Original Article

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The effect of immunosuppressive therapy on the lipid profile of kidney transplant patients: A single-center study

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ABSTRACT

Background: Post-transplant dyslipidemia is common and presents unique management challenges for nephrologists. The most important outcomes of post-transplant dyslipidemia treatment include preserving or improving allograft function and reducing cardiovascular morbidity and mortality. **Aim**: This study aims to assess the impact of immunosuppressive therapy on lipid profiles in kidney transplant patients. **Methods**: This cross-sectional study was conducted at the Renal Transplant and Nephrology Center, Baghdad Medical City, over 15 months. A total of 51 kidney transplant recipients were enrolled, including 34 males (66.7%) and 17 females (33.3%), with an age range of 20 to 60 years and a male-to-female ratio of 2:1. Patient data, including age, gender, medical history (including drug history and pre-transplant dyslipidemia), date of kidney transplantation, donor type (related or unrelated), immunosuppressive regimen, renal function tests, fasting blood sugar, fasting lipid profile, urinalysis, body weight, body mass index (to exclude obese patients), and blood pressure, were recorded using pre-prepared data sheets. Dyslipidemia in kidney transplant recipients was diagnosed based on a fasting lipid profile obtained after 8–12 hours of fasting. **Results**: Among the 51 kidney transplant recipients, 25 patients were on cyclosporine A, 21 on tacrolimus, and five on sirolimus, in addition to mycophenolate mofetil and prednisolone. There were statistically significant differences in serum cholesterol, low-density lipoprotein (LDL), and triglyceride levels among kidney transplant recipients based on their immunosuppressive medication. However, no statistically significant differences were observed in serum high-density lipoprotein (HDL) and very low-density lipoprotein (VLDL) levels. The highest cholesterol, triglyceride, and LDL levels were observed in patients receiving sirolimus, followed by those on cyclosporine A. In contrast, the lowest levels of cholesterol, LDL, and triglycerides were found in patients on tacrolimus therapy. No statistically significant differences in serum lipid levels were observed between males and females, between patients older than 40 years and those younger than 40 years, or between individuals with and without diabetes mellitus. Patients were categorized based on the time since transplantation (one, two, three, four, or five or more years post-transplant), but no statistically significant differences in serum lipid levels were found based on the duration since transplantation. **Conclusion**: Cyclosporine and sirolimus may contribute to dyslipidemia. Among the immunosuppressive therapies studied, sirolimus was associated with the worst lipid profile, while tacrolimus was linked to a more favorable lipid profile compared to both sirolimus and cyclosporine.

Keywords: dyslipidemia, hypertriglyceridemia, transplantation

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INTRODUCTION

At one time, the prevalence of hyperlipidemia, the most common form of dyslipidemia, was estimated to be as high as 80% in kidney transplant recipients.[1] The significant incidence of hyperlipidemia has been documented since 1973.[2] During the post-transplant era with azathioprine and corticosteroids, the prevalence rate was estimated to be between 50% and 78%.[3–5] Hypertriglyceridemia was found to be as common as hypercholesterolemia. However, with the introduction of cyclosporine, hypercholesterolemia became the predominant abnormality,[6] particularly with an elevation in LDL cholesterol levels.[7] Early prevalence estimates of hyperlipidemia exceeding 50% have also been reported in heart transplant recipients (HTRs).[8] In lung transplant recipients, the prevalence of hypercholesterolemia and hypertriglyceridemia has been reported as 32% and 41%, respectively.[9] Estimates of dyslipidemia in liver transplant recipients range from 31% to 51% ,with one study reporting a prevalence of 43%.[10, 11]

The point prevalence of hyperlipidemia is unlikely to change over time following transplantation. In kidney transplant recipients, hyperlipidaemia persists if left untreated. Furthermore, its prevalence may increase over time due to inadequate long-term patient surveillance. Several cumulative factors, including aging, immunosuppression, weight gain, and the onset of diabetes, may contribute to its development.

