# CARDIOVASCULAR RISKFACTORS ANALYSIS IN RENAL TRANSPLANT RECIPIENTS

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# CERTIFICATE

This is to certify that the dissertation titled **"CARDIOVASCULAR RISKFACTORS ANALYSIS IN RENAL TRANSPLANT RECIPIENTS "** is the bonafide original work of **Dr.S.A.K.NOOR MOHAMED,** in partial fulfillment of the requirements for D.M. Branch – III (Nephrology) Examination of the Tamilnadu DR. M.G.R Medical University to be held in AUGUST 2012.

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# DECLARATION

I, **Dr. S.A.K.NOOR MOHAMED,** solemnly declare that dissertation titled **"CARDIOVASCULAR RISKFACTORS ANALYSIS IN RENAL TRANSPLANT RECIPIENTS "** is a bonafide work done by me at Government Stanley Medical College and Hospital during October 2010 to November 2011 under the guidance and supervision of my unit chief **PROF. M.EDWIN FERNANDO M.D., D.M.(Nephrology),** Professor and Head, Department of Nephrology, Government Stanley Medical College and Hospital, Chennai.

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# INTRODUCTION

# INTRODUCTION

Renal transplantation is currently the preferred treatment modality for virtually all suitable candidates with end-stage renal disease. Compared with dialysis, kidney transplantation improves both patient survival and quality of life. Nonetheless, post transplant cardiac complications are associated with increased morbidity and mortality after renal transplantation. When compared with the general population, cardiovascular mortality in transplant recipients is increased by nearly 10-fold among patients within the age range of 35 and 44 and at least doubled among those between the ages of 55 and 64. Although renal transplantation ameliorates cardiovascular disease risk factors by restoring renal function, it introduces new cardiovascular risks derived, in part, from immunosuppressive medications.

# AIM OF THE STUDY

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To analyze the risk factors for cardiovascular disease in the renal transplant recipients

# REVIEW OF LITERATURE

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Successful kidney transplantation has been shown repeatedly to be associated with a reduction in mortality compared with dialysis. Studies suggest that this effect largely may be the result of the reduction in cardiovascular disease(CVD) associated with the improvement in renal function. In a retrospective analysis of the United States Renal Data System data consisting of more than 60,000 adult primary kidney transplant recipients transplanted between 1995 to 2000 and more than 66,000 adult wait-listed patients over the same time period, Meier-Kriesche et al[1] showed a progressive decrease in cardiovascular death rates by renal transplant vintage for diabetic and nondiabetic recipients of both living- and deceased-donor transplants. Although the CVD death rates among transplant recipients were expectedly higher in the early postoperative period, they decreased significantly by 3 months post transplant. On long-term follow-up evaluation, although there seemed to be a modest increase in CVD death rates in the second transplant year, the rates actually remained low even among high CVD risk groups such as those with end-stage renal disease secondary to diabetes mellitus or hypertension. This finding likely reflects the impact of deteriorating transplant function on CVD death rates and is consistent with the relationship between declining renal function and CVD risk observed in non transplant chronic kidney disease[2].Yet despite the well-established survival advantage of transplantation over dialysis, CVD death has emerged

as the most frequent cause of late graft loss. Recognition of CVD risk factors and aggressive management of CVD risk factors should begin in the early posttransplant period and should remain an integral part of long-term care in renal transplant recipients.

## CARDIOVASCULAR RISK FACTORS IN THE RECIPIENTS OF RENAL TRANSPLANTS

Although all the determinants of enhanced CVD risks in renal transplant recipients have not been well defined, both conventional and unconventional risk factors have been suggested to be contributory. The former risks include diabetes mellitus, hypertension, dyslipidemia, obesity, smoking, and family history. The latter risks include pre-existing left ventricular hypertrophy, coronary artery vascular calcification, impaired allograft function, proteinuria, anemia, acute rejection episodes, hyperhomocysteinemia, and inflammatory cytokines[3,4]. More recently, CD4 lymphopenia and cytomegalovirus (CMV) infection also has been suggested to be associated with cardiac complications and atherosclerosis[5,6,7]. Selected CVD risks are discussed here.

## Cardiovascular Risk Factors[8]

### Major Risk Factors for Coronary Heart Disease in ATP III Guidelines

- Cigarette smoking
- Hypertension (ie, blood pressure >140/90 mm Hg or on antihypertensive medication)
- High LDL cholesterol (ie, >159 mg/dL)
- Low HDL cholesterol (ie, <40 mg/dL)
- Family history of premature coronary heart disease (ie, <55years of age in male first-degree relative or <65 years of agein female first-degree relative)
- Age (men >45 years and women >55 years)
- Diabetes

### Predisposing Risk Factors

- Obesity (ie, body mass index >30 kg/m2)
- Abdominal obesity (ie, waist circumference >102 cm, or 40in, for men and >88 cm, or 35 in, for women)
- Physical inactivity
- Family history of premature coronary heart disease

- Ethnic characteristics
- Psychosocial factors
- Nontraditional Biomarkers
- Elevated serum triglycerides
- Small LDL particles
- Elevated serum homocysteine
- Elevated serum lipoprotein(a)
- Prothrombotic factors (eg, fibrinogen)
- Inflammatory markers (eg, C-reactive protein, IL-6, CMV)
- B-type natriuretic peptide/N-terminal pro-atrial natriureticpeptide
- Aldosterone

**Risk Factors Associated with Kidney Disease or Transplant**

- Immunosuppressive agents
- Graft failure
- Graft dysfunction (elevated homocysteinemia, proteinuria, predisposition to vascular calcification)
- Anaemia

| Risk factors for posttransplant cardiovascular disease | |
|---|---|
| **Risk Factor** | **Strength of Evidence** |
| Pretransplant cardiovascular disease | ++++ |
| Diabetes(includingposttransplant diabetes) | ++++ |
| Cigarette smoking | +++ |
| Hyperlipidemia | +++ |
| Hypertension | ++ |
| Platelet and coagulation abnormalities | ++ |
| Allograft dysfunction/rejection | ++ |
| Hypoalbuminemia | ++ |
| Erythrocytosis | + |
| Oxygen free radicals | + |
| Infections | + |
| Increased homocysteine | + |

The determinants that lead to atherosclerosis in renal transplant recipients are similar to those in the general population[9-13]

Risk factors for coronary heart disease after renal transplantation were investigated in a report of 403 patients who received 464 kidney transplants

during a 10-year period[14]. New atherosclerotic complications developed in 16 percent of patients. After accounting for pre-transplant vascular disease, multivariate analysis revealed that the following risk factors were independently associated with post-transplant atherosclerotic cardiovascular disease: Increasing patient age, Diabetesmellitus, Male gender, Cigarette smoking, Hypertension, Elevated serum cholesterol

Similar findings were observed in other studies[13,15,16] including a retrospective analysis of the placebo arm of the Assessment of LEscol in Renal Transplantation (ALERT) study[15].

In multivariate analysis, independent risk factors for myocardial infarction, cardiac death, and noncardiac death were preexisting coronary heart disease (hazard ratio, 3.69), total cholesterol level (HR, 1.55 per 50 mg/dL), and prior acute rejection (HR, 1.58). Age, diabetes, and elevated serum creatinine levels were independent risk factors for cardiac death.

Other possible risk factors include hyperhomocysteinemia, elevated levels of lipoprotein(a), elevated C-reactive protein and interleukin-6 levels, proteinuria, allograft loss, obesity, and rejection[17-22].

Prior to transplantation, most renal allograft recipients are uremic for months or years. One of the features of this syndrome is accelerated atherogenesis. The atherogenic factors contributing to this process prior to transplantation may include hyperhomocysteinemia, hyperfibrinogenemia, increased calcium ingestion, abnormalities of mineral metabolism,

dyslipidemia, and modification of low-density lipoproteins (LDL) by advanced glycosylation end-products (AGE), particularly in diabetics[23-25].In studies evaluating risk factors for post-transplant cardiovascular disease, pre-transplant cardiovascular disease is among the most important determinants[14,2]

## SYSTEMIC HYPERTENSION

Hypertension is an independent risk factor for allograft failure and mortality and is present in 50% to 90% of renal transplant recipients[27,28].The wide range in the frequency may reflect the variable definitions of hypertension, donor source, immunosuppressive medications, time posttransplantation, and level of allograft function. Systolic blood pressure (BP) is highest immediately after transplantation and declines during the first year[27].

The contributory role of calcineurin inhibitors and glucocorticoids in the development of posttransplant hypertension has been well established. In a large randomized trial consisting of more than 400 patients randomized to remain on sirolimus-cyclosporine-steroid (sirolimus-cyclosporine-steroid) or to have cyclosporine withdrawn (sirolimus-steroid) at 3 months, systolic and diastolic BP were significantly lower in the sirolimus-steroid compared with the sirolimus-cyclosporine-steroid groups at the 36-month follow-up evaluation (systolicBP, 131.3 versus 140.1 mm Hg, respectively, and diastolic BP, 76.3 versus 81.2 mm Hg, respectively). Moreover, this difference was

observed despite significantly less use of antihypertensive medication in the sirolimus-steroid group[30].A 3-year observational follow-up evaluation of a European, multicenter, randomized, clinical trial comparing triple therapywith tacrolimus, steroids, and mycophenolate mofetil (MMF) with withdrawal of either steroids or MMF at 3 months after renal transplantation showed that steroid withdrawal was advantageous in reducing hypertension, hyperlipidemia, and diabetes mellitus[31].The mean systolic BP was lower in the steroid-stop group compared with the steroid maintenance groups (steroid stop, 133.6 mm Hg; triple therapy, 136.2 mm Hg; MMF stop, 139.8 mm Hg). The mean diastolic BP was similar in all groups. Renal function was maintained in all groups, and patient and graft survival at 3 years were not compromised by withdrawal of concomitant immunosuppression at 3 months from a tacrolimus-based regimen.

The results of the Collaborative Transplant Study registry suggest that BP control after transplantation is suboptimal. Management of posttransplant hypertension should include attempts to identify and treat the underlying cause, lifestyle modifications, and treatment of associated cardiovascular risk factors. Lifestyle modifications should be similar to those used in the nontransplant population. Potassium-based salt substitutes must be used with caution or should be avoided because of the high incidence of hyperkalemia among patients receiving cyclosporine or tacrolimus immunosuppression.

There is a paucity of controlled clinical trials to determine the superiority of one class of antihypertensive agents over the other in the

transplant setting. In general, there are no absolute contraindications to the use of any antihypertensive agent in renal transplant recipients. All classes of antihypertensives have been used in various combinations with good results. Nondihydropyridine calcium channel blockers and diuretics are used frequently in the early posttransplant period, the former because of their beneficial effect on renal hemodynamics and the latter because of their ability to eliminate salt and water in these subjects who frequently are volume expanded. In a single-center retrospective study to identify ischemic heart disease risk after renal transplantation, Kasiske et al[32] unexpectedly found an association between the use of dihydropyridine calcium channel antagonists and an increased risk of ischemic heart disease. Of interest, the use of dihydropyridine calcium channel blockers in proteinuric chronic kidney disease patients also has been shown to be associated with an increased risk of renal disease progression and death, except when used in conjunction with angiotensin II blockade therapy.[9,33-35].

Although the mechanism(s) for the potential adverse effects of dihydropyridine calcium channel blockers on the cardiovascular risk profile is unclear, the use of amlodipine has been reported to be associated with increased catecholamine levels[36].Although further recommendations await results of large, ongoing, randomized, controlled trials in the general population, monotherapy with dihydropyridine calcium channel antagonists should be used with caution.

The use of angiotensin converting enzyme inhibitor (ACE-I) and angiotensin receptor blocker (ARB) alone or in combination has gained increasing popularity because of their safety, efficacy, and well-established renoprotective, antiproteinuric, and cardioprotective effects. Nonetheless, an increase in serum creatinine level (ie, ≥30% above baseline) associated with their use should alert the clinician of possible transplant renal artery stenosis. Caution should be exercised when used with diuretics because ACE-I or ARB may potentiate volume depletion induced renal hypoperfusion. In patients with slow or delayed graft function, ACE-I and ARB generally are not recommended until allograft function has recovered.Mild to moderate renal allograft dysfunction, however, does not exclude their use if serum potassium and creatinine levels can be monitored closely.

Betablockers should be considered in patients with known coronary artery disease or other atherosclerotic vascular disease whereas alpha2 blockers may be beneficial in patients with benign prostatic hypertrophy and neurogenic bladder. Symptomatic bradycardia and blunting of hypoglycemic unawareness occasionally may limit the use of the former. Although aggressive blood pressure control is vital in reducing cardiovascular morbidities and mortalities as well as improving graft survival, this is not recommended in the early perioperative period because of the risk of precipitating acute tubular necrosis and/or graft thrombosis.

## NEWONSET DIABETESMELLITUSAFTERTRANSPLANTATION

New-onset diabetes mellitus after transplantation (NODAT) is a well-known complication after solid-organ transplantation and has been reported to occur in 4% to 25% of renal transplant recipients. The variation in the reported incidence may be owing to the lack of a universal agreement on the definition of NODAT, the difference in the duration of follow-up evaluation, and the presence of both modifiable and nonmodifiable risks factors. Kidney transplant recipients who developed NODAT are at 2- to 3- fold increased risk of fatal and nonfatal CVD events.[9]

### Potential Risk Factors for NODAT

- African American and Hispanic ethnicities
- Obesity defined as a body mass index of >30 kg/m2
- Increasing age >40 years
- Male gender
- Family history of diabetes among first-degree relatives
- Impaired glucose tolerance before transplantation
- Recipients of deceased donor kidneys
- Hypertriglyceridemia
- Hypertension
- Hepatitis C and CMV infection
- Corticosteroids, tacrolimus, cyclosporine and Sirolimus

- The presence of certain HLA antigens such as A30, B27, and B42
- Increasing HLA mismatches
- Acute rejection history
- Male donor

The antimetabolites azathioprine and MMF have not been shown to be diabetogenic. On the contrary, the concomitant use of MMF has been suggested to mitigate the diabetogenic effect of tacrolimus.[37]It is conceivable that the use of azathioprine or MMF allows clinicians to use lower doses of other diabetogenic immunosuppressive medications. Early clinical trials have suggested that sirolimus is devoid of a diabetogenic effect. However, recent studies in animal models and in recipients of renal transplants have suggested that sirolimus is associated with reduced insulin sensitivity and a defect in the compensatory βcell response.[38,39] Studies in diabetic mice transplanted with islet cells have suggested that sirolimus is associated with reduced islet engraftment and impaired betacell function in transplants.[39]

The management of NODAT should follow the conventional approach for patients with type 2 diabetes mellitus as recommended by many clinical guidelines established by well recognized organizations including the American Diabetes Association Further intervention may include adjustment or modification in immunosuppressive medications and pharmacologic therapy to achieve a target hemoglobin $A_1C$ level of less than 6.5%. Corticosteroid dose reduction has been shown to improve glucose tolerance

significantly during the first year after transplantation.[37] However, any dose reduction should be weighed against the risk of acute rejection. A steroid-sparing regimen or steroid avoidance protocol should be tailored to each individual patient. Tacrolimus to cyclosporine conversion therapy in patients who fail to achieve target glycemic control or in those with difficult to control diabetes has yielded variable results.

When lifestyle modification fails to achieve adequate glycemic control, medical intervention is recommended. Orally administered agents can be used either alone or in combination with other oral agents or insulin. Although oral hypoglycemic agents may be effective in many patients with corticosteroid, cyclosporine, or tacrolimus induced NODAT, insulin therapy may be necessary in up to 40% of patients [40] particularly in the early posttransplant period.

The choice of pharmacologic therapy is based on the potential advantages and disadvantages associated with the different classes of oral agents. Although metformin (a biguanide derivative) is the preferred agent for overweight patients, its use should be avoided in patients with impaired allograft function because of the possibility of lactic acidosis. Care also should be taken when the sulfonylurea derivatives are prescribed to patients with impaired allograft function or to elderly patients because of the increased risk of hypoglycemia. In general, it is best to start with a low dose and titrate upward every 1 to 2 weeks. The nonsulfonylureas meglitinides are insulin secretagogues with a mechanism of action similar to that of the sulfonylureas.

Nonetheless, they have a more rapid onset and shorter duration of action and seemingly lower risks of hypoglycemia and the amount of weight gain.[41] These agents therefore are best suited for patients whose food intake is erratic, elderly patients, and patients with impaired graft function. They are best taken before meals and the dose may be omitted if a meal is skipped.

The thiazolidinedione derivatives are insulin sensitizers that may allow for a reduction in insulin requirement. Potential adverse effects of these agents include weight gain, peripheral edema, anemia, pulmonary edema, and congestive heart failure. The incidence of peripheral edema is increased when thiazolidinedione derivatives are used in combination therapy with insulin. .Drug interactions also should be considered carefully.

The meglitinide derivatives repaglinide and to a lesser extent nateglinide are metabolized through the cytochrome p450 isozyme CYP 3A4, and the glucose level should be monitored closely when the patient also receives a strong inhibitor (eg, cyclosporine, gemfibrozil, or the azole antifungal) or inducer (eg, rifampin, carbamazepine, phenytoin, or St John's wort) of the CYP 3A4 system.[41] The use of gemfibrozil, a CYP 3A4 inhibitor, and repaglinide combination therapy has been shown to dramatically increase the action of the latter, resulting in prolonged hypoglycemia. Co-administration of cyclosporine and repaglinide also has been shown to enhance the blood glucose–lowering effect of repaglinide and increase the risk of hypoglycemia.[42]In contrast, rifampin, a strong inducer of CYP 3A4, considerably decreases the plasma concentration of repaglinide and also

reduces its effects.[43]Although tacrolimus also is metabolized via the CYP 3A4 system and should therefore be susceptible to many drug interactions similar to those of cyclosporine, these interactions are not as well documented. Monitoring of patients with posttransplant diabetes mellitus should include measuring hemoglobin $A_1C$ level every 3 months, and screening for diabetic complications including microalbuminuria, regular ophthalmologic examinations, and regular foot care. In addition, the fasting lipid profile should be measured annually. In transplant recipients with multiple CVD risk factors, more frequent monitoring of the lipid profile should be performed at the discretion of the clinicians.

## POST TRANSPLANT DYSLIPIDEMIA

Dyslipidemia is a common occurrence after transplantation. The hyperlipemic effect of immunosuppressive agents including corticosteroids, cyclosporine, tacrolimus, and sirolimus has been well documented. Although tacrolimus-based therapy has been suggested to be associated with better lipid profiles than cyclosporine-based therapy, sirolimus has been shown to be associated with a significantly greater incidence and severity of dyslipidemia than cyclosporine-based therapy, including higher total cholesterol and triglyceride levels.

Causative Factors for Posttransplant Dyslipidemia

- Sirolimus, corticosteroids, cyclosporine, and tacrolimus
- Age

- Diet
- Rapid weight gain
- Hyperinsulinemia
- Pre-existing hypercholesterolemia
- Allograft dysfunction
- Proteinuria
- betablockers and diuretic therapy

Although hyperlipidemia often improves within the first 6 months after transplantation as the doses of prednisone, cyclosporine/tacrolimus, or sirolimus are reduced, total and low-density lipoprotein (LDL) cholesterol goals as defined by the National Cholesterol Education Program guidelines usually are not achieved and treatment frequently is required. Management of hyperlipidemia includes therapeutic lifestyle changes and pharmacotherapy Statins or the hydroxyl glutaryl (HMG)-CoA reductase inhibitors are the most widely used lipid-lowering agents in both the nontransplant and transplant settings. The clinical benefits of statins have been shown in several large randomized controlled trials including the Heart Protection Study and the Lescol Intervention Prevention Study[44,45].

The Heart Protection Study, the largest study to date, randomized more than 20,000 individuals in the United Kingdom aged 40 to 80 years with total cholesterol levels of greater than 135 mg/dL to receive either simvastatin (40 mg/day) or placebo. At the 5.5-year follow-up evaluation there was a 12%

reduction in total mortality, a 17% reduction in vascular mortality, a 24% reduction in CVD events, a 27% reduction in strokes, and a 16% reduction in noncoronary revascularizations[44]. The study further revealed that statin therapy was beneficial in reducing major vascular events independent of baseline LDL in patients with known coronary artery disease, cerebrovascular disease, peripheral vascular disease, diabetes mellitus, or hypertension.

The beneficial effect of statins was greatest in the lowest LDL subgroups (LDL < 60). Whether this effect can be extrapolated to renal transplant recipients awaits further studies. Results of the Assessment of Lescol in Renal Transplantation study revealed that treatment of renal transplant recipients with fluvastatin over a 5- to 6-year period significantly and safely reduced LDL cholesterol levels. The incidence of major adverse cardiac events also was shown to be reduced, albeit not statistically significant. However, further analysis showed a beneficial effect of early initiation of fluvastatin on outcome the earlier the initiation of therapy, the greater the reduction in cardiac events. For patients initiated on therapy within the first 4years posttransplant, there was a risk reduction of 64% compared with 19% for patients initiated on therapy after 10 years.

