, symptoms of congestive cardiac failure, and the mode of treatment of coronary artery disease were the factors independently associated with decreased survival. Four-year adjusted survival for patients with 50%, 75%, and 95% stenosis was 70%, 68%, and 48%, respectively.[34]

Harding MB and his colleague prospectively studied the prevalence and associated risk factors in patients undergoing routine cardiac catheterization. Out of 1302 patients undergoing abdominal aortogram, 30% of them were found to have renal artery disease and significant renal stenosis was found identified in 18.8 in 15% of the population. Significant unilateral disease was present in 1%, and bilateral disease was present in 4%.[35]

Multivariable predictors included age, severity of coronary artery disease, congestive heart failure, female gender, and peripheral vascular disease. Hypertension was not an associated variable.

In 2005, Mauricio G. Cohen et al conducted a study in 843 patients who underwent abdominal aortography during cardiac catheterization. The aim of the study is to formulate a score which would be able to identify high risk patients. The risk factors included in the predictive score are hypertension, Sex, PAD, 3VD, serum creatinine, number of CV drugs and past CABG.[36]

The low risk score is <5 and it correlated with a probability of significant RAS of 0.6 %. The high risk is score of >18 correlated with a probability of 62.1%.

We did not formulate a predictive score in our study as there is only a small number of study populations.

The luminal diameter of significant renal artery stenosis was ≥50% in our study. The relatively high prevalence in our population may be because of less stringent criteria. Resistant HT, duration of HT correlated significantly with RAS. The clinical presentation of CAD has no significance.

SUMMARY

Prevalence of RAS in patients undergoing cardiac catheterization

Study	No.	HT	CAD	RAS%
Masoomi et al	122	122	95	23%
Rimoldi et al	1403	1403	558	8%
Robbins et al	110	110	69%	20%
Yamashitaq et al	289	138	220	7% (16%) HT
Harding et al	1235	49%	66%	15%
Rath et al I sathyamurthy et al Range 15%-23% Indian study	1000 469	522 287	849 360	10.3%/2.5% 14%(66)
Our study	75	75	71	15(20%)

The predictors in our study are 1) age >55yrs, 2)female sex, 3) stage II HT,5) Resistant HT and heart failure 5) 3VD, 6) duration of HT >5yrs are found statistically significant.

LIMITATIONS OF THE STUDY

The study population was small and it is a single centre cross sectional observational study.

Functional assessment of renal artery stenosis was not done. IVUS and FFR are not in our investigation strategy.

There is a strong correlation of renal artery stenosis and peripheral artery disease but peripheral angio was not part of our study.

Screening for ARAS in normal coronaries was done but it did not yield significantly.

Overestimation of lesion severity was done because it is only a luminogram and orthogonal assessment is not done.

Invasive renal angiography is recommended when clinical suspicion is high and noninvasive testing is inconclusive or inconsistent with the clinical evidence (Hirsch et al., 2006). Routine non invasive screening tests were not our investigation protocol.

Only Hypertensive patients with suspected CAD were included in our study the chances for missing RAS lesions in non hypertensive CAD patients is a major limitation of our study. Hypertension is both a risk factor and consequence of RAS. The causative role was not established. There is selection bias in our study.

CONCLUSION

Renal Angiogram is the gold standard investigation of renal artery stenosis. The guidelines did not recommend routine use of renal angio for screening purpose. But there are high risk predictors which has been the importance of our study to identify patients who will be screened for RAS while doing coronary angiogram for suspected CAD.

Because of oculostenotic reflex of interventional cardiologists there is an overestimation of lesion severity by visual estimation during renal angiography. There is an intention towards stenting of lesions. Large randomized control trials till now did not favor stenting of all lesions. But there are cases that would benefit stenting are cardiac destabilization syndromes, resistant hypertension and chronic renal insufficiency. There is no robust evidence to support this situation too.

Presence of renal artery stenosis is an independent predictor of survival in associated CAD patients. It is an adverse prognostic indicator of atherosclerosis.

In our study the absolute number of renal artery stenosis was found significant. Renal angiography is recommended to this subset of patient with severe CAD as part of screening strategy. The procedure is

found to be safe with no major complications. It can be done with limited fluoroscopy time. The amount of contrast is also very less.

Routine practice of screening RAS and intervention is not cost effective if there is no immediate hazard or risk of progression of ARAS.