Every recipient must have at least one fasting lipid profile, with the first assessment conducted within the first year. An initial review has been suggested as early as three months post-transplantation.[6]

A Canadian commentary on the 2009 KDIGO Clinical Practice Guideline[12] recommends an initial lipid measurement 2–3 months after transplantation, 2–3 months after any treatment modification, and annually thereafter. Older European guidelines 2002 [13] also support annual monitoring. The necessity of repeated lipid level measurements in various forms of chronic renal disease has been questioned, primarily due to a lack of relevant data and clinical trial evidence. A practical approach may involve assessing the transplant recipient's overall cardiac risk profile and reserving lipid monitoring for those considered more susceptible to cardiovascular disease. Notably, persistent graft dysfunction may itself be regarded as a high-risk equivalent.[14] All transplant recipients should consult a dietitian regularly, if not routinely. Reducing cholesterol, saturated fats, and total fat intake is recommended as an initial intervention, particularly for kidney transplant recipients, who inherently have chronic kidney disease (CKD).[15, 16]

Early post-transplant initiation of statin therapy may be more feasible, as patients are often more receptive to new health interventions and medication adjustments during this period. Over time, concerns about long-term issues such as cardiovascular vascular diseases (CVD) may become less prominent, and the introduction of new medications may be perceived as an unnecessary risk or a potential threat to allograft health.[16]

Thanks

MATERIAL AND METHODS

Study Setting and Design

This study was conducted at the nephrology and renal transplant center of a medical city. Ethical approval was obtained from the Board Ethics Committee.

Data collection began in May 2017 and continued until the end of August 2018.

Selection of the Study Sample

Fifty-one transplant recipients were included in this cross-sectional study over a 15-month period. The study population consisted of 34 men and 17 women, with a male-to-female ratio of 2:1. Participants ranged in age from 20 to 60 years.

The cases were documented on a pre-prepared data sheet, including details on the participants' age, gender, medical history (including drug history and pre-transplant dyslipidemia), date of kidney transplantation, type of donor (related or unrelated), immunosuppressive regimen, renal function test results, fasting blood sugar, fasting lipid profile, urinalysis, urine protein-creatinine ratio, body weight, body mass index, and blood pressure.

Inclusion Criteria

Patients with kidney transplantation who have not been on statin therapy for more than three months, either due to intolerance of side effects or discontinuation because of non-adherence.

Exclusion Criteria

1. Kidney transplant patients currently on statin therapy or any medication affecting lipid profiles, other than immunosuppressive regimens (e.g., diuretics or beta-blockers).

2. Patients with chronic allograft dysfunction or proteinuria exceeding 1 g/day, as determined by the urine protein-creatinine ratio.
3. Obese patients.

Standard Regimens

The standard immunosuppressive treatment regimens for kidney transplant recipients at our center included:

- TAC/MMF-MPA/Prednisolone or
- CSA/MMF-MPA/Prednisolone or
- mTORi/MMF-MPA/Prednisolone

All patients were on maintenance immunosuppressive therapy with no recent changes to their regimen.

Definition of Variables

1. A patient was considered diabetic if they had a history of diabetes or a fasting venous plasma glucose evel ≥ 7.0 mmol/L. Additionally, diabetes was diagnosed if the random venous plasma glucose level was ≥ 11.1 mmol/L on two separate occasions.
2. Dyslipidemia was diagnosed if one or more of the following criteria were met: total serum cholesterol (hypercholesterolemia) > 5.2 mmol/L (220 mg/dL), total triglycerides (hypertriglyceridemia) (TG) > 2.26 mmol/L (200 mg/dL), LDL > 100 mg/dL, or HDL < 40 mg/dL in males and < 50 mg/dL in females.[17]
3. Obesity: Body mass index (BMI), calculated as weight in kilograms divided by height in meters squared, is used to assess obesity. For adults, a BMI of 25.0 to 29.9 kg/m2 is classified as overweight, while a BMI of 30 kg/m2 or higher is classified as obese.[18]
4. Chronic allograft dysfunction is associated with various fibrosing and sclerosing changes in the allograft. Fibrosis is multifactorial and represents a final pathway following different types of injury. Using a range of diagnostic criteria, pathologists can and should define specific lesions to identify the pathogenic processes affecting the allograft.
5. Hypertension: According to the new guidelines, blood pressure categories are defined as follows:
   - Normal: Less than 120/80 mm Hg.
   - Elevated: Systolic between 120–129 mm Hg and diastolic less than 80 mm Hg.
   - Stage 1: Systolic between 130–139 mm Hg or diastolic between 80–89 mm Hg.
   - Stage 2: Systolic at least 140 mm Hg or diastolic at least 90 mm Hg.
   - These classifications are based on an average of at least two careful readings taken on two or more separate occasions.[19]

