ORIGINAL ARTICLE

The blood pressure after renal transplantation. A single center experience

Vergoulas G, Miserlis Gr, Karasavvidou F[1], Imvrios G, Katsara I, Georgilas N, Leontsini M[1] , Antoniadis A

Organ Transplant Unit, Hippoktario Hospital, Thessaloniki, Greece
[1] Histopathology Department, Hippokratio Hospital, Thessaloniki, Greece

Post-transplant (pstnt) hypertension is multifactorial and has been connected with increased rate of cardiovascular accidents and decreased graft survival. In this work the clinical factors that may influence pstnt blood pressure were examined. Between 1987 and 1995 the blood pressure of 272 patients (186 male) with renal transplantation (172 from LRD) was investigated retrospectively. Patients' (pts) mean age was 40 years (range 17 - 64). There was at least a six-month follow up with a functioning allograft. Each pt's blood pressure was recorded on the 7th, 15th, 30th pstnt day, on 3rd, 6th pstnt month and on 1st, 2nd, 3rd, 4th and 5th pstnt year. The effect of acute rejection episodes (AR), graft origin (LRD, cadaveric), donor sex, recipient hypertension before transplantation, donor hypertension, recipient sex, cold ischemia time, recipient age, donor age, kind of dialysis before transplantation and primary renal disease on pts' systolic (SBP) and diastolic blood pressure (DBP) during time were investigated. Multivariate repeated measures analysis of variance was used for statistical analysis.

SBP and DBP were 153.68 ±18.54 / 94.40±10.69 mmHg, 142.04±18.77 / 88.96 ± 10.10 mmHg, 134.37 ±16.16 /86.26±8.95 mmHg, 132.48±15.81 /84.72±9.63 mmHg, 134.12 ±15.86 /86.16 ±9.65 mmHg, 133.58±17.35 /85.50±10.00 mmHg, 131.16±15.46/ 83.84± 8.61 mmHg, 131.64±18.2 /84.72±10.28 mmHg, 133.24± 16.20/ 85.22±8.59 mmHg, 134.72±14.22/84.62 ±8.50 mmHg on 7th, 15th, 30th pstnt day, 3rd, 6th pstnt month and 1st, 2nd, 3rd 4th and 5th pstnt year respectively. Recipient's hypertension before transplantation had statistically significant (ss) effect on pts' SBP (p: 0.0005) and DBP (p:0.0005) during the five year follow up. Donor hypertension had ss effect on SBP from the 3rd pstnt month (p: 0.032) to the 4th pstnt year (p:0.038). The effect of AR on SBP was ss from the 1st pstnt month (p:0.003) up to the end of the 3rd year of follow up (p:0.01) and on DBP between 6th pstnt month (p:0.042) and 4th pstnt year (p:0.037). Graft origin (LRD) had ss effect on DBP (p:0.018) during the 1st pstnt month while the kind of dialysis (HD) had ss effect on SBP and DBP during the 1st pstnt month (p:0.004 and p:002 respectively). Donor age had ss effect on SBP from the 6th pstnt month (p:0.014) up to the 4th year of follow up(p:0.049) and on DBP from the 6th pstnt month (p:0.001) to the 5th year of follow up (p:0.024). Recipient age had ss effect on SBP from the 1st pstnt month (p:0.002) up to the 5th pstnt year (p:0.005) and on DBP from the 3rd pstnt year (p:0.019) up to the 5th pstnt year (p:0.008). In conclusion, the factors most significant on posttransplant blood pressure are recipient and donor hypertension before transplantation, recipient and donor age and acute rejection episodes.
Hippokratia 2002, 6 (2): 62-70

Hypertension is a frequent complication of renal insufficiency[1]. Unfortunately the incidence of hypertension does not decrease after transplantation[1,2] and causes shortened graft survival[2,3]. Cardiovascular complications are the most frequent causes of morbidity and mortality following renal transplantation[3,4]. Hypertension has been suggested to be a significant factor for these morbid events[5], although the nature of this relationship has not been completely defined.

There are four subdivisions of cardiovascular disease on patients with renal insufficiency, namely

Poster presentation at the 38th EDTA - ERA Congress, Vienna, June 24-27, 2001

coronary artery disease, left ventricular hypertrophy, cerebrovascular and peripheral vascular disease. Left ventricular hypertrophy begins early in the course of chronic renal failure and tends to increase with increasing dialysis time[6-8]. About three quarters of end-stage renal disease patients starting dialysis therapy have left ventricular hypertrophy, left ventricular dilatation and low fractional shortening[9]. Despite its tendency to regress after renal transplantation[10], its presence during transplantation is an adverse prognostic factor for subsequent patient survival[11].

