The toxic effects of acute mercury vapor inhalation have been described. The clinical picture evolving may be divided into three phases. The initial phase is manifested as a flu-like illness. The intermediate phase involves a period in which severe multi-organ symptoms may manifest. The late phase consists of a period when central nervous symptoms persist. Rare cases with very high acute exposure to mercury vapor have been reported, in which severe respiratory symptoms dominate the clinical picture. The cause of death in all lethal cases is progressive respiratory failure. We experienced a patient with acute respiratory distress syndrome (ARDS) after illicit use of mercury vapor for hemorrhoid treatment; he developed acute chemical pneumonitis following exposure to mercury vapor. Prompt treatment with corticosteroids and penicillamine for acute chemical pneumonitis was instituted; radiologic pulmonary infiltrates disappeared within a week, but late phase neurologic sequelae and pulmonary interstitial fibrosis progressed.

**Key words**: Acute mercury inhalation, Acute respiratory distress syndrome.

**INTRODUCTION**

The toxic effects of acute mercury vapor inhalation have been described. The clinical picture evolving may be divided into three phases. The initial phase is manifested as a flu-like illness. The intermediate phase involves a period in which severe multi-organ symptoms may manifest. The late phase consists of a period when central nervous symptoms persist. Rare cases with very high acute exposure to mercury vapor have been reported, in which severe respiratory symptoms dominate the clinical picture. The cause of death in all lethal cases is progressive respiratory failure. We report the findings in a patient with severe mercury inhalation injury who manifested all the three phases of the clinical picture, including ARDS, and yet survived with treatment using corticosteroid and penicillamine.

**CASE REPORT**

On admission day, a 72-year-old man had attempted illicit use of mercury-lead amalgam to treat hemorrhoids. This procedure was done in a closed room of his house. He became ill with paroxysmal cough, dyspnea, chest pain, nausea and vomiting, but he was unaware of the cause of his illness. He was admitted and then progressively developed dyspnea and respiratory failure. Routine blood chemistries were unremarkable with the exception of arterial blood gases at room air, which revealed pH 7.533, PaO₂ 25 mmHg, and PaCO₂ 21 mmHg. His initial chest mentgenogram was normal. On the second day after exposure, chest radiography revealed bilateral diffuse pulmonary infiltrates (Fig. 1. Top) and bilateral air-space consolidation on high resolution computerized tomography of chest (HRCT), especially in the dependent portion (Fig. 1. Bottom). Transbronchial lung biopsy revealed a chemical pneumonitis, suggestive of pulmonary change of early acute lung injury induced by the exposure to mercury vapors (Fig. 2). The initial urinary mercury concentration was 6402 μg/L. On the third day after exposure to mercury vapor, therapy was initiated with mechanical ventilation and intravenous methylprednisolone pulse (500 mg/day) for 3 days.
followed by oral prednisolone (1 mg/kg) and oral D-
penicillamine (1500 mg/day) for 7 days. The follow-up
chest X-ray showed a much-improved state (Fig. 3. Top)
and follow-up urinary mercury concentration was 25.6
ug/L. Follow-up arterial blood gases at room air revealed
PaO$_2$ 64.9 mmHg, PaCO$_2$ 31 mmHg, pH 7.48. He
improved daily. But on the 16th day after exposure to
mercury vapor, he became disoriented. On physical
examination, his mental status was disoriented and
auscultation revealed fine crackle bilaterally. Brain MRI
showed old cerebral infarction in the right frontal lobe,
and a follow-up HRCT in chest showed interstitial fibrosis
of both lung fields, especially in the dependent portions
(Fig. 3. Bottom). He was treated with second intravenous
methylprednisolone pulse (500 mg/day) for 3 days and then
oral prednisolone (1 mg/kg). Dyspnea improved slightly
but his mental status was aggravated. Pulmonary
symptoms showed a steady state, but mental status did
not improve. He was discharged on the 21st day against
medical advice.

Fig. 1. Top: chest radiograph on admission shows bilat-
eral extensive patchy air-space consolidation.
Bottom: HRCT scan taken 2 days after
exposure shows bilateral air-space consolidation,
especially in dependent portions. Note ground-
glass opacity adjacent to the consolidation.

Fig. 2. There is interstitial edema and small amount of
fibrin deposition. Gray-black granular pigments
suggestive of mercury pigments are noted in the
edematous septae (H&E stain, X200).

Fig. 3. Top: follow-up chest radiograph shows that
bilateral consolidation is markedly improved with
residual opacity in the right lower lung field.
Bottom: follow-up HRCT scan shows mild
peribronchovascular interstitial thickening and mild
interlobular septal thickening, especially in the
dependent portion.
DISCUSSION

Elemental mercury vapor is absorbed rapidly through the alveolar membrane and transported by blood to the brain and other parts of the nervous system. Mercury is rapidly converted to mercuric ions (Hg++)), which are then excreted in the urine and feces. Elimination of elemental mercury occurs primarily in the urine with a half-life of about 60 days. After oxidation, elemental mercury may act as mercuric ion or divalent mercury and thus may be identical to the chemical form that occurs after dissociation of mercuric salts. The danger of mercury vapor is similar to that of ingestion of mercuric chloride or the mercurial diuretics. The same areas of the brain are affected by both inorganic and organic mercury. The tremor, rigidity, truncal unsteadiness and impaired gait may produce a Parkinsonian syndrome suggesting involvement of the basal ganglia and the cerebellum. Involvement of the corpus callosum may be indicated by performance on tests showing no improvement on switching to the preferred from the nonpreferred hand. Defects in memory suggest involvement of the temporal lobe. A urine excretion level of 300mg/L probably represents mercury poisoning; 100 mg/L of mercury in urine requires treatment and levels of 50 mg/L or lower are considered safe. However, urinalysis often yields unreliable results and normal levels have not been clearly established as yet. In our case, the urinary mercury concentration was 6408 mg/L, a level representing acute mercury intoxication.