Measurement of Serum Lipids

The diagnosis of dyslipidemia in SOT recipients typically begins with a lipid profile obtained after an 8–12 hour fast. While non-fasting lipid measurements are sometimes recommended for the general population, transplant recipients are considered a high-risk group for CVD and should therefore undergo fasting lipid assessments.[17]

Statistics

Data analysis was performed using SPSS version 20 (Statistical Package for the Social Sciences) to determine statistical significance among different variables. Descriptive statistics, such as the mean, were used alongside analytical statistics where appropriate. Categorical variables were presented as frequencies (numbers) and proportions (%), while continuous variables were reported as mean and standard deviation (SD).

A P-value of less than 0.05 was considered statistically significant and was calculated using the Pearson chi-square test. In the result tables, percentages were based on rows rather than columns. Results and findings were presented in tables, accompanied by explanatory paragraphs.

RESULTS

This study enrolled 51 kidney transplant recipients, including 34 males (66.7%) and 17 females (33.3%), with an age range of 20 to 60 years. The male-to-female ratio was 2:1.As seen in figure 1, glomerular nephritis and small, sick kidneys are the most frequent causes of end-stage renal disorders, respectively.

Table 1 presents the demographic distribution of patients based on gender, age, type of kidney donor (living-related or living-unrelated), date of transplantation, and the presence of diabetes or hypertension.

Figure 1 show distribution of patients according to the causes of end stage renal diseases

Table 2 show the highest cholesterol levels were observed in patients on sirolimus, followed by those on

cyclosporine, while the lowest levels were found in patients on tacrolimus therapy. The P-value was statistically significant (p = 0.001).

Table 3 presents the mean values of lipid parameters based on the gender of kidney transplant recipients.

Table 4 presents the mean values of lipid parameters based on the age categories of kidney transplant recipients. In this study, 27 patients (52.9%) were younger than 40 years, while 24 patients (47.1%) were 40 years or older

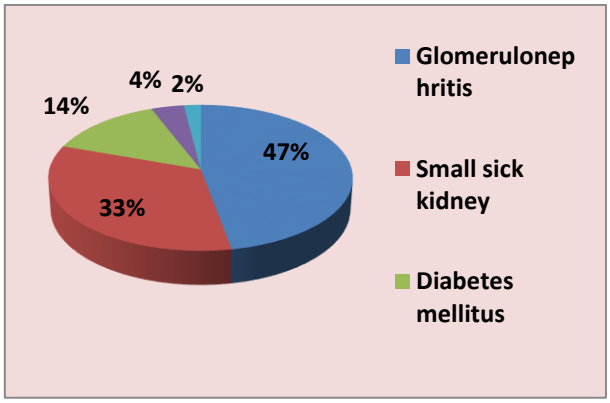

Figure 1: Distribution of patients according to causes of ERSD.

Table 1: Descriptive characteristics of included patients.

Characteristics of patients		No.	%
Age category/year	< 40	27	52.9%
	≥ 40	24	47.1%
Gender	Male	34	66.7%
	Female	17	33.3%
Living relation	Related	31	60.8%
	Unrelated	20	39.2%
Date of transplant/year	One	12	23.5%
	Two	13	25.5%
	Three	12	23.5%
	Four	7	13.7%
	Five and more	7	13.7%
DM	Yes	13	25.5%
	No	38	74.5%
HT	Yes	46	90.2%
	No	5	9.8%
IHD	Yes	0	0.0%
	No	51	100.0%
STROKE	Yes	0	0.0%
	No	51	100.0%

Table 2: Mean values of lipid parameters based on the immunosuppressive medications used (cyclosporine A, tacrolimus, or sirolimus).

