Lp(a) Lipoprotein in Patients with Chronic Renal Failure Treated by Hemodialysis, H. J. Parra, H. Mezdour, C. Cachera, M. Dracq, A. Tacq, and J. C. Fruchart (*Serlia et U. Inserm 279, Institut Pasteur, 1, rue du Pr Calmette, 59019 Lille Cédex, France; Laboratoire de Nephrologie du Centre de l'Hôpital Calmette, rue du Pr J. Leclercq, 59037 Lille Cédex, France)

Patients with chronic renal failure (CRF) who are treated with hemodialysis commonly also have premature and accelerated vascular disease (1), which may derive in part from derangements in concentrations of lipids and lipoproteins in plasma. Hypertriglyceridemia in particular commonly accompanies this condition and is associated with an increased concentration of apolipoprotein B-containing very-low-density lipoproteins in plasma. Recently, clinical interest has been paid to the disturbances of Lp(a) lipoprotein concentrations in relation to cardiovascular disease (2). Lp(a) lipoprotein, a variant of apolipoprotein B-containing lipoproteins, is chemically similar to low-density lipoprotein but immunologically differs from it by containing an additional antigen, the (a)-antigen; the concentration of Lp(a) in serum is genetically determined. Although high concentrations of Lp(a) reportedly are associated with increased risk of coronary artery disease (2) and cerebral infarction (3), to our knowledge such data are not available for patients with CRF who are receiving hemodialysis.

We quantified serum Lp(a) lipoprotein (4) in 71 patients (46 men, 25 women; ages 16–72 years) undergoing chronic hemodialysis for CRF, and in 71 controls matched for sex and age (Table 1). Total cholesterol (TC) and triglycerides (TG) in serum were assayed enzymatically in all subjects. In agreement with previous reports, we found no sex-related difference in either mean or median Lp(a) concentrations and no statistically significant correlation of Lp(a) with either cholesterol, triglycerides, or age. Because the distribution of the Lp(a) concentrations was highly skewed, especially in controls, we used a distribution-free statistical test (Mann–Whitney U-test) to compare the values.

As did others (1), we found that hypertriglyceridemia is the most usual abnormality in hemodialysis patients, whereas hypercholesterolemia is uncommon. However, we also noted that Lp(a) concentrations in patients averaged threefold those of controls. Moreover, compared with controls, more than thrice as many of these hemodialysis patients had high-risk (> 300 mg/L) (2) concentrations of Lp(a). These findings suggest a strong association between increased concentrations of Lp(a) and hemodialysis treatment of CRF. Along with other known risk factors such as hypertriglyceridemia, an increased concentration of Lp(a) may play an important role in accelerating development of atherosclerosis in this condition. Recently, Gurkar et al. (5), using the cholesterol-lowering drugs neomycin and niacin, decreased the concentration of Lp(a) in 14 type II hyperlipoproteinemic subjects by 45%. Such treatment might also be desirable for these hemodialysis patients.

References

Rapid, Qualitative Immunoenzymometric Technique Evaluated for Detecting Choriogonadotropin in Serum and Urine, James H. McBride, Henry Hao, Sharon Higgins, and Denis O. Rodgerson (Cln. Chem. Lab., Dept. of Pathol, UCLA Sch. Med., Los Angeles, CA 90024)

We evaluated the Quidel pregnancy test (Quidel, La Jolla, CA 92037), a "dipstick" immunoenzymometric technique for rapid qualitative detection of human choriogonadotropin (hCG) in serum and urine.

The stated sensitivity of the Quidel pregnancy test is 50 int. units/L, for a 15-min test, but we found we could easily detect hCG at 25 int. units/L, and in some samples at as little as 8 int. units/L.

Specificity and sensitivity for predicting pregnancy from serum results (n = 70) were 95% and 97%, respectively, and 98% and 99%, respectively, for urine test results (n = 40) at a detection limit of 25 int. units/L. For serum, hemolysis. (hemoglobin 15 g/L), lipemia (triglyceride 9.2 g/L), and icteria (bilirubin 200 mg/L) did not interfere in the test for hCG concentrations ranging from < 5 to 200 int. units/L, nor did 3+ hematura or 4+ icerus in urines containing 30 int. units of hCG per liter.

