Brief Report

Acute cyanide poisoning among jewelry and textile industry workers

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Abstract Limited work has focused on occupational exposures that may increase the risk of cyanide poisoning by ingestion. A retrospective chart review of all admissions for acute cyanide poisoning by ingestion for the years 1988 to 2008 was conducted in a tertiary university hospital serving the largest population in the country working in jewelry and textile facilities. Of the 9 patients admitted to the hospital during the study period, 8 (7 males, 1 female; age 36 ± 11 years, mean ± SD) attempted suicide by ingestion of potassium cyanide used in their profession as goldsmiths or textile industry workers. Five patients had severe neurologic impairment and severe metabolic acidosis (pH 7.02 ± 0.08, mean ± SD) with high anion gap (23 ± 4 mmol/L, mean ± SD). Of the 5 severely intoxicated patients, 3 received antidote therapy (sodium thiosulfate or hydroxocobalamin) and resumed full consciousness in less than 8 hours. All patients survived without major sequelae. Cyanide intoxication by ingestion in our patients was mainly suicidal and occurred in specific jobs where potassium cyanide is used. Metabolic acidosis with high anion is a good surrogated marker of severe cyanide poisoning. Sodium thiosulfate and hydroxocobalamin are both safe and effective antidotes.

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1. Introduction

Cyanide inhibits oxidative phosphorylation and causes central nervous system and cardiovascular dysfunction by cellular hypoxia. Cyanide poisoning has a rapidly progressive nature, arising from numerous sources including industrial accidents, food, pharmaceuticals, and fire smoke [1]. Cyanide salts are commonly used in the jewelry industry but are not commonly available to the general population in retail markets. Only approximately 10% of documented cases are suicidal in etiology [2].

Initial clinical manifestations of acute cyanide poisoning are nonspecific, thereby making diagnosis difficult [3,4]. Blood cyanide concentrations should be determined to confirm systemic exposure but are generally not available in due time to be useful for the diagnosis. In addition, blood cyanide concentrations can be misleading or inaccurate [4,5]. Therefore, physicians should be aware of this potentially lethal intoxication, as treating cyanide poisoning effectively means to wage a battle against time.

Victims of acute cyanide poisoning are well known to be susceptible to acid-base disorders due to cellular hypoxia. Cyanide poisoning produces rapid blockade of cellular respiration due to binding to cytochrome a₃, resulting in accumulation of lactate, and lactic acidosis is a recognized hallmark of acute cyanide poisoning in humans [6,7]. In fact, a...
prospective study showed that plasma lactate level of 10 mmol/L or higher is a relatively sensitive marker of cyanide poisoning in fire victims. The most common form of cyanide poisoning occurs by smoke inhalation, whereas less clinical information is available from cyanide poisoning by ingestion [6,8]. A case report of cyanide poisoning by ingestion showed a close correlation between blood cyanide and plasma lactate concentrations [7]. A subsequent study in a limited series of 11 cyanide poisonings resulting from ingestion in all but one patient showed that a plasma lactate of 8 mmol/L is a relatively sensitive marker of cyanide poisoning [8]. However, these were single-center studies precluding any extension to other centers.

In the present study, we report the cases of acute cyanide poisoning by ingestion in our institution, emphasizing the source of exposure and the importance of metabolic acidosis in the assessment of the severity of this intoxication.

2. Methods

We reviewed the records of all patients admitted from 1988 to 2008 at the emergency department (ED) of the Hospital S. João EPE, Porto, Portugal, to identify patients with the discharge diagnosis of acute cyanide poisoning by ingestion. Hospital S. João EPE is a large university hospital located in an urban area serving as a tertiary care center for a local population of approximately 1 million—the largest population in the country working in small- or medium-sized jewelry and textile facilities. The year 2008 had a total of 173,448 ED admissions. Cases of cyanide poisoning involving inhalation of fire smoke were excluded.

Information extracted from hospital records included demographics; circumstances of poisoning and potential sources of exposure to cyanide; clinical presentation; antidotal and supportive treatment; critical laboratory values including acid-base parameters and renal function. Blood cyanide and cyanocobalamin concentrations were not available. Neurologic status was characterized by the Glasgow Coma Scale [9]. Glasgow Coma Scale scores of >12 to <15, 8 to 12, and <8 reflect mild, moderate, and severe neurologic impairment, respectively. Cyanide poisoning was considered as severe if any major organ dysfunction, namely neurologic dysfunction, was present on admission. The relation of acid-base parameters to the severity of poisoning was analyzed. Data were summarized for each patient. No hypothesis testing was undertaken.