The most important causative factor preserving LVH after transplantation is hypertension. Hypertension probably contributes not only to chronic allograft nephropathy but also to accelerated arteriosclerosis and arteriolosclerosis[5]. Blood pressure control is not always feasible and high rates of unsatisfactory blood pressure control have been reported[12]. Because of the above reasons we decided to investigate retrospectively the clinical factors that might influence arterial blood pressure after renal transplantation in an effort to achieve a better posttransplant blood pressure control.

Patients and Methods

From 1.1.1987 to 31.12.1995, three hundred ninety five renal transplantations took place in our center. We recorded retrospectively the blood pressure of 272 patients. From the study were excluded pediatric transplant patients and patients with less than six months follow up. Patients' demographic data are shown in table 1. Haemodialysis was the replacement therapy for 84.2% of the patients before transplantation and CAPD for 15.8%. The immunosuppressive agents used were steroids, azathioprine (AZA), mycophenolate mofetil (MMF), cyclosporine (CsA), antilymphocyte globulin (ALG) and the immunosuppressive protocols used are shown in table 2. The frequency of first transplantation in our sample was 92.5%, of 2nd 7.1% and 3rd 0.4%.

Table 1. Patients' demographic data

Number of patients	272
Male/female	186/86
Recipient mean age (years)	39.99±11.45
range(years)	17.13 – 64.12
Donor mean age (years)	50.05±17.65
range (years)	1.7 – 84.46
Primary renal disease	
Glomerulonephritis	128
Pyelonephritis/interstitial	36
Diabetic nephropathy	12
Polycystic Kidney disease	24
Hypertensive nephropathy	12
Other	33
Unknown etiology	27
Graft origin(LRD/CD)	172/100

Table 2. Immunosuppressive protocols from 1987 to 1995

Cortisol + AZA	0.7%
Cortisol + AZA + CsA	42.4%
Cortisol + AZA + CsA + ALG	12.9%
Cortisol + MMF + CsA	3.0%
Conversion from Aza to MMF	28.8%
CsA discontinuation	3.0%
Aza discontinuation	4.0%
Others	5.2%

Blood pressure was measured in the morning, with the patient at a sitting position. Hypertensive patients were considered to be all patients with a systolic blood pressure and/or diastolic blood pressure over 140/90 mmHg after two or more readings at different time intervals or those taking antihypertensive treatment other than diuretics. Each patient's blood pressure was recorded on the 7th, 15th, 30th posttransplant day, on the 3rd, 6th posttransplant month and on the 1st, 2nd, 3rd, 4th and 5th posttransplant year. Acute rejection episodes (AR), graft origin (LRD or CD), recipient and donor sex, recipient hypertension before transplantation, cold ischemia time, recipient and donor age, donor hypertension, kind of dialysis before transplantation and primary renal disease, were recorded too.

Repeated measures analysis of variance was used to evaluate the effect of the above parameters on the recipient's blood pressure. Acute rejection episodes, graft origin, recipient and donor sex, donor hypertension, recipient hypertension before transplantation, kind of dialysis before transplantation and primary renal disease were considered to be the factors between subjects in the analysis while cold ischemia time, recipient and donor age were the covariates. A value of $p < 0.05$ was considered statistically significant. Quantitative

Table 3. Systolic and diastolic blood pressure during 5 year follow up

Time	SBP (mmHg)	DBP (mmHg)
7th posttransplant day	153.68±18.54	94.40±10.69
15th posttransplant day	142.04±18.77	88.94±10.10
30th posttransplant day	134.37±16.16	86.26±8.95
3rd posttransplant month	132.48±15.81	84.72±9.63
6th posttransplant month	134.12±15.86	86.16±9.65
1st posttransplant year	133.58±17.35	85.50±10.00
2nd posttransplant year	131.16±15.46	83.84±8.61
3rd posttransplant year	131.64±18.2	84.72±10.20
4th posttransplant year	133.24±16.20	85.22±8.59
5th posttransplant year	134.72±14.22	84.62±8.50

results were expressed as Mean±SD. The Statistical Package for Social Sciences (SPSS for windows, version 10) was used.

Results

The SBP and the DBP of the recorded patients from the 7th postoperative day to the end of the 5th year are shown in table 3, figure 1.

The number of hypertensive patients and the frequency of hypertension during time are shown in table 4.

