AN UNUSUAL TOXIC CAUSE OF HEMOLYTIC-UREMIC SYNDROME

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ABSTRACT — Hemolytic uremic syndrome (HUS) has been associated with a variety of infective as well as non-infective causes. HUS as a toxic manifestation of exposure to herbicides/pesticides has not been reported so far in literature. We report a subject who presented with clinical features of features of HUS after intentional suicidal ingestion of the herbicidal agent monochloroacetic acid (MCA). A 55-year-old farmer was admitted with a history of consumption of monochloroacetic acid with vomiting, hematochezia and oligo-anuria. Our investigations revealed severe renal failure, metabolic acidosis, anemia, and thrombocytopenia with evidence of intravascular hemolysis. He was treated for HUS with plasma transfusions and haemodialysis in view of renal failure. During the course of hospital admission he developed acute antero-septal myocardial infarction and subsequently succumbed to the disease. MCA is used as an herbicidal agent and also a bleaching agent for silk worm cocoons. The toxicity of MCA has included metabolic acidosis, rhabdomyolysis and renal failure; however HUS has not been described in the literature. Extra—renal manifestations of HUS such as cardiomyopathy have also been infrequently described. This case is presented to highlight an as yet unknown toxicity of MCA.

KEY WORDS: Monochloroacetic acid, Hemolytic uremic syndrome, Renal failure, Cardiomyopathy

INTRODUCTION

Hemolytic uremic syndrome (HUS) has been associated with a variety of infective as well as non-infective causes (Kulling et al., 1992). HUS as a toxic manifestation of exposure to herbicides/pesticides has not been reported so far in the literature. Monochloroacetic acid (MCA) is used in the agricultural industry as an herbicide and a bleaching agent for silk worm cocoons. Toxic manifestations of MCA have included severe metabolic acidosis, rhabdomyolysis, renal failure and cerebral edema. Hemolytic uremic syndrome so far has not been reported as a toxicity of MCA We report a subject who presented with clinical features of features of HUS after intentional suicidal ingestion of the herbicidal agent monochloroacetic acid (MCA).

MATERIALS AND METHODS

A 55-year-old farmer was brought to the emergency unit in July 2005 with a history of consumption of monochloroacetic acid (MCA), about 50-75 ml in amount, with symptoms of vomiting, hematochezia and oligo-anuria of 3 days duration. The subject had a history of psychiatric illness previously but was not on any medications currently for the same. He was a known hypertensive on therapy with Atenolol. The patient had complained of intense pain in the back and severe headache following ingestion of the acid. Gastric lavage had been administered at a local hospital and he had been referred to our center for further management. At admission, he was confused, tachypnoeic, afebrile with severe palor and icterus. Pulse rate was 102/ min; B.P - 160/100 mm of Hg. Examination of the skin did not reveal any burns, petechiae or purpuric spots. Systemic evaluation was unremarkable except for epigastric tenderness. A rectal examination revealed fresh blood.

RESULTS

Investigations done in the hospital are depicted in Table 1.

Arterial Blood Gas Analysis revealed a pH of 7.43, PCO2 of 22 mm of Hg, PO2 of 151 mm of Hg with HCO3 of 14.5 mEq/L. Serum LDH was 798 U/L

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Monochloroacetic acid (MCA) is used in the agricultural industry as an herbicide and a bleaching agent for silkworm cocoons. There have been case reports of fatal systemic poisoning following skin exposure to MCA (Kulling et al., 1992; Kush et al., 1990). Toxic manifestations have included severe metabolic acidosis, rhabdomyolysis, renal failure and cerebral edema. Hemolytic uremic syndrome so far has not been reported as MCA toxicity. Blood levels of MCA in this patient could not be estimated due to the lack of facilities. The patient presented had evidence of ongoing hemolysis as evidenced by anemia, raised LDH, unconjugated hyperbilirubinemia, and peripheral smear showing schistocytes with normal coagulation parameters. Corrosive acid ingestion has been known to cause a disseminated intravascular coagulation-like picture; however, our patient had normal coagulation parameters thus excluding DIC. Thrombotic thrombocytopenic purpura (TTP) is unlikely due to absence of fever and neurological signs. Hemorrhagic colitis could have been a direct caustic effect of acid ingestion and has not been described per se in HUS.

In addition to its corrosive action, MCA has found to block the Kreb’s cycle (tricarboxylic acid cycle) and may also cause depletion of sulphydryl groups leading to tissue damage. Rat studies using radiolabelled MCA showed a rapid accumulation in kidney, myocardium liver and small intestinal lumen (Kulling et al., 1992). In HUS platelet fibrin thrombi occur in capillaries, arterioles and sometimes arteries in the absence of vascular inflammation. Mesenteric ischemia, pancreatitis and cardiomyopathy with microthrombi in coronary vessels have been reported in various forms. Extra –renal involvement has also been described in classical D+ HUS (Askiti et al., 2004; Thomas et al., 2005). Our patient had classical features of coronary arterial thrombosis with myocardial infarction, confirmed by grossly elevated cardiac specific enzyme Troponin I, although absolute convincing evidence of coronary thrombosis is lacking since neither a

Table 1. Summary of investigations done in the hospital.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (gm/dl)</td>
<td>6.3</td>
<td>7.1</td>
<td>9.3</td>
<td>11.7</td>
<td>10.7</td>
<td>10.7</td>
</tr>
<tr>
<td>Platelet count (c/mm)</td>
<td>89,000</td>
<td>38,000</td>
<td>35,000</td>
<td>45,000</td>
<td>48,000</td>
<td>50,000</td>
</tr>
<tr>
<td>Blood urea (mg/dl)</td>
<td>310</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>S.creat (mg/dl)</td>
<td>9.2</td>
<td>7.9</td>
<td>6.9</td>
<td>5.4</td>
<td>5.6</td>
<td>–</td>
</tr>
<tr>
<td>Serum Na/K (meq/L)</td>
<td>135/5.5</td>
<td>136/4.1</td>
<td>137/3.8</td>
<td>140/3.7</td>
<td>138/3.8</td>
<td>–</td>
</tr>
<tr>
<td>Troponin I (ng/ml)</td>
<td>16.2</td>
<td>–</td>
<td>–</td>
<td>6.6</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Serum Ca (mg/dl)</td>
<td>10.3</td>
<td>–</td>
<td>–</td>
<td>9.7</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Serum Po4 (mg/dl)</td>
<td>6.0</td>
<td>–</td>
<td>–</td>
<td>4.8</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

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coronary angiogram nor autopsy could be performed.

We postulate that direct MCA toxicity could have contributed to multiple organ involvement in this patient. This case is presented to highlight an as yet unknown toxicity of MCA.

REFERENCES