No statin effect on graft loss or on doubling of serum creatinine level was observed[46-48].This finding contrasts with that of Masterson et al[49], who found better renal function at 12 months posttransplant in recipients who received statins compared with those who were not on statin therapy .Despite the well-established efficacy and safety of the use of statins in transplant

recipients clinicians should remain vigilant to the potential drug-drug interactions in transplant patients, who often require multiple medications. The use of statins in the presence of calcineurin inhibitors, particularly cyclosporine, often results in a several-fold increase in statin blood level and an increased risk for myopathy and rhabdomyolysis[50].Cyclosporine increases plasma exposure to fluvastatin by approximately 2-fold, simvastatin (20 mg/day) by 3-fold, atorvastatin by approximately 6-fold, pravastatin by 5- to 23-fold, and lovastatin by up to 20-fold. Approximate therapeutic equivalencies are achieved by 10 mg of atorvastatin, 20 mg of simvastatin, 40 mg of pravastatin, 40 mg of lovastatin, and 80 mg of fluvastatin. At these doses, the LDL cholesterol decrease is approximately 34%, with very little change in highdensity lipoprotein levels[50]. In addition to their lipid-lowering effect, statins may offer protection against CVD via their antiproliferative properties and effects on the reduction of circulating endothelin-1, C-reactive protein (CRP) levels, systolic and diastolic BP, and pulse pressure.

Other classes of lipid-lowering agents include fibric acid derivatives, nicotinic acid, bile acid sequestrants, and the newer lipidlowering agent ezetimibe. Ezetimibe and statin combination therapy can significantly improve cholesterol control because of their complementary mechanism of actions. Ezetimibe blocks intestinal absorption of dietary cholesterol and related phytosterols whereas statin blocks hepatic cholesterol synthesis. The currently available ezetimibe/simvastatin drug combination has been shown to markedly reduce LDL-cholesterol (LDL-C) levels and has been suggested to

represent a valuable option for the management of hyperlipidemia across diverse patient populations[51]. In a cohort study consisting of 40 stable kidney transplant recipients with hypercholesterolemia, 4 weeks of ezetimide therapy significantly lowered total and LDL cholesterol levels[52]. In addition, the drug was found to be more effective when used in combination with a statin. LDL reduction was 24% ± 13% with ezetimide monotherapy versus 41% ± 13% with the statin combination therapy. No significant adverse effects on serum creatinine level, drug level, body weight, or liver function test results were detected. It is likely that ezetimibe also can be used as adjunctive therapy with other lipid-lowering agents in renal transplant recipients with poorly controlled hyperlipidemia on statin monotherapy, although further recommendations await further studies.

To date, no significant drug-to-drug interaction between ezetimibe and calcineurin inhibitors or sirolimus has been reported. Severe hypertriglyceridemia (TGL $\geq$ 500 mg/dL) has been encountered more frequently since the introduction of sirolimus. Management includes sirolimus dose reduction, addition of a fibric acid derivative or nicotinic acid, and, in refractory cases, sirolimus to MMF or tacrolimus switch. Of the major fibric acid medications (bezafibrate, ciprofibrate, fenofibrate, and gemfibrozil), the first 3 have been reported to cause increases in the serum creatinine level in cyclosporine-treated patients, as well as higher plasma homocysteine levels. Although all fibrates in combinations with statins have been associated with creatinine kinase increases with or without overt rhabdomyolysis and

myopathy, gemfibrozil may have a greater risk for the development of myopathy compared with bezafibrate or fenofibrate[50].

Niacin monotherapy has not been reported to cause myopathy, but its combined use with lovastatin, pravastatin, or simvastatin may be associated with rhabdomyolysis. Bile acid sequestrants must be used with caution because of their potential interference with the absorption of other medications vital to the renal transplant recipients.

Suggested guidelines for the treatment of posttransplant dyslipidemia includes all transplant recipients should be regarded as CHD risk equivalent. Goals: LDL ≤ 100 mg/dL (optional ≤ 70 mg/dL), TGL ≤ 200 mg/dL, HDL ≥ 45 mg/dL. has been suggested for very high-risk patients (NCEP, ATP III guidelines). Statins are the most effective drugs and should be the agents of first choice. Start at low dose in patients on cyclosporine and tacrolimus. Monitor for myositis and transaminitis, particularly in those receiving combination therapy. Bile acid sequestrans should probably not be taken at the same time as cyclosporine. Extreme caution should be used with statin and fibrate combination therapy. Consider cholesterol absorption inhibitors in patients intolerant to statins.

**RENAL INSUFFICIENCY**

Renal insufficiency in renal transplant patients is also a significant risk factor for adverse cardiovascular outcomes[53-55].Among nearly 60,000 patients in one study, serum creatinine levels above 1.5 mg/dL (133 µmol/L) at one

year post-transplant were significantly associated with an increased risk for cardiovascular disease[53].In a second study of almost 30,000 renal transplant recipients, a decreased estimated glomerular filtration rate at one year post-transplant correlated with increased risks of acute coronary syndrome and heart failure[54].Among over 1000 placebo-treated patients in the Assessment of LEscol in Renal Transplantation (ALERT) trial, an increased serum creatinine concentration, particularly higher than 2.3 mg/dL (200 micromol/L), was strongly associated with an increased risk of adverse cardiac events and cardiac death[55,56].

**OBESITY**

Obesity trends in transplant recipients tend to mimic the general population, 65 percent of whom are now defined as overweight (body mass index [BMI] 25 to 29.9 kg/m2). In one study from the United Network for Organ Sharing, approximately one-half of patients who underwent kidney transplantation between 1997 and 1999 were obese (BMI 30 to 35) or morbidly obese (BMI $\geq$35)[57].Among kidney transplant recipients, the presence of obesity, particularly within the context of the metabolic syndrome, also appears to be associated with an increased number of adverse cardiovascular events. In one study of 337 renal transplant recipients, one-third of whom had metabolic syndrome, 42 patients experienced an adverse atherosclerotic event at one year post-transplant[58]

Compared to those without metabolic syndrome, a significantly higher incidence of such events was observed among patients with the syndrome (25 versus 7 percent).Obesity also increases the risk of heart failure and atrial fibrillation. This was shown in a single center study in which the five-year incidence of cardiac diagnoses increased from 9 to 30 percent as the BMI quartiles increased from the lowest to highest[59]. This was largely due to increases in the incidence of heart failure and atrial fibrillation.

**HOMOCYSTEINE**

Homocysteine is a nonessential amino acid that is an intermediate in the synthesis of cysteine and a precursor of the essential amino acid, methionine. Since homocysteine is normally cleared by the kidneys, serum concentrations are increased in chronic renal failure[62,63].Another major cause is an absolute or relative deficiency of vitamins B6, B12, and folate, all of which are involved in methionine metabolism. A defect in the methylene tetrahydrofolate reductase enzyme has also been causally associated with hyperhomocysteinemia. The presence of elevated homocysteine levels in peripheral venous blood has been identified as an independent risk factor for ischemic heart disease and stroke in the general population, as well as being a predictor of mortality in patients with coronary heart disease[64,65].

In one prospective study of 207 stable patients, fasting mean total homocysteine levels were significantly higher among patients who experienced a cardiovascular event at follow-up of 21 months (32 versus 18

μmol/L for those without such an event)[66].Among 733 kidney transplant recipients during a six-year period, elevated homocysteine levels were associated with 2.44 times the mortality risk of patients with normal levels (hazards ratio 2.44, CI 95% 1.45-4.12)[67].In patients with end-stage renal disease, hyperhomocysteinemia is closely linked with decreased serum folate and pyridoxine concentrations[63]. Serum homocysteine concentrations often continue to rise after both renal and cardiac transplantation, independent of renal function[68-70].

In one series, serum concentrations of total homocysteine were significantly higher in 120 renal transplant recipients than in 60 healthy controls (mean 19.0 versus 11.6 μmol/L) or 53 patients with chronic renal failure (mean 16.0 μmol/L)[70] . In this report, renal transplant recipients on cyclosporine had significantly higher plasma homocysteine concentrations than those not on cyclosporine. A similar role for cyclosporine was noted in another series[71].however, this association has not been confirmed in other studies[72].Effective reduction in serum homocysteine levels in renal transplant recipients may be obtained with the administration of folic acid and vitamins B6 and B12[73].However, although hyperhomocysteinemia is an independent risk factor for cerebrovascular, peripheral vascular, and coronary heart disease, the effect of lowering homocysteine levels on cardiovascular risk in renal transplant recipients has been unclear

The NIH has sponsored a multicenter randomized control trial (FAVORIT: Folic Acid for Vascular Outcome Reduction In Transplantation),

that has examined the effectiveness of B complex vitamin supplementation on the cardiovascular outcomes in kidney transplant recipients. Preliminary results presented in abstract form indicate that while homocysteine can be effectively lowered in this patient population, no benefit was seen in terms of lowering cardiovascular events or deaths.

**FRAMINGHAM RISK SCORE**

The Framingham risk score has a modest ability to predict cardiovascular outcomes among kidney transplant patients. A prospective cohort evaluation of 540 patients followed for 4.7 years found that the ratio of observed-to-predicted cardiac events was 1.64-fold higher for the group and 2.74-fold higher in those ages 45 to 60 with prior cardiac disease or diabetes mellitus[74].

The Framingham Risk Score is used to estimate the 10-year cardiovascular risk of an individual. The Framingham Risk Score is based on data obtained from the Framingham Heart Study. There are two Framingham Risk Scores, one for men and one for women.

The Framingham Risk Score is one of a number of scoring systems used to determine an individual's chances of developing cardiovascular disease. This means either coronary heart disease or stroke. A number of these scoring systems are available online[75].Cardiovascular risk scoring systems give a best estimate of the probability that a person will develop cardiovascular disease within the next 5 or 10 years. Because they give an

indication of who is most likely to develop cardiovascular disease they also indicate who is most likely to benefit from prevention. For this reason cardiovascular risk scores are used to determine who should be offered preventive drugs such as drugs to lower blood pressure and drugs to lower cholesterol levels.

Because the Framingham Risk Score (or another appropriate scoring system) give a good indication of the likely benefits of prevention, they are useful for both the individual patient and for the clinician in helping decide whether lifestyle modification and preventive medical treatment, and for patient education, by identifying men and women at increased risk for future cardiovascular events[76].The CHD risk at 10 years in percent can be calculated with the help of the Framingham Risk Score. Individuals with low risk have 10% or less CHD risk at 10 years, with intermediate risk 10-20%, and with high risk 20% or more. However it should be remembered that these categorisations are arbitrary.

A more useful metric is to consider the effects of treatment. If 100 persons have a 20% ten-year risk of cardiovascular disease it means that 20 of these 100 individuals will develop cardiovascular disease (coronary heart disease or stroke) in the next 10 years. Eighty of them will not develop cardiovascular disease in the next 10 years. If they were to take a combination of treatments (for example drugs to lower cholesterol levels plus drugs to lower blood pressure) that reduced their risk of cardiovascular disease by half it means that 10 of these 100 individuals would develop cardiovascular

disease in the next 10 years: 90 of them would not develop cardiovascular disease. This means that 10 of these individuals would have avoided cardiovascular disease by taking treatment for 10 years; 10 would get cardiovascular disease whether or not they took treatment; and 80 would not have got cardiovascular disease whether or not they took treatment

## INFLAMMATION AND OXIDATIVE STRESS

Inflammation and oxidative stress, which are prevalent in patients with CKD, are not controlled effectively by dialysis. Simmons et al3 have shown that pretransplant levels of the proinflammatory proteins interleukin-6, tumor necrosis factor-α, and CRP, as well as the oxidative stress markers plasma protein carbonyls and F2-isoprostanes, were increased significantly in CKD patients compared with healthy control subjects. After a successful kidney transplant, there was a rapid and sustained decline in all of these biomarkers, reaching levels of those of controls by 2 months posttransplant.

In a prospective study to determine the incidence and risk factors for ischemic heart disease in renal transplant recipients who were free of vascular disease at enrollment, coronary events were recorded in 7.8% of 344 consecutive renal transplant recipients at a mean follow up period of 72 ± 14 months. In addition to traditional Framingham risk factors, CRP level and hyperhomocysteinemia were found to be independent risk factors for ischemic heart disease events[77]. Increased CRP and other inflammatory markers also have been shown to be associated with an increased risk of all-

cause mortality in renal transplant recipients. In a single-center prospective study consisting of more than 400 consecutive kidney transplant recipients followed up for a median of 7.8 years, Winkelmayer et al[78] showed that patients with a CRP of 0.5 mg/dL or higher had a 53% higher mortality risk compared with patients whose CRP was below that threshold [hazard ratio (HR) = 1.53; 95% confidence interval, 1.01-2.31; *P* = .04]. No associations between CRP and the risk of kidney allograft loss were detected. Recent studies have established a link between inflammation, atherosclerosis, and other manifestations of cardiovascular disease.

Hansson[79]illustrated the similarities between the role of T-cell activation on plaque inflammation and on the alloimmune response. It is conceivable that the dramatic reduction in CVD mortality posttransplant compared with remaining on dialysis is, in part, related to the use of immunosuppressive agents that also are antiinflammatory. The putative role of inflammation in the development of pretransplant and posttransplant morbidity and mortality raises intriguing therapeutic options. Grotz et al[80] hypothesized that aspirin protects allograft function and survival in the context of chronic renal allograft dysfunction because of the similarities between the inflammatory mechanisms underlying atherogenesis and chronic allograft nephropathy. In a retrospective multivariate analysis performed to assess the effect of low-dose aspirin treatment (100 mg/d) on allograft function and survival, the Grotz et al[80] found that low-dose aspirin substantially improved median allograft survival time compared with no

aspirin treatment (low-dose aspirin versus no aspirin, 13.8 ± 2.6 years [n = 205] versus 7.8 ± 0.3 years [n =625], respectively; adjusted relative risk, 0.443; *P* =0 .0001).

Renal allograft function was better preserved in aspirin treated patients, who displayed a slower increase of serum creatinine level and less proteinuria and hematuria during the observation period. The investigators suggested that aspirin should be considered as part of the long-term posttransplant treatment regimen. The failed or failing kidney transplant also has been suggested to be a potential source of chronic inflammation, which contributes to higher morbidity and mortality rates among patients who returned to hemodialysis after failure of their kidney transplant compared with nontransplanted dialysis patients. Lopez-Gomez et al [81] found that hemodialysis patients with a failed kidney transplant in situ commonly suffered from a chronic inflammatory state and that transplant nephrectomy was associated with amelioration of markers of chronic inflammation, including improvement in serum albumin level, prealbumin level, ferritin level, fibrinogen level, CRP level, erythrocyte sedimentation rate, and erythropoietin resistance index. Transplant nephrectomy should be considered in patients with failed kidney transplants, particularly if they show clinical evidence of a chronic inflammatory state.

## PROTEINURIA

Proteinuria has been reported to occur in 9% to 40% of kidney transplant recipients with a functioning allograft [82].As in the nontransplant setting, posttransplantation proteinuria has been shown to be an independent risk factor for CVD. In a retrospective study consisting of more than 500 Caucasian patients who received a deceased-donor renal transplant and had a functioning allograft for longer than a year, Fernandez- Fresnedo et al [83] found that compared with no proteinuria, the presence of persistent proteinuria (defined as urine protein excretion greater than 0.5 g/d for more than 6 months; mean follow-up period, 6.41± 3.6 y) was associated with increased mortality and graft loss (relative risk of death and graft loss [RR], 1.92 and 4.18, respectively), and a higher incidence of CVD (RR, 2.45).

Roodnat et al[84] reported a nearly 2-fold risk of death in renal transplant recipients with a functioning allograft and proteinuria at 1 year compared with those without proteinuria. The literature on the link between proteinuria and increased CVD and related death, and its negative impact on patient and kidney allograft survival, has been increasingly recognized. It is suggested that proteinuria is a biomarker ofsystemic endothelial dysfunction inherent to the atherosclerotic process [85]. Unless contraindicated, ACE-I, ARB, or both should be considered in transplant recipients with microalbuminuria or overt proteinuria because of their well-established renoprotective, antiproteinuric, and cardioprotective effects. Whether the

development of proteinuria associated with sirolimus [86] adversely affects CVD risks currently is unknown and warrants close monitoring

**ANEMIA**

Anemia after renal transplantation has a reported prevalence of 20% to 80% [87]. The wide variation in the prevalence reported in part is owing to the variable definitions of anemia, immunosuppressive medications, time post transplantation, duration of follow-up evaluation, and level of allograft function, among others. In a retrospective study consisting of 92 renal transplant recipients with a functioning allograft at 1 year, post transplant anemia, defined as a hemoglobin level of less than 13 g/dL for men and less than 12 g/dL for women, was found in 35.5% and 25% of patients at months 6 and 12, respectively[88].In a multivariate analysis, the independent predictive factors of anemia at month 6 were erythropoietin level at day 0, cause of end-stage renal disease (polycystic kidney disease versus others), post transplantation recombinant erythropoietin therapy, hematocrit level at month 3, platelets at day 7, and sirolimus therapy. Delayed graft function, renal function at month 12, and anemia at month 6 were independent risk factors for the presence of persistent anemia at 1 year.

In a retrospective study consisting of more than 200 transplant recipients receiving sirolimus, nearly 60% were found to be anemic, a frequency nearly twice that for patients receiving MMF[89]. It has been suggested that sirolimus inhibits erythropoiesis at the level of the

erythropoietin receptor. The binding of erythropoietin to its cytoplasmic receptors leads to the activation of a cascade of phosphorylating enzymes including phosphoinositide 3-kinase, an enzyme responsible for controlling cell survival and cell-cycle progression in several cell lines including erythroid precursors. Sirolimus blocks p70S6-kinase, an enzyme downstream from phosphoinositide 3-kinase, and inhibits basal- as well as erythropoietinstimulated proliferation. Sirolimus, however, does not interfere with the maturation of the J2E erythroid cell line [90]. Suggested causative factors for posttransplant anemia include iron, folate, and B12 deficiency, impaired allograft function, acute rejection epi sodes, recent infection, and medications such as azathioprine, MMF, sirolimus, and ACE-I and ARB. Anemia also has been reported to be more common in African American and female transplant recipients. Similar to the general population and patients with chronic kidney disease, it has shown that anemia adversely affects CVD in kidney transplant recipients. In a multivariate analysis of more than 400 recipients of kidney alone or simultaneous kidney- pancreas transplants, Djamali et al [91] found that diabetic transplant recipients with a hematocrit level greater than 30% were less likely to suffer from a CVD event (myocardial infarction, cardiovascular death, angina, and congestive heart failure) in the first 6 posttransplant months compared with those with a hematocrit level less than or equal to 30%.

In a retrospective study involving consecutive de novo MMF-treated kidney recipients from the Hospital of the University of Pennsylvania

between 1996 and 2002, Imoagene- Oyedeji et al [92] revealed that the cohort with anemia at 12 months, defined as a hemoglobin level of less than 12 g/dL, had inferior patient survival and a higher proportion of cardiovascular deaths (6.3% versus 2.2%) compared with the nonanemic patients.

In a study involving more than 400 kidney transplant recipients, Winkelmayer et al[93] failed to show an association between anemia defined as a hemoglobin level less than 10 g/dL and mortality or graft loss. Among the iron parameters, only the percentage of hypochromic red cells was associated with greater all-cause mortality. The clinical significance and therapeutic implications of these findings remain to be determined. Darbepoetin alfa is an effective and safe alternative to recombinant human erythropoietin treatment for anemic renal transplant recipients. However,it currently is not known whether erythropoiesis-stimulating agents have a beneficial effect on CVD risk factor reduction beyond correction of post transplant anemia alone. Assessment of baseline iron stores at the time of transplantation may be invaluable because iron deficiency is not uncommon in the dialysis population. Profound iron deficiency should be treated with intravenous iron as tolerated. Refractory or severe anemia mandates aggressive evaluation to exclude the possibility of surgical postoperative bleeding, particularly in those with a rapid decrease in hemoglobin and hematocrit levels. Other possibilities include gastrointestinal bleed, tertiary hyperparathyroidism, underlying inflammatory conditions, or parvovirus B19 infection. Erythropoietin-

resistant anemia has been described in patients receiving sirolimus immunosuppression.

**HYPOALBUMINEMIA**

Up to 70% of the dialysis patients have some element of malnutrition and low serum albumin level is a predictor of mortality risk for ESRD patients on dialysis. Approximately 10% of patients exhibit hypoalbuminemia at 1 year and 20% at 10 years after transplantation. Low serum albumin levels may be the result of decreased production and/or increased catabolism [94,95]. Increased urinary protein excretion, especially in patients with chronic transplant nephropathy, may also result in low serum albumin levels. Chronic allograft nephropathy with hypoalbuminemia is associated with decrease in muscle mass, which is reflected in decreases in urinary creatinine excretion. Corticosteroids accelerate the protein catabolic rate and frequently create negative nitrogen balance. Studies by Hoy et al [96].have documented significant increases in the protein catabolic rate, accompanied by decreases in serum albumin levels, in the immediate posttransplant period. Even maintenance low-dose corticosteroid therapy increases protein catabolism and muscle wasting. Severe protein catabolism contributes to poor wound healing and an increased susceptibility to infection[97]. Early nutritional support is indicated in high-risk patients. Assessment of nutritional status by a renal dietitian should be incorporated into the clinic visit. Serum albumin should be monitored annually. Serum prealbumin levels should be measured if albumin levels are low or if clinical findings suggest possible malnutrition. The degree

of protein catabolism can be assessed by the measurement of urea nitrogen appearance. A daily protein intake ranging from 0.55 to 1.0 g/kg has been recommended for stable posttransplant patients.

