Apolipoprotein(a) Phenotype–Associated Decrease in Lipoprotein(a) Plasma Concentrations After Renal Transplantation

Florian Kronenberg, Paul König, Karl Lhotta, Dietmar Öfner, Christoph Sandholzer, Raimund Margreiter, Elisabeth Dosch, Gerd Utermann, Hans Dieplinger

Renal transplantation (RTX) is the best form of renal replacement therapy and should be considered as the treatment of choice for end-stage renal disease (ESRD). In the last 20 years a decrease in deaths by infections has occurred because of the improved management of immunosuppressive and antibiotic therapy. At the same time cardiovascular morbidity has become the most common cause of death in this group of patients. This has been ascribed to the development of hyperlipidemia after RTX (for review, see Reference 2).

Recently, increased plasma concentrations of lipoprotein(a) [Lp(a)] were reported in ESRD patients. The distribution of Lp(a) in the general Caucasian population is highly skewed and extremely broad. Lp(a) levels range from less than 0.1 to more than 100 mg/dL, with a mean and median concentration of approximately 15 and 8 mg/dL, respectively.

The apolipoprotein(a) [apo(a)] gene locus on chromosome 6q2.6-2.7 has been described as the major gene controlling Lp(a) plasma concentrations. More than 30 alleles at this highly polymorphic locus determine a protein size polymorphism of apo(a). An inverse relation exists between Lp(a) plasma concentrations and the molecular weight of the Lp(a) glycoprotein. In healthy Caucasians, about half of the variability in Lp(a) levels can be explained by the apo(a) size polymorphism.

Besides the variation at the apo(a) gene locus, other factors that influence Lp(a) plasma levels have been described. Increased Lp(a) plasma concentrations have been described in acute-phase reactions as well as in case-control studies have found an association between high Lp(a) plasma levels and coronary heart disease (CHD). However, this association was not confirmed by the Helsinki Heart Study and the Physicians’ Health Study. Recently, a multipopulation study showed that the apo(a) size polymorphism determines the risk for CHD through its effect on Lp(a) plasma concentrations.

Some studies described elevated plasma Lp(a) concentrations in patients undergoing hemodialysis. At the same time a prospective study has shown that Lp(a) is an independent risk factor for cardiovascular disease in hemodialysis patients. In one of these reports we have demonstrated that the elevation of Lp(a) is not due to differences in isoform frequencies. The elevated plasma Lp(a) concentrations in ESRD patients are therefore believed to be nongenetic and secondary to the disease.

To further investigate the origin of the elevated Lp(a) plasma concentrations in ESRD, we observed a rapid normalization of Lp(a) levels from an average concentration of 25.9±28.7 mg/dL before to 17.9±25.5 mg/dL 3 weeks after renal transplantation (P<.0001). Only patients with high-molecular-weight phenotypes had a significant decrease in Lp(a) plasma concentrations. This study demonstrates the nongenetic origin of elevated Lp(a) concentrations in ESRD patients, which is obviously caused by the disease. It further confirms a phenotype-associated elevation of Lp(a) concentrations in ESRD. (Arterioscler Thromb. 1994;14:1399-1404.)

Key Words • lipoprotein(a) • apolipoprotein(a) phenotype • atherosclerosis • end-stage renal disease • immunosuppression

Received September 28, 1993; revision accepted March 10, 1994.

From the Institute of Medical Biology and Human Genetics, Innsbruck (F.K., C.S., G.U., H.D.), and the Innsbruck University Hospital, Clinic of Internal Medicine, Department of Clinical Nephrology (P.K., K.L.), Department of Transplant Surgery (D.O., R.M.), and Clinic of Ophthalmology (E.D.) (Austria).

A preliminary report of this data was presented at the Second International Conference on Lipoprotein(a), New Orleans, La, November 12-14, 1992.

Correspondence to Gerd Utermann, MD, Institute of Medical Biology and Human Genetics, University of Innsbruck, Schöpfstr 41, A-6020 Innsbruck, Austria.

© 1994 American Heart Association, Inc.
prospective study the effect of RTX on Lp(a) plasma concentrations in patients with ESRD in a longitudinal study design. Because of the large patient group, it was possible to analyze the changes after transplantation in relation to apo(a) phenotypes.

