

Original Article

Association between acute severe mercury poisoning and multiple organ failure: a case report

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Abstract: Acute mercury poisoning, involving a number of organs, leads to severe dysfunctions, such as acute renal failure (ARF), and even threatens patients' lives. A case of acute severe mercuric chloride (HgCl_2) poisoning with multiple organ failure was reported in this study. A 38-year-old woman orally took about 50 g HgCl_2 powder in 2015, and showed nausea, emesis, clouding of consciousness, lip and nail cyanosis, and dark red bloody fluid from bilateral nostrils. Based on chest and abdominal CT examinations, gastroscopy, and colonoscopy, the patient was found to suffer oral mucosal hyperemia and ulceration, gastrointestinal bleeding (haematemesis and hemaecia), ARF, metabolic acidosis, collapse and shock. Despite assisted respiration and relevant active treatments, the patient's condition deteriorated gradually and she was dead eventually. The study suggests that the best treatments for acute HgCl_2 poisoning accompanied with ARF are early blood purification and mercury elimination on the basis of conventional therapy.

Keywords: Mercuric chloride, poisoning, acute renal failure

Introduction

Mercury is a kind of silver liquid metal and can volatilize at room temperature. Both mercury and its compounds induce intoxication through steam inhalation, skin inhalation, ingestion and injection. Acute mercury poisoning is mainly induced by oral administration or the inhalation of mercuric chloride (HgCl_2) compounds. Its occurrence can cause corrosive stomatitis, gastroenteritis and acute pulmonary edema within a few or dozens of minutes after contacts, accompanied by nausea, emesis, abdominal pain, and diarrhea. Besides, severe cases may simultaneously bear multiple organs injuries, including circulatory failure, gastrointestinal perforation and respiratory failure, as well as acute renal failure (ARF) with liver damage within 3-4 days (within 24 hours in serious cases) and even death despite active treatments [1-3]. Therefore, acute mercury poisoning should be paid more attention due to its damages and high lethality. In order to deepen the understanding of mercury poisoning and its practical significance in clinical work, we cur-

rently reported a severe poisoning case orally taking HgCl_2 who was admitted in our hospital on April 4th 2015.

Case report

A 38-year old female was of HgCl_2 poisoning. She orally took about 50 g HgCl_2 powder at 21:00 on March 31st, 2015 and afterwards developed nausea and emesis. Vomitus was bloody gastric contents which were untreated [4]. The patient was found to be unconscious on the bed by her family members at 01:00 on April 1st, with lip and nail cyanosis and dark red blood released from bilateral nostrils. Then the patient was sent to local municipal hospital at 01:40, with a blood pressure of 50/30 mmHg and 1000 ml hemaecia after examinations. She received not only tracheal intubation to connect breathing machine for assisted respiration, but also the treatments of fluid infusion, expansion, vasopressors, and plasmapheresis. Tracheal intubation was removed when the patient became conscious at 6:30. Around 14:00, she was sent to the affiliated hospital of

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the local medical university to receive milk gastric lavage, mercury removal, acid suppression, liver protection and neuro nutrition. The above therapies were operated in the intensive care unit (ICU) at 20:00 due to the patient's confused consciousness. The patient's blood pressure was 135/83 mmHg, and blood gas analysis (FiO₂ 21%) parameters were: pH 7.24, pO₂ 79.1 mmHg, pCO₂ 26.6 mmHg, and lactic acid 7.5 mmol/L. Blood routine examination showed that white blood cell count was 38.37×10⁹/L, and the percentage of neutrophils was 95%. Moreover, other physiological indexes were as follows: blood myoglobin, 1337.4 ng/ml; creatine kinase, 675.5 U/L; creatine kinase isoenzyme, 105.4 U/L; glutamic-pyruvic transaminase, 135.6 U/L; glutamic oxalacetic transaminase, 1118.5 U/L; and serum amylase, 1217.7 U/L. Bleeding and coagulation function test displayed that prothrombin time and activity were 26.7 s and 23%, respectively, activated partial thromboplastin time (APTT) was 45.7 s, international normalized ratio (INR) was 2.36, and fibrinogen was 0.63 g/L. The patient was diagnosed with acute oral HgCl₂ poisoning, multiple organ dysfunction syndrome (MODS), and digestive tract hemorrhage, so further therapies were implemented, including milk gastric lavage (200 ml/2 h) for the promotion of mercury discharge, hemoperfusion, bedside blood filtration, anti-infection, the scavenging of oxygen free radicals, liver protection, and neuro nutrition. Nonetheless, the patient's clinical condition continued to deteriorate and progressively became severer. Once again, orotracheal intubation was performed, and breathing machine was adopted. The patient was transferred to our hospital for further treatment at 18:50 on April 4th, 2015.