S. lipids	Drug	No.	Mean	Std. Deviation	p-value
Serum cholesterol	Cyclosporine	25	199.76	47.87	0.001
	Tacrolimus	21	153.62	35.48	
	Sirolimmus	5	212.40	40.86	
Serum LDL	Cyclosporine	25	112.03	32.85	0.001
	Tacrolimuse	21	77.41	29.02	
	Sirolimmus	5	123.34	31.40	
Serum HDL	Cyclosporine	25	47.76	13.88	0.3
	Tacrolimus	21	47.41	12.26	
	Sirolimmus	5	38.54	4.01	
Serum VLDL	Cyclosporine	25	37.78	20.60	0.07
	Tacrolimuse	21	31.20	10.95	
Tacrolimus	Sirolimmus	5	50.12	17.37	
Serum triglyceride	Cyclosporine	25	189.00	103.27	0.03
	Tacrolimuse	21	148.90	51.46	
	Sirolimmus	5	253.40	85.78	
	Total	51			

* A significant P-wave value is defined as < 0.05

This study enrolled 51 kidney transplant recipients, including 34 males (66.7%) and 17 females (33.3%), with a male-to-female ratio of 2:1.

Table 5 presents the mean values of lipid parameters based on the number of years since kidney transplantation. Patients were categorized into one, two, three, four, and five or more years post-transplant. The analysis showed no statistically significant differences in serum lipid levels among kidney transplant recipients based on the time since transplantation (P-values ≥ 0.05).

Table 6 presents the mean values of lipid parameters based on the presence or absence of diabetes mellitus among kidney transplant recipients. In this study, 13 patients (25.5%) had diabetes mellitus, while 38 patients (74.5%) did not.

Table 7 shows the overall correlations between fasting glucose levels and serum lipids, including cholesterol, LDL, HDL, VLDL, and triglyceride levels. With each increase in fasting blood sugar, there was an increase in S-LDL by 0.03, S-VLDL by 0.1, and S-triglycerides by 0.1

Table 3: Mean values of lipid parameters by patients' gender.

S.lipids	Gender	No.	Mean	Std. Deviation	p-value
Serum cholesterol	Male	34	177.71	49.95	0.3
	Female	17	190.59	44.75	
Serum LDL	Male	34	93.92	35.20	0.1
	Females	17	108.82	35.58	
Serum HDL	Male	34	44.57	11.15	0.08
	Female	17	51.00	14.78	
Serum VLDL	Male	34	38.91	19.66	0.1
	Female	17	31.01	10.84	
Serum triglyceride	Male	34	190.74	99.37	0.1
	Female	17	154.94	54.30	

** A significant P-wave value is defined as less than 0.05*

Table 4: Mean values of lipid parameters by age category.

S.lipids	Age category	No.	Mean	Std. Deviation	p-value
Serum cholesterol	< 40	27	178.67	54.37	0.6
	≥ 40	24	185.75	41.06	
Serum.LDL	< 40	27	93.30	36.72	0.2
	≥ 40	24	105.17	34.16	
Serum.HDL	< 40	27	48.75	13.44	0.2
	≥ 40	24	44.42	11.66	
Serum.VLDL	< 40	27	33.82	18.31	0.2
	≥ 40	24	39.04	16.53	
Serum triglyceride	< 40	27	169.19	91.77	0.4
	≥ 40	24	189.63	84.27	

**A significant P-wave value is defined as < 0.05*

Table 5: Mean values of lipid parameters by years since transplantation.

Year	S.cholesterol		S.LDL		S.HDL		S.VLDL		S.triglycerides	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
one	189.08	49.71	102.50	36.91	53.21	15.14	36.85	21.94	183.83	109.73
Two	163.77	40.46	84.71	30.38	46.91	15.30	34.32	20.21	160.46	99.72
Three	211.92	54.25	118.58	41.03	42.25	7.80	43.23	16.86	217.17	84.95
Four	154.71	34.11	82.43	20.69	42.57	9.55	28.29	11.08	143.00	57.89
Five and more	179.71	39.101	101.71	34.524	47.00	10.440	35.00	5.627	174.29	28.476
p-value	0.05		0.1		0.2		0.4		0.4	

** A significant P-wave value is defined as less than 0.05*

Table 6: Mean values of lipid parameters by diabetes mellitus status.

