Acute Mercury Poisoning and Mercurial Pneumonitis from Gold Ore Purification

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We describe four men who had symptoms of acute mercury poisoning following exposure to mercury vapor. They were attempting home gold ore purification using a gold-mercury amalgam and sulfuric acid. Three of the four patients required treatment with penicillamine. The clinical and laboratory data are presented along with pulmonary function test results. Long-term follow-up of one patient indicates residual morbidity, with continued reduction in pulmonary diffusing capacity. This suggests permanent impairment of pulmonary function despite prompt chelation therapy. (Chest 1985; 94:554-56)

Long-term mercury poisoning occurs in workers exposed to mercury vapors over a long period. The symptoms usually include, but are not limited to, gingivitis, stomatitis, fine tremor, and irritability. The literature reflects that exposure to this metal has been the source of extreme morbidity.1-4

In contrast to the extensive literature addressing long-term exposure, to our knowledge there is relatively little reported regarding acute toxicity with mercury. Rare cases have been reported of very high exposure to metallic mercury vapor. The organ system that appears to be most affected in these acute exposures is the pulmonary system.

We report four men exposed to mercury vapor while attempting to purify gold ore at home. A gold-mercury amalgam was heated in sulfuric acid, resulting in almost immediate illness in these patients.

CASE REPORTS

Four men, ranging in age from 19 to 58 years, attempted further purification of a piece of gold ore that one of them procured in South America. The gold ore had been partially purified at the source by heating in liquid mercury. In an attempt to separate the gold from the mercury, the men heated the specimen in a solution of sulfuric acid in a small furnace in an unventilated area. Immediately thereafter, the mixture produced noxious green fumes that were irritating to their eyes and respiratory passages. All attempts at further purification were abandoned, and the men sought medical care.

CASE 1

This 19-year-old Hispanic man felt nausea, chest discomfort, and shortness of breath immediately on exposure. He was seen by a physician on the following day and given an unknown over-the-counter preparation. He continued to feel ill and presented to the emergency room of the New York Infirmary-Beekman Downtown Hospital approximately 24 hours after exposure.

On admission, physical examination revealed the following values: blood pressure, 130/100 mm Hg; respiration rate, 18 breaths/min; and temperature, 37.4°C. The physical examination and chest x-ray film showed no abnormalities. Arterial blood gas analysis yielded the following: pH, 7.44; PaO2, 36 mm Hg; and PaCO2, 108 mm Hg on 2-L supplemental oxygen. Because of continuing complaints of chest discomfort, and in view of the history, the patient was admitted for possible mercury vapor intoxication. This diagnosis was confirmed when a random urinary mercury concentration was found to be 24.5 μg/dl (normal <15 μg/dl). Treatment was initiated with penicillamine, 250 mg, orally every six hours. The 24-hour urine mercury excrections, before and after treatment with penicillamine, are given in Table 1. Pulmonary function test results are given in Table 2. The patient's symptoms gradually resolved, and he remained asymptomatic two months after discharge.

CASE 2

This 33-year-old Brazilian man noticed progressive body aches, profound lethargy, headache, shortness of breath, and pleuritic chest pain, as well as nausea and blurring of vision, three hours after exposure. On presentation to the emergency room at New York Infirmary-Beekman Downtown Hospital, the patient appeared

Table 1—24-Hour Urine Mercury Excretion in Three Patients*

<table>
<thead>
<tr>
<th>Time of Sample</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretreatment↑</td>
<td>520</td>
<td>169</td>
<td>NA‡</td>
</tr>
<tr>
<td>Posttreatment↑</td>
<td>640</td>
<td>200</td>
<td>3,600</td>
</tr>
</tbody>
</table>

*All values are in μg/L.
↑Treatment was penicillamine, 250 mg orally every six hours for 24 hours.
‡Not available.

Table 2—Pulmonary Function Test Results in Three Patients Following Acute Exposure to Mercury (% Predicted Values)

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC, L</td>
<td>3.61 (94%)</td>
<td>2.65 (61%)</td>
<td>3.94 (80%)</td>
</tr>
<tr>
<td>FVC, L</td>
<td>3.61 (94%)</td>
<td>2.64 (60%)</td>
<td>2.54 (53%)</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>3.15 (95%)</td>
<td>2.34 (67%)</td>
<td>2.29 (68%)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>87 (87%)</td>
<td>88 (83%)</td>
<td>75 (72%)</td>
</tr>
<tr>
<td>Dsb, ml/min/mm Hg</td>
<td>27.1 (93%)</td>
<td>17.2 (64%)</td>
<td>7.7 (33%)</td>
</tr>
</tbody>
</table>

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flushed, with a labored respiratory rate of 20 breaths/min and blood pressure of 160/100 mm Hg. Bilateral rhonchi were auscultated, and the chest x-ray film was significant for diffuse bilateral interstitial markings, especially in the upper lung fields. His WBC count was $16.9 \times 10^9$/cu mm, and arterial blood gas analysis revealed pH, 7.44; $P_{CO_2}$, 32 mm Hg; and $P_{O_2}$, 76 mm Hg. The alveolar-arterial oxygen gradient was 34 mm Hg (normal <12 mm Hg). Again, because of the history and his symptoms, the patient was admitted to the hospital and started receiving oral penicillinamide, 250 mg every six hours. The 24-hour urine mercury excretion levels before and after treatment with penicillinamide are given in Table 1. Pulmonary function test results are given in Table 2. The patient's symptoms resolved, and he was discharged with no further complaints. However, he later had a recurrence of symptoms and was seen at another hospital, where he received an additional course of chelation therapy. Since then he has returned to Brazil and has been unavailable for follow-up.