At the time of admission, the patient was under drug-induced sedation, and dark red liquid was drained through an indwelling gastric tube. Retention catheterization was conducted. The patient's average urine volume was less than 100 ml per 24 hours and pale red bloody urine occurred. In addition, her excrement was same as before but change in her weight was unknown. Because the patient had been suffered depression 4 months after her delivery, she seldom communicated with others. Her family members acknowledged her postpartum depression, but denied other medical history for her. Physical examination results contained:

T 37.2°C, P 79 times/min, R 17/min and BP 145/90 mmHg. The patient was kept in sedation, and showed the xanthochromia of whole skin, obvious edema, and multiple ecchymoses (such as subcutaneous ecchymosis near puncture point, around 1×1 cm subcutaneous ecchymosis inside the right upper eyelid, 37×28 cm subcutaneous ecchymosis on the left groin, 5×5 cm subcutaneous ecchymosis on the right ilium, and swelling and ecchymosis on the bilateral labium). Besides, the patients also developed congestion and edema of bilateral bulbar conjunctivas, pale palpebral conjunctivas and icteric sclera. Bilateral pupils were of the same size with a diameter of 2 mm; light reflex was slow and corneal reflex disappeared. Plenty of bloody secretions and bright red blood clots were sucked out from nasal cavities, but there was no abnormal secretion in her ears. Meanwhile, orotracheal intubation was fixed and unobstructed with a scale of 21 cm to suck out more bloody secretions. The patient's lips seemed pale but not cyanotic. Lung respiratory sound was thick, while the left side generated more intensive sounds than the right side, and a few diffused moist rales were audible. The heart rate was 79 beats per minute with regular rhythm, but the heart sound was weak. No pathological murmur was audible from each auscultatory valve area. The abdomen was distent with high tension, and bowel sound appeared twice per minute. Moreover, circumferences greatly differed between legs, 48 cm and 32 cm above and below the left knee, and 44.5 cm and 31.5 cm for the other one.

Auxiliary examinations for the patient were carried out in the department. Blood gas analysis (FiO₂ 40%) was also conducted and the results were as follows: pH 7.384, pO₂ 45.5 mmHg, pCO₂ 47.6 mmHg, BE 3.3 mmol/L, and lactic acid 2.9 mmol/L. Blood routine test showed a white blood cell count of 12.95×10⁹/L, a neutrophil percentage of 83.2%, the hemoglobin of 66 g/L, and a platelet count of 5×10⁹/L. Biochemical detection reached a high-sensitivity C-reactive protein (hs-CRP) of 107 mg/L, a blood albumin of 26 g/L, a total bilirubin of 35 μmol/L, a creatinine of 182 μmol/L, a blood calcium of 1.89 mmol/L, and a lactic dehydrogenase of 2752 U/L. Coagulation function test showed a D-dimer ration of 6640 ng/m, a whole blood troponin-I of 0.311 ng/ml, a whole blood myoglobin of 1306 ng/ml, a B-type natri-

