CASE REPORT

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ACUTE MERCURIC CHLORIDE POISONING AT A POTENTIALLY LETHAL DOSE ENDED WITH SURVIVAL: SYMPTOMS, CONCENTRATION IN CEREBROSPINAL FLUID, TREATMENT

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Abstract
This study aims to present a case of acute mercuric chloride poisoning at a potentially lethal dose treated with the antidote – 2,3-dimercapto-1-propanesulfonic acid (DMPS) and continuous renal replacement therapy (CRRT) combined with CytoSorb. A 21-year-old woman was admitted to a hospital with abdominal pain, vomiting, and suspected gastrointestinal bleeding after taking 5000 mg of mercuric chloride for suicidal purposes. Due to the patient deteriorating general condition and multiple organ damage, on the third day she was transported to the Clinic of Anaesthesiology and Intensive Care (CAaIC), Łódź, Poland. Laboratory tests confirmed features of acute kidney injury and high mercury levels in the blood (1051 μg/l) and urine (22 960 μg/l) – DMPS therapy and CRRT combined with CytoSorb were instituted. Due to nervous system complaints (headache, dizziness), a lumbosacral puncture was performed – the mercury concentration in the cerebrospinal fluid (CSF) was 5.45 μg/l. During a colonoscopy, significant diagnostic abnormalities revealed features of colonic mucosal necrosis. The treatment resulted in a decrease in subjective complaints, decreased mercury levels in biological material, and improved parenchymal organ function. On the 15th day of therapy, the patient was transferred to the primary care center for further treatment. The case confirms the possibility of improvement of patient condition following ingestion of a potentially lethal dose (5 g) as a result of the initiation of appropriate therapy even on the third day. The presence of mercury in CSF confirms that inorganic mercury compounds (mercuric chloride) can pass through the blood-brain barrier after oral ingestion. Int J Occup Med Environ Health. 2023;36(5):685–92

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INTRODUCTION

Scientific reports of mercuric chloride poisoning cases relate primarily to the ingestion of the poison for suicidal purposes or accidentally [1–3]. The mechanism of mercuric chloride toxicity is not fully understood. On the one hand, it may result from a direct oxidative effect of mercury ions, which consequently leads to damage to the gastrointestinal mucosa and renal tubules; on the other hand, mercury ions may form many stable complexes with proteins and other compounds (affinity of mercury to sulfhydryl, carboxyl, amino, and amino acids groups) disrupting their functions, thus leading to disruption of biochemical processes in the body [1,4–6]. Material accumulation of inorganic mercury leads to the formation of radicals damaging proteins and DNA (oxidative stress) and to lipid peroxidation. The described mechanisms of toxic action may lead to the development of metabolic acidosis, which may result in severe systemic disorders leading ultimately to the death of the patient [1,5,7].

This study aims to present a case of acute mercuric chloride poisoning in the amount of 5 g successfully treated with a specific antidote – 2,3 dimercapto-1-propanesulfonic acid (DMPS) and continuous renal replacement therapy (CRRT) combined with CytoSorb ended with survival. In the analyzed clinical case, mercury concentration in cerebrospinal fluid (CSF) was monitored in addition to the typical analysis of the examined parameter in blood and urine.

CASE REPORT

A 21-year-old woman was admitted to the surgical ward of a district hospital with suspected gastrointestinal bleeding (upon admission she vomited and reported abdominal pain). During the medical interview, it was established that she had ingested 5 g of mercuric chloride in powder form (suicide attempt). Laboratory and endoscopic diagnostics were implemented (Table 1). Gastrofiberoscopy revealed only erosive lesions without features of perforation or active bleeding into the gastrointestinal tract.