Methods

Patients and Control Subjects

A total of 156 consecutive patients undergoing RTX for ESRD were initially included in this prospective study. In all patients the operative procedure was performed by the same team of surgeons. The study group consisted of 85 men and 71 women with a mean age of 43.0±12.4 (range, 18 to 66) years. The mean duration of dialysis before RTX was 51.0±42.9 (range, 1 to 230) months. The cause of chronic renal failure was chronic glomerulonephritis in 71 patients, chronic pyelonephritis in 16, polycystic kidney disease in 15, diabetic nephropathy in 25, and other reasons in 15; the origin was unknown in 14. Only patients in good physical condition were considered for RTX. Two of the 156 patients were excluded from further analysis, one suffering a fatal stroke during the first postoperative week and the other intraoperatively showing such a marked atherosclerosis that graft vessels could not be linked to the patient’s iliac vessels.

The 256 control subjects were free of renal and liver diseases and were selected from a group of consecutive blood donors from Tyrol. RTX patients and control subjects were comparable with regard to sex (54.5% versus 59.1% men, P=.37) but not to age distribution (45.0±12.4 versus 37.8±11.5 years, P<.001).

Drug Therapy

Treatment of patients before transplantation included calcitriol, calcium acetate, or calcium carbonate and in some cases phosphate binders such as aluminum hydroxide to avoid renal osteodystrophy. For treatment of arterial hypertension, 58% of patients before and 81% of patients after RTX received treatment with β-blockers, calcium antagonists, or angiotensin-converting enzyme inhibitors, alone or in combination. No patient received lipid-lowering drugs before or during the observation period after RTX. Postoperative immunosuppressive therapy consisted of a triple-drug regimen (combination of cyclosporin A [CsA], azathioprine, and prednisone) in 145 patients. Nine patients were treated with a double-drug therapy (azathioprine and prednisone) for the first 10 days because of insufficient urine production during the first postoperative days. Seven patients received only CsA with prednisone during the observation period for a short time of 5 to 32 days.

Triple-Drug Therapy

For the first two administrations, CsA was set at 3 mg/kg body wt per day. When initial urine production was less than 50 mL/h, CsA dosage was kept at 3 mg/kg body wt per day to aim for a whole-blood level of approximately 100 ng/mL (measured with the monoclonal Sandimmun radioimmunoadsay kit from Sandoz). When urine production was greater than 50 mL/h, CsA dosage was raised to 3 mg/kg body wt per day and adjusted accordingly to aim for a whole-blood level of approximately 150 to 200 ng/mL.

Immediately before revascularization, 500 mg methylprednisolone was administered and reduced stepwise to 250, 125, and 100 mg at 24-hour intervals thereafter. Then patients were switched from methylprednisolone to 100 mg prednisone, and the dosage was subsequently reduced by 10 mg/d to 25 mg/d. In the patient group with low initial urine production, prednisone dosage was reduced at 2-day intervals. This dosage was maintained until postoperative day 21, whereupon it was further reduced by 2.5 mg at 2-week intervals to a maintenance dose of 5 to 10 mg/d.

Azathioprine was administered postoperatively at 1 to 1.5 mg/kg body wt per day with regular control of leukocytes and thrombocytes.

Samples

Baseline values of all measured parameters were obtained from a blood sample taken after a 7- to 12-hour preoperative fasting period. The second and third samples were taken after a 12- to 14-hour overnight fast 1 and 3 weeks after RTX, respectively. Additionally, blood samples were taken monthly for a mean period of 14.5 months from a subgroup of 29 patients. Plasma was obtained by addition of heparin followed by low-speed centrifugation. Samples from control subjects and patients were frozen under the same conditions at −80°C and stored for the same time period. All samples withdrawn during the first 3 postoperative weeks were analyzed no later than 6 weeks after transplantation in one assay. Twenty-nine patients with long observation periods (mean, 14.5 months) had all their samples (from day 0 to the end of observation) additionally analyzed in one assay at the end of the study. In these patients the initially measured values of the first 3 weeks were not significantly different from the repeated measurement of the same time period.

Laboratory Procedures

Lp(a) quantification was performed with a double-antibody enzyme-linked immunosorbant assay (ELISA) using an affinity-purified polyclonal rabbit anti-apo(a) antibody for coating and the horseradish peroxidase-conjugated monoclonal antibody 1A2 for detection.36 This antibody does not cross-react with plasminogen. Lp(a)-positive serum from Immuno served as a standard. Lp(a) concentrations are expressed as total Lp(a) lipoprotein mass. Lp(a) values 1 and 3 weeks after RTX were corrected for the individual total protein changes after transplantation, therefore taking a possible effect of postoperative hemodilution or hemococoncentration into account. This is needed to distinguish the metabolic effect of kidney transplantation from that of hemodilution or hemococoncentration. The correction was performed by the formula

$$Lp(a)_{cor} = \frac{TP_r \times Lp(a)_{tot}}{TP_p}$$

where Lp(a)_{cor} is corrected Lp(a) value after transplantation, Lp(a)_{tot} is measured Lp(a) value after transplantation, TP_r is total protein before transplantation, and TP_p is total protein after transplantation.

