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CASE REPORT

Hidden Mercury Poisoning Causing Pulmonary Embolism, Nephrotic Syndrome, and Hypothyroidism

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SUMMARY

Background: **Mercury is a highly toxic heavy metal, and mercury poisoning often presents with different symptoms in multiple systems. While lung and kidney damage have been studied separately, involvement of the endocrine system has rarely been reported. We report on a case with pulmonary embolism, nephrotic syndrome, and hypothyroidism in a woman who used a mercury-containing whitening cream.**
Methods: **Chest enhancement CT; Laboratory tests.**
Results: **Chest enhancement CT suggested the presence of pulmonary embolism. Laboratory tests suggested nephrotic syndrome and hypothyroidism, and blood and urine were positive for mercury. The patient made a full recovery after treatment with immunosuppressants and mercury chelators.**
Conclusions: **This case demonstrates the need for greater public awareness and stricter regulatory action to prevent health hazards associated with mercury in skin-lightening products.**
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KEYWORDS

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CASE REPORT

Whitening creams are cosmetics that are applied to the surface of the skin. They include creams, lotions, ointments, gels, and soaps, and they promise a fairer, smoother complexion [1]. Mercury has the name “melanocyte poison”, which can kill melanocytes and make the skin white, so individual merchants will add “mercury” ingredients to whitening products. Mercury is a highly toxic heavy metal that can damage the lungs, kidneys, digestive tract, skin, nerves, and many other systems, but there are few reports of damage to the endocrine system. Consumers may be unknowingly exposed to the health effects of mercury toxicity in their pursuit of beauty. Due to the lack of specificity in the clinical manifestations of mercury poisoning, it is easy to be misdiagnosed and missed. We therefore describe a case

of chronic mercury poisoning complicated by nephrotic syndrome, pulmonary embolism, and hypothyroidism. The patient is a 37-year-old woman with a long history of cosmetic work. She was admitted to the hospital with left-sided intermittent abdominal pain for 4 days, accompanied by nausea and vomiting, but without significant chest pain or hemoptysis. One day ago, the patient was checked locally for chest enhancement CT results suggesting (Figure 1): 1. inflammation of the lower lobes of both lungs; 2. embolism of the right lower pulmonary artery branch; and 3. a small amount of pleural effusion. She was admitted to the hospital with a preliminary diagnosis of pulmonary embolism and pneumonia. Subcutaneous anticoagulation with low molecular heparin injection of 0.4 mL twice a day and anti-infective treatment with piperacillin-tazobactam 4.5 g IV three times a day were given. After admission to the hospital, laboratory tests were completed: blood test: leukocyte 9.7 x 10^9/L, erythrocyte 5.26 x 10^9/L, platelet 384 x 10^9/L, hemoglobin 160 g/L. Coagulation series: D dimer 4,124 ng/mL, plasma fibrinogen 5.25 g/L. Blood biochemistry: total protein 37.9 g/L, albumin 17.4 g/L. Urinalysis: Urine protein 3+, occult blood+. Hepatitis B, hepatitis C, HIV, female tumor series, rheumatology, and immunology series were normal. Five thyroid function tests: TT4 39.470 nmol/L, TT3 1.110 nmol/L, TSH 14.270 μIU/mL, FT4 8.690 pmol/L, FT3 2.140 pmol/L, diagnosed as hypothyroidism, and levothyroxine sodium tablets 12.5 μg/day were given as replacement therapy. On September 25, 2024, Quantitative analysis of urinary protein: The 24-hour quantitative urinary protein was 19.2 g/24 hour. The patient was given methylprednisolone 40 mg/day and low molecular heparin 5000 iu subcutaneous injection twice a day. The next day, because the patient appeared to have a facial rash, it was changed to prednisone acetate 50 mg/day oral treatment. During the course of treatment, we inadvertently noticed the patient's facial whiteness and traced the patient's medical history and found that the patient had been using White Beauty Cream for five months, so we tested her blood and urine for mercury. On October 2, 2024, the blood mercury level was 11.65 μg/L, and the urinary mercury level was 62.48 μg/g (the reference range is less than 35 μg/g; the urinary mercury level is more reliable for determining clinically significant mercury exposure). Therefore, we considered that the patient had chronic mercury poisoning, and the treatment plan was changed to sodium dimercaptopropyl sulfate injection 0.25 g/day intramuscular mercury repellent therapy once a day for 3 days (days 1 - 3), stopping for 4 days (days 4 - 7), and cycling for 2 cycles, with a total of 6 injections. Since this patient had pulmonary embolism and was on anticoagulants, we could not perform a renal puncture biopsy to clarify the etiology, so we could only empirically give compound cyclophosphamide tablets 50 mg/day. On October 4, 2024, the 24-hour urinary protein quantification was 9.52 g/24 hour. On October 5, 2024, the coagulation series was reviewed: D-dimer 181 ng/mL, plasma fibrinogen 2.71 g/L. Therefore, low molecular heparin was discontinued and rivaroxaban 20 mg/day was given. On October 14, 2024, the 24-hour urinary protein quantification was 3.74 g/24 hour. On October 18, 2024, the urinary mercury was 11.71 μg/g. On October 28, 2024 the 24-hour urinary protein quantification was 0.54 g/24 hour. On November 12, 2024, the urinary mercury was 6.47 μg/g. The trend of urinary mercury levels is visualized in Figure 2. On November 25, 2024, the 24-hour urinary protein quantification was 0.04 g/24 hour. The trend of urinary protein levels is visualized in Figure 3. On November 25, 2024, the five items of thyroid function were FT3 3.9 pmol/L, FT4 17.6 pmol/L, and thyrotropin 5.61 μIU/mL. Cyclophosphamide was stopped (total amount of 4.4 g), and the hormone was reduced to 30 mg/day for 7 days, then to 20 mg/day, and then reduced to 5 mg/day every week. After the above treatment, the patient's urinary mercury and urinary protein became negative, and the thyroid function was generally normalized.

