Tohoku J. Exp. Med., 1997, **182**, 347-352

Case Report

Acute Mercury Poisoning by Intentional Ingestion of Mercuric Chloride

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Department of Chemistry, St. Marianna University School of Medicine, [1]Department of Environmental Health Sciences, Tohoku University Graduate School of Medicine, Sendai 980-77, [2]The Second Department of Surgery, [3]Department of Acute Medicine, and [4]Department of Preventive Medicine, St. Marianna University School of Medicine, Kawasaki 216

YOSHIDA, M., SATOH, H., IGARASHI, M., AKASHI, K., YAMAMURA, Y. and YOSHIDA, K. *Acute Mercury Poisoning by Intentional Ingestion of Mercuric Chloride.* Tohoku J. Exp. Med., 1997, **182** (4), 347-352 —— A 26-year-old woman ingested 0.9 g of mercuric chloride in a suicide attempt and developed hematemesis, melena and acute renal failure. Anuria persisted for 14 days. She was treated by plasma exchange, hemodialysis and peritoneal dialysis in combination with continued dimercaprol chelation. While hemodialysis was ineffective in removing the mercury, plasma exchange effectively eliminated mercury. After two plasma exchange therapies, mercury concentration in the blood decreased linearly on a log scale with half-lives of 23.1 days for whole blood and 19.1 days for plasma, using first-order kinetics. One month after ingestion, renal function recovered to normal as judged by serum creatine and blood urea nitrogen levels, although the β_2-microglobulin level in urine was still elevated. At a follow-up examination four months later, renal function was found to be completely normal. This indicates that the renal damage caused by acute mercuric chloride poisoning may not be permanent. —— mercuric chloride; inorganic mercury poisoning; renal failure; plasma exchange; hemodialysis

Inorganic mercury poisoning usually occurs as a result of occupational exposure. However, accidental, criminal or suicidal ingestion has also produced poisoning at non-occupational settings (Samuels et al. 1982; Worth et al. 1982; Sauder et al. 1988).

Ingested inorganic mercury causes corrosive and necrotic lesions in the upper gastrointestinal tract. After absorption from the gastrointestinal tract, it is

Received December 25, 1996; revision accepted for publication June 13, 1997.

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distributed mainly to the kidney and liver (Berlin 1986). Acute inorganic mercury poisoning causes gastrointestinal symptoms and renal failure. Therefore, the kidney and gastrointestinal tract are the main target organs for inorganic mercury.

Inorganic mercury poisoning has traditionally been treated with chelating agents, dimercaprol (BAL) and D-penicillamine, to eliminate mercury from the body (Berlin 1986). Recently, hemodialysis, hemoperfusion and peritoneal dialysis with concomitant BAL-therapy have been widely used to treat acute mercury intoxication. Nevertheless, there have been few reports on the effectiveness of such treatment in removing mercury from the body (Samuels et al. 1982; Sauder et al. 1988; McLauchlan 1991).

In the present case of acute intoxication by mercuric chloride, we attempted plasma exchange therapy in addition to dialysis. Blood mercury levels and renal function were monitored during the patient's hospital stay and followed-up examinations were done four months later.

Case Report

A 26-year-old woman working at an agricultural research center ingested 0.9 g of mercuric chloride (equivalent to 0.66 g of mercury) in a suicide attempt. Three hours later, she was sent to a local hospital and treated with gastric lavage. She was then transferred to our emergency lifesaving center and placed in the intensive care unit. On admission she had dyspnea, hematemesis, laryngeal edema and melena. Endoscopic examination revealed necrotic lesions of the esophageal and gastric mucus. Her blood pressure was 110/78 mmHg. Heart rate was 112/min and regular. Cardiopulmonary examination revealed no abnormal findings. The patient was intubated because of dyspnea and laryngeal edema, and treated with BAL, 400 mg i.m. per day.

Twenty-four hours after ingestion, the patient became anuric and her condition deteriorated. She was then treated with plasma exchange to remove the mercury. Three days later, progressive increases in serum creatinine and blood urea nitrogen (BUN) levels indicated acute renal failure (Fig. 1A). The patient was treated with hemodialysis six times in total for the acute renal failure, and subsequently with continuous ambulatory peritoneal dialysis (CAPD). The anuria persisted until 14 days after ingestion, when diuresis occurred (Fig. 1C).

Serum creatine and BUN levels returned to normal levels about one month after ingestion, indicating an end of renal failure. However, urinary β_2-microglobulin (β_2-MG) levels had not returned to normal ($<300\ \mu$g/liter) at that time (Fig. 1B). At a follow-up examination about four months later, the patient's renal function was normal and there were no symptoms of poisoning.