Apo(a) phenotyping was performed with sodium dodecyl sulfate–polyacrylamide gel electrophoresis of plasma under reducing conditions followed by immunoblotting using the monoclonal antibody 1A2 for the detection of apo(a) phenotypes.37 Isoforms that did not exactly comigrate with the standards were binned with the closest respective isoform,38 apo(a) phenotypes of plasma from patients and control subjects were determined at the same time and under identical conditions.

ApoB plasma concentrations were measured with a double-antibody ELISA using the same affinity-purified polyclonal antibody against apoB for coating and in a peroxidase-labeled form for detection.17 Plasma concentrations of total and high-density lipoprotein cholesterol (HDL) cholesteral, triglycerides, total protein, and creatinine were determined using commercially available kits from Boehringer Mannheim. Creatinine clearance was calculated from serum creatinine by the formula of Cockcroft and Gault.39

Statistical Analysis

A χ² test was used to compare apo(a) phenotype frequencies between RTX patients and control subjects considering all types. This analysis was performed applying a likelihood ratio χ² test because many empty cells occurred in both groups. In
addition, Pearson’s \( \chi^2 \) test was applied on two subgroups formed by combining subjects with low-molecular-weight (LMW) and high-molecular-weight (HMW) phenotypes as described.\(^{34}\) As in our previous work, we decided a priori to divide apo(a) phenotypes into two subgroups according to the molecular weight of isoforms. The LMW group includes all subjects who had at least one of the isoforms F, B, S1, or S2. The HMW group comprised all subjects with only S3 or S4 isoforms or with a null type (0). Because of the highly skewed distribution of Lp(a) plasma concentrations in patients and control subjects, the nonparametric Wilcoxon rank sum test was performed to compare Lp(a) levels between the RTX and control groups. The paired Wilcoxon test was used for differences in Lp(a) plasma concentrations in the RTX groups before and after RTX. The paired \( t \) test was used to examine differences in Lp(a) plasma concentrations in the RTX groups at various times and to test whether changes in renal parameters were dependent on apo(a) isoforms.

**Results**

Lp(a) concentrations and apo(a) phenotypes were determined in 154 patients with ESRD undergoing RTX and in a control group. As in previous studies Lp(a) levels were significantly higher in the ESRD patients than in control subjects (25.9±28.7 versus 18.4±22.8 mg/dL, \( P=.0012 \)).

The frequency distribution of the apo(a) isoforms in patients undergoing RTX was also significantly different from the control group (\( P<.0001 \)). This difference was caused by fewer LMW phenotypes in the RTX group (14.9% versus 25.8%, \( P<.01 \)) (Table 1). The difference in the apo(a) isoform frequencies between patients and control subjects does not explain the differences in Lp(a) concentrations but is in the opposite direction. Isoforms that in the general population (control subjects) are associated with high Lp(a) concentrations were underrepresented in the patients.

Table 2 shows the mean plasma concentrations of Lp(a) in a control group and in the RTX group before and after RTX. A significant decrease to 17.9±25.5 mg/dL was observed 3 weeks after transplantation (\( P<.0001 \)). Patients with HMW phenotypes showed significantly higher Lp(a) levels before RTX compared with the phenotype-matched control groups (S3, \( P=.0011 \); S4, \( P<.0001 \); S3S4, \( P<.005 \); all HMW phenotypes, \( P<.0001 \)). However, Lp(a) levels in patients with LMW phenotypes were similar to those in control subjects (S2, \( P=.27 \); all LMW phenotypes, \( P=.63 \)).

Three weeks after transplantation, patients with HMW phenotypes showed a significantly larger decrease in Lp(a) than patients with LMW phenotypes (\( P<.0001 \)) (Table 2 and Figure). Lp(a) plasma concentrations of patients with the most common HMW phenotypes, S3, S4, and S3S4, had decreased by 45.2%, 46.2%, and 36.3%, respectively, from the baseline values before transplantation. Lp(a) concentrations in the LMW isoform group S2 remained unchanged. Lp(a) values measured 3 weeks after RTX in all phenotypic groups were almost identical to those of the respective subgroup in the control subjects (S2, \( P=.29 \); S3, \( P=.10 \); S4, \( P=.63 \); S3S4, \( P=.31 \); all phenotypes, \( P=.85 \)) (Table 2).

