CASE REPORT

Acute Respiratory Distress Syndrome Due to Overdose Desferrioxamine: Report of a Child

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Summary

In this article, we present an 18-month-old girl with acute iron poisoning who died from acute respiratory distress syndrome due to overdose of desferrioxamine. Our purpose is to emphasize the importance of close follow-up of children with acute iron poisoning for desferrioxamine toxicity.

Key Words: Iron, Poisoning, Acute respiratory distress syndrome, Desferrioxamine, Death

Introduction

Iron overdose is the most common cause of poisoning death in children (1-3). In this article, we present a child with acute iron poisoning who died from acute respiratory distress syndrome (ARDS) due to overdose of desferrioxamine. Our purpose was to emphasize the importance of close follow-up of children with acute iron poisoning for desferrioxamine toxicity.

Case Report

A previously healthy 18-month-old girl was admitted to our Emergency Department with drug ingestion. She ingested 30 tablets of iron preparation (110 mg/kg/dose elementary iron) one hour before admission to our hospital. She had no complaints. Her temperature was 37.4ºC, with a respiratory rate of 30/min, a pulse rate of 128/min, and arterial blood pressure of 110/70 mmHg. Physical examination was completely normal. On admission laboratory examination revealed normal urine analysis, normal blood count, serum electrolytes, glucose concentration, renal and liver function tests, prothrombin and partial thromboplastine time, and blood gas analysis. Chest x-ray radiography was unremarkable. Abdominal radiography showed widespread radio-opaque appearance (iron deposits) in the intestines (Fig. 1). Abdominal ultrasonography was normal. Serum iron and total iron-binding capacity was not measured due to lack of commercial kits. Serum ferritin level was 7.68 ng/ml (N: 7-140 ng/ml), on the 3rd day of admission. Aside from supportive therapy desferrioxamine (DFO) (15 mg/kg/h continue intravenous infusion) was given for three days. No abnormal clinical and laboratory findings were noted during hospitalization. She was discharged from hospital in good health on the fourth day of admission.

She was readmitted with a 1-day-history of fatigue, loss of appetite and vomiting three days after discharge from the hospital. The temperature was 37ºC, with a respiratory rate of 76/min, a pulse rate of 188/min and arterial blood pressure of 105/70 mmHg. She had tachycardia, tachypnea, subcostal and intercostal retractions, and 1 cm hepatomegaly. Hemoglobin was 12.5 g/dl, leukocyte count was 20,000/mm^3, and thrombocyte count was 415,000/mm^3. On biochemical analysis, blood glucose level was 91 mg/dl; serum sodium 129 mEq/L; potassium 5 mEq/L; chloride 100 mEq/L; blood urea nitrogen 42 mg/dl; creatinine 0.2 mg/dl; calcium 10 mg/dl; phosphorus 3.9 mg/dl; uric acid 3.7 mg/dl; aspartate aminotransferase 33 U/L,

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alanine aminotransferase 24 U/L and alkaline phosphatase 464 U/L. On blood gas analysis, pH was 7.39; HCO_3 22 mmol/L; PO2 21 mmHg and PCO_2 38 mmHg. Serum iron levels and total iron-binding capacity were 5.6 mmol/L (N: 4-33 mmol/L), and 72.5 mmol/L (N: 44.75- 71.60 mmol/L) respectively. Thorax radiography revealed bilateral widespread infiltrates, and decreased inflation, but with normal heart size. Abdominal radiography revealed marked decrease of radio-opaque appearance in the intestines. Echocardiographic examination showed mild pulmonary hypertension. Based on the clinical and laboratory findings, we firstly thought that she had bronchopneumonia, but later ARDS was diagnosed. Ampicillin-sulbactam plus cefotaxime and DFO (10 mg/kg/h continuous intravenous infusion) was initiated because we thought that the duration and dosage of DFO was inadequate. On the 4th day of admission her general condition became worse and hepatomegaly and cardiomegaly developed. She was diagnosed with heart failure and digoxin was initiated and previous antibiotics were replaced with vancomycin and ceftriaxone. Additionally prednisolone and furosemide were given. Because she showed rapid progression, we did not use mechanical ventilation in the pediatric intensive care unit. Despite high-quality supportive care she died on the 4th day of re-admission. Unfortunately, postmortem examination could not be performed because her parents did not give permission.

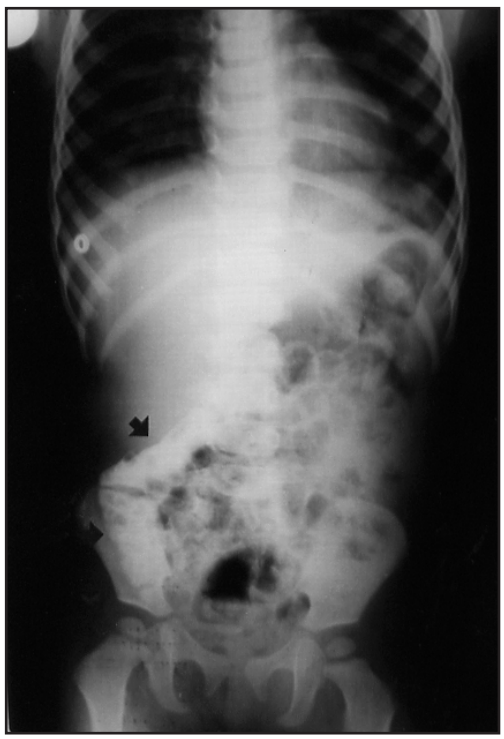

Fig 1: Abdominal radiography shows widespread radio-opaque appearance (iron deposits) in intestines (see arrows)

Discussion

Iron poisoning continues to be a major toxicologic problem, with a major impact on the gastrointestinal and circulatory systems. Although a single value for the toxic dose has not been established, significant gastrointestinal manifestations occur following the ingestion of 20 mg of elemental iron per kilogram of body weight while systemic toxicity may occur following the ingestion of at least 60 mg of elemental iron per kilogram of body weight [2]. Treatment consists of stabilizing vital functions, removing unabsorbed iron from the gastrointestinal tract, and administering intravenous DFO when there are serious clinical symptoms or when a serum iron level > 500 micrograms/dL is measured within 8 hours of ingestion [3, 6]. Despite her normal clinical and laboratory findings we immediately initiated DFO therapy associated with supportive care. We found marked decrease of iron deposits in the intestines and normal serum iron concentration and iron-binding capacity during follow-up.

Recently, Tenenbein et al. [3] reported fatal lung injury in four patients who were treated with continuous intravenous DFO infusions. The patients, aged 19-26 years, had received DFO infusions of 15 mg/kg per h for 65-92 h. Respiratory distress developed after 32-72 h. The patients met clinical, physiological, and necropsy criteria for the diagnosis of ARDS; none had any of the known risk factors for the development of this disorder. They reviewed the records of 43 iron-poisoned patients treated with DFO infusions. No patient treated for less than 24 h had pulmonary complications. However, of the fourteen treated for longer than 24 hour, four developed ARDS and four others had pulmonary edema of other causes. They suggest that the pulmonary complications were caused by continuous infusion of DFO and that the ARDS in these patients was a consequence of free-radical generation. They recommend that DFO infusion should not be administered for longer than 24 h [13]. We

were not aware of this paper during follow-up of our patient. Now, we think that our patient died from ARDS, which was most probably due to overdose DFO infusion. We could not find any other underlying cause leading to death in our patient. On account of this case we would like to emphasize that children with acute iron poisoning should be closely be followed up for toxicity of desferrioxamine.

References

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