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CASE REPORT

Severe glitter poisoning: Case report

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ABSTRACT. Glitter, also referred to as 'bronze dust' or 'gold dust,' is a highly toxic substance typically produced by grinding bronze and combining it with zinc and stearin. It is commonly used in paints to achieve golden tones and is widely employed in crafts, cosmetics, and school projects. Ingestion or inhalation of glitter can lead to potentially fatal poisoning in children. This article describes the case of a 3-year-old girl exposed to glitter who developed acute respiratory distress and altered sensorium, requiring ventilatory support. In cases of glitter ingestion and/or inhalation, bronchoscopy with bronchoalveolar lavage should be performed immediately, even in the absence of respiratory symptoms

Key words: *Glitter; Gold dust; Copper; Bronchoalveolar lavage; D-penicillamine.*

Exposure to toxic substances accounts for 0.3% of pediatric emergency cases, of which 5-10% are considered potentially fatal. Ninety percent of childhood poisonings occur at home, primarily in children aged 1 to 4 years, which coincides with the highest prevalence of domestic accidents.[1,2]

Glitter, also referred to as 'bronze dust' or 'gold dust,' is a highly toxic substance typically produced by grinding bronze (an alloy of copper and tin) and combining it with zinc and stearin. The latter is a fatty acid that acts more as an additive to improve the texture and handling of the product, but it does not have a significant impact on its toxicity. Glitter is used in paints to achieve golden tones and is widely employed in crafts, cosmetics, and school projects. Accidental ingestion or inhalation is rare but potentially fatal in children.[3] We report the case of a 3-year-old girl who presented with respiratory distress and altered consciousness over a short period following exposure.

CLINICAL CASE

A 3-year-old female patient with no significant medical history was accidentally exposed to glitter through inhalation (aspiring a large amount of dust), dermal contact, oral ingestion, and ocular exposure. The glitter was being used by a family member for the commercial decoration of ornaments. She was brought to the emergency department of our hospital, presenting with respiratory distress, cyanosis, vomiting, and abdominal pain. Her vital signs upon admission were: HR 143 bpm, RR 42 bpm, and SpO2 92% (FiO2 0.21). Fig. 1 shows pulmonary involvement on the thoracic X-ray, 2 hours after admission to the Emergency Department (ED). It demonstrated signs of air trapping with bilateral reinforcement of the lung pattern.

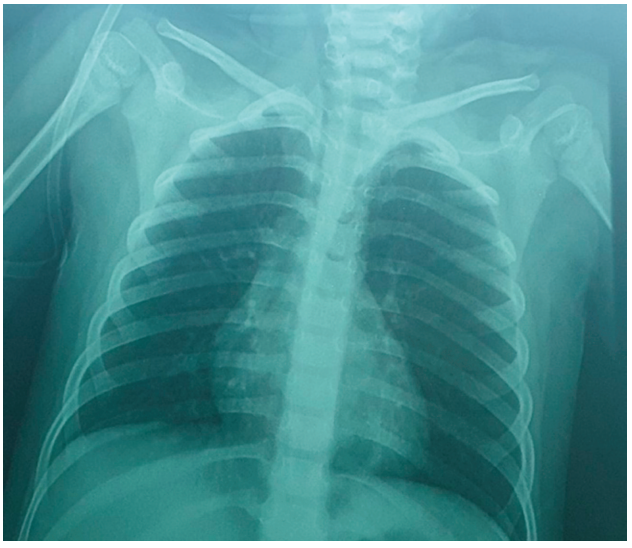

Figure 1. The chest X-ray upon admission showed signs of air trapping and bilateral increased lung markings.

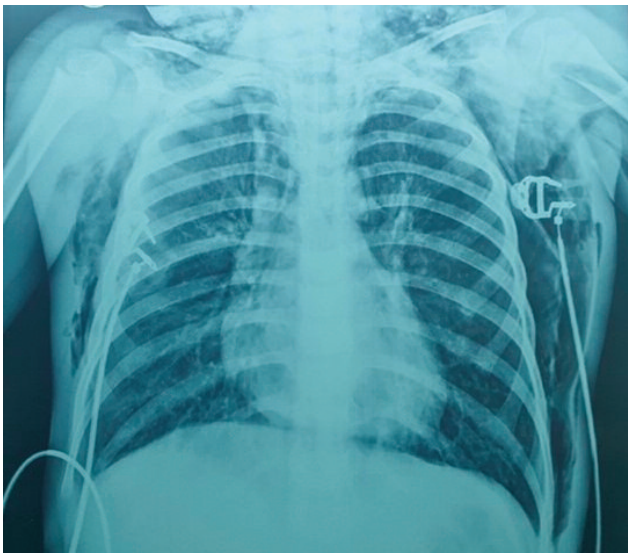

Figure 2. Thoracic X-ray 48 hours after admission to the PICU. Signs of air trapping are observed, along with the presence of bilateral interstitial infiltrates compatible with chemical pneumonitis.