Due to deteriorating general condition and features of multiple organ damage (WBC 24 × 10^3/µl, HGB 9.7 g/dl, UREA 27.63 mmol/l, CREA 884.0 µmol/l, serum amylase 402 u/l, CRP 215 mg/l – day of discharge), she was sent on the third day to the Clinic of Anaesthesiology and Intensive Care (CAaIC), Łódź, Poland, for specific treatment due to clinical symptoms of acute poisoning. On admission, she reported only a slight headache, the patient was in a fairly severe general condition, Glasgow Coma Scale (GCS) 15 pts; respiratory and circulatory efficient, heart rate was 90 bpm; blood pressure was 135/70 mm Hg. On physical examination, abnormalities included auscultation over the lung fields with furcations and thick-bel-

Table 1. Summary of the laboratory tests performed in the patient in district hospital and in the Clinic of Anaesthesiology and Intensive Care (CAaIC) on the admission day (first day of the hospitalization), Łódź, Poland

Parameter	Test result on the first day		Reference range
	district hospital	CAaIC	
White blood cell [×10^3/µl]	27	17.45	4.00–11.00
Hemoglobin [g/dl]	15.4	9.9	12.0–16.0
Urea [mmol/l]	7.30	28.33	2.8–7.2
Creatinine [µmol/l]	209.50	1031.4	49.0–90.0
C-reactive protein [mg/l]	–	211	0.0–5.0
Amylase [u/l]	159	94	22.00–88.0
Procalcitonin [µg/l]	–	17.46	<0.5

lied rales, intermittent moist cough (PCR for SARS CoV-2 infection was negative), abdominal pain in the lower abdomen, and anuria (<0.06 ml/kg/h of urine was recorded in the hourly urine collection bag).

Immediately after admission to the CAaIC, the determined blood and urine mercury concentrations were 1051 μg/l and 22 960 μg/l, respectively. The mercury concentration in CSF on the first day of therapy (DMPS and CRRT) was 5.45 μg/l, whereas on the ninth day (CRRT combined with CytoSorb) it was 1.1 μg/l.

Imaging studies upon admission to the CAaIC showed the following abnormalities:

- Abdominal ultrasound – kidneys with slightly hyperechoic parenchyma and markedly contrasting hypoechoic pyramids (suspicion of acute kidney injury).
- Chest X-ray – in segment 1 of the right lung, in segment 3 of the left lung, in the segment of the uvula, and the left lower lobe, airy areas of thickening of the milky glass type (suspicion of inflammatory changes), presence of fluid in the pleural cavities: right 18 mm, left 27 mm; atelectatic changes above the fluid in the lower lobe of the left lung.
- Colonoscopy – from about 5 cm from the anus extensive inflammatory changes with deep crater-shaped ulcerations covering 75% of the intestinal perimeter with mucosal edema and lumen narrowing. Above, black mucosal necrosis was visible. Due to the risk of perforation, further insertion was abandoned (Figure 1).

During the next 10 days of hospitalization, the patient complained of a slight headache, dizziness, dryness of oral mucous membranes, nausea, belching, and taste disorders – the patient began to report a meaty taste of water and tingling in the left upper limb. On physical examination, the following abnormalities were observed: increased attacks of moist cough, a tendency to severe drug-resistant hypertension (RR 190/100 mm Hg) and slow heart rate – min. 55 bpm despite the pharmacological treatment (among others: antibiotics, red blood cells,

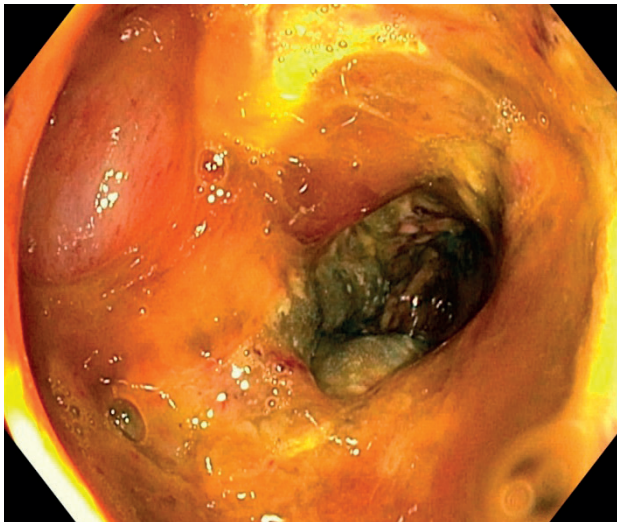

Figure 1. Colonoscopic examination presenting inflammatory and necrotizing changes in the large intestine after mercuric chloride ingestion in the patient in the Clinic of Anaesthesiology and Intensive Care (CAaIC), Łódź, Poland

fresh frozen plasma, protein pomp inhibitors, urapidil, nitroglycerine, ebrantil). The pallor of the skin was observed, the presence of bloody bruises on the upper limbs, transient abdominal bloating, and slight bleeding from the lower gastrointestinal tract and the genital tract.

