SHORT REPORT: A CASE OF HEMOLYSIS RESULTING FROM CONTACT WITH A LONOMIA CATERPILLAR IN SOUTHERN BRAZIL

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Abstract. Contact with a caterpillar of the genus Lonomia can result in a hemorrhagic syndrome. Lonomia obliqua venom activates prothrombin and factor X and promotes fibrinogenolytic activity. Although crude L. obliqua bristle extract can induce hemolytic activity in human and rat erythrocytes, there have been no reports of hemolysis in the cases of human contact. We report a confirmed human case of Lonomia venom-induced hemolysis.

Contact with a caterpillar of the genus Lonomia can result in a hemorrhagic syndrome. This was first described in 1967 by Arocha-Piñango in a case in Venezuela.1 In Brazil, the risk of Lonomia envenomation was considered low until 1989, when a high number of human cases were reported in southern Brazil. In 2004, 364 cases were reported in Brazil. The patients had a hemorrhagic syndrome after skin contact with Lonomia obliqua. Two species of the genus Lonomia are known to cause hemorrhagic syndrome: L. achatous, which is found in Venezuela and northern Brazil, and L. obliqua, which is found in southern Brazil. The symptoms of envenomation typically begin with a burning sensation at the point of contact, followed by erythema and edema. The skin around the site is warm to the touch. Headache, general discomfort, nausea, and vomiting may also occur. The hemorrhagic phenomena may appear as soon as 1 hour after contact or up to 12 hours later. Ecchymoses and hematomas are often seen and may be accompanied by bleeding from various sites. The bleeding is occasionally severe enough to be life threatening. Some patients with hemostatic disorders can develop acute renal failure (ARF).2-4 The L. obliqua venom activates both prothrombin and factor X and promotes fibrinogenolytic activity.5-7 Crude L. obliqua bristle extract has been shown to induce hemolytic activity in human and rat erythrocytes.8 However, there have been no reports of hemolysis in the cases of human contact. We report a confirmed human case of Lonomia venom-induced hemolysis.

A 64-year-old man accidentally came into contact with a caterpillar. Immediately after contact, he experienced burning pain, edema, and erythema at the site of contact (on his right hand). Subsequently, he began vomiting and developed a headache. Eight hours after the accident, he developed hematemesis, bleeding gums, and hematuria. Twenty-four hours after contact (day 1), he sought treatment and was admitted to a hospital where a saline infusion was administered. Laboratory testing showed a hemoglobin level = 11.9 g/dL, hematocrit = 36%, 17,800 leukocytes/mL, 57,000 platelets/mL, serum urea = 137 mg/dL, serum creatinine = 2.6 mg/dL, total bilirubin = 2.6 mg/dL, indirect bilirubin = 2.1 mg/dL, thrombin time = 22.8 seconds, international normalized ratio = 3.54; and activated partial thromboplastin time = 50 seconds. Based on these results, he was diagnosed with caterpillar contact-induced hemorrhagic syndrome accompanied by ARF.

On day 2, he was transferred to Emílio Ribas Institute of Infectology in São Paulo, Brazil. Upon admission, he showed dehydration and jaundice, and his blood pressure was 160/90 mmHg. There was mild edema and erythema in his right hand, without ecchymoses or other bleeding. Laboratory tests showed a hemoglobin level = 9.3 g/dL, hematocrit = 26.6%; urea = 197 mg/dL, serum creatinine = 6.4 mg/dL, fibrinogen = 0.06 g/L, fibrin degradation products = 640 μg/mL, D-dimer = 256 μg/mL, and lactate dehydrogenase = 5,028 mg/dL. He received a specific antivenom (antilomonic serum; Butantan Institute, São Paulo, Brazil) and renal replacement therapy was started. Twenty-four hours after receiving the antivenom, the patient showed a significant improvement in coagulation test results: fibrinogen increased to 1.41 g/L, fibrin degradation products decreased to 320 μg/mL, and D-dimer levels decreased to 16 μg/mL.

Despite the improvement in the coagulation test results and no further signs of hemorrhage, there was a significant decrease in hemoglobin levels (to 5.1 g/dL on the fourth post-incident day), and the patient showed lower platelet levels (18,000/mm³). We suspected hemolysis because the patient had higher serum levels of indirect bilirubin and lactate dehydrogenase (LDH) at admission. Hemolysis was confirmed by the observed levels of serum haptoglobin (10 mg/dL) and plasma free hemoglobin (1,220 μg/L) (Table 1).

On day 20, creatinine levels began to decrease, and hemodialysis was discontinued. By day 25, the patient had significantly lower levels of creatinine and urea (5.6 mg/dL and 91 mg/dL, respectively), and although not considered completely recovered, he was discharged. In a follow-up examination on day 30, a normal creatinine level (1.5 mg/dL) was observed. To rule out possible an underlying predisposition for hemolysis we obtained the following test results: hemoglobin electrophoresis: HbA₁, 97.4% (normal range = 94.5–98.5%), HbA₂, 1.6% (normal range = 1.5–3.5%), HbF 1.1% (normal value < 2 %), HbS absent, glucose-6-phosphate dehydrogenase = 177 mU/10 erythrocytes (normal value = 131 ± 13), haptoglobin = 90 mg/dL (normal range = 30–200); LDH = 390 U/L (normal range = 240–480). Tests results for Heinz bodies and for succrose were negative. Results of a Ham test and a Coombs test were negative.

