

Severe Cadmium Poisoning with Multiple Organ Dysfunction Syndrome Caused by Long-term Betel Quid Chewing: A Case Report

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Abstract

Cadmium (Cd), a toxic heavy metal commonly found in the environment, is mainly absorbed by the body through the respiratory tract, digestive tract and skin. Due to its characteristics of airtight and tasteless, Cd poisoning can be concealed and easily misdiagnosed in hospitals. However, chronic cadmium poisoning may lead to a variety of diseases, severe cases can lead to multiple organ dysfunction syndromes (MODS). To better understand the diagnosis and treatment of Cd poisoning, we report a successful diagnosis and treatment of Cd poisoning in a young man with multiple organ failure.

Keywords

Betel Quid Chewing; Cadmium Poisoning; MODS.

1. Introduction

Cadmium (Cd) is one of the most toxic elements in the world, and human exposure can result in the development of various diseases, including renal failure, immunosuppression, and several types of cancers [1,2]. For nonoccupationally exposed populations, the source of exposure to Cd is mainly through the dietary route. Exposure to Cd can occur through different routes such as water and food. Previous studies have reported that prolonged betel quid chewing can cause cadmium poisoning [3]. Cadmium is also present in tobacco smoke and contributes to cadmium toxicity by inhalation. The human lungs reabsorb 40–60% of the cadmium in tobacco smoke and thus smokers have four to five times higher blood cadmium concentrations than those of non-smokers [4]. Chronic cadmium toxicity has a major toxic effect on the kidneys. Because of the extremely long biological half-life of cadmium (>30 years), prolonged low-concentration exposure leads to excessive accumulation in tissues, especially the renal cortex. Multiple organ dysfunction syndromes (MODS) caused by long-term betel nut chewing is relatively rare and easy to be misdiagnosed.

2. Presentation of case

A 28-year-old male patient complained of abdominal pain with chest tightness for 3 days and dyspnea for 1 day. He was admitted to the hospital on December 25, 2020. The patient developed epigastric pain after drinking a lot of alcohol (approximately 700ml of white wine in total) 3 days before admission, showing paroxysmal pain, dissolving yellow and watery stool 4

times, accompanied by nausea, chest tightness, and appetite decreased significantly. 1 day ago, abdominal pain worsened with dyspnea and nausea. After diagnosis and treatment in the hospital, the patient developed more shortness of breath on the second day after admission and was in critical condition with multiple organ dysfunction, and was transferred to the ICU. Since the illness, my spirits and sleep have been poor, and my urine has decreased. Personal history of oral "betel nut" (every day) for more than ten years; a history of drinking for many years, drinking 1-2 times a week, 2-3 cups of Erguotou each time. No history of special medication.

Physical examination: blood pressure 100/36 mmHg, pulse 97 beats/min, body temperature 36.7°C, breathing 38 beats/min, blood oxygen saturation 94%. Consciousness, irritability, mild yellow discoloration of the sclera, thick breath sounds in both lungs, less dry and wet rales in both lungs, full abdomen, no special physical examination.

Arterial blood gas analysis: oxygen saturation 91.7%, oxygen partial pressure (PO₂) 119 mm Hg (80-100 mm Hg), carbon dioxide partial pressure 19 mm Hg (35-45 mm Hg), whole blood alkali remaining 7.4 mmol/L, blood Potassium 5.6 mmol/L, blood calcium 1.02 mmol/L, lactic acid: 17 mmol/L. Blood routine: white blood cell count 28.96×10⁹/L, red blood cell count 3.80×10¹²/L, absolute lymphocyte value 5.33×10⁹/L, absolute neutrophil value 21.06×10⁹/L, hypersensitive C-reactive protein 66.86mg/L. Type B natriuretic peptide precursor (PRO-BNP): 21525pg/ml; Procalcitonin: 1.226ng/ml; Biochemical set: Alanine aminoconverting enzyme (ALT) 2159U/L, Aspartate aminoconverting enzyme (AST) 3579U/L; Apolipoprotein A (APOA) 0.9g/L, Apolipoprotein B (APOB) 0.47g/L, total cholesterol (TCHOL) 2.69mmol/L, creatine kinase (CK) 507U/L, Creatine kinase isoenzyme (CKMB) 88U/L, α-hydroxybutyrate dehydrogenase (HBDH) 968U/L, high-density lipoprotein C (HDL-c) 0.67mmol/L, total bilirubin (TBIL) 56.1umol/L, creatinine (CREA) 255umol/L, total protein (TP) 59.9g/L, uric acid (URIC) 916umol/L, urea (UREA) 13.6mmol/L. DIC complete set: activated partial thromboplastin time 62.8sec, D-dimer quantitative 6670ng/ml, fibrin degradation product 20.81ug/ml, fibrinogen 1.64g/L, prothrombin time international standard 3.75, prothrombin Time 39.0sec. Three items of myocardial infarction: creatine kinase isoenzyme 8.7ng/ml, high-sensitivity troponin 0.861ng/ml, and myoglobin 602.8ng/ml. β ketone body: β-hydroxybutyric acid 3.37mmol/L. Urine amylase determination: Amylase (AMY) 2210U/L. Urine routine: Urine bilirubin (BIL) 8.5umol/L, occult blood (BLD) 150ERY/uL, white blood cells 325.00/uL. 7 items of immunoglobulin + complement: complement C3: 506 mg/L, complement C4: 72 mg/L. Hepatitis A IgG antibody (+). Anti-EB virus nuclear antigen IgG: positive (40.4), anti-EB virus capsid antigen antibody IgG: positive (55.8).