A psychiatric consultation was carried out (day 4), during which abnormalities included: psychomotor retardation; the patient was hypomimic, emotionally indifferent, answered questions with a delay, and „admits that it is difficult for her to collect thoughts".

During hospitalization, the patient underwent nephrological, surgical, and gastroenterological consultations to determine the diagnostic and therapeutic management. In laboratory tests, in addition to anemia, features of renal and pancreatic organ damage were noted: GGTP 128.9 u/l (range 0.0–38 u/l); ALAT 47.7 u/l (range 0.0–35 u/l); AspAT 45 u/l (range 0.0–35 u/l).

Due to acute kidney injury and anuria on admission to CAaIC, CRRT continuous veno-venous hemodialysis (CVVHD) Ci-Ca was used; the procedure was subsequently converted to continuous veno-venous hemo-

Table 2. Mercury concentration in the biological materials before and after treatment of the patient in the Clinic of Anaesthesiology and Intensive Care (CAaIC), Łódź, Poland

Testing time	Mercury concentration [µg/l]	
	urine[b]	blood (serum/plasma)[c]
Admission to the CAaIC	22 960	1051
Ninth day of therapy[a]	724	580

[a] 2,3-dimercapto-1-propanesulfonic acid (DMPS) *i.v.* + continuous renal replacement therapy 1st–5th day; continuous renal replacement therapy 6th–9th day.
[b] Admissible concentration in biological material, biological exposure index <5 (7 µg/l).
[c] Reference values according to Human Biomonitoring Commission in case of not occupationally exposed children and adults: 5 µg/l (concentration below which there is no risk of adverse health effects).

diafiltration (CVVHDF) Ci-Ca with ultrafiltration to optimize therapy; on the third day of therapy, CytoSorb was added to the artificial kidney system (the procedure lasted 6 h; the study results are in preparation for publication). The average dose of therapy was respectively: 48 ml/kg/h for CVVHD and 63 ml/kg/h for CVVHDF.

Analyzing the entire clinical picture consistent with acute mercuric chloride poisoning and the results of toxicological tests, the specific antidote – DMPS was included. The first dose was administered on the third day after mercuric chloride ingestion, at a dose of 250 mg *i.v.* every 6 h for the next 3 days. Then, the dose was reduced to 250 mg *i.v.* every 12 h, and finally 250 mg *i.v.* every 24 h. On the 15th day of therapy, the patient was transferred to a district hospital in optimal condition for the course of the underlying disease, where the current process of therapy was continued together with pharmacological treatment and haemodialysis.

In addition, symptomatic treatment was provided throughout the hospitalization according to the general principles of intensive therapy, depending on the abnormalities found during the physical examination and laboratory tests.

DISCUSSION

This article presents a case report of acute mercuric chloride poisoning after ingestion of a potentially lethal dose of 5 g. The lethal oral dose for adults ranges 1–4.0 g, although the lowest doses causing toxic symptoms may not exceed 0.5 g [4,8]. The dietary absorption of inorganic mercury compounds in humans is approx. 7–10% [9,10]. In studies conducted on volunteers, the average absorption in the gastrointestinal tract was found to be about 5% of the administered dose [10,11], although oral intake of mercuric chloride in high doses may increase its absorption through the gastrointestinal mucosa due to irritation [4]. The above-mentioned situation may have occurred in the analyzed clinical case considering the symptomatology and the changes found in the performed endoscopic examinations.