The multivariate analysis (table 5) showed that recipients' hypertension before transplantation had ss effect on pts' systolic (p:0.0005) and diastolic (p:0.0005) blood pressure during the entire 5 year follow up. Donor hypertension had ss effect on recipients' SBP from the 3rd pstnt month (p:0.032) to the end of the 4th year (p:0.038), while there was no significant effect on diastolic blood pressure. Acute rejection episodes had ss effect on systolic blood pressure from the first posttransplant month (p:0.003) up to the end of the 3rd year of the follow up (p:0.01). The effect of acute rejection episodes on diastolic blood pressure was ss from the 6th posttransplant month (p:0.042) to the end of the 4th year of follow up (p:0.037). Recipient age showed an ss effect on the systolic blood pressure from the first posttransplant month (p:0.002) up to the 5th posttransplant year (p:0.005) and on the diastolic blood pressure from the 3rd posttransplant year (p:0.019) to the 5th posttransplant year (p:0.008). Donor age had ss effect on systolic blood pressure from the 6th posttransplant month (p:0.014) to the 4th year of follow up (p:0.049) and to the diastolic blood pressure from the 6th posttransplant month (p:0.001) to the 5th year of follow up (p:0.024). Graft origin (LRD) had ss effect on DBP (p:0.018) only during the first posttransplant month. The kind of dialysis (HD or CAPD) had ss effect on blood pressure during the first month after renal transplantation on SBP and DBP (p:0.004 and 0.002 respectively). Primary renal disease, cold ischemia time, recipient and donor sex had no significant impact on recipients' blood pressure.

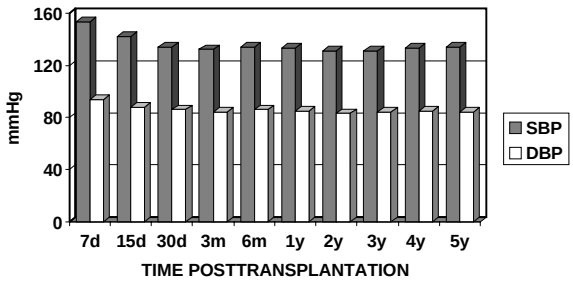

Figure 1. Systolic and diastolic blood pressure during 5 year follow up

The blood pressure load on the heart was defined by the percentage of abnormal readings during time. In tables 6 and 7 are shown the number and the incidence of blood pressure abnormal readings of patients that were hypertensives (156) or normotensives (116) before transplantation.

Discussion

The prevalence of hypertension among patients in haemodialysis and CAPD is high. This hypertension may remit or, ab initio, develop after transplantation because of pathogenic mechanisms entirely different from those responsible for hypertension present pretransplant[13]. Most of our patients were taking cortisol and cyclosporine (table

Table 4. Total number of measured patients, number of hypertensive pts, number of normotensives pts and % of hypertension

Time	total number of pts No	hypertensive pts No / %	normotensive pts No / %
7th po day	271	197 / 72.7	74 / 27.3
15th po day	271	182 / 67.2	89 / 32.8
30th po day	268	167 / 62.3	101 / 37.7
3rd po month	267	161 / 60.3	106 / 39.7
6th po month	261	168 / 64.4	93 / 35.6
1st po year	252	168 / 66.7	84 / 33.3
2nd po year	234	158 / 67.5	76 / 32.5
3rd po year	212	147 / 69.3	65 / 30.7
4th po year	180	119 / 66.1	61 / 33.9
5th po year	170	115 / 67.6	55 /32.4

2, rate 91.1%). These agents have been proved to be major factors influencing arterial blood pressure of renal allograft recipients[14,15].

Recipient hypertension before transplantation has been connected with chronic allograft nephropathy and lower graft survival[16,17]. In our work, recipient and donor hypertension before transplantation proved to be major determinants of the level of blood pressure after transplantation (table 5). The blood pressure load defined by the percentage of abnormal readings[18] during the five year follow up was greater in patients hypertensive before transplantation. Therefore the burden on the heart by the high BP probably was increased in these patients. Having in mind that BP overload is considered to be better determinant of cardiac and

Table 5. Factors that influence significantly the posttransplant blood pressure

Factor	Duration of ss influence on recipients' SBP	Duration of ss influence on recipients' DBP
Recipient hypertension before transplantation	from 1st pstnt month to 5th year (p:0.0005)	from 1st pstnt month to 5th year (p:0.0005)
Donor hypertension	from 3rd pstnt month to 4th year (p:0.032→0.038)	NS effect on DBP
Acute rejection	from 1st pstnt month to 3rd year(p:0.003→0.001)	from 5th pstnt month to 4th year(p:0.042→0.037)
Recipient age	from 1st pstnt month to 5th year(p:0.002→0.005	from 3rd pstnt year to 5th year(p:0.019→0.008)
Donor age	from 6th pstnt month to 4th year(p:0.014→0.049)	from 6th pstnt month to 5th year(p:0.001→0.024)
Graft Origin (CD or LRD)	NS effect on SBP	1st pstnt month (p:0.018)
Kind of dialysis (HD or CAPD)	1st postnt month (p:0.004)	1st postnt month (p:0.002)

Repeated measures analysis of variance
pstnt: posttransplant

Table 6. Number of blood pressure readings above the normal range (SBP>140, DBP > 90 mmHg) from the 7th po day to the 6th posttransplant month in patient's hypertensives or normotensives before transplantation.

