The combination of CK-MB and LD-1 was better than either one alone. When interpreting results from individual patients, calculating probability of AMI from likelihood ratio functions gives much more information than just classifying the results as "positive" or "negative" (3).

References


Recipients of blood transfusions run the risk of potential infection, and several procedures for the identification of high-risk donors are used, including the detection of antibody to human immunodeficiency virus (HIV), hepatitis B core antigen, and the measurement of plasma or serum alanine aminotransferase (ALT; EC 2.6.1.2) as a marker for non-A,non-B (NANB) hepatitis (1). To minimize the risk of infection from HIV when handling plasma or serum, one of the suggested procedures is heat treatment at 56 °C (2, 3).

Published data on the effects of heat treatment on ALT and aspartate aminotransferase (AST; EC 2.6.1.1) are limited. For AST, some reports showed no statistically significant differences after treatment for 0.5 h at 56 °C, although slight reductions of the mean enzyme activities were noted (4–6). For ALT, there is one report of a 63% reduction of enzyme activity in serum after heat treatment for 0.5 h (4).

We measured AST and ALT in plasma after heat treatment at 56 °C for 0.5 and 1 h (3). Heparinized plasma samples (n = 20) free from hemolysis, lipemia, and icterus were obtained from healthy volunteers, and were heated in 1.5-ML polypropylene screw-capped centrifuge tubes in a dry-heating block. To measure absorbance, we used a Roche Cobas-Bio centrifugal analyzer at 37 °C. For plasma AST and ALT measurements we used reagents formulated according to the recommendations of the Deutsche Gesellschaft für Klinische Chemie (DGK) (9) (Boehringer Mannheim, product nos. 158178 and 161071).

In addition, we measured plasma ALT with a reagent formulated according to the International Federation of Clinical Chemistry (IFCC) recommendations (10), differences of ALT activity having been observed between use of the two reagent formulations (11). We used the IFCC reagent (Boehringer Mannheim, product no. 487325) either (a) excluding pyridoxal phosphate or (b) with addition of pyridoxal phosphate, 0.10 mmol/L, and no preincubation step.

For all methods, within-batch precision studies gave CV <1% for assays of a bovine control serum (n = 25). Using the DGKC reagent, we obtained a mean value for plasma AST of 17.3 (SD 6.8) U/L (range, 12–29 U/L). After heat treatment at 56 °C for 0.5 and 1 h, the mean value changed to 19.0 (SD 5.6) and 18.6 (SD 5.3) U/L, respectively, but not statistically significant: P >0.05.

However, ALT activity in plasma was markedly reduced (P <0.01) after heat treatment at 56 °C for 0.5 and 1 h, as assayed with either reagent (Table 1). Without preincubation, adding pyridoxal phosphate to the IFCC reagent did not restore the apparent plasma ALT activity to preheating values. Further studies will be required to demonstrate whether preincubation with pyridoxal phosphate reactive plasma ALT after heat treatment, because the preincubation procedure is known to affect ALT measurements (12).

In conclusion, the effects of heat treatment were much greater than other preanalytical variables described for plasma ALT measurements (13), and alternative procedures should be used for handling "high risk" samples.

Table 1. Effect of Heat Treatment at 56 °C on Plasma ALT (U/L)

<table>
<thead>
<tr>
<th></th>
<th>No treatment</th>
<th>0.5 h</th>
<th>1 h</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DGKC reagent</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>13.4 (4.9)</td>
<td>5.0 (2.8)</td>
<td>5.0 (2.9)</td>
</tr>
<tr>
<td>Range</td>
<td>5–24</td>
<td>1–11</td>
<td>1–13</td>
</tr>
<tr>
<td><strong>IFCC reagent without pyridoxal phosphate</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>16.5 (6.4)</td>
<td>7.3 (3.1)</td>
<td>5.8 (3.0)</td>
</tr>
<tr>
<td>Range</td>
<td>8–32</td>
<td>4–16</td>
<td>3–16</td>
</tr>
<tr>
<td><strong>IFCC reagent with pyridoxal phosphate</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>17.8 (6.8)</td>
<td>6.3 (2.8)</td>
<td>4.7 (2.1)</td>
</tr>
<tr>
<td>Range</td>
<td>8–31</td>
<td>3–12</td>
<td>2–10</td>
</tr>
</tbody>
</table>

References

In screening for a pheochromocytoma, the extraction methods used for assay of urinary catecholamines by HPLC do not eliminate all the chromatographic peaks from antihypertensive drugs (1). We describe here the possible interference by labetalol, captopril, and α-methyldopa in such assays.

Urine was collected for 24 h; catecholamines were extracted as described previously (2) and injected onto a C18 column with phosphate buffer (pH 4.0) containing, per liter, 0.03 mmol of EDTA, 1.0 mmol of octane sulfonic acid, and 8-40 mL of acetonitrile at a flow rate of 0.5-1.0 mL/min for 30-45 min. Peaks were detected with an electrochemical detector at 600 mV with recorder sensitivity set at 12 nA full-scale.

The amount of acetonitrile added to the mobile phase, together with the flow rate, was important in ensuring that norepinephrine (noradrenaline, NA) and epinephrine (adrenaline, A) were separated by at least 3 min on the chromatogram (Figure 1). Peaks from labetalol were often larger than that for NA and difficult to resolve from it. Another peak from labetalol was occasionally seen between A and the internal standard, dihydroxybenzylamine (DHBA).

Figure 2 shows a chromatogram of urine containing α-methyl-NA, a metabolite of α-methyldopa, as well as α-methyldopamine, another metabolite of α-methyldopa, which is carried over from the previous injection. The previous chromatogram run had been ended at 45 min instead of 68 min, the latter being the time necessary for all

References

Lipid-Peroxidation Products and Antioxidants in Plasma of Cigarette Smokers, J. Pré and A. Le Floch
(Biochimie Clinique, UFR de Médecine Paris Nord, 74, rue Marcel Cachin, 93012 Bobigny, France)

Cigarette smoking increases the concentrations of fluorescent lipid-peroxidation products in plasma (1) and decreases the ascorbic acid concentrations (2). We decided to investigate the changes of free alpha-tocopherol concentration and the reactivity of 2-thiobarbituric acid in plasma. Thiocyanate (3), free tocopherol (4), ascorbic acid (5), 2-thiobarbituric acid reactivity (6), and fluorescent liperoxidation products (7) were simultaneously measured in plasma from healthy Caucasian blood donors, divided into either cigarette smokers or nonsmokers. Venous blood was collected in glass tubes containing lithium heparinate (Becton Dickinson Vacutainer System) and assayed within 3 h.

The mean concentrations of free tocopherol in plasma were not significantly different in nonsmokers and cigarette smokers. Thus, unlike ascorbic acid, free tocopherol is not an effective plasma antioxidant. A and B fluoroses (see Table 1) of isopropanolic extracts of plasma are markers of in vivo lipid peroxidation (1). Meanwhile, the increase of A and B fluoroses in smokers is probably, in part, the result of a weak platelet activation. Indeed, it has