Methods

Hemodialysis therapy was performed using a model BK-1.2U hemodialysis

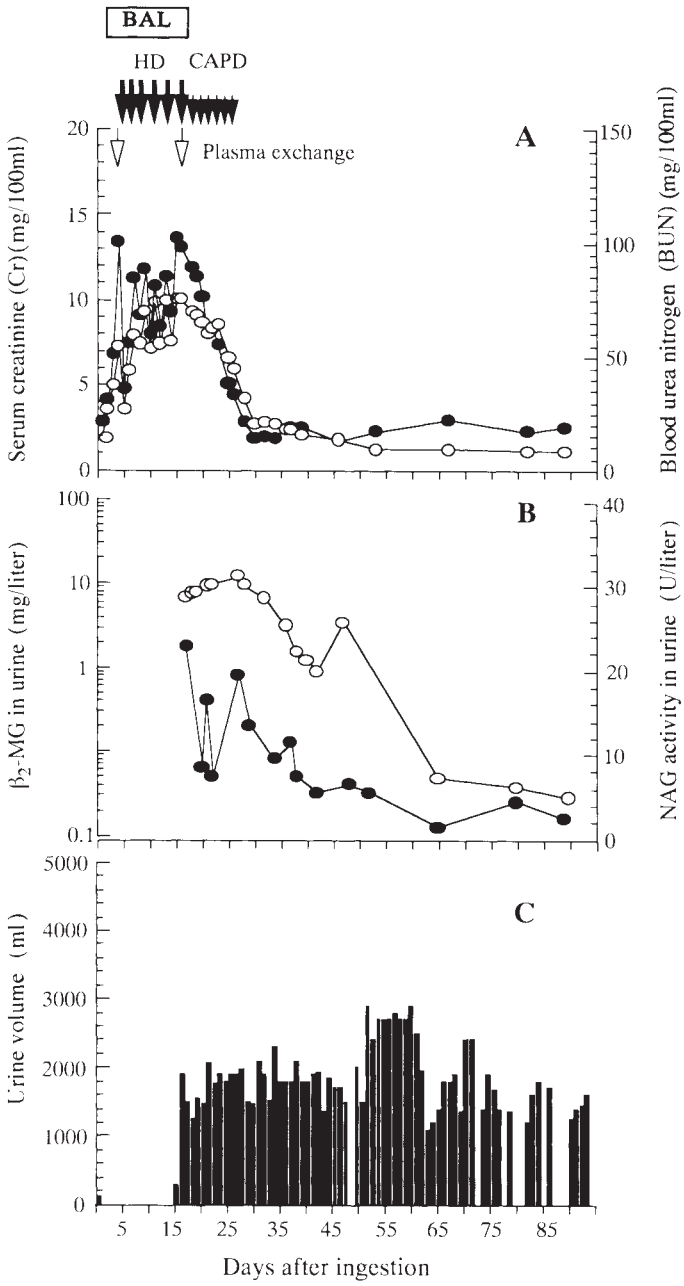

Fig. 1. Results of renal function tests during the hospital stay of a patient who had ingested 0.9 g of mercuric chloride.
(A) Serum creatinine (Cr, ○—○) and blood urea nitrogen (BUN, ●—●) values; (B) urinary β_2-microglobulin (β_2-MG, ○—○) and β-N-acetylglucosaminidase (NAG, ●—●) activity; (C) urine volume. Missing bars indicate days on which urine volume was not measured.

unit (Toray Co., Tokyo) operating at 150 ml/min. Blood samples for mercury analysis were collected at the start and end of hemodialysis.

CAPD was performed with PD-4 1.5 dialysis solution (Baxter Limited, Tokyo), using three 2000 ml exchanges a day.

Mercury concentrations in the blood and urine were determined by cold vapor atomic absorption spectrometry after wet digestion (Yoshida et al. 1986). Samples were digested with nitric acid in Uniseal decomposition vessels (Uniseal Ltd.,

Haifa, Israel). The accuracy of this method was assured by using a reference material, NIST Oyster (SRM 1566, National Institute of Standard and Technology, Washington D.C., USA). The detection limit of the method was 0.5 ng Hg.

Results

Plasma mercury concentrations at the start and end of each hemodialysis and plasma exchange treatment are shown in Tables 1 and 2. Hemodialysis did not significantly decrease plasma mercury concentration. The mercury concentration after hemodialysis was somewhat higher than before on three occasions.

On the other hand, mercury concentration at the end of the first plasma exchange treatment was only about 12% of the value at the start of treatment and was about 67% of the pretreatment value after the second treatment. During the second treatment (a 2.4-liter plasma exchange), approximately 2.0 mg of mercury was removed. The plasma exchange clearance, calculated using the formula proposed by Sauder et al. (1988), was 9.7 ml/min.

Changes in mercury concentration in whole blood, plasma and urine during the patient's hospital stay are shown in Fig. 2. The whole blood mercury concentration decreased rapidly on a log scale over time. The half-life of the whole blood mercury was estimated to be 23.1 days, assuming linear first-order kinetics. The plasma mercury concentration decreased abruptly at first, rebounded slightly after 14 days, and then decreased again at a constant rate. The half-life of the plasma mercury, based on the linear part of the curve, was estimated to be about 19.1 days.