The apo(a) size polymorphism explained 39.0% of the variability in Lp(a) levels before transplantation, and this percent increased to 58.2% after transplantation, reflecting the decrease in the nongenetic effect on Lp(a) level.

In a subgroup of 29 patients with different isoforms, Lp(a) plasma concentrations were followed for 14.5±6.4 months after transplantation. No significant increase was observed when the third-week (15.2±17.9 mg/dL) and 15th-month (15.9±14.6 mg/dL) values were compared.

We also measured the lipoprotein parameters total cholesterol, HDL cholesterol, triglycerides, and apoB as well as the renal parameter creatinine and calculated creatinine clearance (Table 3). The plasma concentrations of cholesterol, which were in the normal range in the patients before RTX, decreased significantly 1 week after RTX and returned to preoperative values 3 weeks after RTX. The HDL cholesterol changes were similar, with a level 3 weeks after RTX being even higher than the preoperative level. Concentrations of apoB and triglycerides did not change. Changes in renal parameters reflected the restored renal function. They were independent of the respective apo(a) isoform (\( P=.87 \) for creatinine; \( P=.22 \) for creatinine clearance) and did not correlate with the decrease in Lp(a) plasma concentrations.

**Discussion**

Patients with ESRD and patients after RTX have an increased risk for CHD.\(^{1,40}\) During recent years Lp(a)

---

**Table 1. Frequencies of Apolipoprotein(a) Phenotypes in Control Subjects and Renal Transplant Patients**

| Phenotype   | Controls | | | Patients | | |
|-------------|----------|--------|--------|----------|--------|
| B           | 2        | 0.8    | ...    | 4        | 0.7    |
| S1          | 2        | 0.8    | ...    | 1        | 0.8    |
| S2          | 27       | 10.5   | 16     | 32       | 12.5   |
| S3          | 32       | 12.5   | 25     | 66       | 19.0   |
| S4          | 94       | 36.7   | 64     | 182      | 41.6   |
| 0           | 16       | 6.2    | 20     | 16       | 13.0   |
| FS4         | 1        | 0.4    | ...    | 1        | 0.6    |
| BS1         | ...      | ...    | 1      | 1        | 0.6    |
| BS3         | 2        | 0.8    | ...    | 1        | 0.6    |
| BS4         | ...      | ...    | 1      | 1        | 0.6    |
| S1S3        | ...      | ...    | 1      | 1        | 0.6    |
| S1S4        | ...      | ...    | 2      | 1        | 1.3    |
| S2S3        | 6        | 3.3    | ...    | 1        | 1      |
| S2S4        | 28       | 10.2   | 2      | 2        | 1.3    |
| S3S4        | 48       | 18.8   | 22     | 2        | 15.4   |

LMW* indicates low-molecular-weight phenotypes; HMW, high-molecular-weight phenotypes. Likelihood ratio \( \chi^2 \) test of LMW vs HMW phenotype subgroups: \( x^2=6.66, df=1, P<.01 \).

*All subjects with any of the isoforms F, B, S1, or S2.
†All subjects with only S3 or S4 isoforms or with null type.
Apo indicates apolipoprotein; RTX, renal transplantation; LMW, low-molecular-weight phenotypes; and HMW, high-molecular-weight phenotypes. Values are mean±SD [median] and expressed as milligrams per deciliter.