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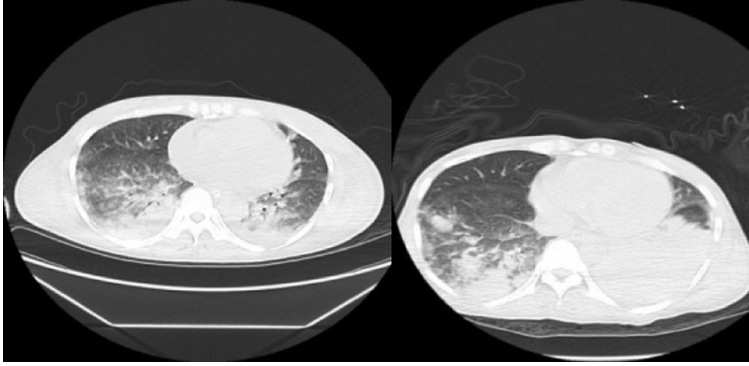


Figure 1. Chest CT examination on April 9th. Various pathological changes were observed, such as pulmonary vascular markings, multiple patches and nodular fuzzy shadow in two lungs, and fluid density shadow in both thoracic cavity.

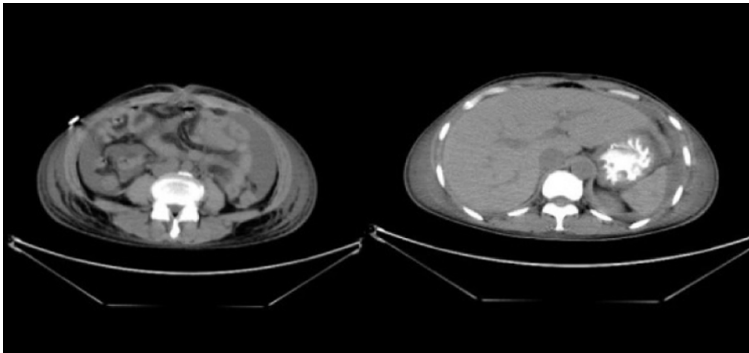


Figure 2. Abdominal CT examination. Several structure disturbances were visible in abdomen, including turbid intraperitoneal mesenteric fat space and fluid density shadow in abdominal cavity, high density shadow in stomach and intestinal tubes, and cystiform high-low hybrid density shadow and flocculent density shadow in pelvic cavity.

uretic peptide (BNP) of 10092 pg/ml, and procalcitonin greater than 200 ng/ml. Five tests on thyroid function revealed a thyronine of 0.49 nmol/L, a thyroxine of 59 nmol/L, FT3 at 2.6 pmol/L, FT4 at 11.81 pmol/L, and a thyroid stimulating hormone (TSH) of 0.41 Uiu/ml. Routine stool test showed positive occult blood. Chest radiography uncovered two pulmonary exudative lesions. Ultrasonic cardiogram implied mild pulmonary valve and mitral valve regurgitation as well as mild to moderate tricuspid valve regurgitation. Bilateral pleural effusion was found through routine examination. Abdominal ultrasound showed a weak echo in gallbladder cavity, resulting in suspicious cholestasis, increased echo of double kidney cortex, and abdominal pelvic effusion.

On admission, the patient was diagnosed with acute severe HgCl₂ poisoning (oral), MODS

(including respiratory failure and ARF), toxic liver injury, hemorrhage in digestive tract, colporrhagia, urethrorrhagia, nasal bleeding, toxic myocardial injury, coagulation disorders, postpartum depression, and subcutaneous ecchymosis [5]. Breathing machine was applied to assist respiration (model: SIMV; parameters: oxygen concentration at 60%, respiratory rate at 16 beats/min, positive end-expiratory pressure at 5 cmH₂O, pressure support at 14 cmH₂O, and tidal volume at 410 ml). Erythrocytes, plasma, and platelets were transfused; furthermore, hemodiafiltration, albumin supplementation, anti-acid (protection of gastrointestinal mucosa), and nutritional support were also implemented for further treatment [6]. Abdominal puncture was performed at 16:50 on April 7th to drain out dark red uncoagulated blood. Abdominal viscera bleeding was suspected. Ascites cultivation was implemented with pan-drug resistant enterococcus faecium. Serum amylase exhibited a continuous elevation, suggesting the occurrence of toxic