A clinical picture evolves that may be divided into the following three phases: 1) The initial phase (first few days after exposure) is manifested as metal fume fever or a flu-like illness characterized by chills, fever, achiness, muscles, dryness in the mouth and throat and headache. Toxic pneumonitis with respiratory failure complicate the picture to revere cases, such as the case presented. The intermediate phase (symptoms present 2 weeks after an accident) can be defined as the period during which severe multiorgan symptoms (central nervous system, respiratory tract, gastrointestinal and renal systems) may be manifested. In the respiratory tract, mercury vapor acts as a direct airway irritant and a cellular poison. In mercury vapour inhalation, death has occurred from respiratory failure. Postmortem studies have shown severe damage to the bronchi and bronchioles with marked alveolar edema. In the presence of necrosis, complications such as interstitial emphysema, pneumomediastinum and pneumothorax can occur. The late phase involves the period when central nervous symptoms persist and other organ symptoms resolve. Thus, acute exposure to elemental mercury and its vapor induce acute inorganic mercury toxicity and can cause long-term, probably irreversible, neurologic sequelae. The clinical course of our case showed all three phases.

The pathologic studies show that the histologic picture varied in accordance with how long each patient survived postinsult. Kanik et al. reported that the pathologic findings in the lungs at autopsy reveal various stages of acute lung injury. In our case, at 5 days postinsult, transbronchial lung biopsy showed early acute lung injury, characterized by slight pneumocyte hyperplasia and intra-alveolar fibrin clot with hyaline membrane formation. The consolidation areas on radiograph were predominantly arranged in edematous interstitium. There was also the deposition of gray-black granular pigments in the alveolar septae, highly suggestive of mercury pigments. A minimal interstitial neutrophilic and mononuclear infiltrate was also present. At 16 days postinsult, HRCT showed moderate airspace consolidation and markedly increased interstitial fibrosis, especially in previously edematous portions. Also, emphysema was present in the upper lung field. Lung biopsy was not performed due to the patient's altered mental status. These histologic changes are similar to changes described in the adult respiratory distress syndrome from other causes.

The therapeutic aspect of our case also deserves mention. Corticosteroids have been used sporadically, as reported in the literature. The response to corticosteroids may have been coincidental given the absence of response in the adult respiratory distress syndrome in previous studies. In contrast, we saw beneficial positive response in our case. It has been suggested that steroids may prevent progression to severe interstitial fibrosis if used in mildly affected patients. But the progression of late stage occurred rapidly as we tapered the steroid.

The effectiveness of chelating agent for mercury-induced pulmonary toxicity remains unclear. Penicillamine is generally accepted as an effective chelating agent for mercury. Dimercaptopropanol (BAL) is also effective; however, penicillamine has the advantage of oral administration and is possibly more potent. D-penicillamine, as an oral compound, may be useful in the less severe symptomatic inorganic and elemental mercury inhalation exposures, but its actual value remains to be determined by clinical studies. D-penicillamine reverses sulfhydryl...
binding in the blood and chelates both mercury and lead. N-acetyl-d,L-penicillamine has been administered successfully to patients with inorganic mercury-induced neuropathies (tremor, ataxia) and chronic elemental mercury toxicity\(^1\). Although chelation therapy has been shown to decrease serum mercury concentration, review of the literature shows that this has no effect on progression of acute lung injury. Jaeger et al\(^1\) postulated that lung tissue damage is complete and that the treatment of serum levels with chelating agents has no effect on the reversal of lung damage. In Rowens et al\(^2\) reports, despite reduction in serum mercury levels with dimercaprol, there was also no reversal in the progression of lung injury and respiratory dysfunction. Although a number of authors have advocated the use of N-acetyl-d,L-penicillamine, this modified chelating agent is not currently available in North America\(^7\). Even though it has been reported successful chelation, the effectiveness of D-penicillamine therapy remains unclear.

In summary, we report a patient with acute respiratory distress syndrome after illicit use of mercury vapor for hemorrhoid treatment, who developed acute chemical pneumonitis.

REFERENCES

1. Ellenhorn MJ. Mercury poisoning In: Ellenhorn MJ, ed. Medical Toxicology. 2nd ed. Baltimore: Williams & Wilkins, 1997:1588-862.
2. Rowens B, Guerrino-Betancourt D, Gottlieb CA, Boyes RJ, Eichenhorn MS. Respiratory failure and death following acute inhalation of mercury vapor. Chest 1991;109:459-90.
3. Kanuon S, Gottlieb CA. A clinical pathologic study of four adult cases of acute mercury inhalation toxicity. Arch Pathol Lab Med 1991;115:56-60.
4. Moromisato D, Anas NG, Goodman G. Mercury inhalation poisoning and acute lung injury in child. Chest 1994;105:12-15.
5. Lüs R, Miller A, Lerman Y. Acute mercury poisoning with severe chronic manifestations. Chest 1985;88:306-309.
6. Jaeger A, Tempe JD, Haegy JM, Lemoy M, Pone A, Mintz JM. Accidental acute mercury vapor poisoning. Vet Hum Toxicol 1979;21:63-63.
7. Lien DC, Toddak DN, Rajani HR, Cook DA, Herbert HA. Accidental inhalation of mercury vapor: Respiratory and toxicologic consequences. Can Med Assoc J 1983;129:591-95.