For serum samples containing hCG concentrations of 1690, 8500, 14 200, and 60 400 int. units/L we observed an increasing intensity of blue color on the dipsticks, with clear differentiation between each, and no excess-antigen effect. We saw no cross reactivity when we tested sera containing, per liter, 236 int. units of lutropin, 1610 milli-int. units of thyrotropin, and 199 int. units of follitropin. Pathological concentrations of alkaline phophatase, 700 and 1000 U/L, were also negative in the test.

Table 1. Concentrations of Lp(a) Lipoprotein and Lipids in Serum of Controls and Hemodialysis Patients

<table>
<thead>
<tr>
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<th>Controls (n = 71)</th>
<th>Hemodialysis patients (n = 71)</th>
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<tbody>
<tr>
<td><strong>Age, years</strong></td>
<td>44.0 ± 14.98</td>
<td>44.03 ± 15.08</td>
</tr>
<tr>
<td><strong>Lp(a), mg/L</strong></td>
<td>125.00 ± 172.80</td>
<td>378.00 ± 463.50</td>
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<tr>
<td><strong>TC, mmol/L</strong></td>
<td>5.93 ± 1.08</td>
<td>5.99 ± 2.06</td>
</tr>
<tr>
<td><strong>TG, mmol/L</strong></td>
<td>1.11 ± 0.39</td>
<td>2.50 ± 1.47</td>
</tr>
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*Values significantly different from controls: P < 0.0001 (Mann–Whitney U-test).
Because an alkaline phosphatase label is used, we investigated the effects of metal ions on enzyme activity. We tested a control urine containing arsenic (1085 μg/L), calcium (455 mg/L), copper (1035 μg/L), lead (750 μg/L), magnesium (75 mg/L), sodium (550 mmol/L), potassium (165 mmol/L), phosphorus (3 g/L), and zinc (11 mg/L), finding no interference by any of these at hCG concentrations of 30 int. units/L. At this same hCG concentration, there was no interference by total catecholamines (580 μg/L), ciprofibrate (3295 μg/L), creatinine (4.8 g/L), delta-aminovaleric acid (49 mg/L), glucose (14 g/L), 5-hydroxyindole-3-acetic acid (72.5 mg/L), 17-ketogenic steroids (47 mg/L), 17-ketosteroids (133 mg/L), metanephrines (1.2 mg/L), uric acid (1090 mg/L), or vanillylmandelic acid (100 mg/L).

Urinary pH (5–8) had no effect on the pregnancy test for urines containing 0, 30, or 100 int. units of hCG per liter. Urea nitrogen, 16 and 32 g/L, did not interfere. A total-protein concentration of 128 g/L or an albumin concentration of 53 g/L in serum did not interfere with results for specimens containing <5 int. units of hCG per liter, nor did urinary protein in a concentration of 13 g/L.

The test dipstick absorbs the color of the drug phenazopyridine (Pyridium®) if it is present in urine specimens. However, it is not difficult to distinguish the color difference between test and reference pads for a hCG concentration of 30 int. units/L when this drug is present.

This test is useful for detection of hCG, both in the clinical laboratory and the hospital emergency room. Further, the test offers the possibility of home use, because it is easy to use, requires no special equipment, and includes a negative reference pad. Also, the test dipsticks may be retained as a permanent record of the test, which can be compared with subsequent results.

