Evaluation of subclinical cardiac damage and its determinants in a cohort of Kidney transplanted patients

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Background

Chronic kidney disease (CKD) is a condition with a relevant cardiovascular (CV) risk. Left ventricular hypertrophy (LVH) is mostly responsible of it and is relevant also after kidney transplantation (KTx). In our study, by means of echocardiography we explored at 1 and 12 months after KTx:

1) the prevalence of LVH; 2) the factors related with left ventricular mass index (LVMI) 3) the factors implicated with the development of LVH during the 1st year of KTx.

Material and Methods

In 121 patients (M=69; mean age 47±12 years) over the 470 transplanted in our unit between April 2004 and July 2013, clinical parameters, blood and urinary samples were collected after an overnight fast at 1 and 12 months after KTx. In addition, at the same time, plasmatic levels of FGF-23, Fetuin-A, 25OH-Vitamin D, PTH, Ca, phosphorus and Osteoprotegerin were assessed. Left ventricular hypertrophy (LVH) was determined by echocardiography, calculating left ventricular mass indexed at the height^{2,7} (LVMI) at 1st and 12th month and defined as LVMI>51g/m^{2,7}.

Parameter	Mean±SD	
	1st mth	12th mth
Dialysis time (mths)	58±54	
Age At Ktx (yrs)	47±12	
LVMI (g/m2,7)	56±19	57±26
BMI (kg/m2)	23±3	23±3
SBP (mmHg)	133±16	134±18
DBP (mmHg)	82±10	83±10
eGFR (ml/min)	60±20	58±19
Creatinine (mg/dl)	1,35±0,42	1,36±0,40
Hb (g/dl)	11,1±1,33	12,9±1,4
Albumin (g/dl)	4,2±0,3	4,4±0,4
Blood Glucose (mg/dl)	82±18	82±18
PTH (pg/ml)	175±145	157±167
Ca (mg/dl)	9,89±0,78	10,02±0,71
P (mg/dl)	2,23±0,69	3,0±0,59
RCP (mg/dl)	0,5±0,7	0,3±0,9
U-Prot (g/24h)	0,24±0,20	0,26±0,74
Fetuin (g/l)	0,34±0,19	0,32±0,08
25-OH-VitD (ng/ml)	12,8±5,7	15,9±7,8
25OH-VitD mean (ng/ml)	13,2±5,3	
FGF-23 (pg/ml)	52,7±68,8	50,2±80,6
OPG (pmol/l)	5,38±2,13	4,99±1,66

Table I: Characteristics of the cohort.: LVMI: left ventricular mass indexed at the height2,7; BMI: Body mass index; eGFR: estimated glomerular filtration rate estimated using MDRD formula; PTH: Paratormone; RCP: Reactive C Protein; U-Prot: protein urinary excretion; FGF-23: Fibroblas grouth factor 23, OPG: Osteoprotegerin; HD: Hemodialysis, PD: Peritoneal Dialysis; LVH: Left ventricular hypertrophy; MMF: Acid mycofenolic

Patients (n)	121
Gender (M/F)	69/52
Type of dialysis (%) (HD/PD)	67/21
Type of KTx (%) (Deceased/Living)	95/26
LVHpos 1st mth (%)	49
LVHpos 12th mth (%)	50
LVH regression (%)	19
LVH new development (%)	15
Previous Steroid Therapy (%)	40
Calcineurin inhibitos therapy (%)	98
Total Antihypertensive drugs (%) 0 1 2 3 >3	 7 32 30 21 10
Beta-blockers (%)	75
RAS-inhibitors (%)	4
Diuretics (%)	8
MMF therapy (%)	90
mTOR inhibitor therapy (%)	5
Cumulative steroids in the 1st year (mg±SD)	2882±967
Vit.D Therapy (Calcifediol/Calcitriol) (%)	0,8/6,5
Restart of dialysis (%)	4

Results

Forty-nine and 50% of patients had LVH at 1st (LVHpos1) and 12th mth (LVHpos12) respectively. At univariate regression, at 1st mth, LVMI correlated directly with age (p=0.0001), baseline serum albumin (p=0.004), FGF-23 (p=0.01), systolic blood pressure (p=0.05), U-Prot-24h (p=0.001) and body mass index (p=0.005). LVMI at 12th mth was directly correlated with age (p=0.005), baseline albumin (p=0.01), OPG (p=0.01), FGF-23 (p=0.05) and with LVMI at 1th mth (p<0.0001). During the first year of KTx, 15% of patients developed LVH whereas 19% had its regression. Once those variables were analyzed in a logistic regression (table III), LVMI at baseline resulted the only modifiable risk factor associated with LVH develop. No influence in LVH development was found for Immunosuppressive and anti-hypertensive therapy both in general and class-specifically. No relations were found with Hb levels and mineral metabolism parameters.

Parameter	r(X,Y)	p
Dialysis time (mths)	0,16	0,03
Age At Ktx (yrs)	0,23	0,001
BMI (Kg/m2)	0,22	0,005
DBP (mmHg)	-0,01	0,88
SBP (mmHg)	0,15	0,05
eGFR (ml/min)	-0,11	0,17
Hb (g/dl)	-0,06	0,47
Albumin (g/dl)	-0,22	0,004
Ca (mg/dl)	-0,06	0,44
P (mg/dl)	-0,28	0,0002
U-Prot (g/24h)	0,24	0,001
Fetuin (g/l)	0,10	0,30
FGF-23 (pg/ml)	0,25	0,01
OPG (pmol/l)	0,11	0,22
25-OH-VitD (ng/ml)	0,00	0,98

Parameter	r(X,Y)	p
Dialysis time (mths)	0,20	0,02
Age At Ktx (yrs)	0,26	0,002
BMI (Kg/m2)	0,16	0,06
LV M /2.7 1	0,44	<0,0001
SBP (mmHg)	0,06	0,50
DBP (mmHg)	-0,09	0,30
eGFR (ml/min)	0,00	0,98
Hb (g/dl)	-0,07	0,41
Albumin (g/dl)	-0,26	0,004
Ca (mg/dl)	0,02	0,81
P (mg/dl)	0,00	0,96
U-Prot (g/24h)	-0,16	0,06
Fetuin (g/l)	0,04	0,73
FGF-23 (pg/ml)	0,20	0,05
OPG (pmol/l)	0,23	0,01
25-OH-VitD (ng/ml)	-0,12	0,21
25OH mean	-0,15	0,20

Table II: Univariate correlations of LVMI at 1st (left).and 1th mth after KTx: LVMI: left ventricular mass indexed at the height2,7; BMI: Body mass index; eGFR: estimated gòlomerular filtration rate estimated using MDRD formula; U-Prot: protein urinary excretion; FGF-23: Fibroblas grouth factor 23, OPG: Osteoprotegerin;

Parameter	p	IC		OR
Age	0,05	1,000	1,295	1,13
LVMI	0,03	0,710	0,996	0,84
Albumin	0,93	0,005	117,73	0,803
FGF-23	0,95	0,977	1,022	0,99
eGFR	0,38	0,941	1,172	1,05

Table III: Logistic regression: evaluation of the factors implicated new development of LVH

Conclusions

The prevalence of LVH is relatively high in KTx patients. Nevertheless, in a small portion of them, we observed an improvement of cardiac abnormalities. Albumin, FGF-23 and BMI seem to be directly related with LVMI. In any case, LVMI at baseline is the only modifiable risk factor able in predicting developing of LVH.

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