3. Results

The sample included 9 patients admitted with acute cyanide poisoning by ingestion (male/female, 7/2; age, 32 ± 15 years) (Table 1). The triggering cause of acute cyanide poisoning was ingestion of potassium cyanide in 8 cases and unintentional intoxication with a jewelry cleaning solution containing sodium cyanide by a 2-year-old female infant. All cyanide poisonings in adult patients (n = 8) were related to suicidal attempts. Six adult patients were jewelry industry workers, working as goldsmiths in a nearby city from Porto. The remaining patients worked in the textile industry.

Individual values on admission to the ED are shown in Table 1. Five patients (cases 2, 3, 4, 6, and 9) presented with severe neurologic impairment, and one of those was in shock (case 6). In these 5 cases, severe metabolic acidosis with high anion gap was invariably present on admission: arterial blood pH 7.02 ± 0.08 (mean ± SD), range 6.90 to 7.10 (reference range, 7.38-7.43); arterial blood bicarbonate 10 ± 4 mmol/L (mean ± SD), range 5 to 12 mmol/L (reference range, 22-26 mmol/L); and anion gap 23 ± 4 mmol/L (mean ± SD), range 24 to 32 mmol/L (reference range, 8-12 mmol/L). Blood lactate levels were only available in 2 patients (cases 6 and 9), 12 and 22 mmol/L, respectively. In the remaining 4 patients (cases 1, 5, 7, and 8), the chief complaints were dizziness, nausea, vomiting, and dyspepsia; and neither major organ dysfunction nor metabolic acidosis was present—arterial blood pH 7.43 ± 0.04 (mean ± SD), range 7.40-7.48 (reference range, 7.38-7.43); bicarbonate 21 ± 3 mmol/L (mean ± SD), range 19-23 mmol/L, (reference range, 22-26 mmol/L).

### Table 1

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Age</th>
<th>Sex</th>
<th>Year of poisoning</th>
<th>Occupational Exposure</th>
<th>GCS score</th>
<th>Arterial pH</th>
<th>Arterial HCO₃</th>
<th>Anion gap</th>
<th>Blood lactate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27</td>
<td>Male</td>
<td>1989</td>
<td>Textile industry</td>
<td>14</td>
<td>7.48</td>
<td>19.6</td>
<td>4</td>
<td>Unknown</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>Female</td>
<td>1990</td>
<td>None</td>
<td>5</td>
<td>6.98</td>
<td>5.2</td>
<td>32</td>
<td>Unknown</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>Male</td>
<td>1991</td>
<td>Jewelry industry</td>
<td>3</td>
<td>7.06</td>
<td>9.1</td>
<td>28</td>
<td>Unknown</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>Male</td>
<td>1995</td>
<td>Jewelry industry</td>
<td>7</td>
<td>6.9</td>
<td>10</td>
<td>26</td>
<td>Unknown</td>
</tr>
<tr>
<td>5</td>
<td>28</td>
<td>Male</td>
<td>1998</td>
<td>Jewelry industry</td>
<td>15</td>
<td>7.41</td>
<td>18.5</td>
<td>6</td>
<td>Unknown</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>Female</td>
<td>2002</td>
<td>Textile industry</td>
<td>6</td>
<td>7.04</td>
<td>12</td>
<td>24</td>
<td>12</td>
</tr>
<tr>
<td>7</td>
<td>39</td>
<td>Male</td>
<td>2003</td>
<td>Jewelry industry</td>
<td>15</td>
<td>7.45</td>
<td>20</td>
<td>14</td>
<td>Unknown</td>
</tr>
<tr>
<td>8</td>
<td>52</td>
<td>Male</td>
<td>2004</td>
<td>Jewelry industry</td>
<td>15</td>
<td>7.4</td>
<td>23</td>
<td>8</td>
<td>2.2</td>
</tr>
<tr>
<td>9</td>
<td>39</td>
<td>Female</td>
<td>2008</td>
<td>Jewelry industry</td>
<td>3</td>
<td>7.1</td>
<td>11</td>
<td>27.8</td>
<td>22</td>
</tr>
</tbody>
</table>

HCO₃ (bicarbonate) is expressed in millimoles per liter; anion gap, millimoles per liter; blood lactate, millimoles per liter. GCS, Glasgow Coma Scale.
On admission, all patients had normal renal function (plasma creatinine, $87 \pm 3 \, \mu\text{mol/L}$ [mean $\pm$ SD]) and toxicology screen was negative. The amount of potassium cyanide ingested was self-reported only in one patient (case 8) who alleged having ingested approximately 1000 mg.

Supportive treatment consisted of (Table 2) ventilatory support and bicarbonate administration in 5 patients (cases 2, 3, 4, 6, and 9); dopamine was administered in 1 patient (case 6).