## LEFT VENTRICULAR HYPERTROPHY AND CARDIO VASCULAR RISK

Left ventricular hypertrophy may be considered either a risk factor for subsequent major cardiovascular events, or a cardiovascular disease itself. Although this is still a topic for debate. LVH is present in 40–60% of renal transplant recipients, and its persistence in the first year after renal transplantation is associated with reduced patient survival [98]. LVH has also been shown to be the strongest predictor of all-cause mortality, together with diabetes[98]. Left ventricular hypertrophy is inversely correlated with renal function [99]. Improved renal function following renal transplantation ameliorates LVH; however, a degree of LVH is often still present in renal transplant recipients and may be exacerbated as graft function declines[100] .Renal dysfunction may increase LVH through hypertension,volume expansion, hyperparathyroidism, and/or altered calcium-phosphate homeostasis[101].Preliminary results from a clinical trial examining the effects of conversion from CNI to sirolimus showed a significant regression of LVH in the majority of renal transplant patients at 1 year after conversion[102] . This regression in LVH occurs mainly by decreasing left ventricular wall thickness, which suggests a nonhemodynamiceffect mechanism of sirolimus of the left ventricular mass [102] .

## HYPERURICEMIA

Renal handling of uric acid is affected by the use of CsA leading to higher serum urate levels in CsA-treated patients[103,104,105]. Asymptomatic hyperuricemia occurs in 55% of patients receiving CsA and in 25% of those taking azathioprine. There is no report of graft failure due to urate nephropathy in the transplanted kidney. Crystal-induced erosive arthritis can occur in these patients. Nonsteroidal anti-inflammatory agents should also be avoided because of potential negative influence on renal hemodynamics and the development of interstitial nephritis. Colchicine is the preferred treatment if symptoms persist. Allopurinol, a xanthine oxidase inhibitor, should be avoided in patients taking azathioprine. Concomitant administration of allopurinol and azathioprine results in marrow suppression and a fourfold increase in immunosuppression

## POST TRANSPLANT ERYTHROCYTOSIS

An increased erythrocyte mass has been demonstrated in some 17% of graft recipients[106]. Erythrocytosis, defined as a hematocrit greater than 52%, most often occurs within the first year after transplantation and may be associated with good allograft function, chronic rejection, transplant renal artery stenosis[107], hydronephrosis, native kidney and hepatic erythropoietin production, and the use of androgenic steroids. In patients with good allograft function, it is postulated that correction of the uremic milieu allows

overzealous red blood cell production, because of a reset marrow response to erythropoietin (EPO) [108,109].

In patients with chronic rejection, renal artery stenosis, and hydronephrosis, intrarenal hypoxemia may stimulate erythropoietin production [110].In most cases, the precise etiology is uncertain, but studies on erythropoietin levels after transplantation indicate that graft function restores the hematopoietic response to normal. The phenomenon usually is self-limited, lasting 3 to 12 months. Low doses of an ACE inhibitor (beginning with 2.5 mg of enalapril per day or 12.5 mg of captopril twice a day) reduces the hematocrit to normal or near normal levels. The effect begins within 6 weeks and is complete in 3 to 6 months.

An association between the ACE inhibitor-induced reduction in hematocrit and a fall in plasma EPO levels has been demonstrated in some studies. Also compatible with an EPO-independent mechanism is the observation that withdrawal of the ACE inhibitor results in a gradual rise in hematocrit without a concurrent elevation in EPO levels. ACE inhibitors can also induce anemia in some renal transplant recipients without erythrocytosis. The mechanism of action is unclear. An alternative to ACE inhibition is theophylline. Theophylline appears to act as an adenosine antagonist in this setting, suggesting that adenosine facilitates both the release and perhaps the bone marrow response to EPO [111-114]. In severe cases (hematocrit >52%), phlebotomy is indicated to prevent thromboembolic complications, which may occur in as many as 20% of patients with erythrocytosis.

## CAROTID INTIMA MEDIA THICKNESS

Links between CIMT and coronary heart disease[115] or stroke[116] [14] are well known. An association between carotid intima media thickness (CIMT) and cardio-vascular risk factors has been demonstrated in several epidemiological studies[117-121]. Ultrasound measurement of the two internal layers of the carotid artery is a validatedtechnique[122]. IMT study has opened a broad field in clinical research because it detects early arterial disease in asymptomatic individuals and is significantly associated to a higher risk of incident myocardial infarction and stroke[123] . IMT measurements could constitute an important tool to identify and target intermediate risk subjects in preventive medicine. In this large cross-sectional study of 5199 subjects, found that the Framingham score and CIMT values were significantly correlated

# MATERIALS AND METHODS

**Study place** **:** Govt.Stanley Medical College & Hospital Nephrology Department, Chennai

**Study period** **:** From October 2010 to November 2011

**Study design** **:** Retrospective Analytical study

**Study population** **:** Cadaver and Live related renal transplant recipients(RTR)

**EXCLUSION CRITERIA**

- Less than one month post transplant
- Less than 18 years of age
- Death due to non cardiac causes during the study
- Graft dysfunction and on maintanance hemodialysis

- Ethical Committee approval from Stanley Medical College, Chennai was obtained for this study.
- All recipients were ABO compatible and cross-match negative and they are followed up regularly in NEPHROLOGY TRANSPLANT OPD.
- Recipients demographic factors like Age, Gender, Occupation, Literacy were noted.
- Nature of donor, post transplant duration, graft function were noted.
- fasting blood samples were drawn to determine serum creatinine, Total cholesterol, Triglycerides, LDL and HDL cholesterol and plasma glucose concentrations.
- Blood pressure was reported as the average of three manual measurements taken at 3-minutes intervals.
- Hypertension was defined by (i) the administration of antihypertensive agents and/or a history of this disorder; (ii) a systolic blood pressure more than 130 mmHg; or (iii) a diastolic blood pressure more than 80 mmHg.
- NODAT was defined as a fasting glucose of more than 126mg/dl on two occasions at any time after transplantation or associated with use of oral hypoglycemic agents and/or insulin, in patients with no prior history of diabetes.

- Weight was measured by the weighing machine which was daily calibrated by a weight of 5 kg, with the subject in light clothing.
- Height was measured by a scale fitted on the wall and the subject standing without shoes. BMI was calculated by the formula weight in kg / height in $m^2$.
- Waist circumference was determined by using a non-stretchable measuring tape midway between the iliac crest and costal margin.
- 2 hours post prandial Blood samples were taken to analyze sugar, hemoglobin, serum albumin,uricacid.
- Diagnosis of Metabolic Syndrome(MS) was made by The National Cholesterol Education Program- Adult Treatment Panel III (NCEP ATP III) criteria such as

(Subjects who had three or more of the risk factors were labelled as MS)

- Fasting blood glucose >110 mg/dl or use of antidiabetic medication,
- Waist circumference >40inch for men and >35inch for women
- (iii) SBP > 130 mmHg and DBP more than 85 mmHg or use of antihypertensive medication,
- (iv) TGL >150mg/dl or specific treatment for this lipid abnormality

- (v) HDL — cholesterol level <40mg/dl in men or <50mg/dl in women or specific treatment for this lipid abnormality,
- Routine urine investigation was done by dipstick method
- Hemoglobin less than 13.5gm/dl in males, less than 12gm/dl in females considered as anaemia and more than 17gm/dl defined as post transplant erythrocytosis
- Framingham risk score was determined by online calculator(hp2010.nhlbihin.net/atpiii/calculator.asp)
- Average carotid intima media thickness was measured by ALOKA ultra sonogram machine.
- Echocardiogram was done to analyze cardiac function, regional wall motion abnormality, left ventricular hypertrophy and ejection fraction

**STATISTICAL METHODOLOGY**

The statistical analysis had been done by using SPSS (Statistical Package on Social Science) version 15.0

The non-parametric model can be used to find out the relationship of categorical variable. One of the method was Pearson's exact Chi-square.

Multi variate analysis was done by MULTIPLE LOGISTIC REGRESSION ANALYSIS

# RESULTS

# RESULTS

Totally 170 recipients are on regular follow up in our department from the period October 2010 to November 2011.

Patients who died in that period and those who are on irregular follow up are excluded from the study

Total patients are divided into groups according to FRAMINGHAM RISK SCORE to predict 10 year ABSOLUTE RISK of coronary heart disease event.

Recipients were fit into risk category of 1-3%, 3-5%, 5-8%, 8-10% with prevalence of 80.6%, 11.8%, 4.7%, 2.9% respectively.

| FRS | NUMBER | PERCENTAGE |
|---|---|---|
| 1 – 3% | 137 | 80.60% |
| 3- 5% | 20 | 11.80% |
| 5- 8% | 8 | 4.70% |
| 8- 10% | 5 | 2.90% |

The following recipient's variables are compared with risk groups

1. Age
2. Sex
3. Donor type
4. Post transplant duration in months
5. Graft dysfunction
6. Body mass index
7. New Onset Diabetes After Transplantation(NODAT)
8. Systemic hypertension
9. Total Cholesterol
10. HDL cholesterol
11. LDL cholesterol
12. Triglycerides
13. Immunosuppressive drugs
14. ECHO-cardiac function
15. ECHO-regional wall motion abnormality
16. ECHO-left ventricular hypertrophy
17. ECHO-ejection fraction
18. Urine routine
19. Hemoglobin
20. Serum albumin
21. Serum uricacid
22. Carotid intima media thickness

All the data are collected in the master sheet and statistically analyzed

| AGE(YEARS) | NUMBER | PERCENTAGE |
|---|---|---|
| <40 | 144 | 84.7% |
| >40 | 26 | 15.3% |

AGE * FRS

| variable | PREVALANCE | | | | p-Value |
|---|---|---|---|---|---|
| Age in years | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| Below 40 | 78.9% | 8.3% | 2.8% | 0% | |
| Above 40 | 34.6% | 30.8% | 15.4% | 19.2% | 0.001 |

Increased recipient age significantly associated with high cardiovascular risk factor score, p- Value .001

| SEX | NUMBER | PERCENTAGE |
|---|---|---|
| MALE | 124 | 72.94% |
| FEMALE | 46 | 27.06% |

SEX * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| SEX | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| MALE | 79.8% | 12.9% | 4.0% | 3.2% | |
| FEMALE | 82.6% | 8.7% | 6.5% | 2.2% | 0.893 |

There was no gender predisposition towards cardio vascular risks p-Value 0.8

| DONOR | NUMBER | PERCENTAGE |
|---|---|---|
| LIVE | 142 | 83.52% |
| CADAVER | 28 | 16.48% |

DONOR * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| DONOR | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| LIVE | 83.1% | 12.0% | 2.8% | 2.1% | 0.026 |
| CADAVER | 67.8% | 10.7% | 14.3% | 7.1% | |

Increased cardio vascular risk score associated with cadaveric graft recipients which was statistically significant, p-Value 0.026

| POST TRANSPLANT DURATION | NUMBER | PERCENTAGE |
|---|---|---|
| <12 MONTHS | 29 | 17.05% |
| 13 – 24 MONTHS | 32 | 18.82% |
| 25 – 48 MONTHS | 33 | 19.42% |
| >48 MONTHS | 76 | 44.71% |

POST TRANSPLANT DURATION* FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| Post transplant duration in months | FRSGroup | | | | 0.273 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| <12 | 86.2% | 10.3% | 3.4% | 0 % | |
| 13-24 | 71.9% | 15.6% | 9.4% | 3.1% | |
| 25-48 | 78.7% | 9.1% | 9.1% | 3.0% | |
| >48 | 82.9% | 11.8% | 1.3% | 3.9% | |

Duration of post transplant period was not significantly associated with Cardiovascular risk score

| GRAFT | NUMBER | PERCENTAGE |
|---|---|---|
| DYSFUNCTION | 88 | 51.75% |
| NORMAL FUNCTION | 82 | 48.25% |

GRAFT DYSFUNCTION * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| GRAFT DYSFUNCTION | FRSGroup | | | | 0.458 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO | 83.0% | 9.8% | 6.1% | 1.2% | |
| YES | 78.4% | 13.6% | 3.4% | 4.5% | |

Higher risk value for graft dysfunction recipients

Influence of graft dysfunction over cardio vascular risk score- not significant

p-Value 0.458

| BMI | NUMBER | PERCENTAGE |
|---|---|---|
| UNDERWEIGHT | 28 | 16.47% |
| NORMAL WEIGHT | 105 | 61.76% |
| OVERWEIGHT | 30 | 17.64% |
| OBESE | 7 | 4.13% |

BODY MASS INDEX * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| BMI | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| UNDER WEIGHT | 82.1% | 7.1% | 10.7% | 0 % | |
| NORMAL | 81.9% | 10.5% | 2.9% | 4.8% | |
| OVER WEIGHT | 76.6% | 20.0% | 3.3% | 0% | 0.120 |
| OBESEITY I | 71.4% | 14.3% | 14.3% | 0% | |

Influence of BMI over cardio vascular risk score not statistically significant

| METABOLIC SYNDROME | NUMBER | PERCENTAGE |
|---|---|---|
| NO | 117 | 68.82% |
| YES | 53 | 31.18% |

METABOLIC SYNDROME * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| METABOLIC SYNDROME | FRSGroup | | | | 0.001 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO | 89.7% | 9.4% | 0% | 0.9% | |
| YES | 60.4% | 17.0% | 15.1% | 7.5% | |

Presence of metabolic syndrome had significant association with high cardio vascular risk score

| NODAT | NUMBER | PERCENTAGE |
|---|---|---|
| NO | 136 | 79.42% |
| YES | 33 | 19.41% |
| PRE TRANSPLANT DM | 2 | 1.17% |

**NEW ONSET DIABETES AFTER TRASPLANTATION * FRS**

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| NODAT | FRSGroup | | | | 0.001 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO | 90.3% | 8.1% | 0.7% | 0.7% | |
| YES | 42.5% | 27.3% | 18.2% | 12.1% | |
| Pre transplant DM | 50% | 0% | 50% | 0% | |

NODAT had significant correlation with cardio vascular risk score

| SHT | NUMBER | PERCENTAGE |
|---|---|---|
| NO | 34 | 20% |
| YES | 136 | 80% |

SYSTEMIC HYPERTENSION * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| SYSTEMIC HYPERTENSION | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO | 84.8% | 6.1% | 3.0% | 6.1% | |
| YES | 79.5% | 13.1% | 5.1% | 2.2% | 0.496 |

There was a non significant , but increasing risk score in those with SHT

| IMMUNOSUPPRESSION | NUMBER | PERCENTAGE |
|---|---|---|
| AZATHIOPRINE<br>PREDNISOLONE | 65 | 38.22% |
| MMF<br>PREDNISOLONE | 27 | 15.88% |
| CYCLOSPORINE<br>AZATHIOPRINE<br>PREDNISOLONE | 3 | 1.76% |
| CYCLOSPORINE<br>MMF<br>PREDNISOLONE | 4 | 2.35% |
| TACROLIMUS<br>AZATHIOPRINE<br>PREDNISOLONE | 11 | 6.47% |
| TACROLIMUS<br>MMF<br>PREDNISOLONE | 57 | 33.52% |
| SIROLIMUS<br>MMF<br>PREDNISOLONE | 3 | 1.76% |

IMMUNO SUPPRESSIVE DRUGS * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| **IMMUNO SUPPRESSIVE DRUGS** | **FRSGroup** | | | | 0.001 |
| | **1-3%** | **3-5%** | **5-8%** | **8-10%** | |
| AZATHIOPRINE PREDNISOLONE | 85.5% | 8.2% | 0% | 3.3% | |
| MMF PREDNISOLONE | 73.3% | 26.7% | 0% | 0% | |
| CYCLOSPORINE AZATHIOPRINE PREDNISOLONE | 100% | 0% | 0% | 0% | |
| CYCLOSPORINE MMF PREDNISOLONE | 50.0% | 25.0% | 25.0% | 0% | |
| TACROLIMUS AZATHIOPRINE PREDNISOLONE | 100% | 0% | 0% | 0% | |
| TACROLIMUS MMF PREDNISOLONE | 77.2% | 10.5% | 10.5% | 1.8% | |
| SIROLIMUS MMF PREDNISOLONE | 33.3% | 0% | 33.3% | 33.4% | |

Among immunosuppressive drugs, Cyclosporine, Tacrolimus, Sirolimus. Prednisolone and MMF significantly associated with high cardio vascular risk score

| ECHO-FUNCTION | NUMBER | PERCENTAGE |
|---|---|---|
| DIASTOLIC DYSFUNCTION | 15 | 8.82% |
| NORMAL FUNCTION | 155 | 91.18% |

ECHO FUNCTION * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| ECHO FUNCTION | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NORMAL | 82.6% | 11.6% | 5.2% | 0.6% | 0.001 |
| DIASTOLIC DYSFUNCTION | 60.0% | 13.3% | 0% | 26.7% | |

Cardiac dysfunction was present in 15 (8.82%) recipients, all had diastolic dysfunction, none had systolic dysfunction and significantly associated with cardiovascular risk score

| ECHO- LVH | NUMBER | PERCENTAGE |
|---|---|---|
| NO | 131 | 77.06% |
| YES | 39 | 22.94% |

LEFT VENTRICULAR HYPERTROPHY * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| LVH | FRSGroup | | | | 0.243 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO | 84.0% | 9.9% | 3.8% | 2.3% | |
| YES | 69.3% | 17.9% | 7.7% | 5.1% | |

Even though statistically not significant, high risk score was observed in patients with LVH .

| **TOTAL CHOLESTEROL (mg/dl)** | **NUMBER** | **PERCENTAGE** |
|---|---|---|
| <200 | 139 | 81.77% |
| >200% | 31 | 18.23% |

TOTAL CHOLESTEROL * FRS

| **variable** | **PREVALENCE** | | | | p-Value |
|---|---|---|---|---|---|
| TOTAL CHOLESTEROL (mg/dl) | **FRSGroup** | | | | 0.001 |
| | **1-3%** | **3-5%** | **5-8%** | **8-10%** | |
| <200 | 84.9% | 11.5% | 2.9% | 0.7% | |
| >200 | 61.3% | 12.9% | 12.9% | 12.9% | |

High cholesterol was significantly correlated with cardio vascular risk score

| HDL | NUMBER | PERCENTAGE |
|---|---|---|
| LOW | 31 | 26.47% |
| NORMAL | 128 | 75.29% |
| HIGH | 11 | 6.47% |

HDL CHOLESTEROL * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| HDL CHOLESTEROL | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| LOW | 66.6% | 25.0% | 6.3% | 3.1% | 0.019 |
| NORMAL | 72.8% | 9.1% | 9.1% | 9.1% | |
| HIGH | 85.8% | 8.7% | 3.1% | 2.4% | |

Low HDL-cholesterol was significantly correlated with cardio vascular risk score

| TGL (mg/dl) | NUMBER | PERCENTAGE |
|---|---|---|
| <100 | 135 | 79.42% |
| >100 | 35 | 20.58% |

LDL CHOLESTEROL * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| LDL CHOLESTROL (mg/dl) | FRSGroup | | | | 0.012 |
| | **1-3%** | **3-5%** | **5-8%** | **8-10%** | |
| <100 | 85.2% | 10.4% | 3.0% | 1.5% | |
| >100 | 62.9% | 17.1% | 11.4% | 8.6% | |

High LDL- cholesterol was significantly correlated with cardio vascular risk score

| TGL (mg/dl) | NUMBER | PERCENTAGE |
|---|---|---|
| <200 | 160 | 94.12% |
| >200 | 10 | 5.88% |

TGL * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| TGL (mg/dl) | FRSGroup | | | | 0.004 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| <200 | 82.7% | 11.2% | 4.3% | 1.9% | |
| >200 | 44.4% | 22.2% | 11.2% | 22.2% | |

High TGL was significantly correlated with cardio vascular risk score

| URINE ROUTINE | NUMBER | PERCENTAGE |
|---|---|---|
| NO PROTEINURIA | 139 | 81.76% |
| PROTEINURIA | 31 | 18.24% |

URINE ROUTINE * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| URINE ROUTINE | FRSGroup | | | | 0.001 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| NO PROTEINURIA | 86.3% | 9.4% | 2.9% | 1.4% | |
| PROTEINURIA | 54.9% | 22.6% | 12.9% | 9.7% | |

Proteinuria was significantly associated with cardio vascular risk score

| HEMOGLOBIN | NUMBER | PERCENTAGE |
|---|---|---|
| NORMAL | 128 | 75.29% |
| PTE | 15 | 8.82% |
| ANEMIA | 27 | 15.88% |

HEMOGLOBIN * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| HEMOGLOBIN | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| ANEMIA | 55.5% | 25.9% | 14.8% | 3.7% | 0.014 |
| NORMAL | 86.6% | 8.7% | 2.4% | 2.4% | |
| PTE | 75.1% | 12.5% | 6.3% | 6.3% | |

Both Anaemia and post transplant erythrocytosis had significant cardio vascular risk score

| S. ALBUMIN (gm/dl) | NUMBER | PERCENTAGE |
|---|---|---|
| >3.5 | 127 | 74.71% |
| <3.5 | 43 | 25.29% |

SERUM ALBUMIN * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| SERUM ALBUMIN (gm/dl) | FRSGroup | | | | 0.679 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| >3.5 | 82.7% | 10.2% | 4.7% | 2.4% | |
| <3.5 | 74.5% | 16.3% | 4.7% | 4.7% | |

Hypoalbuminemia had no significant association with cardio vascular risk score

| URIC ACID mg/dl | NUMBER | PERCENTAGE |
|---|---|---|
| <6 | 131 | 77.05% |
| >6 | 39 | 22.95% |

URIC ACID * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| URIC ACID (mg/dl) | FRSGroup | | | | |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| < 6.0 | 82.3% | 10.8% | 4.6% | 2.3% | |
| > 6.0 | 75.0% | 15.0% | 5.0% | 5.0% | 0.481 |

Influence of serum uric acid over cardio vascular risk score not statistically significant

| CIMT(mm) | NUMBER | PERCENTAGE |
|---|---|---|
| <1.1 | 158 | 92.95% |
| ≥1.1 | 12 | 7.05% |

CAROTID INTIMA MEDIA THICKNESS * FRS

| variable | PREVALENCE | | | | p-Value |
|---|---|---|---|---|---|
| CAROTID INTIMA MEDIA THICKNESS (mm) | FRSGroup | | | | 0.001 |
| | 1-3% | 3-5% | 5-8% | 8-10% | |
| <1.1 | 81.0% | 12.0% | 4.4% | 2.5% | |
| ≥1.1 | 72.7% | 9.1% | 9.1% | 9.1% | |

Carotid intima media thickness had statistically significance correlation with high cardio vascular risk score

# DISCUSSION

# DISCUSSION

Cardiovascular mortality is increased in patients with chronic kidney disease. Mortality from cardiovascular disease is10–20 times higher among individuals treated with dialysis, as compared to general population[124].1 The incidence of cardiovascular disease in kidney transplant patients is nearly twice that of the general population[125]. Even young transplant recipients (aged 35–45 years) experienced an almost 10-fold increase in cardiovascular disease-related mortality.