Ingestion of inorganic mercury salts causes: salivation, burning in the esophagus, vomiting, bloody diarrhea, necrosis of the intestinal mucosa, and damage to renal function leading to anuria and uremia [5,8]. A case with vomiting, diarrhea, stabbing abdominal pain, oesophageal pain, ulceration, hemorrhage along the entire gastrointestinal tract, and acute renal failure was reported in the literature. A post-mortem examination revealed macroscopic renal changes (swelling and pallor) and abscesses of the occipital lobe of the brain and cerebellum [6]. The authors of another report described the case of a woman who ingested about 30 mg/kg of mercuric chloride and developed severe abdominal pain, diarrhea, nausea, and vomiting as well as acute renal failure manifested by oliguria, proteinuria, hematuria, and presence

of granular rolls in urine [12]. The described complications related to gastrointestinal irritation of mercuric chloride as well as a renal failure were also observed in the patient. The kidneys are the main site of mercury accumulation after exposure to its inorganic compounds. The mechanism of mercury accumulation in the kidneys is the binding of metal ions to low molecular weight proteins (metallothioneins) via the cysteine sulfhydryl group. These proteins with protective functions against harmful metal cations are mainly located in the cytosol of hepatocytes, and nephrons, but also enterocytes and neurons (intestine and brain) [11]. In a study on the acute toxicity of mercuric chloride in mice given this poison once by intraperitoneal route, damage to proximal renal tubules was observed, accompanied by a dose-dependent induction of stress proteins (heat shock proteins [HSP] 2, HSP60, HSP72 and glucose-regulated protein [GRP] 75) [13]. Other cases have reported the presence of nephropathy, which was histopathologically defined as foci of alkaline cells with a thickened basement membrane and reduced cytosol, located within the ileum of proximal tubules; vitreous rollers were found in some ileal tubules [14].

The elevated blood pressure observed in the patient was most probably related to acute renal failure, although a toxic effect of mercuric chloride on the myocardium cannot be excluded. The mechanism of toxic action of the poison on the myocardium is not fully understood, most probably it is related to lipid peroxidation (accumulation of hydrocarbons, aldehydes, and hydroxyaldehydes), as well as to increased oxidative stress due to loss of metallothionein function and accumulation of free radicals (reactive oxygen species). In addition, mercury can also induce disturbances in the activity of numerous important enzymes-oxidoreductases, including superoxide dismutase (SOD) [15].

In the analyzed clinical case, an increase in biochemical markers correlating with liver cell damage (ALAT, AspAT, GGTP) was noted. In experimental studies on rats that were administered intragastrically mercuric chloride, liver damage was confirmed, measured by the activity of indicator enzymes ALAT and GGTP; moreover, a decrease in the reduced glutathione/oxidized glutathione (GSH/GSSG) marker of oxidative stress and an increase in the antioxidant enzymes glutathione transferase (GST), SOD and glucose-6-phosphate dehydrogenase (G6PD) [11,16,17].

The inflammatory changes described in the chest X-ray, accompanied by the symptoms reported by the patient in the form of increased productive cough, should rather be associated with respiratory tract infection, on the other hand, mercury may have an immunosuppressive effect and lead to an increased incidence of infections [11,18].

The normocytic anemia observed in laboratory studies is most likely secondary and results from severe hemorrhagic gastrointestinal injury due to the corrosive effect of the poison [1,5].

During hospitalization, the presence of mercury in the CSF was confirmed on the third day after the poison intake, and the patient-reported symptoms of dizziness, slow thinking processes, and tingling of the upper limbs, which may indicate neurotoxic effects of inorganic mercury compounds. The repeated assessment of mercury concentration in the CSF on the ninth day of therapy revealed a decrease in the poison concentration. The analysis of medical records from the in-patient observation in the district hospital (2 days of hospitalization) showed that the above-mentioned symptoms did not occur immediately after mercuric chloride ingestion by the patient. The results of the study may indicate that under conditions of acute poisoning, the blood-brain barrier is not or only very slowly penetrated by inorganic mercury compounds at an early stage, and therefore neurological symptoms are absent or, if present, only in the form of subtle disturbances. It is only after several days (in the analyzed case on the third day after intake), when mercury undergoes numerous transfor-

mations in metabolic cycles, that headaches, coordination disorders, and tremors may appear. A study by Sabbe et al. [19], describing a fatal case of a patient after mercuric chloride ingestion, also points to a weaker distribution of inorganic mercury to nervous tissue; the highest concentration was confirmed in gastric mucosa at 6624 μg/g, while concentrations of this poison in the brain and cerebellar tissues were much lower at 1.4 and 1.2 μg/g, respectively. In an experimental study in which mercuric chloride solution was administered subcutaneously to rats for 6 weeks, only 0.01% of the total dose administered was deposited in the brain and about 3% in the kidneys [10].