**Kinetic Amylase Kit Adapted to the Technicon RA-1000, Ian M. Barlow, Stephen P. Harrison, John R. Minler, and Miles O. Sykes (Dept. of Biochem., Bradford Royal Infirmary, Duckworth Lane, Bradford, BD9 6RJ, U.K.; address correspondence to S. P. H.)**

The "Phadebas" kinetic kit (Pharmacia Diagnostics AB, Box 17, S-751 03 Uppsala, Sweden) for estimating α-amylase (EC 3.2.1.1) in serum, urine, and other body fluids consists of one lyophilized reagent, and the method is that outlined by Marshall et al. (1). The substrate, a blocked starch, is hydrolyzed by the amylase in the sample and yields fragments that can be attacked by glucoamylase (glucan 1,4-α-glucosidase EC 3.2.1.3) to form glucose. Glucose is then converted by hexokinase (EC 2.7.1.1) to glucose 6-phosphate, which is oxidized by glucose-6-phosphate dehydrogenase (EC 1.1.1.49) in the presence of NAD⁺. The increase in absorbance of the NADH produced, which is related to the amylase activity in the sample, is measured at 340 nm.

For use with the RA-1000, the reagent was reconstituted according to the manufacturer's instructions, and after addition of two drops of wetting agent W was found to be stable for two days at 4 °C.

"Phadebas Humylase control (H)" was used to standardize the assay. This is analyzed in triplicate and the mean absorbance change per minute is used to calculate the unit factor as follows:

\[
\text{Unit factor} = \frac{\text{quoted Humylase value}}{(\Delta A/\text{min}) \times \text{calib. factor} (7890)}
\]

This factor is checked, along with a reaction-rate water blank, for each new batch of reagent. The linear range extends to 2500 U/L, as compared with 1500 U/L when the kit is used manually. The CV for 15 determinations at 148 U/L was 2.5% within-batch, 3.5% between-batch; at 695 U/L these were 1.1% and 1.7%, respectively. Comparison of results obtained with the RA-1000 and the "Phadebas" tablet method gave the regression equation: [kinetic amylase method] = 1.00 [tablet amylase method] + 8.5 U/L (r = 0.99). Bilirubin, hemoglobin, or lipemia do not affect the assay. Glucose gives a positive interference but, before this becomes significant (>30 mmol/L), the linearity parameters in the RA-1000 software detect a deviation from linear absorbance indicated by an "L" flag.

We have been using the assay routinely for over 12 months and it has performed well in external quality control schemes. The assay is quick (6 min throughput) and the current cost compares favorably with that for other kits.

**Settings for the RA-1000 are as follows:**

- **Type**: 0
- **Range high**: 2500
- **% Sample vol**: 5
- **Cal factor**: 7890
- **Filter pos**: 1 WL 340
- **Reagent rate**: 0.0008
- **Delay**: 2.00
- **Normal low**: 70
- **% Reagent vol**: 70
- **Normal high**: 300
- **Units**: 3 U/L
- **Slope**: 1.00
- **Unit factor**: 1.247
- **Intercept**: 0
- **Decimal point**: 0
- **C1*10E-6**: 3.43
- **C2*10E-6**: 99999.99
- **RBL low**: 0.15
- **D1*10E-6**: 2.33
- **RBL high**: 0.50
- **Range low**: 0
- **Delta #**: 0.010

*These values should be established by each laboratory.

**Reference**


**Serum Amylase and γ-Glutamyltransferase Assay in the Abbott "Vision" System, Edward A. Sasse and Jill B. Edwards (Dept. of Pathol., Medical College of Wisconsin, Milwaukee, WI 53226)**

We have examined the performance of the α-amylase (EC 3.2.1.1) and γ-glutamyltransferase (GGT, EC 2.3.2.2) assays in the Abbott "Vision," a fully automated chemistry analyzer designed primarily for use in physicians' offices and satellite laboratories (1). The spectrophotometric analyzer system, which is based on the concept of two-dimensional centrifugation and which utilizes self-contained unit-dose test packs, has been evaluated previously for several other assays (2, 3).

The substrate used in the Vision amylase assay is p-nitrophenyl-α-b-maltoheptaoside-blocked. The non-reducing terminal sugar of the substrate has been chemically blocked to prevent initial hydrolysis by exo-glycosidases. Amylase in the patient's sample initiates hydrolysis of the substrate, which is further rapidly hydrolyzed to p-nitrophenol and glucose by α-glucosidase and glucoamylase in the reagent.

The rate of formation of p-nitrophenol, measured spectrophotometrically, is directly proportional to the amount of amylase present.