The clinical course after Lonomia accident is probably related to the intensity of the contact, the particular larvae stage, and the type of immediate medical treatment provided.9,10 The bleeding syndrome may begin as soon as within a few hours or up to several days after the accident and can be severe enough to be life threatening.2 Early laboratory coagulation tests invariably showed prolonged prothrombin,

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We believe that cases with significantly envenomed hemolysis-related ARF is caused by hemoglobin, which is a renal biopsy on this patient. It is therefore likely that he had developed acute tubular necrosis. The etiology of ARF is multifactorial. Although this patient never developed hemodynamic instability, he had severe hemorrhagic syndrome. Hemolysis is another leading cause of acute tubular necrosis. Hemolysis-related ARF is caused by hemoglobin, which is highly nephrotoxic, especially in volume-depleted patients. We cannot rule out a direct nephrotoxic effect of the venom. Another possible explanation for the ARF might be massive DIC-related deposition of microthrombi in glomeruli. However, although most of the patients in the study conducted by Zannin and others developed severe coagulopathy with pronounced fibrinogen reduction, none had renal failure. We can postulate that among the many possible causes of ARF, the etiology of the present case was hemolysis-related acute tubular necrosis or hemolysis-related DIC.

A remarkable feature in this case was the rapid development of severe anuric ARF after venom inoculation. Surprisingly, despite the fact that this patient was treated with hemodialysis for 24 days, he did not completely recover renal function until 1 month after the accident. We did not perform a renal biopsy on this patient. It is therefore likely that he had developed acute tubular necrosis. The etiology of ARF is multifactorial. Although this patient never developed hemo-

dynamic instability, he had severe hemorrhagic syndrome. Hemolysis is another leading cause of acute tubular necrosis. Hemolysis-related ARF is caused by hemoglobin, which is highly nephrotoxic, especially in volume-depleted patients. We cannot rule out a direct nephrotoxic effect of the venom. Another possible explanation for the ARF might be massive DIC-related deposition of microthrombi in glomeruli. However, although most of the patients in the study conducted by Zannin and others developed severe coagulopathy with pronounced fibrinogen reduction, none had renal failure. We can postulate that among the many possible causes of ARF, the etiology of the present case was hemolysis-related acute tubular necrosis or hemolysis-related DIC.

In conclusion, although Lononmia-induced hemolysis has previously been reported in an animal model, the case herein reported represents the first confirmed instance of such hemolysis in a human patient.

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**Table 1**

Summary of laboratory findings*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>10</th>
<th>25</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>11.9</td>
<td>9.3</td>
<td>8.3</td>
<td>5.1</td>
<td>8.9</td>
<td>8.4</td>
<td>8.0</td>
<td>8.6</td>
<td>12–16 g/dL</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>36</td>
<td>26.6</td>
<td>22.9</td>
<td>15</td>
<td>26</td>
<td>25</td>
<td>24</td>
<td>25</td>
<td>37–42%</td>
</tr>
<tr>
<td>Platelets</td>
<td>57,000</td>
<td>45,000</td>
<td>18,000</td>
<td>34,000</td>
<td>34,000</td>
<td>68,000</td>
<td>252,000</td>
<td>280,000</td>
<td>150–400 × 10^11/mm^3</td>
</tr>
<tr>
<td>Urea</td>
<td>137</td>
<td>197</td>
<td>157</td>
<td>169</td>
<td>110</td>
<td>136</td>
<td>160</td>
<td>91</td>
<td>7–50 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>4.6</td>
<td>6.4</td>
<td>7.1</td>
<td>6.4</td>
<td>4.3</td>
<td>6.5</td>
<td>8.4</td>
<td>5.6</td>
<td>0.6–1.3 mg/dL</td>
</tr>
<tr>
<td>LDH</td>
<td>25.6</td>
<td>120</td>
<td>640</td>
<td>320</td>
<td>256</td>
<td>16</td>
<td>12</td>
<td>11</td>
<td>120–360 U/L</td>
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<tr>
<td>Platelet count</td>
<td>–</td>
<td>0.4</td>
<td>1.6</td>
<td>1.4</td>
<td>5.9</td>
<td></td>
<td></td>
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<td>0.5–1.5%</td>
</tr>
<tr>
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<td>1.41</td>
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<td></td>
<td></td>
<td>2.05–2.59 g/L</td>
</tr>
<tr>
<td>Reticulocyte</td>
<td>–</td>
<td>640</td>
<td>320</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 2.5 µg/mL</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>10</td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.5 µg/mL</td>
</tr>
<tr>
<td>Creatinine</td>
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<td></td>
<td></td>
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<td>30–200 mg/dL</td>
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<tr>
<td>Hemoglobin</td>
<td>1,220</td>
<td>830</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 50 mg/L</td>
</tr>
</tbody>
</table>

**Parameter**
- *LDH = lactate dehydrogenase; TB/IB = total bilirubin/indirect bilirubin; PTT = partial thromboplastin time (activated); PT = prothrombin time; PA = prothrombin activity; FDP = fibrin degradation products.**
REFERENCES