Determination of mercury concentrations in blood and urine on the third day after intake was elevated and was: 1051 μg/l and 22 960 μg/l. The highest blood mercury concentration in a patient after ingestion of an inorganic mercury combination, that is mercury sulfide, was described by Dargan et al [1]. In the third hour after ingestion, the concentration of the above-mentioned xenobiotic was 15 580 μg/l, and the patient developed life-threatening symptoms of respiratory and circulatory failure with signs of gastrointestinal irritation. Based on the literature data, it can be assumed that mercury concentrations in urine exceeding 100 μg/l are indicative of poisoning, and symptoms of nervous system damage are also noted in the clinical picture [4]. The authors of another report indicate the possibility of clinical symptoms of poisoning at mercury concentrations >300 μg/l in urine and >200 μg/l in blood [20]. The assessment of mercury concentration in urine is important to evaluate the effectiveness of chelation therapy, but it should be emphasized that no correlation is found between mercury concentration in blood or urine and clinical symptoms of poisoning [4,5].

The patient was not decontaminated in the gastrointestinal tract or given activated charcoal. The use of the procedures mentioned above in poisoning with inorganic mercury compounds is not recommended due to possible damage to the gastrointestinal tract and doubts about the effectiveness of absorption of the poison by carbon; the administration of carbon favors the provocation of vomiting, prevents the control of bleeding and subsequent endoscopic assessment [3,4]. On the other hand, the authors of other reports indicate that the above-mentioned procedures may be performed only when the time from poisoning is no longer than 1 h and the dose is potentially lethal. Inorganic mercury compounds are poorly absorbed from the gastrointestinal tract and for this reason, gastric lavage applied very early after poisoning can eliminate a large part of this substance. In this case, these time criteria were not met.

In the course of poisoning with inorganic mercury compounds, thiol chelating agents such as DMPS are recommended. This antidote administered *i.v.* or orally competes with natural -SH groups included in proteins, effectively eliminating mercury from tissues and accelerating its excretion in urine [5].

It is also possible to remove inorganic mercury from the blood by extracorporeal elimination procedures. In the literature, clinical cases of poisoning with inorganic mercury combinations treated with combined therapy have been described: British anti-Lewisite (BAL) with hemodialysis, BAL with peritoneal dialysis, DMPS with dialysis, BAL with hemoperfusion, BAL/DMPS with plasmapheresis, hemodialysis, and hemodiafiltration [1,5,21,22], as well as a case of combined DMPS therapy with CVVHDF [1]. In the majority of literature data, the efficacy of extracorporeal elimination procedures was debatable except for the last report in which the authors used combined therapy with hemodiafiltration (the type of filter used during therapy enabled to remove from the body about 12.7% of the absorbed dose of the poison, mostly during the first 72 h of therapy). The patient was successfully treated with renal replacement therapy initially with CVVHD Ci-Ca;

the procedure was subsequently converted to CVVHDF Ci-Ca with ultrafiltration to optimize the therapy, on the third day of therapy CytoSorb was added to the artificial kidney system (this is the first report of the use of the above-mentioned filter in renal replacement therapy in this type of poisoning, study results are under preparation for publication).

The conversion from CVVHD to CVVHDF was aimed to increase the elimination of the poison by using the convection mechanism simultaneously with the previously used diffusion in extracorporeal therapy.

CytoSorb was added to the CVVHDF system due to the unsatisfactory dynamics of toxin elimination despite the escalation of the dose of extracorporeal treatment and potential mechanisms of elimination after obtaining information from the manufacturer of CytoSorb about the potential ability to bind mercury.

CONCLUSIONS

The described case, although quite typical in terms of symptoms occurring as a result of acute oral exposure to mercury chloride, confirms the possibility of improvement of the patient clinical condition after ingestion of a potentially lethal dose (5 g) as a result of the initiation of appropriate therapy, even on the third day after ingestion. The confirmed presence of mercury in CSF confirms the possibility that inorganic mercury compounds (mercuric chloride) can pass through the blood-brain barrier after oral intake.

Author contributions

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Statistical analysis: Anna Krakowiak, Łukasz Sadowski
Interpretation of results: Anna Krakowiak, Waldemar Machała
References: Łukasz Sadowski

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