S. LIPIDS	DM	No.	Mean	SD	p-value
Serum cholesterol	Yes	13	180.46	40.40	0.8
	No	38	182.53	51.10	
Serum LDL	Yes	13	102.00	29.15	0.7
	No	38	97.82	37.96	
Serum HDL	Yes	13	45.38	9.32	0.6
	No	38	47.17	13.75	
Serum VLDL	Yes	13	34.91	12.50	0.7
	No	38	36.75	19.05	
Serum triglyceride	Yes	13	175.23	63.62	0.8
	No	38	180.03	95.69	

** A significant P-wave value is defined as less than 0.05*

Table 7: Correlation between measured parameters.

		FBS	S.cholesterol	S.LDL	S.HDL	S.VLDL	S.triglycerides
FBS	R	1	-0.03	0.03	-0.3	0.10	0.1
	P-value		0.8	0.8	0.08	0.4	0.4

** A significant P-wave value is defined as less than 0.05*

DISCUSSION

Hypertension is common after transplantation and was observed in 90% of renal transplant recipients in this study. These findings are consistent with those of Kasiske BL et al. and Premasathian NC et al., which indicate that hypertension may affect 60%–90% of kidney transplant recipients.[20, 21] The wide range in prevalence may reflect variations in the definitions of hypertension, donor sources, immunosuppressive medications, time since transplantation, and level of allograft function. Typically, systolic blood pressure is highest immediately after transplantation and decreases during the first year.

In this study, the post-transplant drug regimen consisted of prednisolone and either MMF or MPA, combined with cyclosporine A, tacrolimus, or sirolimus. There were statistically significant differences in serum cholesterol and LDL levels among kidney transplant recipients based on immunosuppressive medications (cyclosporine, tacrolimus, sirolimus). The mean s. cholesterol levels were 199.76 mg/dL for cyclosporine, 153.62 mg/dL for tacrolimus, and 212.40 mg/dL for sirolimus, with an SD of 47.87, 35.48, and 40.86, respectively.

The highest cholesterol levels were observed in patients on sirolimus, followed by those on cyclosporine, while the lowest levels were found in patients on tacrolimus therapy. The P-value was statistically significant (p = 0.001).

The mean s. LDL levels in patients on cyclosporine, tacrolimus, and sirolimus were 112.03 mg/dL, 77.41 mg/dL, and 123.34 mg/dL, respectively, with an SD of 32.85, 29.02, and 31.40. The highest LDL levels were observed in patients on sirolimus, followed by those on cyclosporine, while the lowest levels were found in patients on tacrolimus therapy. The P-value was statistically significant (p = 0.001).

These findings may be consistent with other studies. For example, the Symphony study by Claes K et al. reported higher LDL cholesterol levels in patients receiving sirolimus therapy.

The mean s. triglyceride levels in patients on cyclosporine, tacrolimus, and sirolimus were 189 mg/dL, 148.90 mg/dL, and 253.40 mg/dL, respectively, with an SD of 103.27, 51.46, and 85.78. The highest triglyceride levels were observed in patients on sirolimus, followed by those on cyclosporine, while the lowest levels were found in patients on tacrolimus therapy. The P-value was statistically significant (p = 0.03). Claes K et al. reported that sirolimus is more strongly associated with hypertriglyceridemia than hypercholesterolemia, even at lower drug exposure levels.[22]

Morrisett JD et al. concluded that sirolimus alters the insulin signaling pathway, leading to increased adipose tissue lipase activity and/or decreased lipoprotein lipase activity. This results in increased hepatic triglyceride synthesis, elevated VLDL secretion, and heightened hypertriglyceridemia.[23] Although S. HDL levels were lowest in patients on sirolimus therapy and highest in those on cyclosporine and tacrolimus therapy, the results were not statistically significant (P = 0.3).

Thus, HDL levels also tend to be affected by immunosuppressive medications. This is consistent with findings by Ettinger WH et al., which reported low HDL levels following kidney transplantation.[24]

Serum VLDL mean levels were lowest in patients on tacrolimus and highest in those on sirolimus; however, these results were not statistically significant (p = 0.07).

Among the study participants, 13 patients (25.5%) had diabetes mellitus, while 38 patients (74.5%) did not. The diabetic group included both individuals with pre-transplant diabetes and those who developed new-onset diabetes after transplantation (NODAT).