DISCUSSION

Epidemiological data show that mercury poisoning is mainly caused by occupational exposure to mercury, abuse of mercury-containing compounds, or skin-lightening cosmetics. Mercury and its compounds cannot exist as free mercury ions or inorganic mercury salts after being absorbed into the human body through the respiratory, digestive, and skin routes, but are combined with sulfhydryl-containing biomolecules [e.g., glutathione (GSH), cysteine (Cys), homocysteine (Hcy), N-acetyl cysteine (NAC)] and albumin, etc. [2]. All forms of mercury are nephrotoxic, and inorganic mercury, which is the most toxic form of mercury in cosmetics, accumulates in the kidneys in about 80% of cases after entering the human body; therefore, the kidneys are the most frequently involved target organ in mercury poisoning [3]. Several studies have shown that long-term exposure to mercury-containing preparations can lead to kidney damage, and the most common types of kidney pathology reported in the literature are membranous nephropathy and microscopic lesion nephropathy. Therefore, in female patients with renal disease, it is important to ask about the history of use of skin-lightening and expectorant cosmetics and to monitor urinary mercury, if necessary, especially if skin-lightening and expectorant effects are evident. In our patient, the retrospective patient's history was free of any other common secondary causes of nephropathy, and she had never used potentially pathogenic medications. After the presence of mercury poisoning and the treatment of mercury expulsion, the patient's urinary mercury decreased from 62.48 ug/g to 6.47 ug/g, and his urinary albumin decreased from 9.52 g/24 hour to 0.04 g/24 hour, thus confirming the etiology of this nephrotic syndrome as chronic mercury poisoning.

Chronic exposure to or ingestion of inorganic mercury

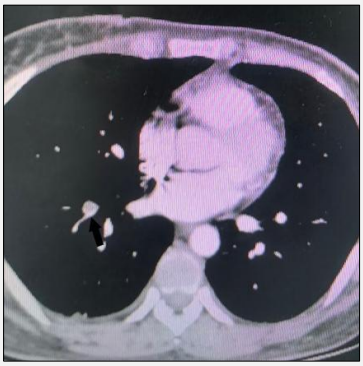

Figure 1. Chest enhancement CT suggesting embolism of the right lower pulmonary artery branch.