acute pancreatitis. Chest CT examination on April 9th (**Figure 1**) revealed increased pulmonary vascular markings, multiple patches, nodular fuzzy shadow, consolidation shadow and stripe shadow in two lungs. Besides, fluid density shadow was visible on both sides of thoracic cavity and adjacent lung tissues. Abdominal CT examination (**Figure 2**) demonstrated structure disturbance of intestinal canal in abdomen. Intraperitoneal mesenteric fat space was turbid, and a large amount of fluid density shadow was visible in abdominal cavity. In addition, multiple high density shadows were visible in stomach and partial intestinal tubes. Cystiform high-low hybrid density shadow was observed in pelvic cavity, in which flocculent density shadow was visible. According to gastroscopy on April 10th (**Figure 3**), large pieces of mucosa exfoliation were observed in upper jaw when pipe reached oral cavity, which

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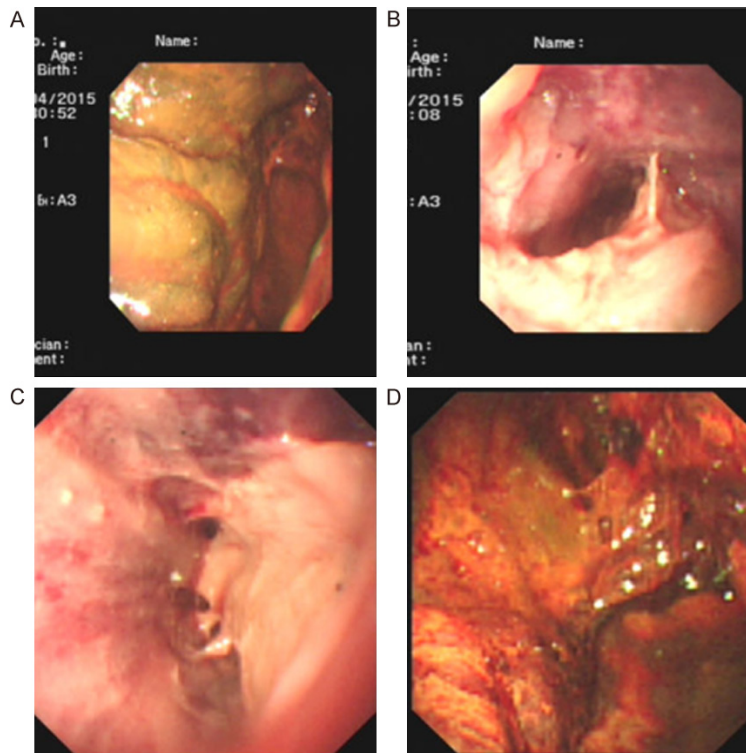


Figure 3. The results of gastroscopy on April 10th. The gastric body was covered by lot of black substances, namely HgCl₂, and bloodstain (A). Meanwhile, plenty of mucosa exfoliation and erosion were observed in upper jaw, esophagus (B, C) and gastric body (D).

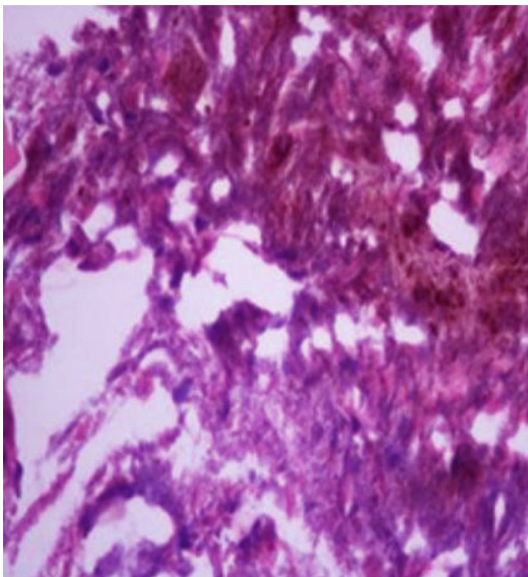


Figure 4. Pathological diagnosis confirmed the large amount of mucosa exfoliation in upper jaw. In addition, inflammatory cells' infiltration was also identified.