<table>
<thead>
<tr>
<th>Apo(a) Type*</th>
<th>n</th>
<th>Before RTX</th>
<th>1 Week After RTX</th>
<th>3 Weeks After RTX</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>S2</td>
<td>16</td>
<td>35.1±35.9[15.3]</td>
<td>35.9±39.8[14.2]</td>
<td>36.2±33.5[22.7]</td>
<td>42.9±24.6<a href="n=27">41.3</a></td>
</tr>
<tr>
<td>S3</td>
<td>25</td>
<td>29.0±26.3[16.9]</td>
<td>25.5±23.0[16.0]</td>
<td>15.9±12.9[11.1]</td>
<td>13.4±17.6<a href="n=32">5.1</a></td>
</tr>
<tr>
<td>S4</td>
<td>64</td>
<td>21.0±18.7[15.6]</td>
<td>14.2±15.9[9.1]†#</td>
<td>11.3±14.4[6.2]†#</td>
<td>8.5±7.2<a href="n=94">6.9</a></td>
</tr>
<tr>
<td>S3S4</td>
<td>22</td>
<td>35.8±29.2[25.9]</td>
<td>29.3±25.0[16.1]†</td>
<td>22.8±28.2[11.6]†</td>
<td>16.4±17.3<a href="n=48">8.1</a></td>
</tr>
<tr>
<td>0</td>
<td>20</td>
<td>1.7±1.5[1.6]†</td>
<td>1.6±0.9[1.5]</td>
<td>1.6±0.9[1.4]</td>
<td>0.9±0.4<a href="n=16">0.9</a></td>
</tr>
<tr>
<td>LMW††</td>
<td>23</td>
<td>47.4±42.0[42.8]</td>
<td>41.4±37.7[35.1]††</td>
<td>44.6±40.6[30.9]†</td>
<td>40.6±29.5<a href="n=66">37.1</a></td>
</tr>
<tr>
<td>HMW‡‡</td>
<td>131</td>
<td>21.6±23.1[12.2]‡</td>
<td>16.7±19.8[9.1]‡#</td>
<td>12.3±16.7[6.2]‡#</td>
<td>10.6±13.0<a href="n=190">6.0</a></td>
</tr>
<tr>
<td>All</td>
<td>154</td>
<td>25.9±28.7[14.6]‡</td>
<td>20.9±25.4[10.1]#</td>
<td>17.9±25.5[7.6]**</td>
<td>18.4±22.8<a href="n=256">8.2</a></td>
</tr>
</tbody>
</table>

$P<.001$. Two recent sib-pair linkage studies have shown that the apo(a) gene determines more than 90% of the variation in plasma Lp(a) concentrations in healthy Caucasian populations.\textsuperscript{10,12} Our study demonstrates that elevated Lp(a) plasma concentrations in ESRD patients are nongenetic and secondary to renal insufficiency because restoration of kidney function completely normalizes Lp(a) plasma levels. Furthermore, we observed a marked increase in the fraction of the variability of Lp(a) plasma concentrations explained by the apo(a) size polymorphism after RTX (from 39.0% to 58.2%), probably reflecting a decrease in the nongenetic effect of Lp(a) levels. This observation therefore also suggests that the elevated Lp(a) levels are a direct or indirect consequence of the kidney disease. Our finding is in agreement with a recent report in which a decrease in Lp(a) levels was observed in a small group of 20 patients without considering apo(a) types.\textsuperscript{41} However, in a further study elevated Lp(a) concentrations were measured in an RTX group 36 months after RTX.\textsuperscript{42} The latter study was designed as a cross-sectional study with unknown Lp(a) plasma concentrations before RTX. In our study, after 3 weeks all phenotype groups reached the average Lp(a) plasma levels characteristic for control subjects of the same phenotype. Considering the results from our previous study\textsuperscript{17} in which we found an elevation of Lp(a) in ESRD patients only with HMW and not with LMW phenotypes, it is not surprising that only patients with HMW phenotypes had a significant decrease in Lp(a) concentrations, whereas patients with LMW phenotypes showed no significant difference after RTX (Figure). The underlying mechanism for this phenotype-associated difference is unclear.

We also considered the possible influence of immuno-suppressive therapy on the observed changes in Lp(a) concentrations. In a cross-sectional study it has recently been established as an independent risk factor for CHD in different ethnic groups\textsuperscript{30,34} and in hemodialysis patients.\textsuperscript{35}

![Line graph](http://atvb.ahajournals.org/)