CASE 3
This 54-year-old white man presented to the New York Infirmary-Beekman Downtown Hospital approximately 32 hours after exposure complaining of generalized weakness, dry cough, myalgias, shortness of breath, wheezing, and hemoptysis. He had been seen at another hospital, where he was treated for a presumed upper respiratory tract infection. Examination disclosed the following: blood pressure 122/68 mm Hg; pulse rate, 110 beats/min; respiration rate, 20 breaths/min; temperature, 39.4°C. He had bilaterally diminished breath sounds and coarse crackles throughout all lung fields. A chest x-ray film showed a bilateral interstitial infiltrate pattern. The WBC count was 21.9 $\times 10^9$ cu mm. Analysis of arterial blood gases revealed pH, 7.46; $P_{CO_2}$, 32 mm Hg; $P_{O_2}$, 46 mm Hg on room air. The A-a gradient was 64 mm Hg. The blood serum mercury level on admission was 13.4 $\mu$g/dl (normal <1 $\mu$g/dl).

The 24-hour urine mercury excretion levels before and after treatment are given in Table 1. The results of pulmonary function tests are given in Table 2. The patient's hospital course was complicated by persistent hypoxemia, fever, and psychosis. He required transfer to the Medical Intensive Care Unit for supportive care but did not require intubation. He received a course of dimercaprol, 4 mg/kg im, every six hours for the first 24 hours, then every 12 hours for the second 24 hours, then once daily for three additional days. He was also given iv Solu-Medrol infusion at 2 g/day for two days, which was stopped before discharge and changed to prednisone, 20 mg orally three times a day. The patient's symptoms gradually improved, and he was discharged, with follow-up on an outpatient basis. At two months after discharge, he continued to have decreased exercise tolerance and dyspnea on exertion. A random urine mercury level was found to be 88 $\mu$g/dl (normal <15 $\mu$g/dl), with a corresponding serum level of 2.5 $\mu$g/dl (normal <1 $\mu$g/dl). Results of outpatient pulmonary function tests are given in Table 2.

CASE 4
This 46-year-old white man was seen at the emergency room of a local hospital complaining of nausea, shortness of breath, fever, and laryngitis. He was treated for a presumed viral syndrome. He returned to that emergency room the following day with worsening of symptoms but refused admission. He has since refused further follow-up. It is unclear whether he ever had a chest x-ray examination, and his associates were unable to specify the hospital at which he received treatment.

DISCUSSION
Poisoning from inhalation of mercury vapor is well documented in the literature. In the majority of these cases there was long-term exposure. It is believed that oxidation of mercury to mercurous and mercuric ions results in toxic products. The reports of inhalation of mercury vapor resulting in acute poisoning, however, are rare.

The spectrum of symptoms resulting from long-term exposure is broad indeed, ranging from gingivitis and oral stomatitis to irritability and insomnia, the latter being referred to as mercurial erethism. However, in acute poisoning it is frequently the lungs that are most severely affected. In our four patients, too, it was pulmonary complaints that prompted their seeking medical attention.
Bronchiolitis and diffuse pulmonary infiltrates\textsuperscript{5,6} have heretofore been reported as pulmonary complications in acute intoxication. However, a radiographic pattern of interstitial lung disease has been reported in only two previous studies.\textsuperscript{5,7} In addition, these two reports present evidence that the patients involved in the mercury exposure had pulmonary function test results consistent with mild obstructive disease.

In our patients three of four were symptomatic enough to warrant pulmonary function testing. Of those, two had marked reduction in a variety of pulmonary measurements. However, in these cases no evidence of obstructive disease was found. In case 2, the FEV\textsubscript{1}/FVC was 88 percent, but the FEV\textsubscript{1} and FVC were severely reduced, suggesting a restrictive pattern. Studies of diffusion capacity showed a Dsb of 64 percent predicted, which is consistent with restrictive lung disease. Essentially the same picture is presented for patient 3. The potential for long-term residual lung disease in our patients could not be evaluated, since all but one patient refused further follow-up. Patient 3 did continue to have moderately decreased diffusing capacity three months after admission; however, no further follow-up has been obtained.

Our patients reported the use of sulfuric acid in their home purification process. This has heretofore not been associated with gold ore purification and mercury exposure. Certainly, noxious fumes such as sulfur dioxide and sulfuric acid could have contributed to some of their acute complaints. However, their role, if any, in the continued pulmonary complaints is difficult to evaluate. We believe that these compounds played a more immediate role and probably contributed nothing to the overall clinical course.

The treatment of mercury poisoning with either dimercaprol or penicillamine has been shown repeatedly to aid in the clearance of the toxic metal. In our patients, urinary excretion of mercury was dramatically improved following the traditional dosing regimen with these agents. We think that the prompt treatment with chelating agents in acute exposures, as well as the use of IV corticosteroids in severe cases, may prevent symptoms of long-term mercury poisoning, which can result from a single, brief exposure to the agent.\textsuperscript{6}

**CONCLUSION**

We reported the cases of four men who, while attempting home gold ore purification with mercury and sulfuric acid, experienced acute onset of a variety of symptoms. These symptoms ranged from minimal shortness of breath and cough to severe hypoxemia requiring intensive care monitoring. The most severely affected patient demonstrated mild interstitial lung disease both radiographically and on pulmonary function testing. We recommend that in such cases physicians initiate prompt chelation therapy and evaluate the pulmonary function status in these patients. Long-term follow-up is mandatory to determine whether the interstitial disease is reversible or chronic following acute exposure. Unfortunately, it appears that patients who engage in such activities are often uncooperative in seeking medical follow-up.

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**REFERENCES**