was later identified through pathological diagnosis (**Figure 4**); mucosal exfoliation and ero-

sion were also visible in intact esophagus with bloodstain. Large pieces of mucosa exfoliation were observed in gastric body when pipe reached gastric cavity; gastric body was covered by lots of black substances and adhered by bloodstain; gastric antrum deformation, mucosal erosion or exfoliation, and pylorus deformation were visible as well. At the descending part of duodenum, mucosa seemed to be more smooth than that at stomach and esophagus, and erosion, namely anabrosis, was invisible. Consequently, erosive gastritis and esophagitis were determined through endoscopic diagnosis. Pathological results showed that there were three pieces of lesser curvature sides in gastric body, namely grey shapeless tissues (about 0.2 cm in diameter). Pathological diagnosis based on light microscope indicated three pi-

eces of lesser curvature sides in gastric body and a large amount of coagulative necrosis (**Figure 5**). Colonoscopy on April 10th (**Figure 6**) displayed that congestive spots and fresh blood residual were scattered on the mucosas of cecum, ascending colon, transverse colon, descending colon and sigmoid colon (especially at ileocecal junction, namely rectum) when pipe reached ileocecal junction after entering 80 cm. The 24-hour urine protein was 1.6 g/L. Electrocardiograph (ECG) revealed it was normal with sinus rhythm. Cardiac ultrasound unveiled mild mitral regurgitation.

Blood anaerobic bacteria culture was conducted using pan-drug resistant acinetobacter baumannii on April 12th and urine culture was performed under the same conditions on April 14th. Cytomegalovirus antibody was inspected with IgG at a dosage of 92.4 U/ml on April 14th.

Despite active treatments, jaundice and infection still became more severer [7]. The patient's condition had deteriorated despite the therapies of plasma exchange and anti-infection (meropenem, Tigecycline and genciclovir). Biochemical re-examination showed hs-CRP at

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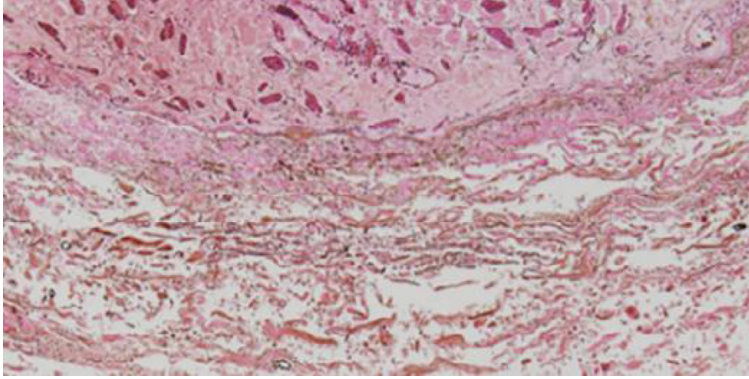


Figure 5. Pathological diagnosis identified a great deal of coagulative necrosis in gastric body and three pieces of lesser curvature sides ($\times 200$).