Due to the progression of respiratory symptoms, the patient was admitted to the Pediatric Intensive Care Unit (PICU). Pulmonary involvement was predominantly on the right side (Fig. 2), requiring initial oxygen therapy via a reservoir mask and antibiotic treatment with clarithromycin and ceftriaxone, later switched to clindamycin. 24 hours after her admission to the PICU, the patient required mechanical ventilation for 7 days due to respiratory failure and a decline in her level of consciousness. Laboratory results showed WBC 25,200/mm³, HCT 36%, HGB 12.2 g/dL, PLT 434,000/mm³ and ABG 7.28/48/55/22.6/-4.1.

Over the following 72 hours, a hemolytic anemia was observed, with a HCT 28%, HBG 9.3 g/dL, TB 2.3 mg/dL and IB 2 mg/dL. Subsequently, the antibiotic regimen was changed to piperacillin-tazobactam combined with amikacin, and furosemide was introduced. The patient never required the administration of vasoactive drugs. Among the complications, she developed extensive subcutaneous emphysema in the chest and neck, along with a right-sided grade I pneumothorax.

As a result of symptomatic improvement, the patient was transferred to the Pediatric Inpatient Unit, where clinical and laboratory monitoring continued. The CBC and renal and liver functions were normal. The serum copper level was 152 µg/dL (normal value [NV]: 88-158 µg/dL), urinary copper or *cupruria* was 68 µg over 24 hours (NV: 20-87 µg), and serum zinc was 141 µg/dL (NV: 60-150 µg/dL). Given the results of the metal assays, chelation therapy was not initiated. The patient was discharged 14 days after admission, with normalized laboratory values and plans for follow-up by the Pulmonology Service due to lung sequelae. A chest X-ray and computed tomography (CT) scan were performed 3 months after exposure, revealing findings consistent with bronchiectasis in both lung bases (Fig. 3).

DISCUSSION

Copper is an essential antioxidant micronutrient involved in erythropoiesis and iron metabolism, primarily found in the liver, brain, kidneys, and heart. In addition, it plays a key role in the immune system, protects against cellular damage by free radicals, participates in the mitochondrial electron

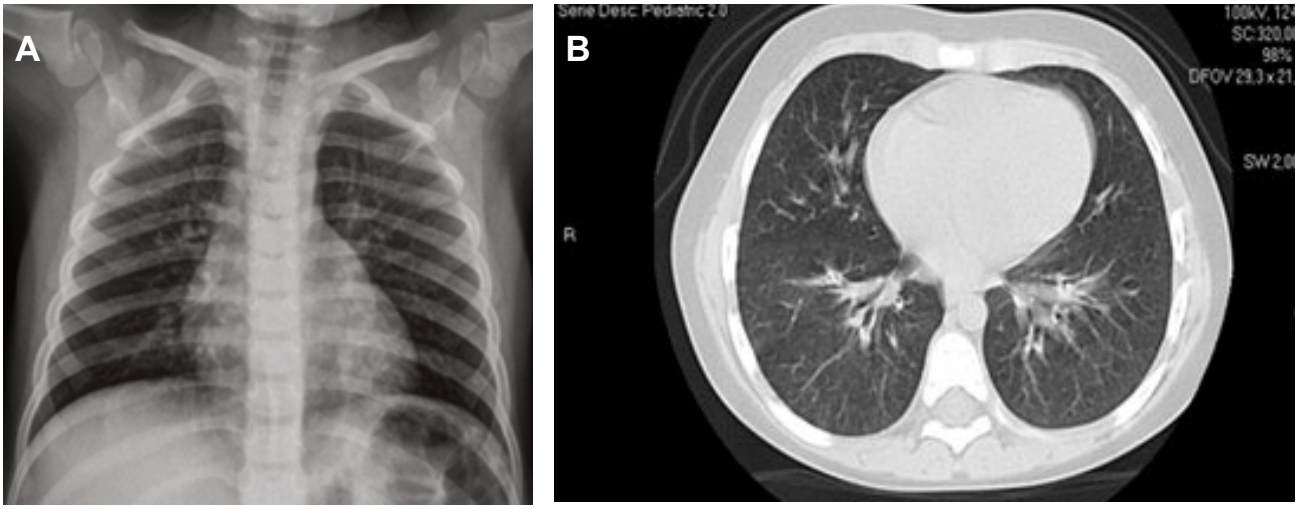

Figure 3. Chest X-ray (A) and CT (B) scan performed 3 months after exposure to glitter. Signs of bronchiectasis can be observed in both lung bases.

transport chain, and is crucial for the synthesis of collagen, elastin, and other biomolecules. However, copper can be highly toxic, and in its Cu^{2+} form, it has the potential to generate free radicals, leading to oxidative stress-induced damage.[3]

Glitter poisoning is infrequent, with only a few reports in the literature. In exposed children, symptoms are primarily determined by copper toxicity. Since fecal and biliary excretion accounts for 80% of ingested copper elimination, systemic toxicity after ingestion is uncommon. Approximately 18% of copper is absorbed and transported via the bloodstream to the liver, where it is stored. Around 2-4% is excreted through urine.[3,4] Gastrointestinal manifestations include nausea, greenish vomiting, abdominal pain, diarrhea, and toxic hepatitis with centrolobular necrosis. Renal involvement can lead to oliguria, hematuria, cylindruria, and may result in acute tubular necrosis. Other clinical manifestations include hemolytic anemia within the first 24 hours, tachycardia, hypotension, methemoglobinemia, rhabdomyolysis, pulmonary edema, seizures, coma, multiorgan failure, and death.[5]