The stent outcome is determined by trans-stenotic gradient. If the systolic gradient is greater than 21 mmHg it predicts control of blood pressure after stenting with unilateral RAS. The diameter stenosis 50% by angiography and renal fractional flow reserve do not predict outcomes after renal artery stenting.[10]

RECOMMENDATION

- ❖ Routine screening of Renal Artery stenosis is not recommended
- ❖ The prevalence is significantly higher if hypertensive population with CAD is screened for ARAS
- ❖ Female sex, elderly age, severe HT, triple vessel disease are significant association with ARAS
- ❖ The presence of renal artery stenosis is a bad prognostic sign
- ❖ Intense medical management should be the prime treatment strategy.
- ❖ The role of renal artery stenting is still unresolved

It is recommended to screen for ARAS in hypertensive patients with multiple risk factors and multivessel disease.

It is necessary to detect ARAS to prevent ischemic nephropathy a reversible cause of chronic renal failure at an early stage.

There is incomplete evidence to support revascularization in multi-drug-resistant renovascular hypertension, advanced CKD (stages 4–5), or steadily deteriorating renal function, which is also true for the vast majority of ARAS patients who present with asymptomatic chronic kidney disease or hypertension and severe RAS.

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PROFORMA

NAME: **AGE/SEX:** **HT:** **WT:**
BMI:

CD NO. **IP/OP no.** **CCL NO.**

D.O.A: **D.O.D:** **D.O.E:**

PRESENTATION: Chest pain: Pedal edema:

Dyspnoea: Abd distension:

Syncope: Oliguria:

Palpitation: Fatigue:

Other:

PAST ILLNESS: HT : onset: controlled:

Diabetes

CAD:

PAD: CVA:

Medical History : No of drugs for HT: ACE/ARB: Compliance:

PERSONAL HISTORY: Smoking:

CLINICAL FEATURES: GE –

BP: PULSE:

CVS:

RS:

ABD: CNS:

Clinical Diagnosis: CAD:

HT:

Accelerated /Resistant/Malignant:

Heart failure/ Pulmonary edema:

Investigations: Hb: TC: DC: P L E M

Urine albumin:

RBS: Bl Urea: Sr. Creatinine:

GFR:

Lipid profile:

ECG: Rate: Rhythm: P wave: PR int:

QRSD: QRS axis: ST: T :

LVH:

ECHO data:

LVID d: LVID s: RWMA: AW/IW LVEF:

IVS d: IVSs PW: RWT: LV mass:

USG/Doppler Abdomen:

Coronary Angiogram:

1)SVD/ DVD/ TVD/ Left Main

Renal Angiogram :

1) unilateral/Bilateral

2) % stenosis:

MASTER CHART

S.No	CD No	Age	sex	Smoking	diabetes	Hyper lipedeima	stage1	stage2	resistant	acc/malignant	duration	Duration in months	CSA	UA/NSTEMI	STEMI	HF/PE	SVD	2VD	3VD	LMCA	Normal	(U/L	B/L
1	302458	58	M				yes				2y	24		yes			yes						
2	302521	41	M	yes				yes			6m	6			yes			yes					
3	302578	65	M				yes				3y	36		yes			Yes						
4	303452	52	F		yes	yes		yes			6y	72		yes				yes					
5	303774	37	M				yes			yes	4m	4	yes								Yes		
6	303983	64	F					yes			5y	60		yes		yes			yes			yes	
7	304115	57	M	yes			yes				1y	12			yes		yes						
8	304522	63	F					yes	yes		10y	120			yes		yes						
9	304658	68	M		yes	yes		yes			5y	60			yes	yes	yes			yes			
10	304856	55	F				yes			yes	2y	24			yes			yes					
11	304966	69	M	yes	yes	yes		yes			8y	96			yes		yes						
12	304987	73	M	yes				yes			10y	120			yes				yes			yes	
13	305554	62	M				yes				2y	24		yes			yes						
14	305587	38	F				yes				3y	36			yes			yes					
15	305630	60	M	yes			yes				1y	12			yes		yes						
16	305755	58	M					yes			5y	60			yes			yes					
17	305861	67	F		yes	yes	yes				7y	84			yes		yes						
18	305910	57	M	yes				yes			4m	4			yes	yes		yes					
19	305934	51	F				yes				2y	24		yes					yes				
20	306005	72	M					yes			5y	60	yes					yes					