Time		7th po d	15th po d	30th po d	3rd po m	6th po m
Patient No		156	155	153	149	144
	SBP	40	43	29	24	26
Hypertensives	DBP	10	9	6	16	11
	SBP+DBP	85	40	27	19	23
% of readings with hypertension		86.5	59.3	40.5	39.5	41.6
Patient No		99	98	98	97	96
	SBP	21	16	8	4	10
Normotensives	DBP	1	8	7	4	11
	SBP+DBP	33	16	11	7	10
% of readings with hypertension		55.5	40.8	26.5	15.4	32.2

pod: postoperative day
pom: postoperative month

Table 7. Number of blood pressure readings above the normal range (SBP>140,DBP>90 mmHg) from the 1st to the 5th posttransplant year in patient's hypertensives or normotensives before renal transplantation

Time		1st poy	2nd poy	3rd poy	4th poy	5th poy
Patient No		141	127	109	98	94
	SBP	22	20	16	15	15
Hypertensives	DBP	7	11	10	7	6
	SBP+DBP	27	17	13	8	9
% of readings with hypertension		39.7	37.8	35.8	30.6	31.9
Patient No		97	90	84	70	67
	SBP	18	14	5	8	12
Normotensives	DBP	18	15	4	7	10
	SBP+DBP	13	10	11	3	7
% of readings with hypertension		50.5	43.3	23.8	25.7	43.2

poy:postoperative year

vascular abnormalities than the casual readings of BP[19], these findings are important because it is already known that, in patients with hypertension, chronic BP overload induces myocardial and vascular damage. The increase of the blood pressure load, in the group of normotensive patients before transplantation, found at the end of the follow up, needs further analysis in correlation with donor hypertension, body weight changes and patient compliance during time[5,17,20] .

It has been demonstrated that essential hypertension disappeares after transplantation of a kidney coming from a normotensive donor[21]. This observation supports primary important of a kidney interaction between systemic mechanisms and a genetically predisposed kidney in the pathogenesis of essential hypertension. In correlation with the above, one might predict that transplantation from a hypertensive donor would result in an increased prevalence of hypertension in the allograft recipient. We found a ss influence of donor hypertension on recipients' systolic blood pressure after transplantation (table 5), while there was no effect on recipients' DBP. Probably the increased systolic arterial blood pressure correlated with the decreased graft and patient survival we recorded in patients with a donor hypertensive allograft[17]. Our findings are in agreement with other clinical and experimental studies supporting the fact that hypertensive donors can cause post-transplant hypertension[22-24].

Acute rejection episodes were found to have statistically significant effect on the levels of systolic and diastolic blood pressure (table 5). It has already been reported that acute rejection episodes, especially with major vascular components and microvascular endothelial damage, could lead to acute recurrence or development of hypertension[14]. The acute rejection effect on arterial blood pressure could possibly be connected with the lower graft and patient survival already reported[17]. It is known that acute rejection episodes are associated with chronic allograft nephropathy and one could argue that hypertension in this setting is immunologically mediated[26-27]. The separate analysis, by Opelz et al, performed on recipients who were rejection free suggested that hypertension in these patients was not a consequence of the host's alloimmune response and that arterial blood pressure was associated with long-term outcome even in the absence of rejection[3]. This observation suggested a causal relationship between hypertension and chronic renal damage but already has been proposed that, even in these cases, hypertension activates inflammatory effector mechanisms[1]. Recently has been proposed that hypertension of the recipient acts together with alloantigen – dependent factors on the expression of growth factors in the graft, responsible for the morphological changes observed in chronic allograft nephropathy, particularly the proliferation of vascular smooth muscle cells, leading to neointimal proliferation[28,29].

Recently, in addition to hypertension, other non-immunological factors such as age, gender and race have been implicated as risk factors for chronic graft loss[30]. Our multivariate analysis showed that recipient and donor age (Table 5) had statistically significant impact on recipient's blood pressure while sex had no impact on it.

Recurrent primary renal disease is an unusual cause of posttransplant hypertension although recurrent FSG and uremic hemolytic syndrome have been associated with quite severe hypertension[31]. In our work primary renal disease was not found to affect posttransplant blood pressure and this is in agreement with the work of Warholm et al[32].