Risk factors for diabetes among immunosuppressive therapies include corticosteroids and the calcineurin inhibitors tacrolimus and, to a lesser extent, cyclosporine.[25] Neither azathioprine nor MMF is diabetogenic. In fact, MMF may even mitigate the diabetogenic effects of tacrolimus, possibly by enabling clinicians to use lower doses.

Turk TR et al. reported that NODAT occurs in 4%–25% of renal transplant recipients. The variation in incidence may be attributed to differences in definitions, duration of follow-up, and the presence of both modifiable and non-modifiable risk factors. Major risk factors for NODAT include African American and Hispanic ethnicity (compared to Caucasians or Asians), obesity (BMI of 30 kg/m2), age over 40 years, a family history of diabetes among first-degree relatives, impaired glucose tolerance before transplantation, or the presence of other components of metabolic syndrome. Additional risk factors include receiving a deceased donor kidney, hepatitis C infection, and immunosuppressive therapies.[25]

Pham PT identified additional potential risk factors for the development of NODAT, including the presence of certain human leukocyte antigen (HLA) antigens (such

as A30, B27, and B42), an increasing number of HLA mismatches, a history of acute rejection, cytomegalovirus (CMV) infection, and male gender of both the recipient and donor.Polycystic kidney disease has also been suggested as a risk factor for post-transplant diabetes in some studies, although findings remain inconsistent.[26]

In this study, there were no statistically significant differences in serum lipid levels between patients with and without diabetes mellitus. Additionally, no significant variations were observed in serum lipid levels between male and female kidney transplant recipients or between recipients younger than 40 and those older than 40. Patients were categorized based on the time since transplantation into one, two, three, four, and five or more years post-transplant. However, serum lipid levels did not show statistically significant differences based on the transplantation date.

CONCLUSIONS

1. Dyslipidemia is a common complication after kidney transplantation, even when allograft function is normal or near normal.
2. Cyclosporine and sirolimus may contribute to the development of dyslipidemia.
3. Sirolimus is associated with the worst lipid profile, while tacrolimus is linked to a better lipid profile compared to both sirolimus and cyclosporine therapy.
4. The effects of tacrolimus on lipid metabolism are generally similar to those of cyclosporine, so it remains unclear why tacrolimus is associated with less hyperlipidemia.

Recommendations

1. Statin therapy has been the primary focus in KT recipients; however, the role of proper dietary guidance and adjuvant pharmacological or non-pharmacological interventions should not be overlooked.
2. At all stages of treatment, appropriate monitoring for the side effects of immunosuppressive medications should be implemented to maximize benefits while minimizing adverse effects.
3. Attention to dyslipidemia is essential, as interventions have been shown to reduce cardiac events in clinical trials specific to the transplant population.
4. All patients should be encouraged to adopt a healthy diet and engage in regular physical activity.
5. Every transplant recipient should have regular consultations with a nutritionist and undergo periodic lipid profile monitoring, especially during the first year post-transplant when immunosuppressive drug doses are highest. An initial review has been suggested as early as three months after transplantation.

Limitations of the Study

1. Single-center study: This study was conducted at a single kidney transplant center in Baghdad (Medical City) because, at the time of the study, the only other center, Al-Karama Teaching Hospital, was closed.
2. Limited number of patients on sirolimus: Only five patients in the study were on sirolimus because it is not a primary drug used in kidney transplant recipients. The main immunosuppressive drugs for kidney transplant recipients are mycophenolate mofetil, cyclosporine, and prednisolone. Additionally, the use of sirolimus is generally low due to its side effects in kidney transplant recipients.
3. The study found that glomerulonephritis was the most common cause of renal transplantation. This is because most patients at the kidney transplant center who were eligible for transplantation had glomerulonephritis. Although diabetes is the most common cause of ERSD, diabetic patients are not necessarily the most frequently referred for kidney transplantation. Many diabetic patients may not be suitable candidates due to comorbidities and associated conditions such as peripheral vascular disease, ischemic heart disease, diabetic foot, and active infections.
4. Lifestyle factors may be challenging to assess in this patient group and should be explored in a separate study.

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