21	306114	69	F					yes			10y	120			yes		yes					yes	
22	306184	55	M	yes				yes			2y	24		yes			yes						
23	306235	62	F		yes	yes	yes				5y	60							yes				
24	306278	68	M	yes			yes		yes		12y	144	yes		yes	yes		yes				yes	
25	306450	57	M					yes			6m	6			yes						yes		
26	306564	40	F				yes				4m	4			yes				yes				
27	306700	63	M	yes			yes				5m	5			yes		yes						
28	306787	58	F					yes			1y	12			yes				yes				
29	306865	80	M					yes			4y	48			yes		yes						
30	306921	68	M		yes	yes		yes			8y	96		yes				yes				yes	
31	306954	57	M				yes				2y	24		yes			yes						
32	306888	49	M	yes				yes			1y	12			yes	yes					yes		
33	307024	66	F				yes				2y	24			yes		yes						
34	307058	46	M					yes			1y	12			yes				yes				
35	307070	67	M				yes				2y	24		yes			yes						
36	307104	72	F		yes	yes		yes	yes	yes	4y	48			yes	yes	yes					yes	yes
37	307245	66	M					yes			3y	36			yes			yes				yes	
38	307268	43	F				yes				1y	12	yes						yes				
39	307312	58	F		yes	yes		yes			2y	24			yes		yes						
40	307354	68	M	yes			yes				6y	72			yes						yes		
41	307458	47	F				yes				1y	12		yes		yes			yes				
42	307493	72	M					yes	yes		4y	48	yes						yes				
43	307512	57	M					yes			1y	12	yes		yes		yes						
44	307654	54	M				yes				3m	3			yes			Yes					
45	307688	70	F		yes	yes	yes				7y	84			yes	yes			yes			yes	yes
46	307701	65	M					yes			5y	60		yes			yes						

47	307763	45	M	yes			yes				1y	12			yes			yes					
48	307823	55	M				yes				3m	3		yes			yes						
49	307885	66	F					yes			6m	6	yes				yes			yes			
50	307915	71	M					yes			4m	4		yes			yes						
51	307969	55	M				yes				1y	12			yes		yes						
52	308007	57	F		yes	yes	yes				6y	72			yes		yes						
53	308045	67	M	yes				yes			3y	36			yes				yes				
54	308086	48	F				yes				6m	6		yes				yes					
55	308146	58	M	yes	yes	yes	yes				5y	60					yes						
56	308176	62	F		yes	yes		yes			4y	48	yes			yes			yes			yes	
57	308190	59	F				yes				4y	48	yes				yes						
58	308212	70	M	yes	yes			yes			6y	72			yes				yes			yes	
59	308263	66	M				yes				4m	4			yes	yes		Yes					
60	308302	50	M	yes			yes				1y	12			yes	yes	yes						
61	308355	57	M					yes			2y	24			yes		yes						
62	308391	70	F		yes	yes		yes			4y	48	yes				yes						
63	308411	58	M	yes				yes	yes		2y	24		yes		yes		yes				yes	
64	308420	55	M				yes				1y	12			yes		yes						
65	308578	60	F		yes			yes			2y	24			yes			yes					
66	308756	57	M	yes			yes				4m	4			yes		yes						
67	308863	58	M				yes				3y	36			yes		yes						
68	308901	65	F					yes		Yes	6m	6		yes		yes			yes			yes	
69	308944	63	M	yes	yes		yes				2y	24		yes				yes					
70	308960	70	F					yes			5y	60		yes		yes			yes			yes	
71	308986	57	M		yes		yes				2y	24		yes			yes						
72	309008	34	M				yes				3m	3		yes		yes			yes				

73	309156	60	M		yes		yes				1y	12	yes				yes						
74	309267	59	M	yes				yes			1y	12			yes		yes						
75	309756	65	M		yes		yes				3y	36		yes				yes					

Ref. No. 23308/E4/2/2013

Govt. Rajaji Hospital,
Madurai.20. Dated: 24.12.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for December 2013
Approved list -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 18.12.2013, Wednesday at 10.00 am to 12.00.noon at the Anaethesia Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. I. Jeyaraj, M.S., (Anatomy) Cell.No 9566211947	Director & Professor Institute of Anatomy /V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048	Professor of Medicine Madurai Medical College	Member
6. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031	Professor & H.O.D of Surgery i/c Madurai Medical College	Member
7. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
8. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
9. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Project was approved by the committee

..2..