We found that the kind of dialysis before transplantation, namely CAPD, was associated with ss lower blood pressure (systolic and diastolic) during the first postoperative month after transplantation. Possibly the ss lower levels of blood pressure of patients on CAPD are related with the ss greater loss of body weight when compared with the patients on haemodialysis[33]. Graft origin (LRD) was found to have a significant effect on recipients' diastolic blood pressure during the 1st pstnt month. This finding needs further analysis. The only comment we can do is the fact that our LRDs were ss older than the cadaveric donors.

The complex nature of post-transplant hypertension has made it difficult to discern if its occurrence is the cause or the consequence of chronic allograft disfunction. The possibility remains that the two processes are not mutually exclusive and coexist. However, post-transplant hypertension has a negative impact on long-term allograft survival[3,34-36]. We already know that acute rejection episodes and recipient and donor hypertension cause lower graft and patient survival[17] and higher levels of blood pressure. According to these we should have lower levels of blood pressure and lower frequency of hypertension with advancing time due to hypertensive graft and patient loss. The arterial blood pressure and the frequency of hypertension noticed in our patients was higher in the first postransplant month after which it was fairly stable during the five year follow up (tables 3 and 4, figure 1). The higher levels of blood pressure during the

first posttransplant month is possibly due to fluid overload during the transplant procedure, graft dysfunction, acute rejection episodes, steroid dose and higher cyclosporine levels. The stable percentage of hypertensive patients after the first posttransplant month is connected with the appearance of new hypertensive patients (tables 6 and 7) and the most probable factors implicated are immunossupression, chronic allograft nephropathy and body weight changes. In the same setting we can explain the loss of influence of acute rejection on post-transplant hypertension after the forth year of follow up.

The development of uremic cardiomyopathy in patients with end stage renal disease is explained by hypertension, anemia, hypoalbuminemia, hyperparathyroidism, diabetes mellitus and uremia[37,38]. All manifestations of uremic cardiomyopathy (LV hypertrophy, LV dilatation, systolic dysfunction) are improved by renal transplantation, particularly systolic dysfunction[10]. In spite of this, LVH is common in these patients. Hypertension is among the factors that perpetuate it, in the process of transplantation[39]. In addition antirejection therapy (corticosteroids and cyclosporine), could also be involved in the development of LVH[40,41]. Several experimental studies have documented the growth-stimulating effect of angiotensin II on myocardial cells[42,43]. Cardiac complications are the main cause of death in renal transplant recipients and left ventricular hypertrophy is considered a major idependent risk factor[44-46].

For many years, we considered appropriate to maintain the arterial blood pressure at the level of 140/90 mmHg or lower. According to the Sixth Report of the Joint National Commission, optimal blood pressure is considered to be < 120/80 mmHg based on an average of two or more recordings[47]. According to this, the frequency of hypertension after transplantation is much higher than that reported by us.

We have already reconsidered our policy about the blood pressure levels that must be attained. Most antihypertensive agents seem to be effective in lowering blood pressure in renal transplant recipients and no single antihypertensive agent has been found to be more efficacious than the others[14]. Drug toxicities and interactions, recipient and donor hypertension history, age, as well as post-transplant pathology[4,48] must guide the use of the antihypertensive agents. Recently it was reported that angiotensin converting enzyme inhibitors decrease left ventricular mass in renal transplantation patients with hypertension and LVH and ACE gene polymorphism may predict the beneficial effect of the therapy[49]. The role of angiotensin II type 1 inhibitors is not known. Immunosuppression protocols that minimize the rejection episodes, the use of steroids and cyclosporine, combined with diet, exercise and weight loss may help to reduce the prevalence of posttransplant hypertension. Perhaps we have to inform aged patients or patients with pre-transplant hypertension when there is a case of hypertensive kidney and ask their consent or avoid them.

ΠΕΡΙΛΗΨΗ

***Γ. Βέργουλας, Γρ. Μυσερλής, Φ. Καρασαββίδου, Γ. Ιμβριος, Ι. Κατσάρα, Ν. Γεωργιλάς, Μ. Λεοντσίνη, Α. Αντωνιάδης*. Η αρτηριακή πίεση μετά τη μεταμόσχευση νεφρού. Εμπειρία ενός κέντρου.** Ιπποκράτεια 2002, 6 (2): 62-70