Table 1. *Plasma mercury concentrations at the start and end of hemodialysis treatments*

Hemodialysis	Hg concentration in plasma (ng/ml)	
	Start	End
1st	2030	2090
2nd	2240	1830
3rd	1520	1860
4th	1250	1430

Table 2. *Plasma mercury concentrations before and after plasma exchange treatments*

Plasma exchange	Hg concentration in plasma (ng/ml)		
	Before	After	Exchange[a]
1st	4680	563	n.d.[b]
2nd	1140	762	785

[a]Hg concentration in the exchanged plasma; [b]n.d., not determined.

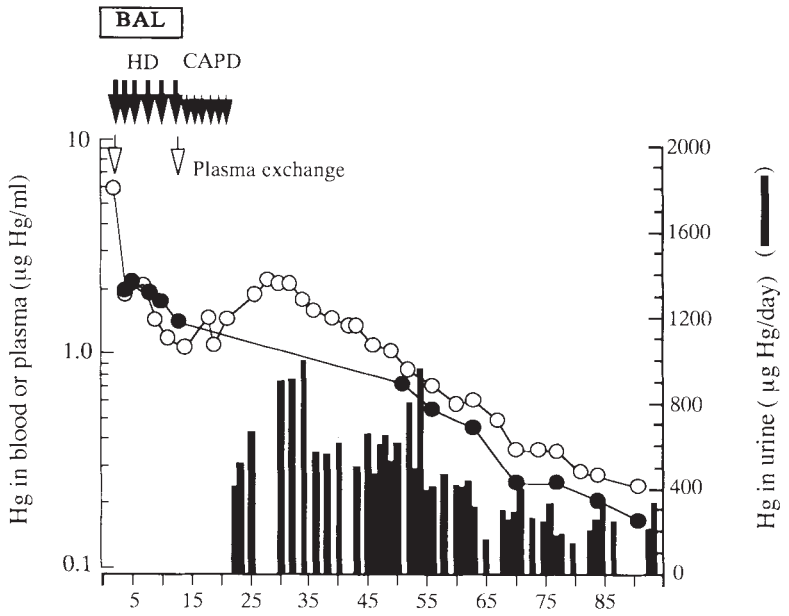

Fig. 2. Time courses of blood (●—●) or plasma (○—○) mercury concentrations and urinary mercury excretion. Mercury levels were not determined for all urine samples.

Discussion

The estimated lethal dose of mercuric compounds for an adult is 0.2-1.0 g (Mogos 1975; Clarkson 1989). The mechanism of the toxicity is tubular cell damage and consequent acute renal failure (Clarkson 1989).

After ingesting 0.9 g of mercuric chloride, the patient in the current study exhibited the typical signs and symptoms of acute inorganic mercury toxicity i. e., burning in the mouth and throat, corrosive trauma to the gastrointestinal tract and rapid development of renal failure. The patient was treated with hemodialysis, CAPD and plasma exchange combined with continued BAL chelation.

Hemodialysis, hemoperfusion and peritoneal dialysis have been reported to have little efficacy or to be completely ineffective in removing mercury. In our patient, the mercury concentration at the end of hemodialysis treatments was higher than the concentration at the start. The results indicate that the mercury clearance was negative and that the hemodialysis was ineffective in eliminating mercury. The higher mercury concentrations at the end of treatment may be attributable to hemoconcentration during hemodialysis.

In contrast, the clearance value for the second plasma exchange treatment was approximately 10 ml/min, though this value was slightly lower than the 17.3 ml/min obtained by Sauder et al. (1988). Mercury elimination by plasma exchange proved much more effective than hemodialysis therapy in acute inorganic mercury poisoning.

A rebound of plasma mercury concentration owing to the release of mercury

from the tissue was observed following the second plasma exchange therapy. This phenomenon suggests that repeated plasma exchange may be effective in removing mercury that was deposited in the tissues.

The mercury concentration in whole blood decreased linearly on a log scale. The half-life of whole blood mercury was calculated to be about 23.1 days. On the other hand, the half-life of plasma mercury was 19.1 days. Yamamura et al. (1991) reported that the half-life of plasma mercury in chronic mercury poisoning due to exposure to mercury vapor was triphasic: 10, 28 and 105 days, respectively. The half-life of the second phase is comparable with the value obtained for acute inorganic mercury poisoning in the present study.

Renal function improved gradually upon resumption of urination. Serum creatinine, BUN and urinary β-N-acetylglucosaminidase activity returned to the normal values about one month after ingestion, but the urinary β_2-MG levels, reflecting impairment of tubular function (Pence et al. 1977), were higher than normal for a longer period. Nevertheless, the renal failure was resolved completely four months later, indicating that renal failure caused by acute inorganic mercury poisoning produced no permanent renal damage.

Acknowledgment

The help by Dr. J. Raymond during preparation of the manuscript was appreciated.

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