Plasma Glutamic Oxalacetic Transaminase as Related to Liver Lesions from Histomoniasis in Turkeys

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SUMMARY

Glutamic oxalacetic transaminase (GOT) was elevated significantly 11 days later in all turkeys inoculated with Histomonas meleagridis. The number of liver lesions was correlated significantly with GOT level. The lesions were both small and large, the small lesions being most abundant, and the large lesions being aggregated small lesions.

Sulfuride, a histomonastat, reduced the number of liver lesions along with a significant lowering of plasma GOT level.

Plasma GOT level can be used clinically as an indicator of the course of histomoniasis and would be useful for quick and accurate screening of potential histomonastats.

INTRODUCTION

It has been reported that the level of glutamic oxalacetic transaminase (GOT) in turkeys rises with histomoniasis (1,3) and that the GOT level might be used to evaluate the efficacy of therapeutic and prophylactic agents against histomoniasis (3). The present study investigated the relation of GOT level to number of liver lesions.

MATERIALS AND METHODS

Day-old Wrolstad White turkey poults were obtained from commercial hatcheries. They were inoculated intranasally with Newcastle vaccine (B1 types, B1 strain) and fed a commercial turkey grower. A like treatment was given Nicholas Broad Breasted White turkey poults, used in the fourth experiment.

 Infective histomonads used in all experiments were cultured in vitro in a modified DeVolt's medium (5). The pathogenicity of

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Table 1. Relationship of plasma glutamic oxalacetic transaminase levels to liver lesions in turkeys infected with *Histomonas meleagridis*.

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Treatment</th>
<th>Turkey(^{A}) (no.)</th>
<th>Age of turkeys (days)</th>
<th>Av and range GOT levels (mU/ml)</th>
<th>Av and range liver lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Infected</td>
<td>12</td>
<td>15</td>
<td>172 (138-215)</td>
<td>395 (169-632)</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>6</td>
<td>15</td>
<td>77</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>Infected</td>
<td>12</td>
<td>30</td>
<td>161 (125-200)</td>
<td>236 (252-470)</td>
</tr>
<tr>
<td></td>
<td>Infected (untreated)</td>
<td>12</td>
<td>30</td>
<td>75 (66-83)</td>
<td>400 (210-585)</td>
</tr>
<tr>
<td>2</td>
<td>Control</td>
<td>6</td>
<td>30</td>
<td>74</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>Control(^B)</td>
<td>3</td>
<td>30</td>
<td>74</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>Infected (untreated)</td>
<td>12</td>
<td>15</td>
<td>161 (119-207)</td>
<td>336 (191-441)</td>
</tr>
<tr>
<td>3</td>
<td>Infected (treated)(^C)</td>
<td>12</td>
<td>15</td>
<td>118 (95-159)</td>
<td>228 (74-431)</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>6</td>
<td>15</td>
<td>79 (72-85)</td>
<td>137 (62-325)</td>
</tr>
<tr>
<td>4</td>
<td>Infected</td>
<td>12</td>
<td>30</td>
<td>271 (184-478)</td>
<td>612 (312-997)</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>6</td>
<td>30</td>
<td>75 (67-83)</td>
<td>--</td>
</tr>
</tbody>
</table>

\(^A\) Wrolstad White breed used in Experiments 1–3, Nicholas Broad Breasted White breed used in Experiment 4.  
\(^B\) Saline-injected  
\(^C\) Salfuride, 10 ppm in feed.
the strain was verified initially with a dose of 30,000 histomonads inoculated intrarectally into 2-month-old male turkeys. Eleven days later they were sacrificed, and histomonads plus associated bacteria were isolated from the ceca. The livers showed an erosion with a remarkable lympholytic reaction and some cellular necrosis, thus verifying the pathogenicity of the strain. Two to 4 drops of cecal contents from these birds were inoculated into six tubes of medium. Subcultures were made every 5 days.