Name of P.G.	Course	Name of the Project	Remarks
Dr.S. Sathish Kumar	P.G in M.D., D.M., Cardiology, Madurai Medical College, Madurai & Government Rajaji Hospital, Madurai.	Study on prevalence and predictors of renal artery stenosis in hypertensive patients undergoing coronary angiography at Government Rajaji Hospital, Madurai-20.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

24/12/13

To
The above Applicant
-thro. Head of the Department concerned

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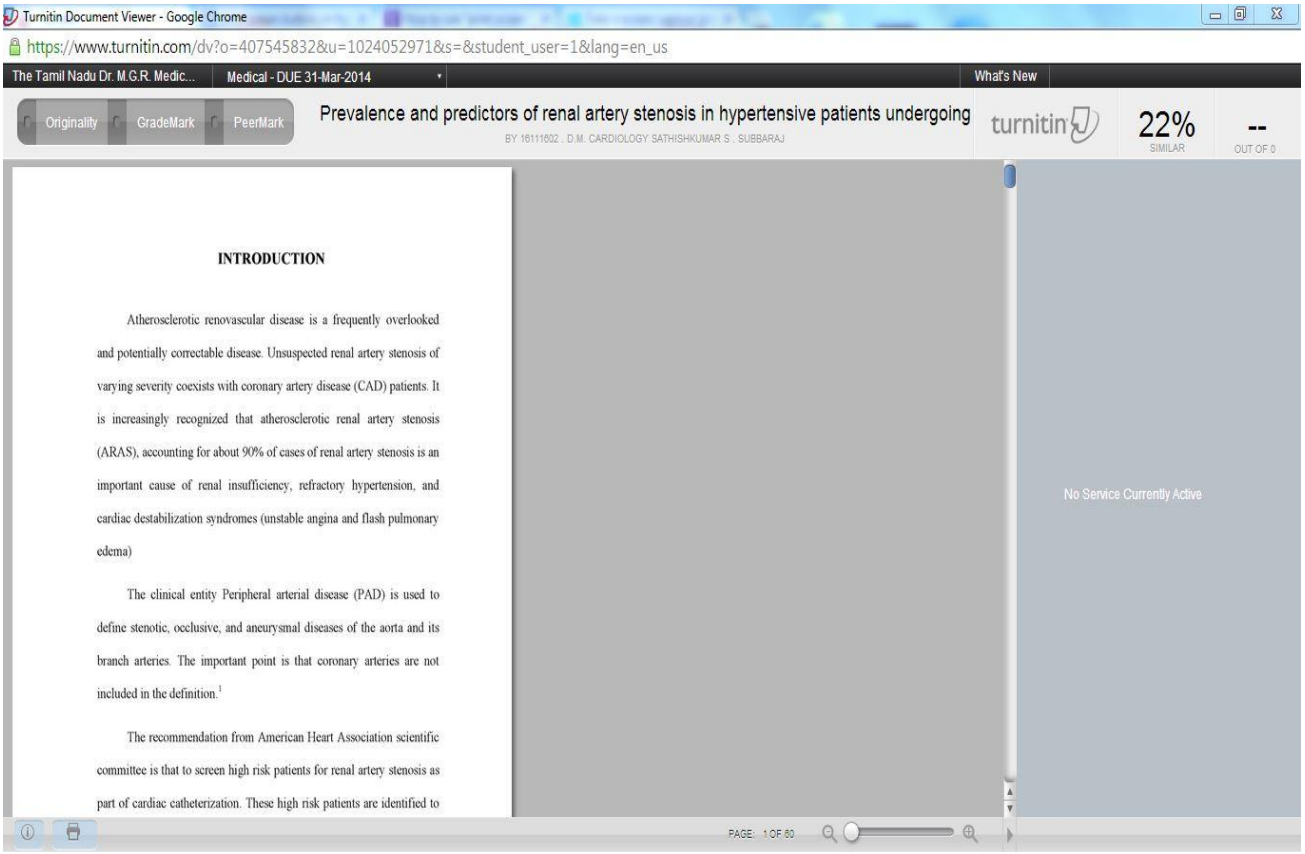
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INTRODUCTION
Atherosclerotic renovascular disease is a frequently overlooked and potentially correctable disease. Unsuspected renal artery stenosis of varying severity coexists with coronary artery disease (CAD) patients. It is increasingly recognized that atherosclerotic renal artery stenosis (ARAS), accounting for about 90% of cases of renal artery stenosis is an important cause of renal insufficiency, refractory hypertension, and cardiac destabilization syndromes (unstable angina and flash pulmonary edema)
The clinical entity Peripheral arterial disease (PAD) is used to define stenotic, occlusive, and aneurysmal diseases of the aorta and its branch arteries. The important point is that coronary arteries are not included in the definition.[1]
The recommendation from American Heart Association scientific committee is that to screen high risk patients for renal artery stenosis as part of cardiac catheterization. These high risk patients are identified to
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