Acute toxicity from the inhalation of copper dust or vapors causes irritation of the upper respiratory tract, accompanied by odynophagia, irritative cough, and sinusitis. In the lower respiratory tract, chemical pneumonitis may occur, depending on the particle size inhaled, which ranges from 3 to 70 microns in the case of glitter. Inhaling these particles may also lead to gastrointestinal irritation, followed by systemic absorption. The presence of vomiting and abdominal pain suggests the passage of copper into the digestive tract, and an elevation in copper levels (*cupremia*) indicates the absorption of copper across the gastrointestinal mucosa and the alveolocapillary barrier into the bloodstream. It can also result in hepatic and/or renal failure.[6]

Gosselin et al. (1984) reported the case of a 2-year-old child who suffered fatal poisoning from gold dust, presenting with fever, gastrointestinal symptoms, renal damage, and severe pneumonitis on autopsy.[7] There is limited literature on the inhalation of gold dust; however, cases of powder or talc aspiration in infants have been reported, with rapid progression to severe pulmonary illness and a mortality rate of 23%.[8-11] These authors report a similar clinical picture in the inhalation of other types of dust, similar to that described with the aspiration of glitter.

Naturally present zinc from food sources does not appear to cause zinc toxicity. However, excessive zinc intake from supplements can lead to adverse health effects, including gastrointestinal distress, nausea, dizziness, headaches, and loss of appetite. Zinc doses exceeding 200 mg can induce vomiting, and gastrointestinal discomfort has been reported with doses as low as 50 mg. A few cases of severe zinc toxicity have been documented following the ingestion of non-food items containing zinc. Chronic and substantial ingestion of zinc-containing coins may result in copper deficiency anemia due to zinc-induced toxicity, particularly in individuals with metal pica. Notably, hematological and neurological manifestations of zinc toxicity resulting from the ingestion of coins or denture cream have improved with the removal of the zinc source and copper supplementation. The inhalation of zinc oxide fumes, common among welders and others working in environments where zinc is heated to high temperatures, can cause a characteristic acute respiratory illness of short duration. This condition is marked by fever and flu-like symptoms and is typically associated with the development of tolerance to continued exposure, with recurrence upon subsequent re-exposure. A second, potentially lethal respiratory condition can arise from exposure to zinc chloride, often from smoke bombs used in crowd control. In such cases, the respiratory distress is severe.[12]

We must distinguish between this patient's exposure to ultra-fine glitter and other larger forms of glitter that become trapped in the upper respiratory tract (trachea and bronchi) without reaching the distal airways. Inhalation of these types of glitter may cause coughing but does not lead to systemic absorption. Due to its toxicity, the sale of glitter should be avoided as a preventive measure.[13] In our clinical case, the primary route of exposure was inhalation, leading to a progressive respiratory condition that required ventilatory support. A latency period of several hours between inhalation and the onset of respiratory symptoms is typically observed. These manifestations, the rapid progression of the clinical course, and the radiological changes observed are similar to those described in the inhalation of other types of dust.

In children with respiratory distress, treatment should include supportive measures such as parenteral hydration, corticosteroids, and bronchodilators. Immediate intubation and bronchoalveolar lavage (BAL) improve the patient's prognosis. Although the number of reported cases where this procedure was carried out is limited, the literature supports its urgent indication in children, even in the absence of respiratory symptoms.[14] This is because BAL can reduce the toxic burden in the lungs and mitigate potential complications.[15] In our patient, it could not be performed due to the unavailability of a specialist, a situation not uncommon in our setting.

The use of chelating agents, such as D-penicillamine, is recommended in cases of acute copper poisoning with evidence of systemic involvement. In asymptomatic patients, chelation therapy should only be initiated after confirming specific laboratory findings. Copper and zinc levels in blood or urine should be monitored, along with a complete laboratory analysis (including a complete blood count, electrolytes, liver and renal function tests), and a chest X-ray in all patients.

Given the hazardous nature of glitter, provincial laws in Argentina have prohibited its sale to minors and its use or handling in educational institutions since 2014. We believe it is essential to implement these regulations nationwide due to the potentially lethal effects on children. Additionally, it is crucial to educate adults on keeping the substance out of children's reach, thereby strengthening preventive measures.[16]

CONCLUSIONS

Exposure to glitter in children can lead to severe poisoning with a potentially fatal outcome. Due to the clinical presentation of our patient, advanced supportive measures were employed, and immediate BAL was recommended. However, this procedure could not be performed due to a lack of specialized personnel at our hospital. Current literature suggests performing this procedure urgently in order to improve the prognosis of poisoned patients.

Conflicts of interest

The authors declare no conflicts of interest.

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