

Arterial Hypertension in Kidney Transplant Recipients

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Abstract

Cardio vascular disease are known to be the first mortality cause in kidney transplantation (KT). HT in KT recipient (KTR) leads to a high cardio-vascular mortality and also to a short kidney survival. (1)

Introduction

Arterial hypertension (HT) is common in renal transplant recipients (RTRs) and it is reported to occur from 50% to 80 % of cases. HT in KTR is considered when Systolic Blood pressure/Diastolic blood pressure is > 130/80 mmHg (KDIGO 2012).(1)

We report here the prevalence of HT in RTRs in our kidney transplantation center and it's predictive factors.

Methods

Our report is about KTR in one KT department (La Rabta Hospital Tunis Tunisia) performed from 2011 to 2018. We study here the prevalence of HT in KTR in our KT department.

Evaluation of blood pressure was done at 1 year after KT based on office blood pressure measurement and HT was considered when Systolic Blood pressure/Diastolic blood pressure was > 130/80 mmHg (KDIGO 2012).(1)

We performed a comparative study of 2 groups (HT recipients and normotensive recipients) in order to determine predictive factors for HT in KTR. The software SPSS 19.0 (IBM Corp, Armonk, NY) was used for statistical analysis. Categorical data were assessed by the chi-square test. A p value <0.05 indicated a significant difference

Results

This study is about 78 KTR from a living kidney donor in 97.5 % of cases and deceased donor in 2,5 % of cases and it is about 51 males and 27 females with a mean age of 34±7 years old.

After KT, HT was noted in 46 patients (59.7 %) and 32 patients were normotensive (41%). Among normotensive patients, we noted that 6 patients (18%) had HT before KT which disappeared after KT.

Among HT patients, HT was not controlled in 22% of cases

(based on target blood pressure levels ≤ 130/80 mm Hg). All these patients had at least 2 or 3 anti-hypertensive medications. In our report of 78 KTR, some factors were noted: dyslipidaemia in 2% of cases, obesity and overweight in 24% of cases and donor HT in 4 % of cases. Delayed graft function was noted in 6 cases (8%) and immunosuppressive therapy was based on steroids in all cases (Prednisone 10 mg/day reached at the first month and maintained for all patients), ciclosporin in 14 patients (18%), sirolimus in 10 patients (12%) and tacrolimus in 70 % of cases.

Our comparative study showed that many factors can be predictive of HT after KT such as HT before KT, diabetes, tabaco, delayed graft function, creatinine at 1 year after KT > 15 mg/l, immunosuppressive medicament such ciclosporin use and donor factors (age donor > 50 years old). All these correlations are statistically significant. (Table 1)

Predictive factors	Recipients without HT N: 32 (41%)	Recipients with HT N: 46 (59.7%)	P value
HT before KT	6 (18%)	40 (86%)	<0.05
Diabetes	0 (0%)	21 (45%)	<0.05
HT in donor	3 (9%)	0 (0%)	not S
Tabaco	6 (18%)	31 (67%)	0.03
Donor age > 50 years old	5 (3%)	22 (47%)	<0.05
Delayed graft function	0 (0%)	6 (13%)	<0.05
Creatinin at 1 year > 15 mg/l	4 (12%)	14 (30%)	<0.05
Ciclosporin use	4 (12 %)	10 (22%)	0.01

Table 1 : Predictive factors of HT- comparative study

Discussion

Our data show that HT had a high prevalence in kidney recipient (59.7%). However, some factors lead to a relatively controlled prevalence of HT in our patients compared to other reports.

These factors are: the relative eviction of ciclosporin use, the low dose of steroids reached early after KT. In the other hand and despite HT guidelines in KT recipients and in chronic kidney disease patients (KDIGO 2012 and ESC/ESH 2018), blood pressure control in our study was not optimal in 22% of HT recipients. (1,2)

Our comparative study showed that many factors can be significantly predictive of HT after KT such as HT before KT, diabetes, tobacco, delayed graft function, creatinine at 1 year after KT > 15 mg/l, immunosuppressive medication such as ciclosporin use and donor factors (age donor > 50 years old). All these correlations are statistically significant (table 1). Most of these factors are reported by authors to be HT predictive factors in KTR (3). Some of these factors can be prevented in KTR in order to control HT prevalence after KT to reduce cardiovascular mortality and to improve graft survival.

Conclusions

All these numerous factors showed that HT mechanisms in KTR are variable and sometimes specific to KTR, the mechanisms include Angiotensin axis stimulation, Reduction of GFR (which is frequent in these populations), Calcineurin inhibitor use especially ciclosporin use, Steroids use, Sodium retention (Only one function kidney), Systemic arterial resistant. (4)

Some of these factors can be prevented in KTR in order to control HT prevalence after KT to reduce cardiovascular mortality and to improve graft survival.

Optimizing treatment should be encouraged by more action on the other risk factors and intensifying treatment.

References

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