Our study analyzed the relationships among traditional and transplant specific risk factors and 10 year cardiovascular risk estimated by Framingham risk score

Overall 170 recipients who were on regular follow up in our department were included in this analysis.

Total patients are divided into groups according to FRAMINGHAM RISK SCORE to predict 10 year ABSOLUTE RISK of coronary heart disease event. Recipients were fit into risk category of 1-3%, 3-5%, 5-8%, 8-10%

We found that 80.6% of recipients had 1-3% of 10 year CV risk, 11.8% had 3-5% of 10 year CV risk,4.7% had 5-8% of 10 year CV risk and 2.9% had 8-10% of 10 year CV risk.

Among the traditional risk factors, age > 40years was found to be statistically significant risk factor for 10 year CV risk. Almost 15.3% of the total study population was >40 years of which 19.2% had 10 year CV risk of

8-10%. This was in comparison to study done by deMattos AM et al [13] who showed that Age > 45 years had hazard ratio of 1.57(95% CI 0.99-2.38). In our study increased age was to be independent risk factor for higher CV risk in the multi variate analysis

Although male gender was proposed as one of the traditional risk factor in many studies. In our study 72.9% of the population were males and there was no statistically significant higher CV risk in the males.

In our study, 83.5% had live related donor and 16.48% had cadaver donors, Lentine et al [126] and deMattos AM et al[13] found an increased risk of cardiovascular events among deceased donor recipients. In our study 7.1% of deceased donor recipients had 8-10% of 10 year CV risk.

In a study by kasiske et al [127] showed a markedly increased risk of acute myocardial infarction early after transplantation, less than three months post-surgery where as in our study cardiovascular risk was not significantly associated with post transplant duration though had a score of < 3%,higher score (>3%) was observed in those with more than 4 years of post transplant period

Renal insufficiency in renal transplant patients is a significant risk factor for adverse cardiovascular outcomes, Serum creatinine levels above 1.5 mg/dL (133 µmol/L) at one year post-transplant were significantly associated with an increased risk for cardiovascular disease by Meier-Kriesche HU et al[53] .In a second study done in 30,000 renal transplant recipients by Abbott KC et al [54], a decreased estimated glomerular filtration

rate at one year post-transplant correlated with increased risks of acute coronary syndrome and heart failure .

In the Assessment of LEscol in Renal Transplantation (ALERT) trial[55], an increased serum creatinine concentration, particularly higher than 2.3 mg/dL (200 micromol/L), was strongly associated with an increased risk of adverse cardiac events and cardiac death by Fellström B et al [56] In our study graft dysfunction was present in 51.75% and among them 4.5% had non significant but increased 10 year CV risks compared to those who had normal graft function in which only 1.2% had a risk score of 8-10%

In study by Courivaud C et al [58] , one-third of them had metabolic syndrome, and significant higher incidence of cardio vascular events was observed among patients with this syndrome, in our study prevalence of metabolic syndrome was 31.8% of which 7.5% had a CV risk score of 8-10% compared to 0.9% in the group without MS

The reported incidence of NEW ONSET DIABETES AFTER TRASPLANTATION(NODAT) in renal transplant recipients is 4% to 25%, in our study it the incidence of NODAT was 19.41%. Ojo AO et al [9] stated that kidney transplant recipients who developed NODAT are at 2- to 3- fold increased risk of fatal and nonfatal CVD events. we also observed statistically significant relation between NODAT and higher cardiovascular risk. In our study among the 19.41% of patients with NODAT,12.1% had a CV risk of 8-10% compared to 0.7% in those without NODAT.

Hypertension is present in 50% to 90% of renal transplant recipients stated by Kasiske et al[27]. Though Systemic Hypertension was regarded as one of the modifiable riskfactor of CV risks compared to general population ,renal transplant recipients had higher prevalence of SHT and because of its universal distribution deMattos AM et al[13] did not find any correlation between SHT and cardiovascular risk in this study. In our study SHT was present in 80% of the patients and there was no statistically significant relation between SHT and higher CV risks

By Gonyea JE et al [128] and Moore R et al [129] from single- and multi-center reports estimate that, by one year post-transplant, 80 to 90 percent of adult recipients have total cholesterol levels >200 mg/dL , and 90 to 97 percent have LDL levels >100 mg/dL . In the study by Kasiske B et al [130], elevated serum cholesterol was found to be risk factor for CVevents. In our study prevalence of total cholesterol levels >200 mg/dL was 18.23%, LDL levels >100 mg/dL was 20.58%, triglyceride levels>200 mg/dL was 5.88%. All dyslipidemia was significantly associated with higher cardiovascular risk score and serum cholesterol levels >200 mg/dL was found to be independent risk factor for higher CV risk in the multi variate analysis.

Among the immunosuppressive groups though there are no literature support for direct correlation between immunosuppressive drugs and CV risk,in our study we found higher CV risks among patients with Azathioprine and Prednisolone combination(3.3% had risk of 8-10%) followed by Tacrolimus, MMF, and Prednisolone combination(10.5% had risk of 5-8%).This may be due to prevalence of other riskfactors like Tacrolimus

induced higher incidence of NODAT and those with Azathioprine and Prednisolone had longer post transplant duration.

On cardiac evaluation ,8.8% had diastolic dysfunction , 22.9% had LVH and none of them had systolic dysfunction or Regional Wall Motion Abnormality. In comparison to studies done in general population, those with diastolic dysfunction had statistically significant correlation with higher CV risks.Those with LVH had non significant higher CV risks(5.1% Vs 2.3%) compared to those without LVH.

Prevalence of proteinuria among recipients in our study was 18.24% which is significantly associated high cardio vascular risk score .In study by peddi VR et al [82] Proteinuria has been reported to occur in 9% to 40% of kidney transplant recipients, Fernandez-Fresnedo et al[83] found that compared with no proteinuria, the presence of persistent proteinuria was associated with increased mortality, graft loss and a higher incidence of CVD

Prevalence of anemia in this study was 15.88%,where as post transplant erythrocytosis was 8.8%,the reported prevalence among renal transplant recipients of 20% to 80% by Afzali B et al [87], Report from Vlahakos DV et al [131] PTE is affecting 8 to 15 percent of kidney transplant Recipients. In our study both anaemia and post transplant erythrocytosis associated with higher cardio vascular risk score which was statistically significant in compare to study by Imoagene- Oyedeji et al [92] revealed that the cohort with anemia at 12 months, defined as a hemoglobin level of less than 12 g/dL, had inferior patient survival and a higher proportion of

cardiovascular deaths (6.3% versus 2.2%) compared with the nonanemic patients

Approximately 10% of patients exhibit hypoalbuminemia at 1 year and 20% at 10 years after transplantation as reported by Guijarro C et al [94] in our study prevalence of hypoalbuminemia was 25.29% and had no correlation with cardio vascular risks.

The incidence of hyperuricemia in renal transplant recipients was 84 percent in those treated with cyclosporine versus 30 percent in patients treated with azathioprine and prednisone reported by Lin HY et al [103], in our study prevalence of hyperuricemia was 22.95%,33 percent in those treated with cyclosporine versus 9.3 percent in patients treated with azathioprine and prednisone. There was no significant correlation between hyperuricemia and high CV risks.

Among the non invasive investigation, Carotid intima media thickness (CIMT)was found to be independent values were significantly correlated in general population studied .Pierre JT et al[132] , compared CIMT and Framingham CV risk score , in his study showedThe Framingham score and CIMT values were non-linearly related (coefficients of determination $R^2$ were 19% and 20% in men, 28% and 29% in women, for subjects with and without personal history of cardio-vascular disease, respectively). In our study CIMT of $\geq$ 1.1mm was present in 7% of the recipients and among them 9.1% had CV risk score of 8-10% compared to 2.5% in those with CIMT <1.1mm and is statistically significant.

In summary, univariate analysis showed significant correlation between factors such as increased age, cadaveric graft recipients, metabolic syndrome, NODAT, high serum cholesterol, high LDL. high TGL,diastolic dysfunction, proteinuria, anaemia, post trasplant erythrocytosis and high carotid intima media thickness

In multi variate regression analysis revealed that the following risk factors were independently associated with post-transplant cardiovascular disease:

INCREASING AGE

NEW ONSET DIABETES AFTER TRASPLANTATION

ELEVATED SERUM CHOLESTROL

PROTEINURIA

Similar to study done by Kasiske BL et al [13] which revealed Increasing patient age, Diabetes Mellitus, Male sex, Cigarette smoking, Hypertension, Elevated serum cholesterol have risk factors for CVD.

The risk factors should be managed accordingly to reduce the cardiovascular events in the renal transplant recipients, so that the longevity of both grtaft and the patient is enhanced.

# CONCLUSION

# CONCLUSION

According to Univariate analysis following variables were concluded as cardiovascular risk factors

- INCREASED AGE,
- CADAVERIC GRAFT RECIPIENTS,
- METABOLIC SYNDROME,
- NEW ONSET DIABETES AFTER TRANSPLANTATION,
- ELEVATED SERUM CHOLESTEROL,
- ELEVATED LDL CHOLESTEROL,
- ELEVATED TGL CHOLESTEROL,
- DIASTOLIC DYSFUNCTION,
- PROTEINURIA,
- ANEMIA,
- POST TRANSPLANT ERYTHROCYTOSIS and
- HIGH CAROTID INTIMA MEDIA THICKNESS.

INDEPENDENT risk factors derived from multivariate analysis were

- INCREASING AGE
- NEW ONSET DIABETES AFTER TRANSPLANTATION
- ELEVATED SERUM CHOLESTEROL
- PROTEINURIA

- All transplant recipients should currently be considered as coronary heart disease risk.

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# ANNEXURES

- **PROFORMA**
- **MASTER CHART**
- **ETHICAL COMMITTEE APPROVAL FORM**

# PROFORMA

**CARDIOVASCULAR RISKFACTORS ANALYSIS IN RENAL TRANSPLANT RECIPIENTS**

Name :

Age :

Sex :

Neph no:

Donor:

Date of surgery:

Post transplant duration:

DGF :

Weight:

height:

BMI :

Waist circumference:

NODAT:

SHT :

Total cholesterol:

LDL :

Triglycerides(TGL):

HDL :

Present creatinine:

Urine routine:

Hemoglobin :

Serum albumin :

S .uricacid :

Carotid intima media thickness:

ECHOFunction :

RWMA :

LVH :

EF :

Immunosuppressive drugs :