Three patients (cases 3, 6, and 9) presented with severe neurologic impairment and received antidote treatment—25% solution of sodium thiosulfate in 2 patients (cases 3 and 6: 1.65 mL/kg and 50 mL of sodium thiosulfate, respectively) and hydroxocobalamin in 1 patient (case 9: 5 g of hydroxocobalamin). They all resumed full consciousness in less than 8 hours after admission to the ED. No adverse events were reported. These 3 patients were hospitalized in intermediate care unit, without any organ support therapy. The remaining 2 patients (cases 2 and 4) with severe poisoning were managed without antidote treatment and were hospitalized in intensive care unit for at least 72 hours, requiring oxygen, endotracheal intubation, mechanical ventilation, and cardiovascular support. All patients survived without evident sequelae.

### 4. Discussion

Cyanide salts are commonly used in jewelry, usually to perform a “gold stripping process.” This procedure involves the use of potassium cyanide, hydrogen peroxide (35%), and water. The gold is stripped and made brighter in appearance (1% of gold is lost in the process). This process is used by almost every company in the gold manufacturing industry, especially in smaller and medium-sized facilities. Few reports described cyanide overdoses in which the main source of exposure was related to the jewelry industry [10]. Here, we report 6 cases of acute cyanide poisoning resulting from ingestion of potassium cyanide salt used in the workplace as a jewelry polish cleaner solution (cases 3, 4, 5, 7, 8, and 9). These patients ingested a potassium cyanide containing solution, similar to the “gold stripping process” solution, in suicidal attempts. The dose ingested was only obtained from one patient (case 9). This patient presented with severe neurologic impairment and was treated successfully with hydroxocobalamin. Ingestion of approximately 50-100 mg cyanide salt was reported to be followed by almost instantaneous collapse [11]. Two jewelry industry workers (cases 3 and 4) were admitted with severe neurologic impairment. Finally, the other 3 jewelry industry workers (cases 5, 7, and 8) were admitted with acute onset of dizziness, nausea, vomiting, and dyspepsia after ingestion of a potassium cyanide containing solution. No major organ dysfunction was present.

Two cases occurred in textile industry workers who had free access to potassium cyanide in their workplaces (cases 1 and 6).

Finally, a 2-year-old female infant ingested an undetermined quantity of a metal polish cleaner solution containing sodium cyanide (case 2).

In critically ill patients, acid-base disturbances are nonspecific. However, our data suggest that acid-base disturbances may help physicians to ascertain the severity and prognosis of cyanide overdose. As described by Baud et al [8], serial plasma lactate concentrations can be used as a marker of cyanide toxicity and of adequacy of treatment. In the present study, we report the association between the clinical severity of the intoxication and the degree of the acid-base disturbance. As shown in Table 1, patients admitted to the ED with severe neurologic impairment presented on admission with metabolic acidosis with high anion gap. On the other hand, patients admitted in the ED without neurologic dysfunction presented with normal acid-base parameters. Therefore, as elevated plasma lactate concentrations are a relatively sensitive marker of cyanide poisoning, also arterial pH

### Table 2

**Summary of cases of cyanide poisoning treated with supportive and antidote treatment**

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Supportive treatment</th>
<th>Antidote treatment</th>
<th>GCS on admission</th>
<th>Time to resume GCS = 15</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ventilatory support</td>
<td>Sodium thiosulfate</td>
<td>Hydroxocobalamin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bicarbonate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dopamine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>15</td>
</tr>
<tr>
<td>6</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>15</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>15</td>
</tr>
<tr>
<td>9</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>3</td>
</tr>
</tbody>
</table>

Time to resume GCS = 15 indicates the time between admission to the ED and resuming full consciousness.
value and bicarbonate concentration may help physicians
to classify and to predict the severity of cyanide overdose.
Indeed, a significant positive correlation between the
values of anion gap and the plasma lactate concentrations
and an inverse significant correlation between the arterial
pH and blood cyanide have been shown [8].

Reports do exist of survival of cyanide poisoning with
supportive care only, suggesting that the use of an antidote
may be required only in cases of severe intoxication [12-14].
In the present study, antidote therapy was administered in an
unpredictable way and not always guided by the clinical
severity of the intoxication. Retrospectively, we observed
that cyanide antidotes were not always available in our
hospital (case 2). However, we might say that patients
submitted to antidote therapy (cases 3, 6, and 9) had a faster
neurologic recovery compared to those patients with similar
neurologic impairment treated only with supportive therapy
(cases 2 and 7), as shown in Table 2.

Limitations of this study include its retrospective nature,
the relatively small heterogeneous sample, and the absence
of confirmatory blood cyanide concentrations, preventing us
to draw definitive conclusions. However, we might say that
physicians must be aware that cyanide is available to the
jewelry and textile industry workers and also that metabolic
acidosis with increased anion gap is a good surrogated
marker of the severity of this intoxication.

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