Line graph shows lipoprotein(a) [Lp(a)] changes over time after renal transplantation in three patients with low-molecular-weight phenotypes (c) and three patients with high-molecular-weight phenotypes (o). The apolipoprotein(a) phenotype and Lp(a) plasma concentration before transplantation were as follows: S2, 20 mg/dL (patient a); S2, 58 mg/dL (patient b); S2, 15 mg/dL (patient c); S3, 21 mg/dL (patient e); S3S4, 90 mg/dL (patient f); and S4, 18 mg/dL (patient g).
been reported that Lp(a) levels increase after RTX as a result of CsA therapy. However, no significant changes of Lp(a) have been observed in a prospective longitudinal study in patients with cardiac or lung transplantation. No evidence for an increase was found in the present study, which was performed in a much larger group of patients and in a longitudinal study design with known pretransplant Lp(a) values. The decline of Lp(a) values to type-specific concentrations after RTX is considered as a particularly strong argument against an Lp(a)-raising effect of CsA. By the same argument we do not believe that the decrease in Lp(a) concentration after RTX is caused by the therapy. Furthermore, we have direct and indirect evidence to argue against immunosuppressive therapy having an effect on the Lp(a) decrease. First, Lp(a) decreased in patients with HMW but not with LMW phenotypes although both groups received the same immunosuppressive therapy. Second, patients under different drug therapies (azathioprine plus prednisone versus triple-drug therapy) showed the same decrease in Lp(a) after transplantation. Third, for control we also analyzed eight patients who underwent ceratoplastic surgery and therefore had to be treated with CsA (four patients) or with the same prednisone regimen as RTX patients (four patients) to prevent rejection. None of them showed a decrease in Lp(a) plasma levels (data not shown). Therefore, it is reasonable to conclude that neither CsA nor prednisone is responsible for the decrease of Lp(a) in RTX patients. Fourth, we observed seven transplanted patients who temporarily discontinued azathioprine. None of them showed a significant elevation in Lp(a). Fifth, it is unlikely that the Lp(a) decrease is a combination effect of CsA, azathioprine, and prednisone, because it also occurs in patients with the combination of CsA plus prednisone or azathioprine plus prednisone.

Finally, we observed a significant difference in the distribution of apo(a) isoforms between patients undergoing RTX and control subjects ($P<.0001$). The former had significantly fewer LMW phenotypes than the control subjects (14.9% versus 25.8%, $P<.01$). This result was surprising and needs some consideration. As in recent studies the control subjects in the present study were from the same geographical area as the patients and were lined up for this and other studies that were performed at the same time. The 25.8% of LMW phenotypes are representative and not significantly different from other control groups from recent studies, in which we found 26.5%, 26.9%, 29.4%, 30.9%, 31.9%, and 32.1% of patients with LMW phenotypes. If we had compared the LMW frequency in the RTX group with these other control groups, the difference between patients and control subjects would be even higher. These observations might suggest that selection against patients with LMW phenotypes had occurred. One possible selection mechanism might be death from atherosclerotic complications and exclusion from transplantation of patients with LMW phenotypes and corresponding high Lp(a) levels. In line with this, the RTX patients differ from a general hemodialysis group in physical health. Only patients in good physical condition were included in the RTX group. Moreover, RTX patients underwent dialysis treatment for a very long time (on average 51.0 months). It should also be considered that the exposure of ESRD patients with LMW isoforms to plasma Lp(a) concentrations around 30 mg/dL lasted for their entire life. Such a plasma concentration represents a higher risk for premature atherosclerosis. In contrast, patients with HMW isoforms acquired their high Lp(a) levels only with the development of their chronic renal insufficiency. Even then, their average Lp(a) levels were lower than in patients with LMW isoforms. Therefore, more patients with LMW phenotypes might have suffered from early atherosclerotic complications than patients with HMW phenotypes, explaining their underrepresentation in the RTX group.

Although we found a distinct normalization of the atherogenetic Lp(a) plasma levels after transplantation, this patient group is still at high risk for atherosclerosis. Thus, the assumed beneficial effect of lowering Lp(a) does not prevent the development of atherosclerotic disease in this group. Our study clearly demonstrates a significant decrease of Lp(a) after RTX. Whether this effect is beneficial remains to be determined.

Acknowledgments

This study was supported by grants from the Austrian Fonds zur Förderung der wissenschaftlichen Forschung (S-4604 and P-10090) to Hans Dieplinger and Gerd Utermann. The expert technical assistance of Eva-Maria Lobentanz, Linda Fineder, Martina Urbanek, and the nursing staffs from the Department of Transplant Surgery and the outpatient clinic of the Department of Clinical Nephrology is appreciated.

References


Apolipoprotein(a) phenotype-associated decrease in lipoprotein(a) plasma concentrations after renal transplantation.

F Kronenberg, P König, K Lhotta, D Ofner, C Sandholzer, R Margreiter, E Dosch, G Utermann and H Dieplinger

_Arterioscler Thromb Vasc Biol_. 1994;14:1399-1404
doi: 10.1161/01.ATV.14.9.1399

_Arteriosclerosis, Thrombosis, and Vascular Biology_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 1994 American Heart Association, Inc. All rights reserved.

Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://atvb.ahajournals.org/content/14/9/1399

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Arteriosclerosis, Thrombosis, and Vascular Biology_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:

http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Arteriosclerosis, Thrombosis, and Vascular Biology_ is online at:

http://atvb.ahajournals.org/subscriptions/