| s.no | name | Nep. No. | age | sex | donor | p.tx.duration | DGF | dis.creatinine | present.creat | graftdysfunction | BMI | MS | NODAT | SHT | Immunosuppression | function | RWMA | LVH | EF | TC | HDL | LDL | TGL | FRS | u.routine | HCT | s.albumin | uricacid | CIMT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | abdulrahim | 1118/09 | 30 | M | C | 14 | N | 1.7 | 1.2 | N | U | N | Y | N | TMP | nrl | nil | N | 68 | 215 | 88 | 111 | 85 | 1 | nrl | nrl | m | l | l |
| 2 | amthulvali | 1243/09 | 21 | F | C | 14 | N | 1.2 | 1.2 | N | nrl | N | N | Y | TMP | nrl | nil | N | 63 | 188 | 49 | 104 | 176 | 1 | nrl | nrl | m | l | l |
| 3 | anandhan | 144/07 | 33 | M | L | 55 | Y | 1.9 | 2.5 | Y | ob1 | Y | N | Y | MP | nrl | nil | Y | 70 | 170 | 36 | 100 | 198 | 1 | nrl | nrl | m | l | l |
| 4 | anbalagan | 950/07 | 38 | M | L | 44 | N | 1 | 2.2 | Y | ov | Y | N | Y | AP | nrl | nil | N | 65 | 168 | 46 | 86 | 60 | 5 | pro | ane | l | m | l |
| 5 | arjunan | 242/09 | 36 | M | L | 30 | N | 1 | 2.2 | Y | U | N | N | Y | TMP | nrl | nil | N | 68 | 140 | 29 | 90 | 102 | 3 | nrl | nrl | m | l | l |
| 6 | ashokkumar | 522/07 | 36 | M | L | 50 | Y | 2 | 3 | Y | ov | Y | Y | Y | MP | nrl | nil | Y | 66 | 180 | 54 | 101 | 125 | 5 | nrl | nrl | m | l | l |
| 7 | anitha | 562/02 | 26 | F | L | 117 | N | 1 | 1 | N | nrl | N | N | N | AP | nrl | nil | N | 72 | 151 | 48 | 70 | 104 | 1 | nrl | nrl | m | l | l |
| 8 | ayubkhan | 270/08 | 24 | M | L | 41 | N | 1 | 3 | Y | nrl | N | N | Y | MP | nrl | nil | Y | 70 | 185 | 41 | 114 | 108 | 1 | pro | nrl | m | m | l |
| 9 | babu | 1081/01 | 21 | M | L | 114 | N | 0.6 | 1 | N | nrl | N | N | Y | AP | anrl | nil | N | 66 | 148 | 41 | 154 | 197 | 2 | nrl | nrl | m | l | l |
| 10 | balaji | 930/04 | 26 | M | L | 82 | N | 1.2 | 1 | N | ob1 | Y | N | Y | AP | nrl | nil | N | 65 | 130 | 46 | 86 | 183 | 2 | nrl | nrl | m | l | l |
| 11 | balakrishnan | 1352/08 | 34 | M | C | 33 | Y | 2.9 | 1.4 | N | ov | N | Y | Y | TMP | nrl | nil | N | 72 | 151 | 47 | 98 | 140 | 2 | nrl | ane | l | l | l |
| 12 | balasubramani | 266/03 | 39 | M | L | 101 | N | 1.5 | 1.5 | N | nrl | N | N | Y | AP | nrl | nil | Y | 68 | 168 | 49 | 90 | 107 | 3 | nrl | nrl | m | l | l |
| 13 | balraj | 1203/09 | 20 | M | L | 19 | N | 1 | 1.2 | N | nrl | N | N | Y | TAP | nrl | nil | N | 66 | 180 | 50 | 149 | 183 | 1 | nrl | pte | m | l | l |
| 14 | bavanandhi | 48/99 | 44 | M | L | 144 | N | 1 | 2.6 | Y | nrl | N | N | Y | AP | nrl | nil | Y | 72 | 151 | 58 | 119 | 119 | 5 | nrl | nrl | m | l | l |
| 15 | baskar | 223/06 | 37 | M | L | 62 | N | 1.2 | 2.5 | Y | U | N | N | Y | AP | nrl | nil | N | 65 | 185 | 47 | 107 | 107 | 3 | nrl | nrl | m | m | l |
| 16 | buhari | 002/02 | 33 | M | L | 120 | N | 1 | 2 | Y | ov | Y | N | Y | AP | nrl | nil | N | 63 | 180 | 49 | 100 | 100 | 1 | pro | nrl | m | m | m |
| 17 | bhuwaneshwari | 429/06 | 25 | F | L | 63 | N | 0.8 | 1.8 | Y | ov | Y | N | N | AP | nrl | nil | N | 70 | 140 | 31 | 120 | 87 | 5 | nrl | nrl | l | m | l |
| 18 | chandrasekar | 2035/05 | 30 | M | L | 67 | N | 1 | 2 | Y | nrl | N | N | Y | AP | nrl | nil | N | 63 | 151 | 31 | 100 | 190 | 3 | nrl | nrl | m | l | l |
| 19 | chakkaravarthy | 604/97 | 58 | M | L | 341 | N | 1 | 1.6 | Y | ov | N | N | N | AP | nrl | nil | N | 70 | 185 | 39 | 98 | 198 | 3 | nrl | ane | m | l | l |
| 20 | chengamuthu | 558/08 | 29 | M | L | 36 | N | 1 | 2.2 | Y | nrl | N | N | N | AP | nrl | nil | N | 65 | 180 | 47 | 80 | 256 | 1 | nrl | nrl | m | l | l |
| 21 | das prakash | 49/09 | 37 | M | C | 19 | N | 1.2 | 2 | N | nrl | Y | N | Y | TMP | nrl | nil | N | 68 | 151 | 50 | 101 | 119 | 5 | pro | nrl | m | m | l |
| 22 | dheenadayalan | 569/09 | 37 | M | L | 23 | N | 1.4 | 2 | Y | nrl | N | N | Y | TMP | nrl | nil | N | 66 | 151 | 58 | 70 | 107 | 3 | pro | nrl | m | m | l |
| 23 | devi | 332/09 | 30 | F | C | 18 | N | 1.2 | 2 | Y | U | N | N | Y | TMP | nrl | nil | N | 72 | 185 | 47 | 114 | 183 | 1 | nrl | ane | m | l | l |
| 24 | devaraj | 902/09 | 48 | F | C | 22 | N | more | 1.4 | N | nrl | Y | Y | Y | TMP | anrl | nil | Y | 65 | 220 | 49 | 124 | 140 | 3 | pro | nrl | l | m | l |
| 25 | devaki | 111/09 | 23 | F | L | 29 | N | 1.5 | more | N | U | N | N | Y | AP | nrl | nil | N | 70 | 174 | 36 | 88 | 120 | 3 | nrl | nrl | m | l | l |
| 26 | devika | 442/10 | 42 | F | L | 17 | N | 0.9 | 0.9 | N | nrl | N | N | Y | MP | nrl | nil | N | 65 | 188 | 38 | 76 | 120 | 5 | pro | nrl | l | l | l |
| 27 | dhanalakshmi | 261/05 | 40 | F | L | 22 | N | 1 | 1 | N | ov | N | N | N | AP | nrl | nil | N | 68 | 180 | 58 | 120 | 119 | 5 | nrl | nrl | m | l | l |
| 28 | dyson | 239/07 | 20 | F | L | 53 | N | 1 | 3 | Y | nrl | N | N | N | TMP | nrl | nil | N | 66 | 151 | 47 | 68 | 107 | 1 | nrl | nrl | m | l | l |
| 29 | elavarasan | 166/09 | 23 | M | C | 20 | N | 1 | 1.7 | N | nrl | Y | Y | Y | TMP | nrl | nil | N | 70 | 185 | 39 | 90 | 100 | 9 | pro | nrl | m | l | l |
| 30 | elumalai.k | 84/02 | 28 | M | L | 114 | N | 1 | 3 | Y | U | N | N | Y | AP | nrl | nil | N | 60 | 156 | 36 | 101 | 87 | 2 | nrl | ane | m | m | l |
| 31 | elumalai.r | 79/07 | 22 | M | L | 248 | N | 0.8 | 1.5 | N | ov | N | N | Y | AP | nrl | nil | N | 66 | 148 | 55 | 70 | 78 | 2 | nrl | nrl | m | l | l |
| 32 | fathima | 928/09 | 26 | F | L | 17 | N | 0.9 | 2.6 | Y | U | N | N | Y | TAP | nrl | nil | N | 72 | 215 | 39 | 114 | 154 | 2 | nrl | ane | l | l | l |
| 33 | gandhi | 196/06 | 36 | M | L | 54 | N | 1 | 1.7 | N | nrl | Y | Y | N | AP | nrl | nil | N | 58 | 178 | 48 | 154 | 168 | 1 | pro | nrl | m | l | l |
| 34 | giri | 949/10 | 23 | M | L | 19 | N | 1.3 | 2.5 | Y | U | Y | Y | Y | TMP | nrl | nil | N | 65 | 180 | 45 | 105 | 107 | 8 | pro | ane | m | m | l |
| 35 | gomathi | 1037/02 | 37 | F | L | 99 | N | 1 | 2 | N | nrl | N | N | N | AP | nrl | nil | N | 68 | 151 | 50 | 76 | 100 | 1 | nrl | nrl | m | l | l |
| 36 | gopal | 1106/09 | 36 | M | L | 24 | N | 1.4 | 3 | Y | ov | Y | Y | Y | CMP | nrl | nil | Y | 66 | 185 | 58 | 99 | 87 | 5 | pro | ane | l | l | l |
| 37 | gopikrishnan | 1185/08 | 42 | M | C | 26 | Y | 1.4 | 1.4 | N | nrl | Y | N | Y | TMP | nrl | nil | N | 70 | 210 | 37 | 126 | 158 | 8 | nrl | nrl | m | l | l |
| 38 | jayakumar | 2746/09 | 31 | M | C | 24 | N | 1.2 | 1.4 | N | U | N | N | N | TMP | nrl | nil | N | 65 | 144 | 49 | 74 | 112 | 1 | nrl | pte | m | m | l |
| 39 | jeyaprakasham | 1235/09 | 32 | M | L | 19 | N | 0.8 | 1.6 | Y | ov | Y | Y | Y | TMP | nrl | nil | N | 68 | 201 | 52 | 93 | 126 | 5 | nrl | nrl | m | l | l |
| 40 | jeevarathinam | 355/05 | 28 | M | L | 75 | N | 1.3 | 2 | Y | nrl | N | Y | Y | AP | nrl | nil | N | 66 | 218 | 46 | 73 | 124 | 1 | nrl | ane | m | l | l |
| 41 | julian | 773/09 | 42 | M | L | 25 | N | 1 | 2.2 | Y | nrl | Y | Y | N | SMP | anrl | nil | Y | 66 | 180 | 40 | 110 | 134 | 10 | pro | pte | l | m | l |
| 42 | kamaraj | 227/09 | 27 | M | L | 31 | N | 1 | 2 | Y | nrl | N | N | Y | TMP | nrl | nil | N | 70 | 151 | 47 | 80 | 102 | 1 | nrl | nrl | m | l | l |
| 43 | karthikeyan | 22/06 | 34 | M | L | 29 | N | 1.4 | 2.6 | Y | U | Y | Y | Y | TMP | nrl | nil | N | 60 | 185 | 40 | 61 | 212 | 5 | pro | ane | m | l | l |
| 44 | kasirajan | 119/06 | 32 | M | L | 67 | N | 1 | 2 | Y | nrl | N | N | Y | AP | nrl | nil | Y | 68 | 176 | 56 | 78 | 123 | 1 | nrl | nrl | m | l | l |
| 45 | kathiravan | 114/06 | 43 | M | L | 67 | N | more | 1.3 | N | ov | Y | N | Y | MP | nrl | nil | N | 65 | 154 | 40 | 110 | 107 | 5 | nrl | pte | l | l | l |
| 46 | kathirvel | 662/07 | 31 | M | L | 20 | Y | 1.7 | 1.7 | Y | nrl | Y | Y | Y | CAP | nrl | nil | N | 62 | 223 | 39 | 145 | 100 | 1 | nrl | pte | m | l | m |
| 47 | kumar.w | 94/10 | 33 | M | L | 17 | N | 0.7 | 1.7 | N | nrl | N | N | Y | TMP | nrl | nil | N | 66 | 180 | 36 | 54 | 87 | 6 | nrl | nrl | l | m | l |
| 48 | kumar.c | 1312/09 | 33 | M | C | 16 | N | 1.2 | 2 | Y | U | N | N | N | TMP | nrl | nil | N | 70 | 205 | 45 | 125 | 198 | 1 | nrl | nrl | l | m | l |
| 49 | kumaresan | 1103/02 | 32 | M | L | 99 | N | 1.5 | 1.5 | Y | nrl | N | N | Y | AP | nrl | nil | N | 60 | 164 | 40 | 101 | 240 | 7 | nrl | ane | l | l | l |
| 50 | kumaravel | 118/06 | 29 | M | L | 65 | N | 1 | 1.5 | N | nrl | N | N | Y | MP | nrl | nil | N | 65 | 180 | 52 | 70 | 130 | 1 | nrl | nrl | m | l | l |
| 51 | lakshmi | 756/07 | 50 | F | L | 42 | Y | 2.7 | 1.5 | N | nrl | N | Y | Y | MP | nrl | nil | N | 63 | 151 | 48 | 114 | 119 | 5 | nrl | ane | m | l | l |
| 52 | logeshwaran | 97/07 | 28 | M | L | 54 | N | 1 | 1.6 | Y | U | N | N | Y | MP | nrl | nil | N | 70 | 185 | 32 | 154 | 107 | 1 | nrl | nrl | m | l | l |
| 53 | lillythresa | 1373/08 | 31 | F | C | 33 | Y | 1.8 | 1.6 | N | ob1 | Y | N | Y | TMP | nrl | nil | N | 65 | 152 | 55 | 77 | 168 | 1 | nrl | nrl | m | l | l |
| 54 | madanagopal | 221/06 | 42 | M | L | 59 | N | 1 | 1.5 | N | nrl | N | N | Y | AP | nrl | nil | Y | 68 | 180 | 50 | 119 | 85 | 3 | nrl | nrl | l | l | l |
| 55 | malathi | 349/04 | 27 | F | L | 84 | N | 1 | 1 | N | nrl | N | N | Y | AP | nrl | nil | N | 66 | 151 | 31 | 107 | 146 | 1 | nrl | nrl | l | l | l |
| 56 | manikandan | 1337/07 | 25 | M | L | 45 | N | 1 | 1 | N | nrl | Y | N | Y | AP | nrl | nil | N | 72 | 185 | 39 | 100 | 183 | 1 | nrl | nrl | m | l | l |
| 57 | mary | 265/04 | 26 | F | L | 89 | N | 1.2 | 1.3 | N | nrl | N | N | Y | AP | nrl | nil | N | 65 | 201 | 47 | 120 | 126 | 1 | nrl | nrl | m | l | l |
| 58 | marimuthu | 617/02 | 35 | M | L | 120 | N | more | 2.2 | Y | nrl | Y | Y | Y | MP | nrl | nil | N | 68 | 218 | 31 | 96 | 124 | 5 | nrl | nrl | m | l | m |
| 59 | manjunathan | 1167/02 | 28 | M | L | 95 | N | 1.2 | 3 | Y | U | N | N | N | AP | nrl | nil | N | 66 | 180 | 68 | 70 | 134 | 1 | pro | ane | m | l | l |
| 60 | menaga | 464/06 | 28 | F | L | 65 | N | 1 | 1.7 | Y | nrl | N | N | Y | AP | nrl | nil | N | 70 | 151 | 55 | 47 | 102 | 1 | nrl | nrl | m | m | l |
| 61 | menaha | 769/09 | 52 | F | L | 27 | Y | 1.6 | 1.6 | N | nrl | Y | Y | Y | CMP | nrl | nil | Y | 65 | 185 | 40 | 79 | 107 | 8 | nrl | ane | m | l | l |
| 62 | mercyannamary | 496/07 | 47 | F | L | 51 | Y | 1.7 | 2.2 | Y | ob1 | Y | Y | Y | MP | nrl | nil | Y | 68 | 170 | 36 | 88 | 100 | 5 | pro | ane | m | m | l |
| 63 | mohaan | 1160/07 | 35 | M | L | 47 | N | 1 | 2 | Y | nrl | N | N | Y | MP | nrl | nil | N | 66 | 126 | 56 | 80 | 87 | 1 | nrl | nrl | l | l | l |
| 64 | mohan | 1308/08 | 40 | M | L | 35 | N | more | 1.6 | N | ov | Y | N | Y | TAP | nrl | nil | N | 58 | 174 | 32 | 54 | 200 | 8 | nrl | nrl | l | l | l |
| 65 | murali | 1368/02 | 27 | M | L | 106 | N | more | 1.8 | Y | nrl | Y | Y | Y | AP | nrl | nil | Y | 62 | 188 | 48 | 101 | 134 | 1 | nrl | nrl | m | m | l |
| 66 | murugesan | 1106/06 | 27 | M | L | 58 | N | 1.3 | 3 | Y | nrl | N | N | Y | MP | nrl | nil | N | 66 | 170 | 46 | 70 | 110 | 1 | nrl | pte | m | l | m |
| 67 | nagaraj | 316/07 | 36 | M | L | 46 | N | more | 1.6 | N | nrl | Y | N | Y | MP | nrl | nil | N | 63 | 126 | 58 | 116 | 126 | 3 | nrl | nrl | m | l | l |
| 68 | nainarmohamed | 2000/05 | 31 | M | L | 66 | N | 1 | 1.7 | Y | nrl | N | N | Y | MP | nrl | nil | Y | 70 | 170 | 47 | 100 | 124 | 1 | pro | nrl | l | l | l |
| 69 | nazirali | 1242/09 | 27 | M | C | 15 | Y | 2 | 1.6 | N | ov | Y | N | Y | TMP | nrl | nil | N | 65 | 126 | 49 | 70 | 134 | 1 | nrl | nrl | m | l | l |
| 70 | navaneetham | 296/97 | 37 | F | L | 164 | N | 1 | 1.4 | N | nrl | N | N | N | AP | nrl | nil | N | 68 | 172 | 31 | 119 | 102 | 3 | nrl | nrl | m | l | l |
| 71 | nirmala | 1221/07 | 36 | F | C | 18 | Y | 1.3 | 2 | Y | nrl | N | N | N | TMP | nrl | nil | N | 66 | 168 | 31 | 107 | 230 | 1 | pro | ane | m | m | l |
| 72 | palani | 467/09 | 30 | M | L | 30 | N | 1.5 | 2.5 | Y | nrl | N | N | Y | MP | nrl | nil | N | 72 | 174 | 39 | 100 | 140 | 1 | nrl | nrl | l | l | l |
| 73 | poonguzali | 691/02 | 29 | F | L | 130 | N | 0.8 | 2.8 | Y | nrl | N | N | N | AP | nrl | nil | N | 66 | 188 | 47 | 120 | 158 | 1 | pro | nrl | m | l | l |
| 74 | prasanth | 17/10 | 30 | M | L | 20 | N | 1.4 | 1.6 | N | nrl | Y | N | Y | TMP | nrl | nil | N | 70 | 220 | 55 | 134 | 173 | 1 | nrl | nrl | m | l | m |
| 75 | prema | 1433/08 | 36 | F | C | 22 | N | more | 1.2 | N | ob1 | Y | Y | Y | TMP | nrl | nil | Y | 65 | 190 | 68 | 132 | 124 | 8 | pro | ane | l | l | m |
| 76 | priya | 758/00 | 33 | F | L | 130 | N | 0.6 | 1.5 | Y | ov | N | N | N | AP | nrl | nil | N | 68 | 143 | 44 | 90 | 132 | 1 | nrl | nrl | m | l | l |
| 77 | raja | 1174/06 | 28 | M | L | 52 | N | 1 | 7.5 | Y | U | N | N | Y | AP | nrl | nil | N | 66 | 140 | 48 | 112 | 160 | 1 | nrl | nrl | m | m | l |
| 78 | rajkamal | 525/08 | 24 | M | L | 40 | Y | 1.7 | 2.5 | Y | nrl | N | N | Y | AP | nrl | nil | N | 68 | 151 | 32 | 101 | 100 | 6 | pro | ane | m | m | l |
| 79 | rajmohan | 501/06 | 39 | M | L | 63 | Y | 1.8 | 10.5 | Y | nrl | N | N | Y | MP | nrl | nil | Y | 65 | 185 | 47 | 70 | 102 | 3 | nrl | nrl | l | m | l |
| 80 | raman | 566/05 | 30 | M | L | 72 | N | 1 | 3 | Y | nrl | N | N | Y | MP | nrl | nil | N | 58 | 180 | 49 | 114 | 80 | 1 | pro | ane | m | l | l |
| 81 | ramachandran | 1130/07 | 41 | M | L | 46 | Y | 2.8 | 3 | Y | nrl | N | N | N | AP | nrl | nil | N | 62 | 151 | 36 | 154 | 109 | 3 | nrl | nrl | l | l | l |
| 82 | ramamoorthy | 382/04 | 60 | M | L | 88 | Y | 1.8 | 3 | Y | nrl | Y | Y | Y | AP | anrl | nil | N | 66 | 180 | 46 | 130 | 71 | 10 | pro | nrl | m | l | l |
| 83 | ramaamoorthy | 420/08 | 42 | M | L | 40 | Y | 1.6 | 1.6 | N | ov | Y | N | Y | MP | nrl | nil | N | 63 | 151 | 58 | 79 | 90 | 3 | nrl | nrl | m | l | l |
| 84 | ramesh | 776/04 | 33 | M | L | 84 | Y | 3.2 | 2 | Y | U | N | N | Y | MP | nrl | nil | Y | 70 | 185 | 47 | 88 | 190 | 1 | nrl | nrl | m | l | l |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 85 | ramesh | 1308/07 | 23 | M | L | 44 | Y | 1 | 2.2 | Y | nrl | N | N | Y | MP | nrl | nil | N | 62 | 148 | 49 | 80 | 198 | 1 | nrl | nrl | l | l | l |
| 86 | ramesh | 999/09 | 26 | M | L | 22 | Y | 1.6 | 2.2 | Y | nrl | N | N | Y | TMP | nrl | nil | Y | 65 | 166 | 31 | 54 | 200 | 1 | nrl | nrl | m | l | l |
| 87 | ramesh | 1075/10 | 33 | M | L | 13 | N | 1.5 | 1.7 | N | nrl | N | N | Y | TMP | nrl | nil | N | 68 | 181 | 31 | 101 | 108 | 1 | nrl | nrl | l | m | m |
| 88 | ravi | 30/07 | 20 | M | L | 48 | Y | 1.9 | 1.5 | N | nrl | N | N | N | AP | nrl | nil | N | 70 | 140 | 39 | 70 | 148 | 1 | nrl | nrl | m | l | l |
| 89 | ravi | 573/07 | 37 | M | L | 50 | N | 1 | 1.8 | Y | nrl | N | N | Y | MP | nrl | nil | N | 65 | 151 | 47 | 101 | 117 | 3 | nrl | nrl | m | l | l |
| 90 | ravichandran | 173/04 | 37 | M | L | 90 | N | 1.4 | 3.5 | Y | nrl | N | N | Y | MP | nrl | nil | N | 68 | 185 | 55 | 98 | 96 | 8 | nrl | nrl | m | l | l |
| 91 | renuga | 001/07 | 38 | F | C | 29 | N | 1.2 | 1 | N | nrl | N | N | Y | TMP | nrl | nil | N | 66 | 180 | 68 | 81 | 81 | 3 | nrl | nrl | m | l | l |
| 92 | revathy | 756/09 | 26 | F | C | 22 | N | 1 | 1 | N | nrl | Y | N | Y | TMP | nrl | nil | N | 63 | 151 | 44 | 89 | 67 | 1 | nrl | nrl | m | m | l |
| 93 | riyaz ali | 806/09 | 26 | M | C | 20 | N | 1.3 | 1.4 | N | nrl | Y | Y | Y | TMP | nrl | nil | N | 70 | 158 | 48 | 117 | 138 | 1 | nrl | pte | m | l | l |
| 94 | sabeetha | 1174/07 | 30 | F | L | 39 | N | 1 | 1.6 | N | nrl | N | N | Y | MP | nrl | nil | N | 65 | 201 | 32 | 98 | 156 | 1 | nrl | nrl | l | l | l |
| 95 | sadhasivam | 812/07 | 34 | M | L | 50 | N | 1.3 | 1.8 | Y | nrl | N | N | Y | CMP | anrl | nil | N | 68 | 218 | 31 | 98 | 111 | 7 | nrl | nrl | l | l | l |
| 96 | sakthivel | 572/05 | 31 | M | L | 71 | N | 1.4 | 1.6 | N | nrl | N | N | Y | AP | nrl | nil | N | 66 | 180 | 68 | 81 | 198 | 1 | nrl | nrl | m | m | l |
| 97 | sakthivel | 859/04 | 40 | M | L | 84 | N | more | 1.2 | N | ov | Y | Y | Y | AP | anrl | nil | N | 72 | 151 | 34 | 67 | 230 | 3 | pro | nrl | l | l | l |
| 98 | samregis | 1105/06 | 26 | M | L | 59 | N | 1 | 2 | Y | nrl | N | N | Y | SMP | nrl | nil | N | 70 | 185 | 46 | 101 | 80 | 1 | nrl | nrl | m | l | l |
| 99 | saranya | 448/05 | 21 | F | L | 74 | N | 1 | 2.2 | Y | nrl | N | N | Y | TMP | nrl | nil | N | 65 | 180 | 47 | 70 | 112 | 1 | nrl | nrl | m | l | l |
| 100 | sarasu | 361/07 | 36 | F | L | 53 | N | 1 | 2 | Y | nrl | Y | N | Y | MP | nrl | nil | N | 68 | 151 | 49 | 114 | 232 | 3 | nrl | nrl | m | l | l |
| 101 | sathishkumar | 1273/06 | 25 | M | L | 57 | N | 1 | 3 | Y | ob1 | Y | N | Y | TMP | nrl | nil | Y | 66 | 185 | 31 | 154 | 146 | 1 | pro | nrl | l | m | m |
| 102 | santhanakumar | 717/07 | 27 | M | L | 47 | N | 1.5 | 3 | Y | nrl | N | N | Y | MP | nrl | nil | N | 68 | 160 | 31 | 137 | 248 | 1 | nrl | nrl | m | l | l |
| 103 | sathya | 255/10 | 30 | F | L | 42 | N | 1 | 1.3 | N | nrl | N | Y | Y | AP | nrl | nil | N | 66 | 180 | 39 | 79 | 120 | 1 | nrl | nrl | m | l | l |
| 104 | selvaraj | 846/07 | 62 | M | L | 238 | N | 1 | 2 | Y | nrl | N | Y | N | CP | nrl | nil | N | 63 | 151 | 47 | 88 | 119 | 10 | nrl | ane | m | l | m |
| 105 | senthilraja | 666/02 | 31 | M | L | 105 | N | more | 1.7 | Y | ov | Y | Y | Y | AP | nrl | nil | N | 63 | 185 | 48 | 80 | 107 | 1 | nrl | nrl | m | l | l |
| 106 | seshadari | 425/04 | 46 | M | L | 89 | Y | 1.8 | 2.5 | Y | nrl | N | N | Y | MP | nrl | nil | Y | 70 | 142 | 56 | 54 | 100 | 5 | nrl | nrl | m | m | l |
| 107 | shankar | 5049/89 | 53 | M | L | 269 | N | 1.4 | 2.8 | Y | nrl | N | N | Y | MP | nrl | nil | N | 65 | 140 | 36 | 101 | 87 | 5 | nrl | nrl | l | m | l |
| 108 | shankar | 388/09 | 33 | M | L | 18 | N | 1 | 1.4 | N | nrl | N | Y | Y | TMP | anrl | nil | N | 68 | 151 | 38 | 70 | 140 | 1 | nrl | nrl | m | l | m |
| 109 | shanmugam | 940/07 | 30 | M | L | 47 | N | 1 | 1.2 | N | U | Y | Pre dm | Y | AP | anrl | nil | Y | 66 | 185 | 42 | 90 | 120 | 1 | nrl | nrl | m | l | l |
| 110 | sharbudeen | 725/07 | 29 | M | L | 49 | N | 1 | 1.7 | Y | nrl | Y | N | Y | AP | nrl | nil | Y | 72 | 180 | 46 | 73 | 120 | 1 | nrl | nrl | m | l | l |
| 111 | simsone | 905/06 | 30 | M | L | 65 | N | 1 | 2 | Y | nrl | Y | N | Y | AP | nrl | nil | N | 66 | 151 | 58 | 100 | 119 | 1 | nrl | nrl | l | l | l |
| 112 | srinivasan | 784/07 | 28 | M | L | 48 | Y | 1.5 | 2 | Y | U | N | N | Y | AP | nrl | nil | N | 66 | 180 | 47 | 110 | 107 | 1 | nrl | nrl | m | l | l |
| 113 | subramanian | 1313/04 | 47 | M | L | 78 | N | 1 | 2.2 | Y | nrl | N | N | Y | AP | nrl | nil | N | 65 | 162 | 49 | 95 | 100 | 5 | nrl | ane | l | l | l |
| 114 | subramanian | 341/09 | 49 | M | C | 24 | N | 1 | 1.2 | N | U | Y | Y | Y | TMP | nrl | nil | N | 58 | 201 | 48 | 124 | 87 | 9 | nrl | nrl | m | l | l |
| 115 | syedmusthafa | 68/06 | 28 | M | L | 64 | Y | 1.5 | 2 | Y | nrl | N | N | Y | AP | nrl | nil | N | 65 | 218 | 32 | 119 | 90 | 1 | nrl | nrl | m | l | l |
| 116 | tamilarasan | 1334/06 | 25 | M | L | 57 | N | 1 | 1.5 | Y | nrl | N | N | Y | AP | nrl | nil | N | 70 | 180 | 55 | 107 | 120 | 1 | nrl | nrl | m | l | l |
| 117 | thagira | 643/03 | 30 | F | L | 95 | N | 1 | 1 | N | ov | N | N | N | AP | nrl | nil | N | 65 | 151 | 46 | 100 | 119 | 1 | nrl | nrl | m | l | l |
| 118 | thangaraj | 1195/08 | 34 | M | L | 35 | N | 1.3 | 2 | Y | nrl | N | N | Y | MP | nrl | nil | N | 68 | 185 | 39 | 120 | 107 | 3 | nrl | nrl | m | l | l |
| 119 | thilagam | 1314/09 | 31 | F | L | 21 | N | 1.3 | 1.5 | N | ob1 | Y | N | Y | CAP | nrl | nil | Y | 66 | 146 | 55 | 110 | 100 | 1 | pro | ane | m | l | l |
| 120 | thirunavukarasu | 215/02 | 42 | M | L | 96 | Y | 1 | 1.8 | Y | nrl | N | N | Y | MP | nrl | nil | N | 63 | 176 | 46 | 100 | 87 | 3 | nrl | nrl | m | l | m |
| 121 | thulasi | 67/09 | 21 | F | L | 30 | N | 1 | 1.2 | N | nrl | N | N | Y | TAP | nrl | nil | N | 70 | 153 | 48 | 87 | 117 | 1 | nrl | nrl | m | l | l |
| 122 | umamaheswaran | 840/03 | 34 | M | L | 128 | N | 0.8 | 1 | N | nrl | Y | Y | N | AP | nrl | nil | N | 65 | 126 | 32 | 117 | 98 | 5 | nrl | nrl | m | l | l |
| 123 | usha | 351/05 | 19 | F | L | 76 | N | 1 | 1 | N | nrl | N | N | N | AP | nrl | nil | N | 68 | 201 | 55 | 75 | 81 | 1 | nrl | nrl | l | l | l |
| 124 | varadhan | 420/00 | 39 | M | L | 124 | N | 1 | 2 | N | nrl | N | N | Y | AP | nrl | nil | Y | 66 | 218 | 45 | 100 | 67 | 5 | nrl | nrl | m | l | l |
| 125 | venkatash | 169/09 | 31 | M | L | 31 | N | 0.9 | 1.5 | Y | U | Y | Pre dm | N | TMP | nrl | nil | N | 72 | 180 | 31 | 98 | 138 | 8 | nrl | pte | l | l | l |
| 126 | vijaya | 896/04 | 47 | F | L | 86 | N | 1 | 1.5 | N | ov | N | N | Y | AP | nrl | nil | N | 68 | 151 | 39 | 81 | 100 | 3 | nrl | ane | m | l | l |
| 127 | vijayakumar | 587/02 | 36 | M | L | 67 | N | 1 | 2.3 | Y | nrl | Y | Y | Y | SMP | nrl | nil | N | 66 | 185 | 47 | 67 | 111 | 8 | pro | ane | m | m | l |
| 128 | vijayalakshmi | 370/06 | 42 | F | L | 64 | N | 1 | 1 | N | ov | Y | N | N | MP | nrl | nil | N | 65 | 180 | 31 | 79 | 198 | 3 | nrl | nrl | m | l | l |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 129 | vijayapandian | 1071/08 | 31 | M | L | 35 | N | 1 | 2.7 | Y | nrl | Y | N | Y | CMP | nrl | nil | Y | 68 | 151 | 68 | 88 | 80 | 1 | pro | ane | l | l | l |
| 130 | vishwanathan | 421/04 | 39 | M | L | 86 | Y | 1.8 | 2.7 | Y | nrl | N | N | N | AP | nrl | nil | N | 70 | 185 | 46 | 80 | 88 | 3 | nrl | nrl | m | l | l |
| 131 | vinayagam | 943/01 | 37 | M | L | 104 | N | 1 | 1.3 | N | nrl | N | N | N | AP | nrl | nil | N | 65 | 184 | 58 | 54 | 124 | 3 | nrl | nrl | m | l | l |
| 132 | vinothkumar | 1247/06 | 21 | M | L | 16 | Y | 1.7 | 1.5 | N | nrl | Y | N | Y | TAP | nrl | nil | Y | 68 | 146 | 47 | 101 | 232 | 1 | nrl | nrl | m | l | l |
| 133 | vivekanandan | 572/09 | 21 | M | L | 24 | N | 1 | 1.2 | N | U | N | N | Y | CAP | nrl | nil | N | 66 | 189 | 49 | 70 | 146 | 1 | nrl | nrl | l | l | l |
| 134 | rajasekaran | 191/11 | 30 | M | L | 4 | N | 0.9 | 1 | N | nrl | N | N | Y | TMP | nrl | nil | N | 68 | 132 | 31 | 40 | 85 | 1 | nrl | nrl | m | m | l |
| 135 | hari | 803/11 | 30 | M | L | 1 | N | 1 | 1 | N | nrl | N | N | N | TMP | nrl | nil | N | 66 | 154 | 31 | 111 | 176 | 1 | nrl | nrl | l | m | l |
| 136 | pandian | 490/11 | 26 | M | L | 1 | N | 1 | 1 | N | nrl | N | N | Y | TAP | nrl | nil | Y | 65 | 215 | 39 | 104 | 190 | 1 | nrl | nrl | l | l | l |
| 137 | perumal | 460/10 | 25 | M | C | 6 | N | 0.8 | 1.2 | N | U | N | N | Y | TMP | nrl | nil | N | 68 | 188 | 47 | 100 | 198 | 1 | nrl | nrl | l | l | l |
| 138 | syedmoosa | 775/11 | 25 | M | L | 1 | N | 1 | 1 | N | nrl | N | N | Y | TMP | nrl | nil | N | 66 | 170 | 55 | 86 | 102 | 1 | nrl | nrl | m | l | l |
| 139 | seethalakshmi | 1246/10 | 36 | F | L | 9 | N | 0.8 | 1 | N | ov | N | N | N | TMP | nrl | nil | N | 68 | 154 | 68 | 90 | 366 | 3 | nrl | pte | l | l | l |
| 140 | kalpana | 887/10 | 25 | F | L | 8 | Y | 1.7 | 2 | Y | nrl | N | N | Y | TMP | nrl | nil | Y | 65 | 140 | 44 | 101 | 198 | 1 | nrl | pte | m | m | l |
| 141 | jayaprakash | 855/10 | 26 | M | L | 13 | N | 1 | 1 | Y | U | N | N | Y | TAP | anrl | nil | N | 63 | 140 | 48 | 70 | 190 | 1 | nrl | nrl | m | m | l |
| 142 | megala | 787/11 | 32 | F | L | 2 | N | 1 | 1.2 | N | nrl | N | N | Y | TAP | nrl | nil | N | 70 | 151 | 32 | 114 | 183 | 1 | nrl | pte | m | l | l |
| 143 | suresh | 816/10 | 35 | M | L | 11 | N | 1 | 1 | N | ov | N | N | Y | TMP | nrl | nil | Y | 65 | 185 | 38 | 154 | 140 | 3 | nrl | nrl | m | m | l |
| 144 | sulaiman | 630/10 | 29 | M | L | 11 | N | 0.9 | 1 | N | ov | N | N | Y | TMP | nrl | nil | Y | 68 | 180 | 46 | 79 | 190 | 1 | nrl | pte | m | l | l |
| 145 | ramamoorthy | 610/06 | 35 | M | L | 61 | N | 1 | 1.4 | N | nrl | N | N | Y | AP | nrl | nil | N | 66 | 151 | 47 | 88 | 198 | 3 | nrl | nrl | m | l | l |
| 146 | babu | 934/10 | 37 | M | L | 8 | N | 1 | 1.2 | N | nrl | N | Y | Y | TMP | anrl | nil | N | 72 | 180 | 49 | 80 | 119 | 5 | nrl | pte | m | l | l |
| 147 | narasingam | 1367/09 | 22 | M | C | 12 | Y | 1.2 | 1.4 | N | nrl | N | N | Y | TMP | anrl | nil | Y | 55 | 151 | 31 | 54 | 107 | 1 | nrl | nrl | m | m | l |
| 148 | mohamedrafi | 717/10 | 22 | M | L | 10 | N | 0.9 | 1.2 | N | nrl | N | N | Y | TMP | nrl | nil | N | 68 | 185 | 47 | 101 | 100 | 1 | nrl | pte | l | m | l |
| 149 | murugan | 1241/10 | 39 | M | C | 3 | N | 1 | 1.4 | N | nrl | N | N | Y | TMP | nrl | nil | Y | 70 | 178 | 49 | 70 | 87 | 5 | nrl | nrl | m | m | l |
| 150 | poonkodi | 304/10 | 45 | F | C | 8 | N | 0.9 | 1 | N | ov | Y | Y | Y | TMP | nrl | nil | Y | 65 | 188 | 31 | 100 | 120 | 8 | pro | nrl | m | l | l |
| 151 | vinothkumar | 1573/10 | 20 | M | L | 3 | N | 1 | 1.7 | Y | U | N | N | N | TAP | nrl | nil | N | 68 | 202 | 31 | 120 | 119 | 1 | nrl | nrl | m | m | l |
| 152 | sajeevan | 702/10 | 32 | M | C | 7 | N | 1 | 2 | Y | U | N | Y | N | TMP | anrl | nil | N | 66 | 211 | 39 | 79 | 107 | 5 | pro | ane | l | l | l |
| 153 | antonyjaganraj | 1241/10 | 29 | M | L | 11 | Y | 1 | 1 | N | ov | Y | N | Y | TMP | nrl | nil | Y | 65 | 156 | 47 | 88 | 100 | 1 | nrl | nrl | m | l | l |
| 154 | dhanalakshmi | 1054/10 | 35 | F | L | 10 | N | 0.9 | 1 | N | ov | Y | Y | N | TAP | nrl | nil | N | 68 | 200 | 34 | 80 | 120 | 3 | nrl | nrl | m | l | l |
| 155 | rameshkumar | 816/04 | 25 | M | L | 83 | N | 1 | 2 | Y | nrl | N | N | Y | AP | nrl | nil | Y | 66 | 166 | 47 | 54 | 119 | 2 | nrl | nrl | m | l | l |
| 156 | rajeshwari | 545/10 | 32 | F | L | 10 | N | 1 | 1 | Y | nrl | N | N | Y | TAP | nrl | nil | N | 68 | 178 | 49 | 101 | 107 | 2 | nrl | nrl | m | m | l |
| 157 | saraswathy | 1322/10 | 29 | F | L | 5 | N | 1 | 1.2 | N | ov | N | N | Y | TMP | nrl | nil | N | 60 | 215 | 38 | 70 | 100 | 2 | nrl | nrl | m | l | l |
| 158 | alli | 752/10 | 45 | F | C | 4 | N | 1 | 1 | N | ov | Y | N | Y | TMP | nrl | nil | N | 62 | 188 | 46 | 120 | 120 | 3 | nrl | nrl | m | l | l |
| 159 | arumugam | 1105/09 | 32 | M | C | 12 | N | 1 | 2 | Y | U | N | N | N | TMP | anrl | nil | Y | 58 | 170 | 58 | 108 | 120 | 1 | pro | ane | l | m | l |
| 160 | sathishkannan | 1186/10 | 35 | M | L | 7 | N | 0.8 | 1.7 | Y | nrl | N | N | Y | TMP | anrl | nil | N | 66 | 200 | 47 | 110 | 140 | 1 | nrl | nrl | m | m | l |
| 161 | suresh | 126/11 | 35 | M | L | 2 | N | 1 | 1.7 | Y | ov | N | N | Y | TMP | nrl | nil | N | 64 | 140 | 49 | 79 | 183 | 4 | nrl | pte | m | l | m |
| 162 | kavitha | 571/11 | 28 | F | L | 4 | Y | 1 | 1 | N | ov | N | N | Y | TMP | nrl | nil | Y | 68 | 151 | 31 | 88 | 190 | 1 | nrl | nrl | l | l | l |
| 163 | periyasamy | 1353/08 | 28 | M | C | 12 | N | 1 | 2 | Y | U | N | N | Y | TMP | nrl | nil | Y | 66 | 185 | 31 | 80 | 198 | 1 | pro | nrl | l | l | l |
| 164 | venkatesan | 334/03 | 30 | M | L | 96 | N | 1 | 2.5 | Y | nrl | N | N | Y | AP | nrl | nil | Y | 70 | 180 | 39 | 54 | 268 | 1 | nrl | nrl | m | l | l |
| 165 | kesavajothi | 101/03 | 35 | M | L | 96 | N | 1 | 1.4 | N | nrl | N | N | Y | AP | nrl | nil | N | 65 | 151 | 47 | 101 | 120 | 3 | nrl | nrl | m | l | l |
| 166 | ranjithkumar | 37/05 | 33 | M | L | 72 | N | 1 | 1.8 | Y | nrl | N | N | Y | AP | nrl | nil | N | 68 | 185 | 55 | 70 | 119 | 3 | nrl | nrl | m | l | l |
| 167 | geetha | 287/11 | 23 | F | L | 3 | N | 0.8 | 1 | N | nrl | N | N | Y | TMP | nrl | nil | N | 66 | 162 | 68 | 114 | 107 | 4 | nrl | nrl | m | l | l |
| 168 | stephen | 1367/04 | 48 | M | L | 93 | N | 1 | 1.5 | N | U | N | N | Y | AP | nrl | nil | N | 68 | 201 | 44 | 154 | 100 | 1 | nrl | nrl | m | l | l |
| 169 | abdulmunaf | 9918/96 | 53 | M | C | 188 | N | 0.9 | 2 | Y | nrl | Y | N | Y | AP | anrl | nil | N | 50 | 218 | 48 | 130 | 190 | 10 | nrl | nrl | m | l | l |
| 170 | suja | 818/09 | 26 | F | C | 10 | N | 1 | 1.7 | Y | U | N | N | Y | TMP | nrl | nil | N | 70 | 180 | 32 | 89 | 198 | 1 | nrl | pte | m | l | l |
| | | | years | M=male<br>F=female | L=live<br>C=cadaver | months | N=no,Y=yes | mg/dl | mg/dl | N=no,Y=yes | under weight<br>nrl=normal<br>ov=over weight<br>ob1=obesity1 | N=no,Y=yes | | | A=azathioprine<br>P=prednisolone<br>M=MMF<br>T=tacrolimus<br>C=cyclosporine<br>S=sirolimus | nrl=normal<br>anrl=abnormal | nil=no | N=no,Y=yes | % | mg/dl | mg/dl | mg/dl | mg/dl | % | nrl=normal<br>pro=proteinuria | nrl=normal<br>ane=anemia<br>pte=post transp<br>lanteryrhtrocytosis | m=>3.5gm<br>l=<3.5gm | m=>6mg<br>l=<6mg | m=≥1.1mm<br>l=<1.1mm |

# INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Cardivascular Riskfactors analysis in renal Transplant Recipients

Principal Investigator : Dr.S.A.K. Noor Mohamed

Designation : PG in D.M.(Nephro)

Department : Department of Nephrology
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 12.10.2011 at the Modernized Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

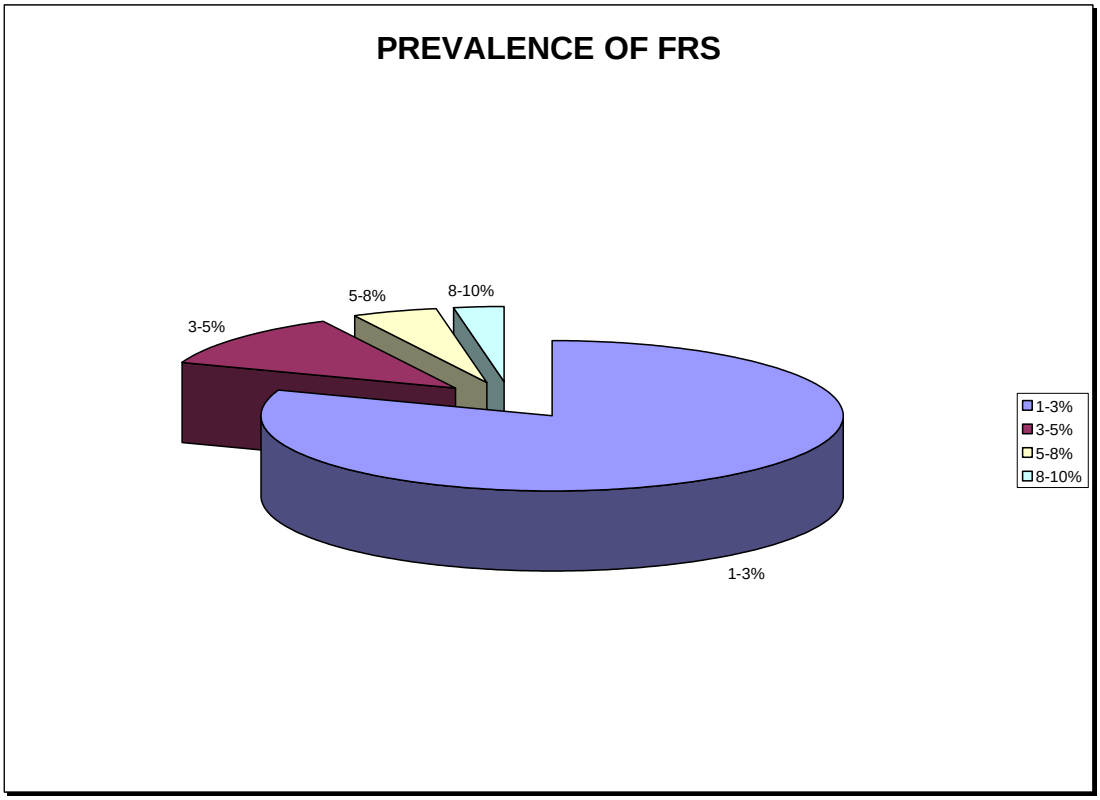
The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