Imaging examination: cardiac color Doppler ultrasound: LVEF 70%; right heart enlargement; tricuspid regurgitation (medium), pulmonary hypertension (light). Color Doppler ultrasound of the abdomen at the bedside: fluid in the abdominal cavity. Chest + whole abdomen CT scan + enhanced + three-dimensional reconstruction: 1. A little exudative change in the lower lobes of both lungs; enlarged heart; 2. Exudation around the pancreas and blurred fat gap, please combine with clinical laboratory examinations to exclude pancreatitis; 3. There are more translucent gas shadows in the anterior abdomen, temporarily consider gas accumulation in the colon; 4. Mild fatty liver; 5. Small kidney stones. All of above, the patient suffered from severe MODS.

In terms of treatment, oxygen inhalation, correction of internal environment disorders, infusion of fresh frozen plasma, norepinephrine, injections to maintain blood pressure, tracheal intubation and ventilator assisted ventilation, continuous liver and kidney replacement therapy (CRRT, Gambro, PrismaFlex, Sweden) Improve liver and kidney function. The patient has multiple organ failure (heart, liver, kidney, circulation, blood coagulation, breathing). Considering that the cause of MODS is unknown in the patient, the blood and urine poisons, sleeping drugs and heavy metals have been tested, and the relevant indicators of poisoning have been comprehensively checked and randomized Urinary cadmium is elevated, and the

urinary cadmium concentration is 89.02 µg/g. Consider heavy metal cadmium poisoning. The multidisciplinary joint consultation of the whole hospital and the consultation with the director of the Toxicology Department of Guangzhou Twelfth People's Hospital are requested to consider that the patient's current causes of multiple organ failure and severe internal environmental disorders are related to acute alcoholism and chronic cadmium poisoning, and continue to provide continuous liver and kidney replacement Treatment (CRRT, Gambro, PrismaFlex, Sweden) improves liver and kidney function and organ function support, and treatments such as anti-infection, volume expansion and fluid supplementation, and nutritional support. After that, the patient's circulation gradually improves, severe acidosis is corrected, and the heart, kidney and coagulation functions are significantly improved, But the separation of bile enzymes occurred in the liver, and the condition is still serious. After plasma exchange and liver protection treatment, the patient's liver function gradually improved, and the overall condition became stable. After contacting the gastroenterologist, it is recommended to transfer to a specialist to continue treatment.

3. Discussion

Acute cadmium poisoning causes gastrointestinal tract erosion, pulmonary, hepatic or renal injury and coma, depending on the route of poisoning [5]. Cd is transported from the gastrointestinal tract or lungs to the plasma, where it binds to albumin, and a small amount is secreted from the liver to bile. In addition to albumin, a large amount of Cd forms a complex with metallothionein (MT), which is easily filtered by the glomerulus, and with the help of the ZIP8 transporter located at the top of renal tubular cells, it acts in the proximal and distal tubules through adsorption and endocytosis To be reabsorbed, which is a complicated process [6,7]. Cd toxicity induces tissue injury through creating oxidative stress, changing in DNA expression [8], Therefore, the toxic effects of Cd poisoning are through multiple systems and pathways. The organs failures in those patients are associated with the cardiovascular system, renal, hepatic and coating, which made diagnosis difficult and management complex. Cd is transported throughout the body, through oral ingestion or inhalation of Cd, especially stored in liver and kidney [9,10]. These two organs absorb approximately 50% of the Cd burden. The long biological half-life of Cd in humans (10–30 years) can cause bone disease, nephrotoxicity, carcinogenicity, and many other poisoning effects [9-11]. he most severely damaged organs of patients precisely are liver and kidney, which lead to their slow recovery. There is no consensus of opinion in the literature regarding treatment of Cd toxicity.

The previous study investigates the potential for human exposure to Cd through the habit of chewing betel quids, and some of the adverse health effects observed may be exacerbated through increased exposure arising from chewing betel quids. Furthermore, since these toxic elements can readily pass from the mother to the foetus, betel quid chewing by pregnant women may be a particular health concern [12]. Our case developed early abdominal pain, diarrhea, nausea, and dyspnea after long-term chewing of betel quid chewing and smoking cadmium poisoning, and finally progressed to MODS. Betel quid chewing has been practised for hundreds of years; it is a very common social habit in East and South Asian countries. More than 200 million of the East and South Asian population chew betel quid daily [13]. However, these cadmium poisonings from chronic betel quid chewing are often overlooked, although they may be a contributing factor to the development of the disease. The common strategy for heavy metal poisoning is mostly chelation therapy to promote heavy metal excretion. However, efforts to find a safe and effective drug for removing Cd from kidneys have largely failed. The chelators for Cd toxicity are themselves described to have different safety and efficacy [14].

4. Conclusion

The results of this study allow us to conclude that our patient's symptoms are caused by chronic cadmium poisoning, and the root cause is long-term betel quid chewing. However, it is very difficult for us to suspect cadmium poisoning based on the patient's symptoms and past history, and then it is confirmed by hematuria test and detailed medical history. Therefore, we suggest that we should expand our clinical thinking when diagnosing multiple organ failure in young patients. Clinicians should diligently draw up personal history to diagnose potential fatal poisoning early.

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