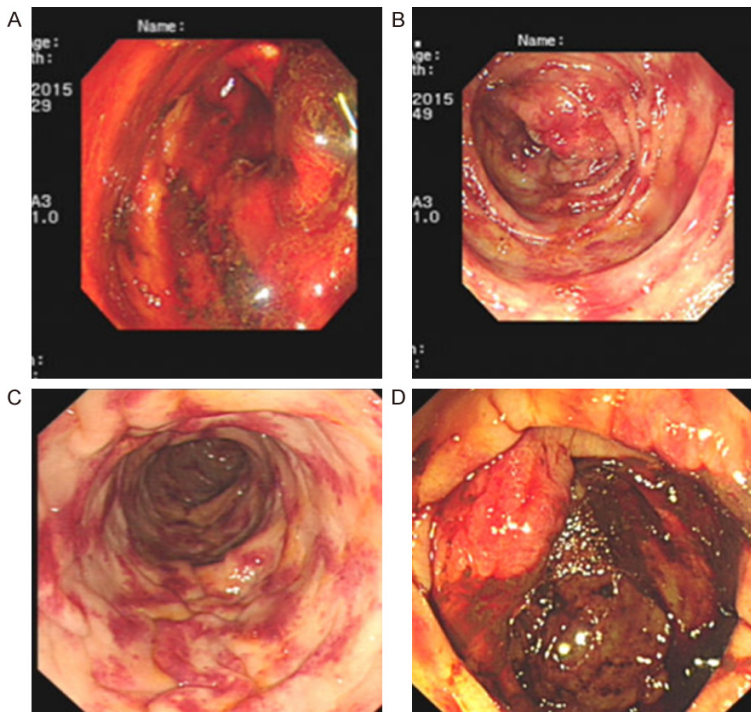


Figure 6. The results of colonoscopy examination on April 10th. It was apparent that congestive spots and fresh blood residual were scattered on the mucosa of enteric cavity (A). Besides, mucosa erosion was also observed in enteric cavity (B-D).

380 mg/L, total bilirubin at 507.1 $\mu\text{mol/L}$, and direct bilirubin at 284.9 $\mu\text{mol/L}$. BNP was greater than 35000 pg/ml persistently. The patient died of the illness on the 17th day after positive therapy.

Discussion

Mercury is the only liquid metal on earth. Besides, there also exist organic mercury and

inorganic mercury in nature. In 1950s, people began to realize the toxicity of mercury and its compounds because of “Minamata disease”. In addition, a large number of clinical data and experiences have been accumulated in the long course of preventing mercury poisoning, revealing a reality that mercury could damage multiple organs. Inorganic mercury is usually pulverescent, and mainly includes mercury chloride (HgCl_2). Digestive tract and skin absorption is the common way of poisoning, due to some inappropriate behavior like suicide or wrong oral administration [8-12]. It has been reported that HgCl_2 is easily soluble in water [13], and can induce erosive lesions in gastrointestinal mucosa after oral administration, which is mainly featured by chemical necrotizing gastroenteritis, abdominal pain, diarrhea (watery stool and bloody stool), and even collapse and shock [14, 15]. The clinical manifestations of acute mercury poisoning were associated with dosages and individual differences. For the case with oral HgCl_2 poisoning in our report, all of the above mentioned manifestations were observed, including oral mucosal hyperemia and ulceration, gastrointestinal bleeding (haematemesis and hema-fecia), ARF, metabolic acidosis, collapse and shock, which were basically consistent with the results obtained in previous literature.

The combination of mercury ions with hemoglobin or plasma proteins could form a diffusion of mercury, which, through blood flow, could be distributed in various tissues and organs such as liver, large intestine and small intestine, especially kidney. Therefore, patients with acute mercury poisoning are frequently accom-

panied with function failures in multiples organs. Moreover, the combination of mercury ion with sulfhydryl enzyme in body may conduce to the degeneration and necrosis of renal proximal convoluted tubule, the blockage of kidney tubule and renal parenchymal lesion, thereby resulting in oliguric ARF. In addition, mercury ions could also inactivate cytochrome oxidase and pyruvate kinase, and affect functional groups, impeding cell biological activities and normal metabolism and then leading to cell degeneration and necrosis. In our study, the patient presented multiple organ failures, which were manifested by ARF, gastrointestinal bleeding and deep coma, and such failures acted as the major cause for poisoning-related deaths.

The best treatment method for acute HgCl_2 poisoning accompanied with ARF refers to blood purification and mercury elimination as early as possible on the basis of conventional therapy [16, 17]. The patient did not receive blood purification in the local hospital, and her condition was significantly improved after plasmapheresis in our hospital. In addition, mercury elimination among cases suffering serious damage of renal function might aggravate renal injury, so it has been suggested that HgCl_2 should be eliminated after the recovery of renal function. Gastric lavage should be carried out prudentially for patients with oral HgCl_2 poisoning, and the best time for such operation is within 1 hour after poisoning; otherwise, it might exacerbate digestive tract injury.

In summary, mercury poisoning has attracted more and more attentions in recent years. The symptoms of mercury poisoning should be eliminated in time, through early detection, cutting off drug source and operating mercury treatment. For cases with severe mercury poisoning, mercury elimination should be performed at once to reduce the toxicity. In addition, more attention should be paid to organ protection to reduce death rate.

Disclosure of conflict of interest

None.

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