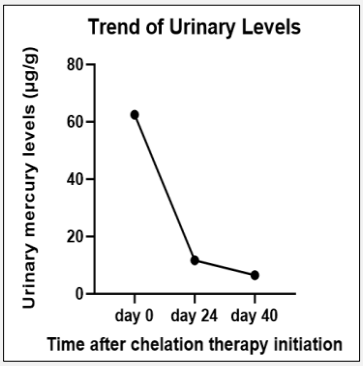

Figure 2. The trend of urinary mercury levels.

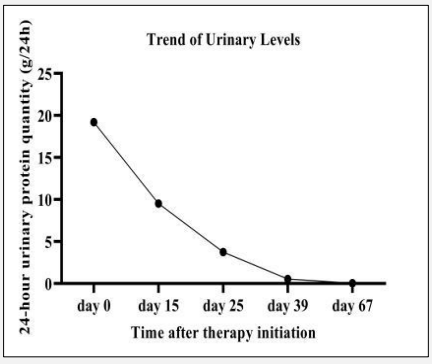

Figure 3. The trend of urinary protein levels.

may lead to inflammation of the pulmonary vasculature, which in turn damages endothelial cells, and this damage may promote thrombosis leading to pulmonary embolism. Chronic mercury poisoning may also have a secondary hypercoagulable state associated with impaired thrombomodulin/protein C systems and an imbalance in fibrinogen activators. Chronic mercury poisoning can lead to kidney damage, and nephrotic syndrome is also a factor in the hypercoagulable state of the blood. Currently, most reports of pulmonary embolism due to mercury poisoning are of self-administered intravenous mercury injection or ruptured thermometers, and reports of pulmonary embolism due to chronic mercury poisoning from the use of whitening creams are rare [4, 5]. It is worth noting that the clinical manifestations of mercury poisoning are often nonspecific, such as weakness, insomnia, tremors, etc., and are easily overlooked or misdiagnosed, thus delaying treatment. Therefore, patients with unexplained pulmonary embolism, especially with chronic kidney disease or hypercoagulable states, should be questioned in detail about their history of mercury exposure, including the use of cosmetics, in order to exclude the possibility of mercury poisoning. Mercury can cause hypothyroidism through several pathways. First, mercury is a known promoter of autoimmune responses and may induce autoimmune thyroiditis (e.g., Hashimoto's thyroiditis), and chronic thyroid inflammation can eventually progress to hypothyroidism [6,7]. Secondly, mercury induces the production of oxygen-free radicals in thyroid follicular cells, causing oxidative damage and thus interfering with thyroid hormone synthesis and secretion [8,9]. In addition, the genotoxicity of mercury may affect the normal metabolism and repair capacity of thyroid cells [10]. Therefore, mercury exposure may impair thyroid function through mechanisms such as autoimmune attack and oxidative stress, and become one of the potential triggers of hypothyroidism. Currently, animal experiments have confirmed that the thyroid gland is characterized by the absorption and accumulation of mercury [11-13], but since it is impossible to fully simulate the characteristics of long-term low-dose exposure and bioaccumulation in human beings, it is difficult to directly transfer these results to human beings, and therefore, domestic and international reports of hypothyroidism caused by mercury poisoning are rare. In our patient, thyroid function returned to normal after a short period of mercury-lowering therapy without hormone supplementation and replacement therapy. We hypothesized that this impairment or suppression of function may be reversible, at least in the short term. It remains to be seen whether the damage or suppression of thyroid function caused by long-term accumulation of mercury or its compounds in the thyroid gland is reversible.

CONCLUSION

This case presents a patient with atypical symptoms and multi-system damage on admission, with clinical manifestations involving thrombosis, nephropathy, and endocrine system, suggesting that physicians need to be on high alert for the possibility of mercury poisoning or other heavy metal poisoning. However, there are some areas for improvement, as the patient was on anticoagulation therapy and had a high risk of bleeding, a renal puncture biopsy could not be performed to clarify the pathologic diagnosis. Finally, we should increase our health education efforts to remind patients to stay away from substandard whitening products, which is an important measure to prevent the recurrence of similar diseases.

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Ethical Approval:
This study was approved by the ethics committee of North China University of Science and Technology Affiliated Hospital. All procedures performed in the studies were in accordance with the ethical standards. Informed consent was obtained.

Declaration of Interest:
No conflicts of interest.

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