Η υπέρταση μετά τη μεταμόσχευση είναι πολυπαραγοντική και έχει συνδεθεί με αυξημένα ποσοστά καρδιαγγειακών επεισοδίων και ελαττωμένη επιβίωση του μοσχεύματος. Στην εργασία αυτή μελετήθηκαν οι κλινικοί παράγοντες που μπορεί να επηρεάσουν την αρτηριακή πίεση μετά τη μεταμόσχευση. Μεταξύ 1987 και 1995 μελετήθηκε αναδρομικά η αρτηριακή πίεση 272 ασθενών (186 άνδρες) που έλαβαν νεφρικό μόσχευμα (172 από ζωντανό δότη). Η μέση ηλικία των ασθενών (pts) ήταν 40 έτη (διακύμανση 17 - 64). Υπήρχε τουλάχιστον έξη μηνών παρακολούθηση με λειτουργούν νεφρικό μόσχευμα. Η αρτηριακή πίεση κάθε ασθενούς καταγράφηκε την 7η, 15η, 30η pstnt ημέρα, τον 3ο, 6ο pstnt μήνα και το 1ο, 2ο, 3ο, 4ο και 5ο pstnt έτος. Μελετήθηκε η επίδραση των επεισοδίων οξείας απόρριψης (AR), της προέλευση του μοσχεύματος (από συγγενή ζωντανό δότη ή πτωματικό), του φύλου του δότη – λήπτη, της υπέρτασης του λήπτη πριν τη μεταμόσχευση, της υπέρτασης του δότη, του χρόνου ψυχρής ισχαιμίας, της ηλικίας του λήπτη και του δότη, του είδος κάθαρσης πριν από τη μεταμόσχευση και της πρωτοπαθούς νεφρικής νόσου στη συστολική (SBP) και διαστολική αρτηριακή πίεση (DBP) του λήπτη στη διάρκεια του χρόνου παρακολούθησης. Έγινε πολυπαραγοντική στατιστική ανάλυση επαναλαμβανομένων μετρήσεων. Η SBP and DBP ήταν 153.68 ±18.54 / 94.40±10.69 mmHg, 142.04±18.77/88.96± 10.10 mmHg, 134.37 ±16.16 /86.26±8.95 mmHg, 132.48±15.81/84.72±9.63 mmHg, 134.12 ±15.86 / 86.16 ±9.65 mmHg, 133.58±17.35/85.50±10.00 mmHg, 131.16±15.46/ 83.84± 8.61 mmHg, 131.64±18.2/84.72±10.28 mmHg, 133.24± 16.20/ 85.22±8.59 mmHg, 134.72±14.22/84.62±8.50 mmHg την 7η, 15η, 30η pstnt ημέρα, τον 3ο, 6ο pstnt

μήνα και το 1ο, 2ο, 3ο 4ο and 5ο pstnt έτος αντίστοιχα. Η υπέρταση του λήπτη πριν από τη μεταμόσχευση είχε στατιστικά σημαντική (ss) επίδραση στην SBP (p:0.0005) και DBP (p:0.0005) των ασθενών κατά την παρακολούθηση των 5 ετών. Η υπέρταση του δότη είχε ss επίδραση στη SBP από τον 3ο pstnt μήνα (p:0.032) μέχρι το 4ο pstnt έτος (p:0.038). Η δράση της AR στη SBP ήταν ss από τον 1ο pstnt μήνα (p:0.003) μέχρι το τέλος του 3ου έτους της παρακολούθησης (p:0.01) και στη DBP από τον 6ο pstnt μήνα (p:0.042) μέχρι το 2ο pstnt έτος (p:0.037). Η προέλευση του μοσχεύματος (LRD v CD) είχε ss επίδραση στη DBP (p:0.018) κατά τον 1ο pstnt μήνα ενώ το είδος της κάθαρσης (HD v CAPD) είχε ss επίδραση στη SBP και DBP κατά τη διάρκεια του 1st pstnt μήνα (p:0.004 και p:002 αντίστοιχα). Η ηλικία του δότη είχε ss επίδραση στη SBP από τον 6ο pstnt μήνα (p:0.014) μέχρι το 4ο έτος της παρακολούθησης (p:0.049) και στη DBP από τον 6ο pstnt μήνα (p:0.001) μέχρι το 5ο έτος της παρακολούθησης (p:0.024). Η ηλικία του λήπτη είχε ss επίδραση στη SBP από τον 1ο pstnt μήνα (p:0.002) μέχρι το 5th pstnt έτος (p:0.005) και στη DBP από το 3ο pstnt έτος (p:0.019) μέχρι το 5ο pstnt έτος (p:0.008). Συμπερασματικά, οι κλινικοί παράγοντες με την πλέον σημαντική επίδραση στην αρτηριακή πίεση μετά τη μεταμόσχευση είναι η υπέρταση του δότη και του λήπτη πριν από τη μεταμόσχευση, η ηλικία δότη και λήπτη κατά τη μεταμόσχευση και τα επεισόδια οξείας απόρριψης.