Duplicate determinations of GOT level were made on the plasma from each bird with a commercial assay kit (Boehringer-Mannheim Corporation, New York, N.Y.).

"Significant" as used herein is at $P<.05$.

**Experiment 1.** Eighteen 15-day-old poults were anesthetized and the ceca exposed by surgical means. Ten thousand histomonads were inoculated into the left cecal lumen of 12 poults, and 6 poult controls were injected with 1% saline.

Eleven days postinoculation all birds were decapitated, and 20 ml of blood from each bird was collected in beakers previously washed with 1% heparin. The heparinized blood was centrifuged, and the plasma was collected and stored in small vials at $-20\,^\circ$C. The liver of each bird was removed, photographed, and stored in 5% formalin. Later, the number and size (small and large) of liver lesions were enumerated. Small lesions were 1 mm or less in diameter; large lesions were greater than 1 mm.

**Experiment 2.** Eighteen 30-day-old poults were treated as in Experiment 1 except that the controls were 3 additional poults not injected with saline.

**Experiment 3.** Thirty 15-day-old poults were treated as follows: 12 were infected as previously described, 12 were infected and treated with Salfuride [3,5-dinitrosalicylic acid (5-nitrofurfurylidene) hydrazide, Salsbury Laboratories, Charles City, Iowa], and 6 were controls. The basal ration and Salfuride-treated feed were prepared as follows. The basal ration was ground to pass a sieve opening of 0.208 mm and then divided into two parts. One part was fed to all birds for 1 week before inoculation with histomonads. The second part was mixed with the drug to 10 ppm, which would give partial protection against histomoniasis. To obtain this concentration 14.9 kg of basal ration was mixed with 150 mg of drug contained in 100 mg of starch carrier.

Beginning on the 8th day postinoculation, the treated group was given the medicated feed for 4 days. On the 11th day all birds
in the experiment were necropsied, the plasma was collected, and liver damage was assessed.

**Experiment 4.** This experiment was a replicate of Experiment 2 except that the Nicholas breed of poults and a different subculture of histomonads were used.

**RESULTS**

**Experiment 1.** The GOT level in the plasma was significantly correlated positively with the number of small liver lesions in turkeys inoculated at 15 days of age. No liver lesions were seen in the controls. The average plasma level of GOT was significantly lower in the controls than in the infected turkeys (Table 1).

**Experiment 2.** GOT levels were significantly correlated positively with the number of small and large liver lesions in turkeys inoculated at 30 days of age. The average GOT level was significantly higher in the infected group than in the controls (Table 1). Average GOT level did not differ significantly between controls receiving saline injections and controls not injected.

**Experiment 3.** The average level of GOT was significantly higher in the untreated infected turkeys than all other groups (Table 1). GOT levels were significantly correlated positively with the number of liver lesions. Also, there were significantly fewer lesions and a lower average GOT level in the infected and Sulfuride-treated group than in the untreated infected group.

**Experiment 4.** The infected Nicholas poults had a significantly higher average GOT level than either their controls or the infected Wrolstad poults (Table 1).

**DISCUSSION**

GOT activity is a good index of liver function because any disturbance in the liver elevates GOT levels in the blood (4). GOT levels are elevated in humans with chronic or acute hepatitis (6,7) and in hamsters with tumors of the liver (2).

Our studies thus suggest that liver damage causes elevated GOT level in the plasma of turkeys with histomoniasis.

The high GOT levels in the fourth experiment may be related either to the change in breed of turkeys or to a change in pathogenicity of the histomonads during subculturing.

The broad range of GOT values in infected turkeys is related to a similar range in the number of small and large liver lesions. Apparently, birds of a given breed differ in resistance to histomoniasis.
Quick screening of histomonastats

At present, researchers rely on the number of liver lesions at necropsy as an index of severity of histomoniasis. The present study suggests that GOT levels would be a convenient and accurate method for evaluating the extent of the disease either without sacrificing the host or at necropsy.

REFERENCES


ACKNOWLEDGMENT

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