12/10/11

MEMBER SECRETARY,
IEC, SMC, CHENNAI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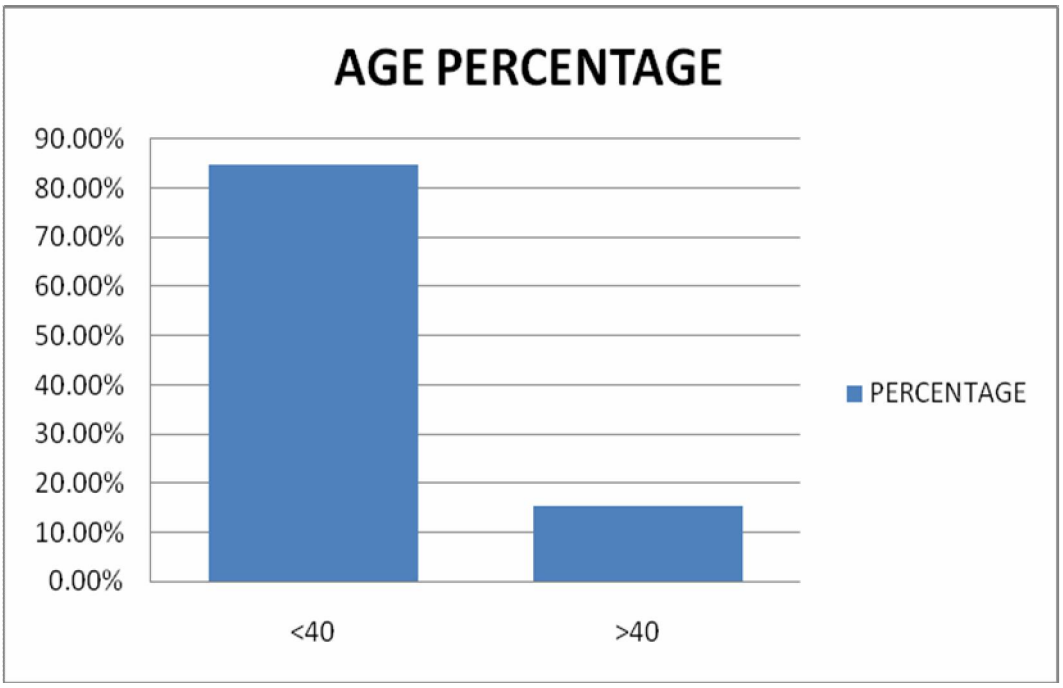
8-10%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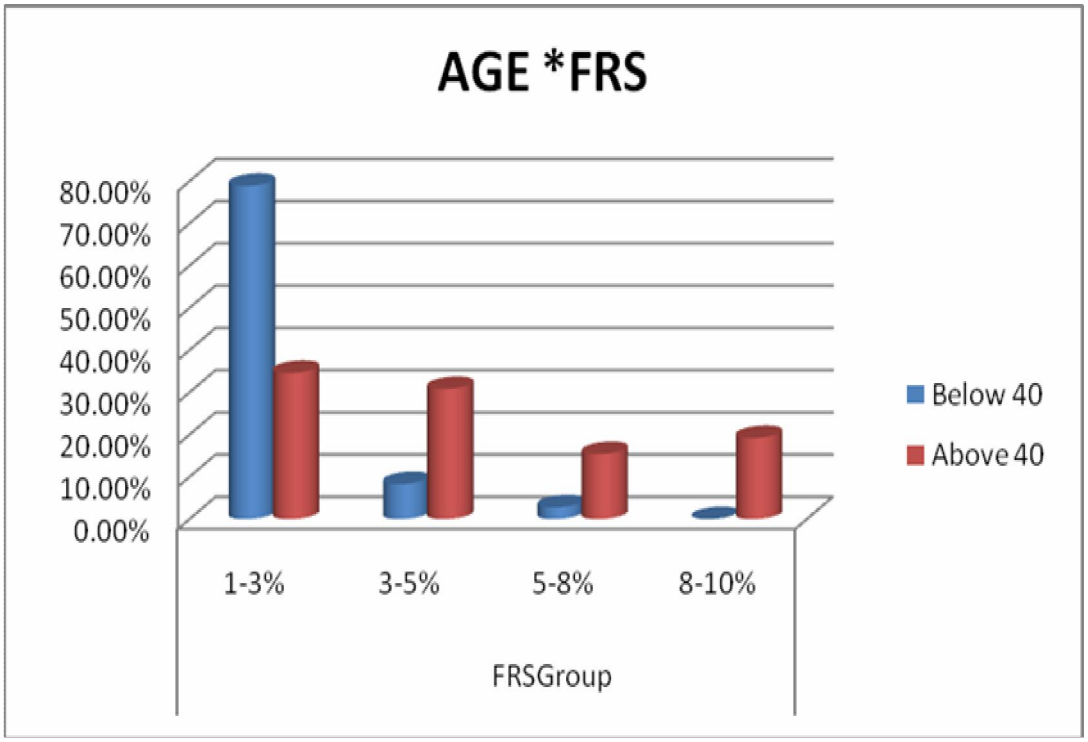
PERCENTAGE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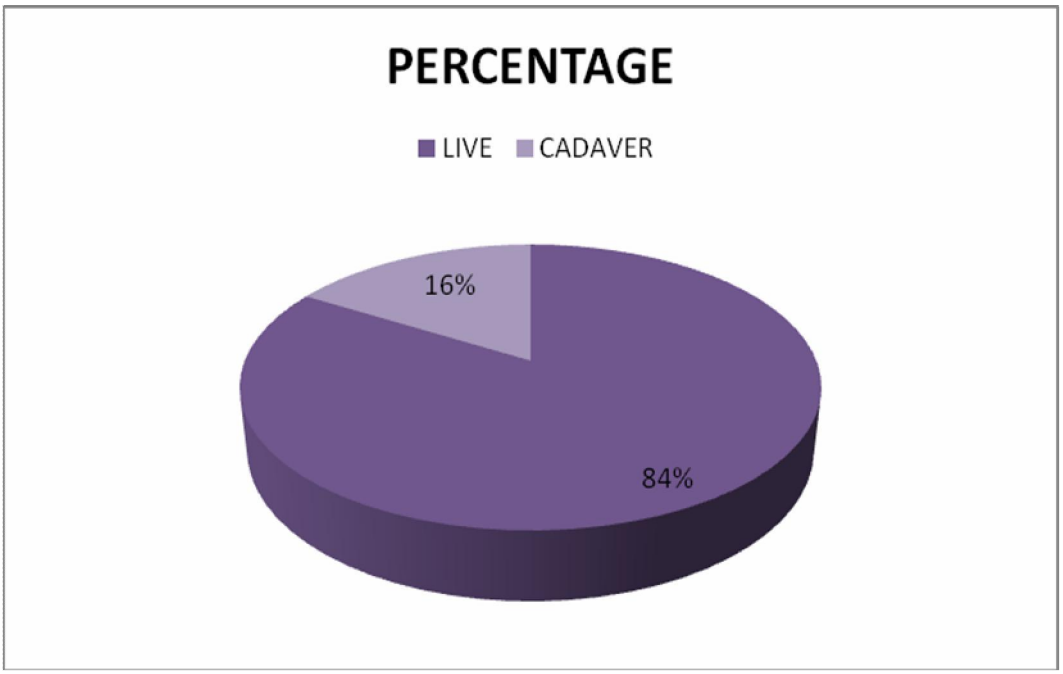
FRSGroup
Below 40
Above 40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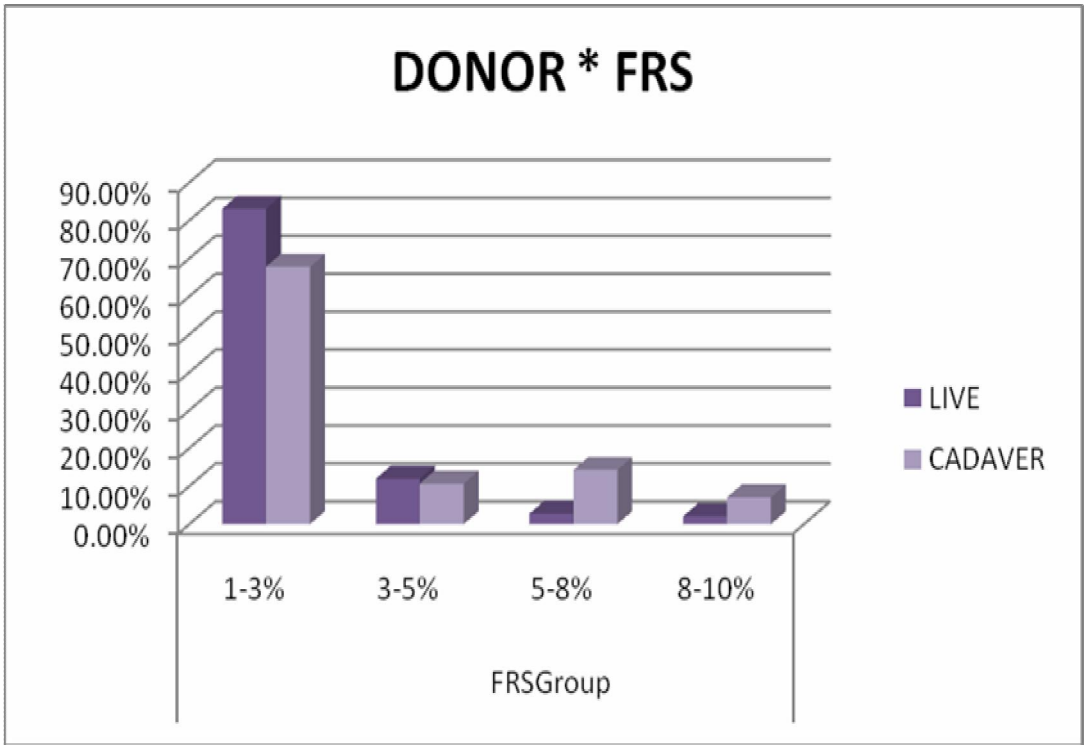
DONOR * FRS
90.00%
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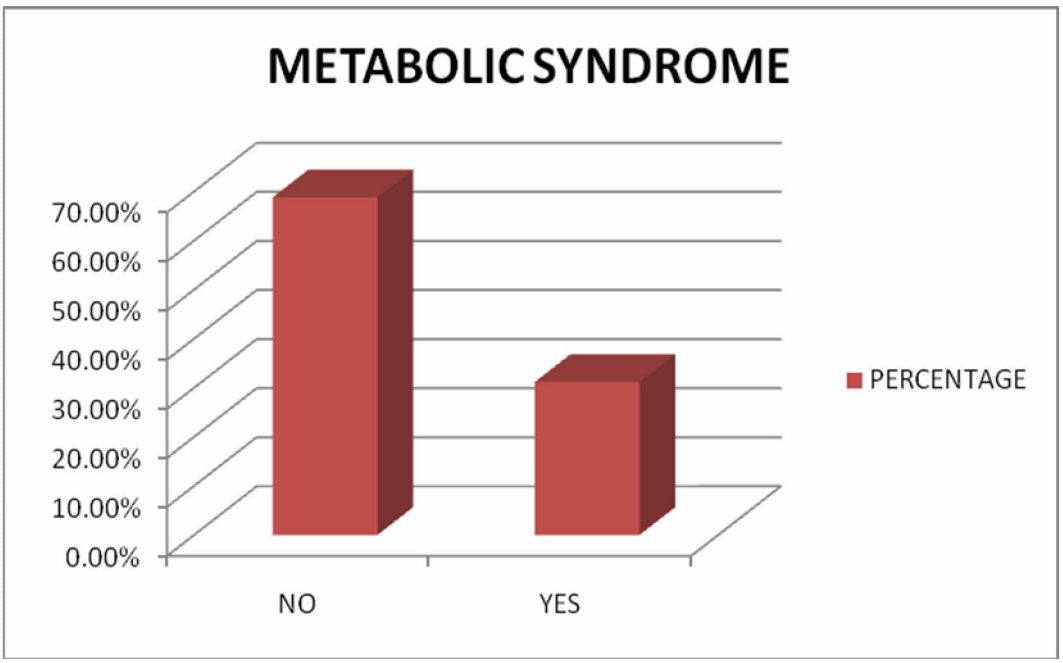
FRSGroup
LIVE
CADAVER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10.00%
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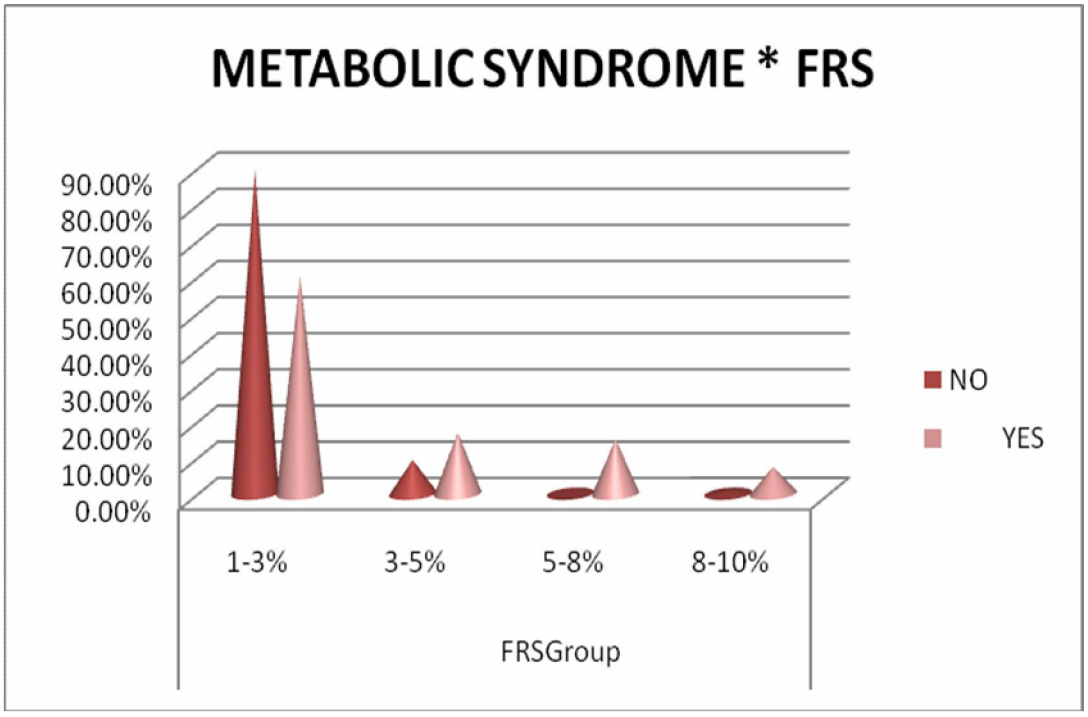
NO
YES
PERCENTAGE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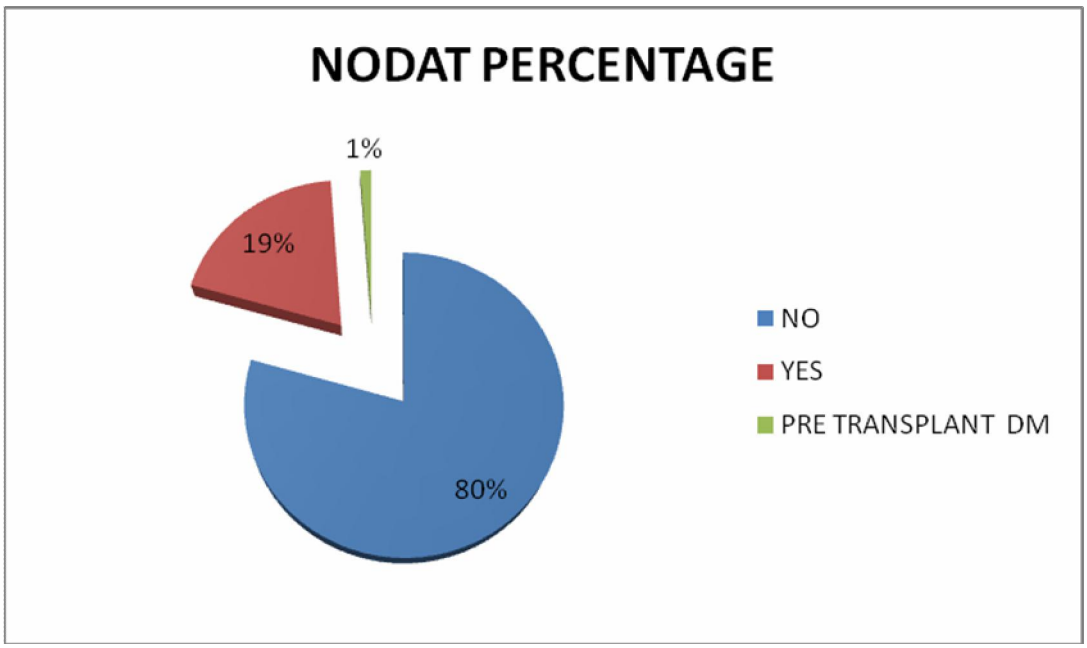
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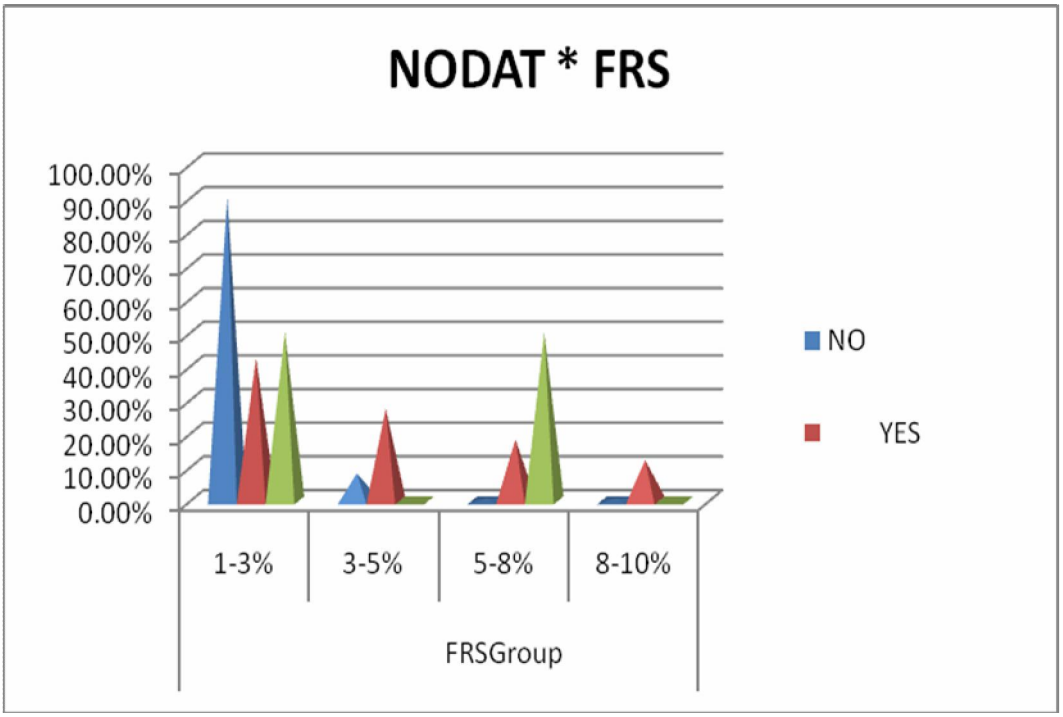
NO
YES
PRE TRANSPLANT DM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1-3%
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5-8%
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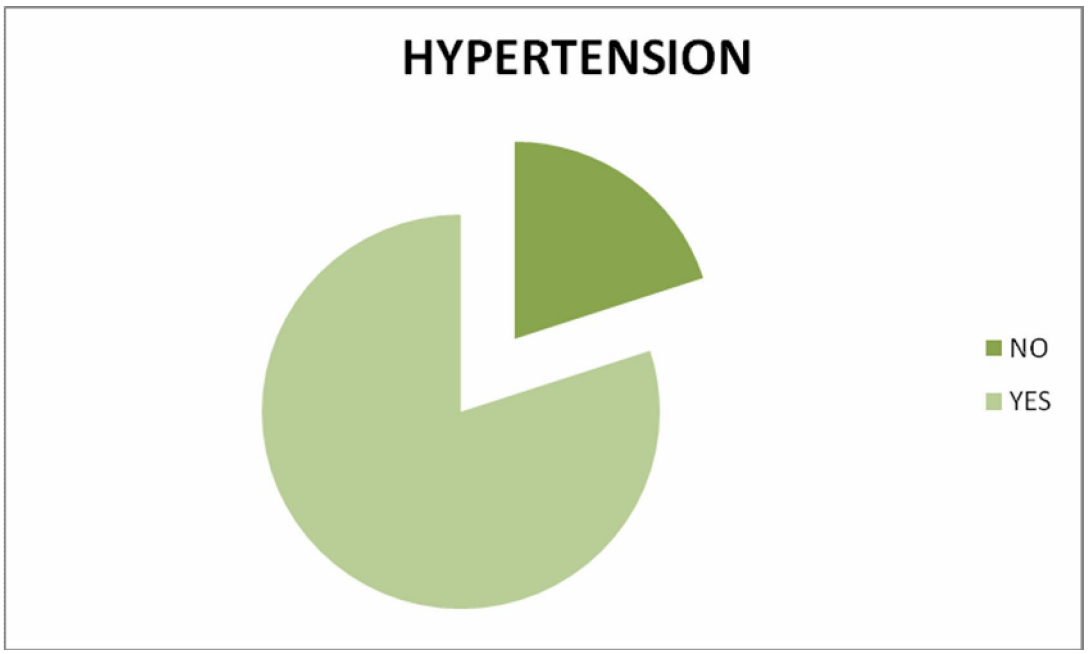
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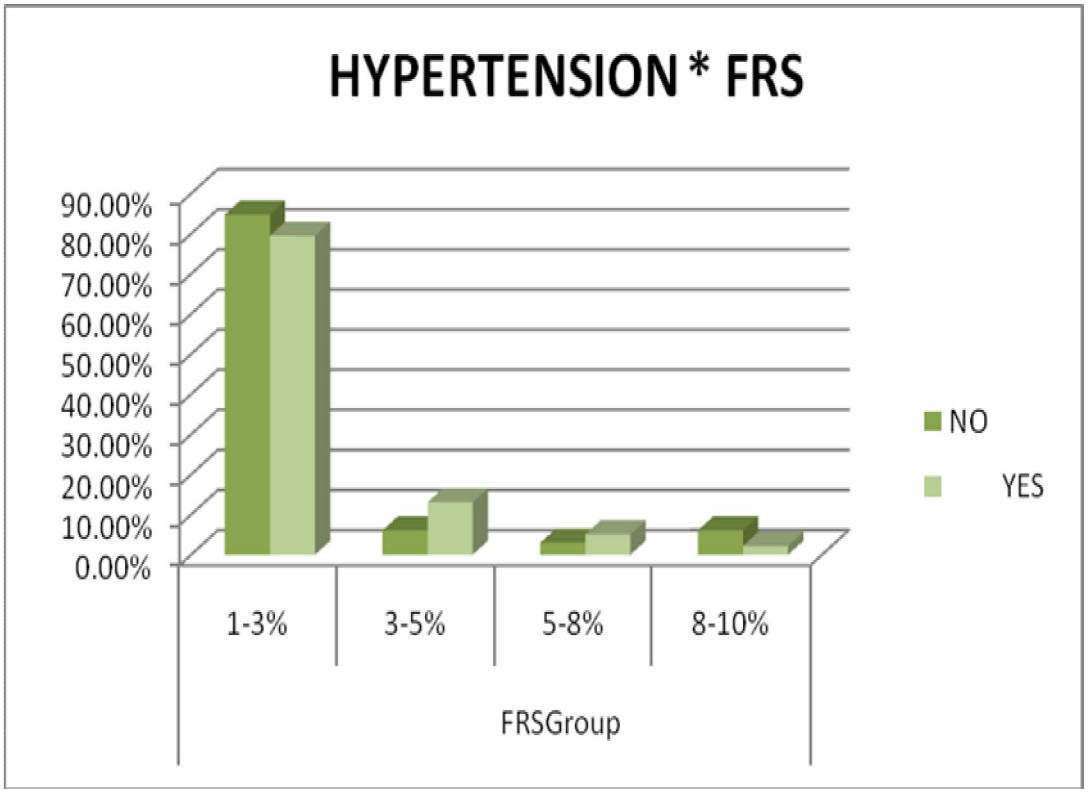
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5-8%
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FRSGroup
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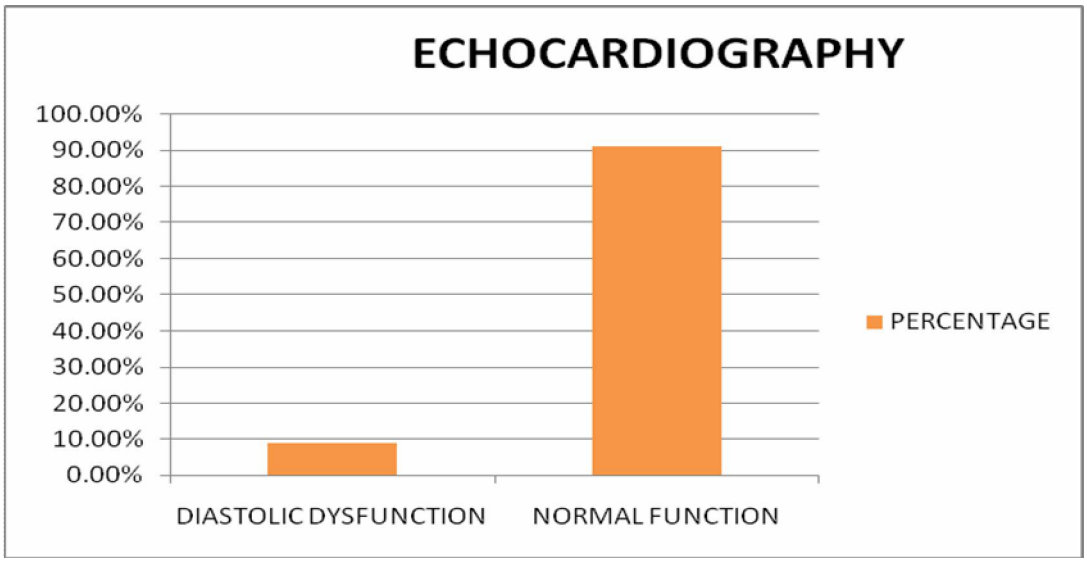
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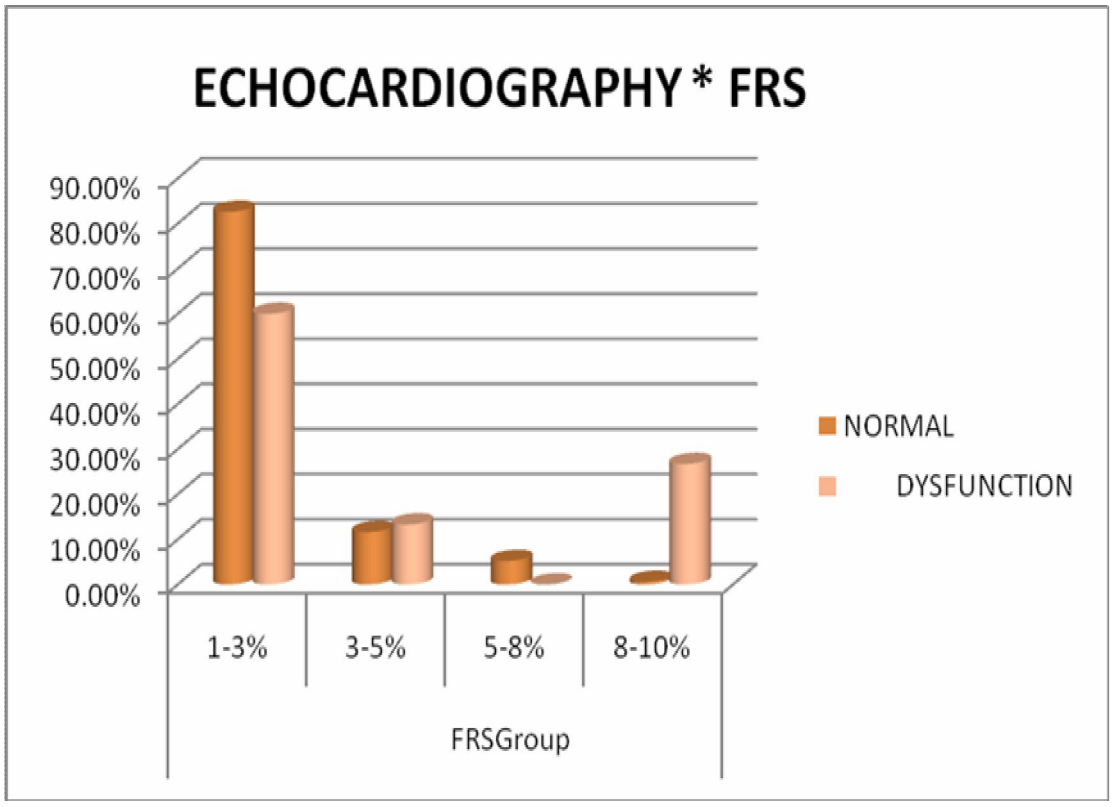
DIASTOLIC DYSFUNCTION
NORMAL FUNCTION
PERCENTAGE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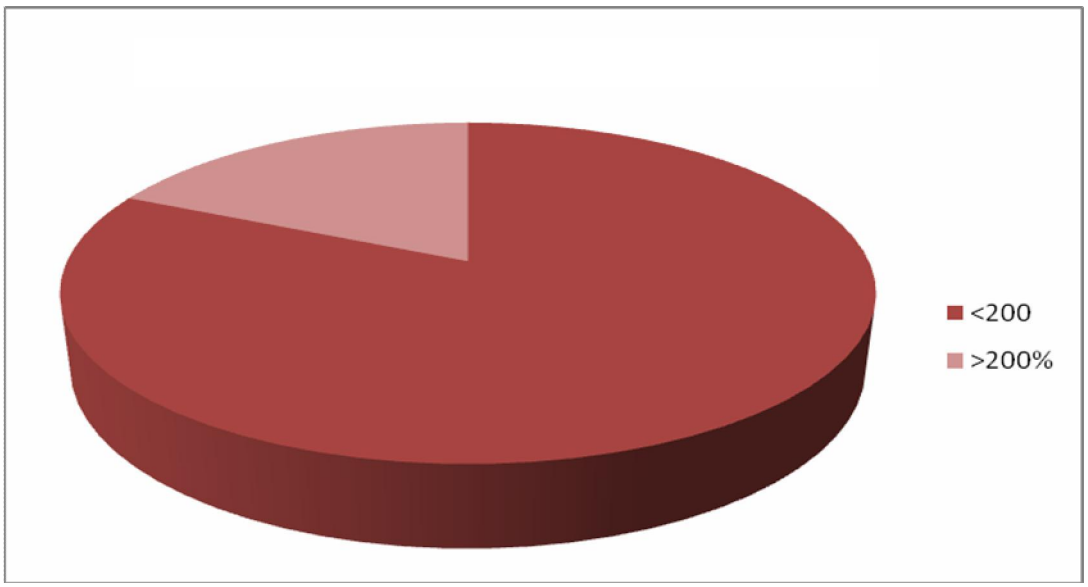
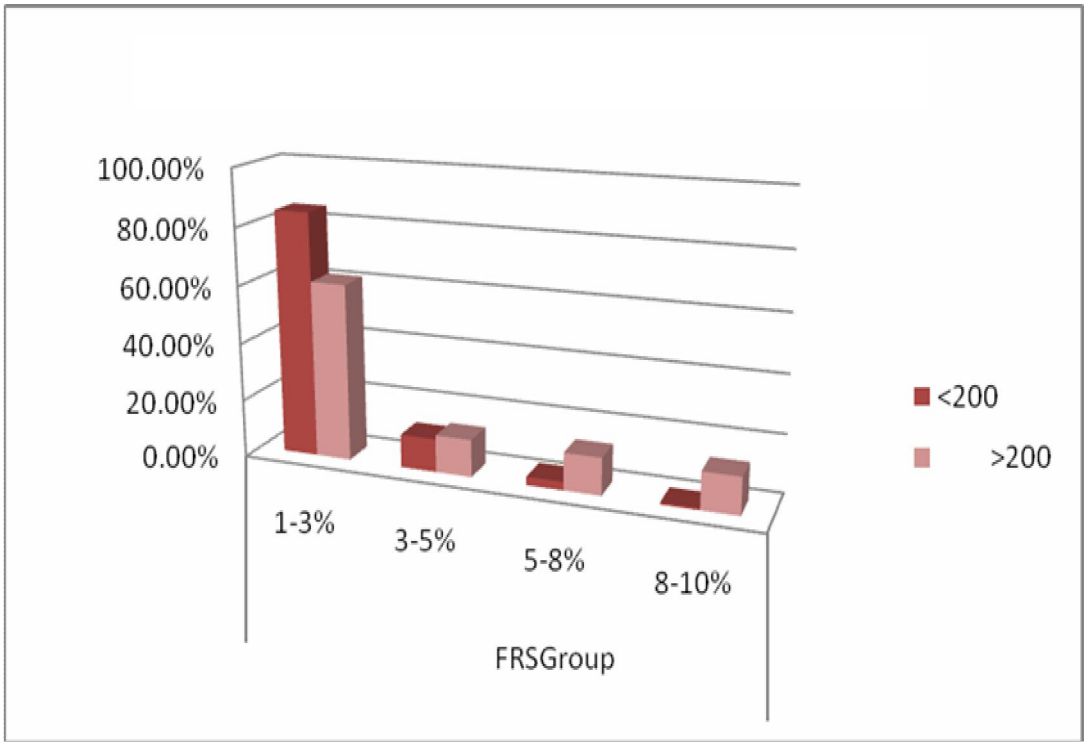
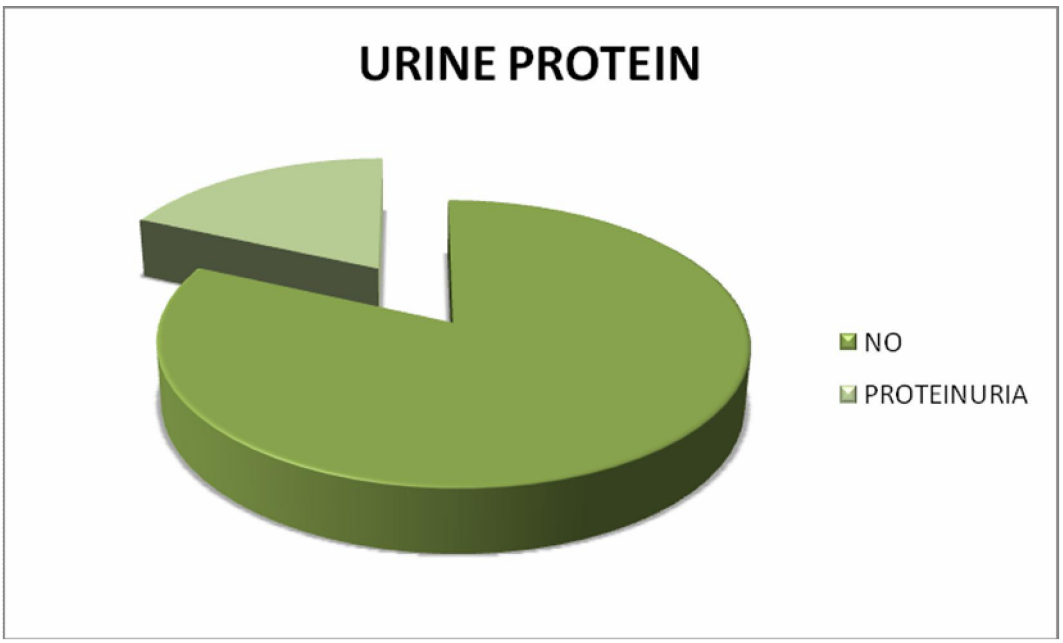
FRSGroup
NORMAL
DYSFUNCTION