REFERENCES

1. Zeier M, Mandelbaum A, Ritz E. Hypertension in the transplanted patient. Nephron 1998; 80: 257-268
2. Peschke B, Scheuerman EH, Geiger H, Bolscher S, Kachel HG, Lenz T. Hypertension is associated with hyperlipidemia, coronary heart disease and chronic graft failure in kidney transplant recipients. Clin Nephrol 1999; 51: 290-295
3. Opelz G, Wujciak T, Ritz E, et al. Association of chronic kidney graft failure with recipient blood pressure. Kidney Int 1998; 53: 217-222
4. Kasiske BL. Cardiovascular disease after renal transplantation. Seminars in Nephrology 2000; 20: 176-187
5. Kasiske BL. Risk factors for accelerated arteriosclerosis in renal transplant recipients. Am J Med 1988; 84: 985-992
6. Levin A, Singer J, Thompson CR, Ross H, Lewis M. Prevalent left ventricular hypertrophy in the dialysis population: identifying opportunities for intervention. Am J Kidney Dis 1996; 27: 347-354
7. Harnett JS, Kent GM, Foley RN, Parfrey PS. Cardiac function and haematocrit level. Am J Kidney Dis 1995; 25(Suppl 1): S3 – S7
8. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE. Long – term evolution of cardiomyopathy in dialysis patients. Kidney Int 1998; 54: 1720 – 1725
9. Foley RN, Parfrey PS, Harnett JD, et al. Clinical and echocardiografic disease in patients starting end-stage renal disease therapy. Kidney Int 1995; 47: 186-192
10. Parfrey PS, Harnett JD, Foley RN, et al. Impact of renal transplantation on uremic cardiomyopathy. Transplantation 1995; 60: 908 – 914
11. McGregor E, Stewart G, Rodger RSC, Jardine AG. Early echocardiografic changes and survival following renal transplantation. Nephrol Dial Transplant 2000; 15: 93 – 98
12. Stewart GA, Tan CC, Rodger RSC, et al. Graft and patient survival following renal transplantation: new targets for blood pressure control. In: Timio M, Wizeman V, Venanzi S. Eds. Cardionephrology 5. Consenza: Editoriale Bios, 1999; 357-361
13. Luke RG and Curtis JJ. Biology and treatment of transplant hypertension. In Laragh JH and Brenner BM (eds), Hypertension : Pathophysiology, diagnosis and management. Raven Press Ltd, NY, 1995; pp: 2471-2483
14. Vergoulas G. Hypertension and renal transplantation. Hippokratia 2001; 5: 51-60
15. Mimram A, Mourad G, Ribstein J, Halimi JM. Cyclosporine – Associated Hypertension. In Laragh JH and Brenner BM (eds). Hypertension: Pathophysiology, Diagnosis and Management. 2nd ed, Raven Press, New York, 1995; pp 2459 - 2469
16. Frei U, Schindler R, Wieters D, Groven V, Brunkhost R, Koch KM. Pre-transplant hypertension: a major risk factor for chronic progressive renal allograft dysfunction. Nephrol Dial Transplant 1995; 10: 1206-1211
17. Vergoulas G, Myserlis Gr, Leontsini M, et al. Influence of clinical parameters on five-year patient and graft survival after first renal transplantation. Transplantation 2002; 74(4): 448(abstr)
18. Zacharias PK, Sheps SG, Ilstrup DM, et al. Blood pressure load, a better determinant of hypertension. Mayo Clin Proc 1988; 63: 1085-1091
19. White WB. Blood pressure load and target organ effects in patients with essential hypertension. J Hypertens 1991; 9 (Suppl 8): S39-S41
20. Vergoulas G. Quality of life in patients with renal transplantation. Hippokrartia 2002; 6 (Suppl 1): 91-98
21. Curtis JJ, Luke RG, Harriet ChB, et al. Remission of essential hypertension after renal transplantation. J Am Soc Nephrol 2000; 12: 2404-2412
22. Uber A and Retting R. Pathogenesis of primary hypertension – Lessons from renal transplanation studies. kidney Int 1996; 55 (Suppl 55): S-42 – S-45,
23. Guidi E, Bianchi G, Rivolta E, et al. Hypertension in man with a kidney transplant: Role of familial vs other factors. Nephron 1985; 41: 14-21,
24. Strandgaard S, Hansen U. Hypertension in renal allograft recipients may be conveyed by cadaveric

