Low Aspartate Transaminase Activity in Serum of Patients Undergoing Chronic Hemodialysis

Paul L. Wolf, Dorothy Williams, Norman Coplon, and Alan S. Coulson

Glutamic-oxalacetic transaminase (aspartate transaminase) activity is decreased in serum of patients undergoing long-term hemodialysis. The reason may be the repeated dialysis, pyridoxine depletion, or both.

Additional Keyphrases pyridoxine deficiency • SGOT • results compared for automated and manual methods

In the course of following the routine serum chemical values of patients undergoing chronic hemodialysis in the renal-care unit, we noted that 11 of 19 patients had little or no activity recorded for glutamic-oxalacetic transaminase (AST, aspartate transaminase; L-aspartate:2-oxoglutarate aminotransferase, EC 2.6.1.1).

Materials and Methods

Serum, 2 ml, was assayed colorimetrically with the “SMA 12/60 AutoAnalyzer” (Technicon Instruments Corp., Tarrytown, N.Y. 10591). Colorimetric determination (at 45°C) of AST with the SMA 12/60 is based on the coupling reaction of a diazonium salt of N-butyl-4-methoxymetanilamide with oxaloacetic acid produced when AST acts on ketoglutarate. AST activity of the same serum specimen was determined manually by an enzymatic ultraviolet technique (1). Normal values range up to 40 U. The lactic acid content of each serum was determined by the following procedure. Lactate is reduced to pyruvate by adding excess NAD. Glycine-hydrazine buffer is added, and pyruvic acid hydrazine is formed. This is a quantitative conversion, and the amount of NADH formed is measured on the Beckman DU at a wavelength of 340 nm (9).

Results

By both the automated and manual techniques, values for AST were abnormally low (or zero) in this group of repeatedly dialyzed patients (Table 1).

Discussion

Possibly, AST activity is low because the patients had abnormally high serum lactate concentrations, which caused a rapid consumption of NADH co-enzyme in the chemical laboratory test and resulted in artificially low AST. This phenomenon occurs in cases of beriberi, diabetic ketoacidosis, or severe liver disease (2). Accordingly, the patients' serum lactate values were determined, but in only one patient was it greater than normal (3).

A second possible explanation for the depressed AST activity may be that the enzyme is lost in the course of dialysis.

Finally, pyridoxal phosphate and pyridoxamine phosphate serve as co-enzymes for AST. It has been established that long-term hemodialysis may result in plasma depletion of a number of vitamins. Depletion of folic acid may result in megaloblastic anemia in chronic hemodialysis patients (4). A recent report demonstrated low vitamin C levels in 10 patients on long-term hemodialysis (5). Patients dialed three times a week suffered a loss of 100 mg of ascorbic acid per 8-h dialysis period; there were petechiae near the site of the shunt. Supplementing pyridoxine in the diet results in a 25–50% increase in serum AST activity (6). Low AST serum activities are found in pregnancy, which may be associated with low pyridoxine concentrations (7). Thus, a possible depletion of plasma pyridoxine during long-term hemodialysis may result in low AST activity.

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Table 1. Effect of Prolonged Dialysis on Serum AST Activity and Lactate in 11 Patients

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Type of dialysis and hours (2x/week)</th>
<th>SMA 12/50 AST, U&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Manual AST</th>
<th>Lactate&lt;sup&gt;b&lt;/sup&gt; mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic glomerulonephritis</td>
<td>Travenol coil 21h</td>
<td>Dec 17-24</td>
<td>Dec 24-31</td>
<td>Jan 1-8</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Dow 27h</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Chronic glomerulonephritis</td>
<td>Dow 30h</td>
<td></td>
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<tr>
<td>Chronic glomerulonephritis</td>
<td>Dow or Kiil 26h</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Renal carcinoma</td>
<td>Dow or Kiil 20h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Dow 18h</td>
<td></td>
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<tr>
<td>Hypertensive nephropathy</td>
<td>Dow 24h</td>
<td></td>
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<tr>
<td>Malignant hypertension</td>
<td>Kiil 20h</td>
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<tr>
<td>Alport's disease</td>
<td>Dow or Kiil 36h</td>
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<tr>
<td>Chronic membranous glomerulonephritis</td>
<td>Dow or Kiil 24h</td>
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<tr>
<td>Chronic glomerulonephritis</td>
<td>Dow 24h</td>
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</table>

<sup>a</sup> normal = 40 U.<br>
<sup>b</sup> normal = 0.613–1.777.

<sup>+</sup> n.d. = not done.

References


A Linear Absorbance Converter for Continuous-Flow Colorimeters

Leonard Walker and Elias Amador<sup>1</sup>

The photocell transmittance output from continuous-flow colorimeters can be converted into linear absorbance by means of a simple solid-state circuit. The circuit includes a field effect transistor operational amplifier and a logarithmic operator, plus several stabilizing circuit networks. The working range is 1.2 absorbance units, which for most applications is an improvement of 50% over the unmodified colorimeter, at wavelengths of 420 to 700 nm. The output can drive the conventional AutoAnalyzer recorder, or a 2-input, low-cost recorder such as the Heath Kit IR-18M.

Additional Keyphrases AutoAnalyzer colorimeter

The widely used "AutoAnalyzer I" colorimeter (Technicon Instrument Corp., Tarrytown, N. Y. 10591) provides output in transmittance, which is a logarithmic function of absorbance. We have designed a linear absorbance converter for the AutoAnalyzer I colorimeter that produces peak heights that are directly proportional to the absorbance of the sample.

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