## TOTAL CHOLESTEROL LEVELS




## TOTAL CHOLESTEROL LEVELS * FRS

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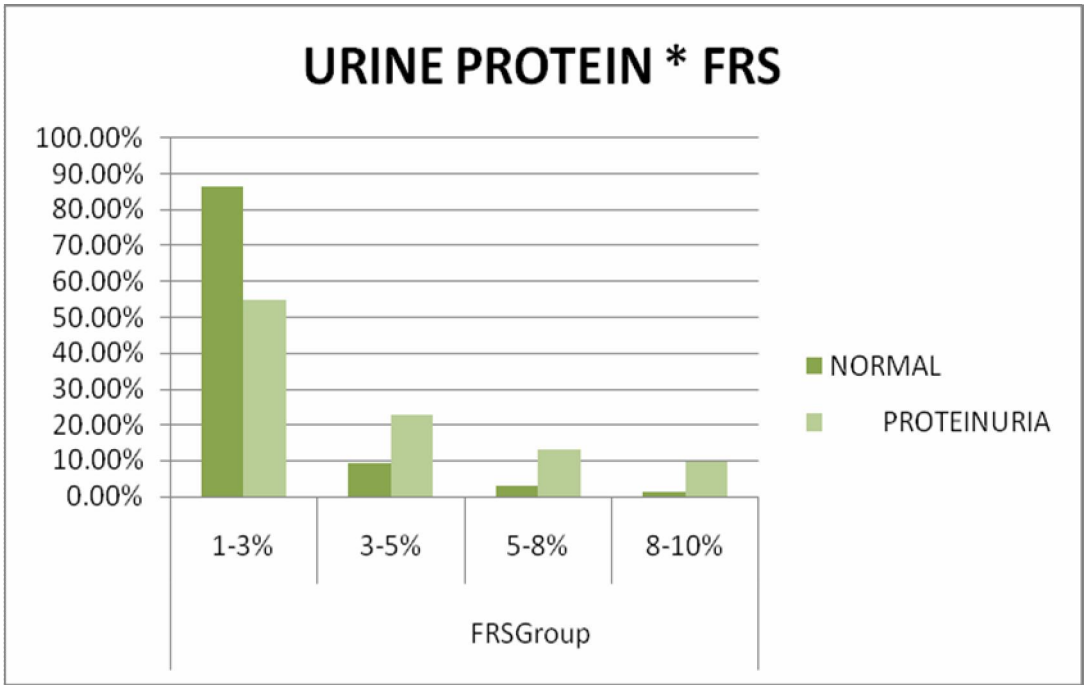
PROTEINURIA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8-10%
FRSGroup
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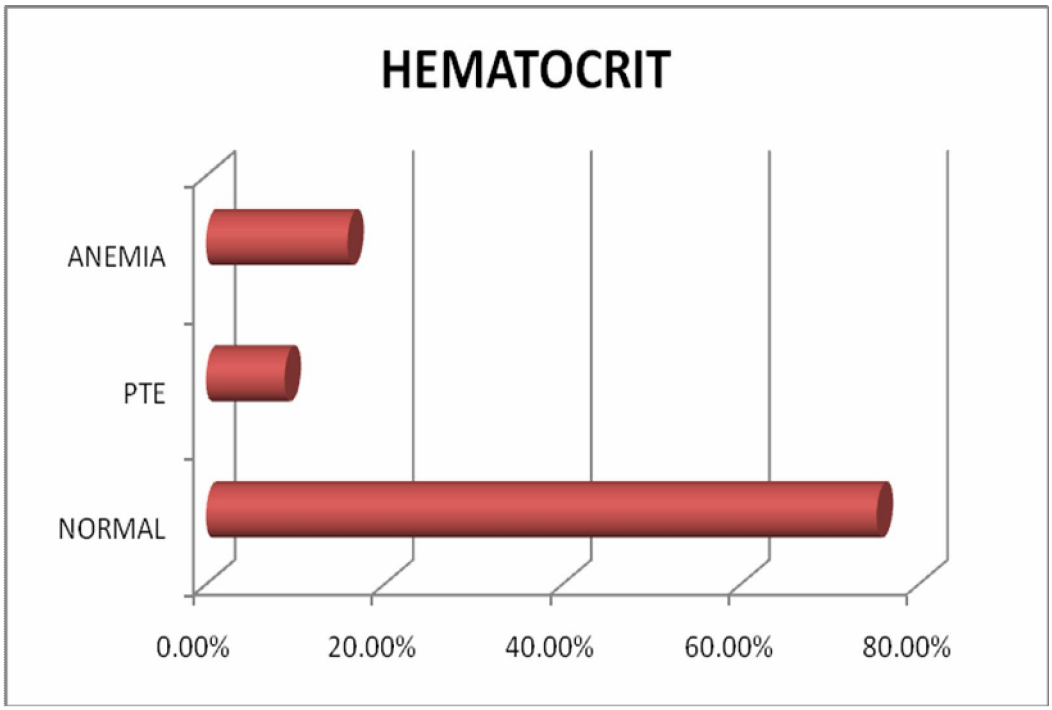
PROTEINURIA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PTE
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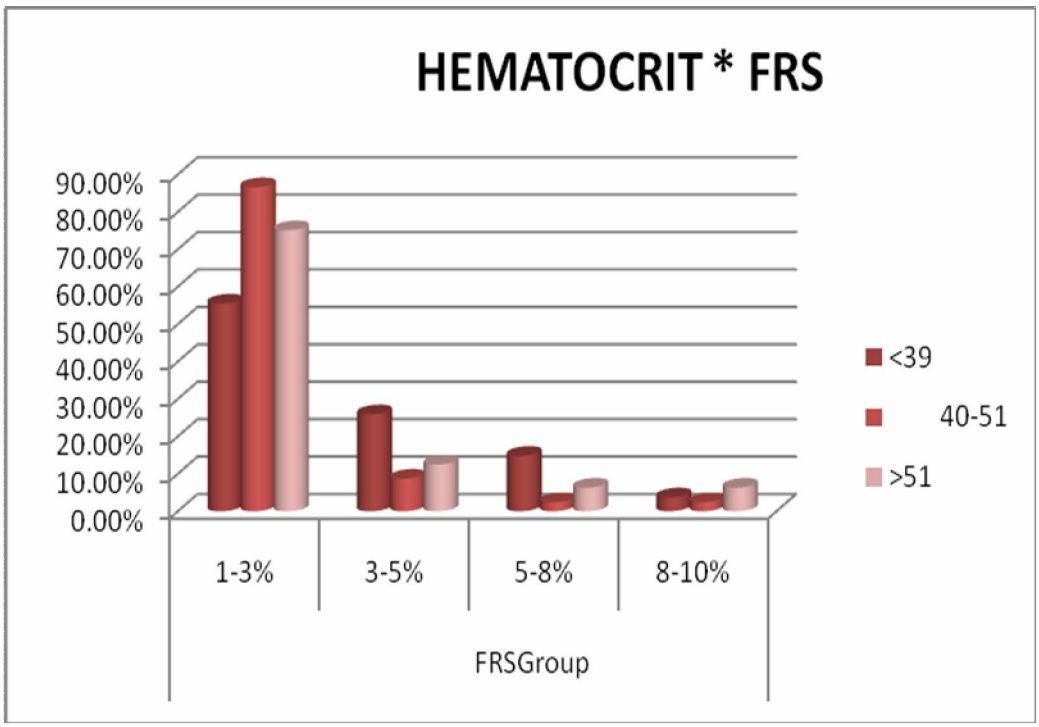
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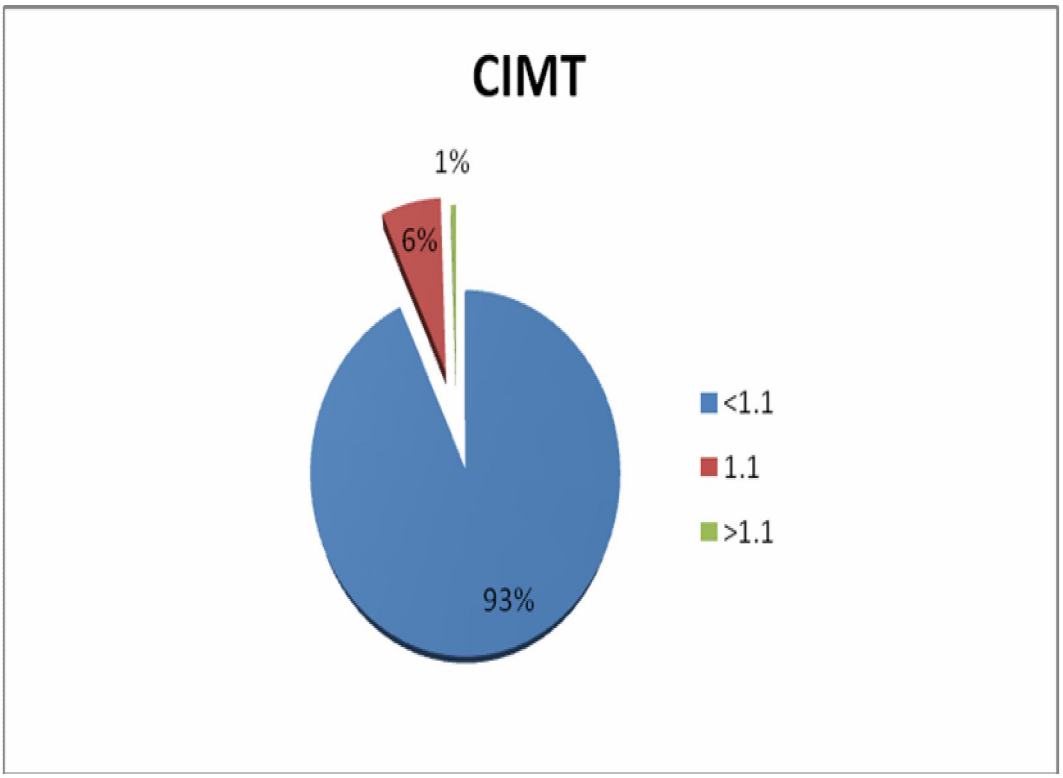
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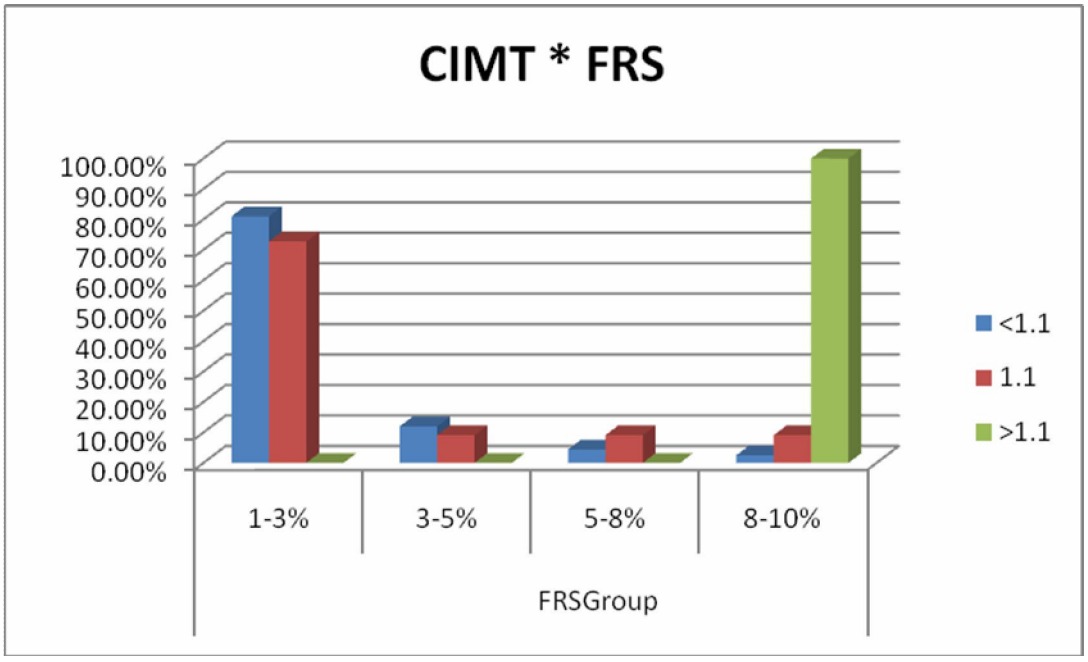
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