kidneys from donors with subarachnoid haemorrhage. Br Med J 1986; 292: 1041-1044
25. Basadonna GP, Matas AJ, Gillingham KJ, et al. Early versus late acute renal allograft rejection: Impact on chronic rejection. Transplantation 1993; 55:993-995
26. Surmani N, Cacciarrelli TV, Georgi B. Contribution of acute rejection to renal allograft loss from chronic rejection. Transplant Proc 1993; 25:2259-2260
27. Luft FC, Haller H. Hypertension – induced renal injury: Is mechanically mediated interstitial inflammation involved? Nephrol Dial Transplant 1995; 10: 9-11
28. Schindler R, Tanriver Y, Tulius S, Neuhaus P, Frei U. Chronic allograft nephropathy in the rat is aggravated by hypertension and improved by ACE-inhibition (abstaract). European Society of Organ Transplantation, Oslo, 1999
29. Schindler R, Tanriver Y, Frei U. Hypertension and allograft nephropathy – cause, consequence, or both? Nephrol Dial Transplant 2000; 15: 8 – 10
30. Brenner BM, Milford EL. Nephron underscoring: A programmed cause of chronic renal allograft failure. Am J Kidney Dis 1993; 21(Suppl 2): 66-72
31. Remuzzi G, Bertoni T. Renal vascular and thrombotic effects of cyclosporine. Am J Kidney Dis 1989; 13: 261-272
32. Warholm C, Wilczek H, Petterson E. Hypertension two years after renal transplantation. Transpl Int 1995; 8:286-292
33. Vergoulas G, Miserlis Gr, Gakis D, et al. Body weight changes of patients on CAPD or HD during the immediate kidney posttransplant period. Sixty first Scientific Meeting of Greek Society Of Nephrology, Athens, 2001 March, abstract book pp 75-76
34. Kasiske BL. Possible causes and consequences of hypertension in stable renal transplant patients. Transplantation 1987; 44:639-643
35. Fernandez – Fresnedo G, Palomar R, Escallada R, et al. Hypertension and long – term allograft survival: effect of early glomerular filtration rate. Nephrol Dial Transplant 2001; 16 (Suppl 1): 105 –109
36. Sanders CE, Curtis JJ. Role of hypertension in chronic renal allograft dysfunction. Kidney Int 1995; 48 (Suppl 52): S-43 – S-47
37. Harnett JD, Foley RN, Kent GM, Barre PE, Murrey DC, Parfrey PS. Congestive heart failure in dialysis patients: prevalence, incidence, prognosis and risk factors. Kidney Int 1995; 47: 884-890
38. Weisenee D, Low-Friedrch I, Richle M, Bereiter – Hahn J, Schoeppe W. In vitro approach to " uremic cardiomyopathy ". Nephron 1993; 65: 392
39. Lipkin GW, Tucker B, Giles M, Raine AE. Ambulatory blood pressure and left ventricular mass in cyclosporin- and non-cyclosporine –treated renal transplant recipients. J Hypertension 1993; 11: 439-442
40. Fishel RS, Eisenberg S, Shai S-Y, Redden RA, Bernstein KE, Berk KC. Glucorticoids induce angiotesnin –converting enzyme expression in vascular smooth muscle. Hypertension 1995; 25: 343-349
41. Ventura HO, Lavie CJ, Messerli FH, et al. Cardiovascular adaptation to cyclosporine-induced hypertension. J Hum Hypertens 1994; 8: 233-237
42. Sadoshima JI, Izumo S. Moelcular characterization of angiotensin II – induced hypertrophy of cardiac myocites and hyperplasia of cardiac fibroblasts: Critical role of the AT1 receptor subtype. Circ Res 1993; 73: 413-423
43. Brilla CG, Zhou G, Matsubara L, Weber KT. Collagen metabolism in cultured adult rat cardiac fibroblasts: response to angiotensin II and aldosterone. J Mol Cell Cardiol 1994; 26: 809-820
44. First. Long - term complications after renal transplantation. Am J Kidney Dis 1993; 22: 477-486
45. US Renal Data System. USRDS: Annual Data Report. The National Institutes of Health, National Institute of Diabetes and Digestive and Kidneey Diseases: Causes of death. Am J Kidney Dis 1994; 24 (Suppl 2): S88-S95
46. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiografically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990; 322: 1561-1566
47. The sixth report of the Joint National Committee on prevention, detection evaluation and treatment of high blood pressure. Ann Int Med 1997, 157: 2413 – 2446
48. Vergoulas G, Miserlis Gr, Papanikolaou V, et al. Angiotensin II type 1 receptor antagonists reduce proteinuria of hypertensive renal transplant recipients. Hippokratia 2001; 4: 165 – 171
49. Hernandez D, Lacalzada J, Salido E, et al. Regression of left ventricular hypertrophy by lisinopril after renal transplantation: Role of ACE gene polymorhpism. Kidney Int 2000; 58: 889-897

Corresponding author:
Vergoulas G
Associate Director
53 Alkminis str
542 49 Thessaloniki
Greece
tel.: +302310-302.311
e-mail: geover@otenet.gr

Αλληλογραφία
Γ. Βέργουλας
Αλκμήνης 53
Θεσσαλονίκη, 542 49
τηλ.: 2310.302.311
e-mail: geover@otenet.gr