Original Article

Acute Iron Ingestion

Tansu Sipahi, Cemsit Karakurt, Arzu Bakirtas and Betül Tavil

Dr. Sami Ulus Children's Hospital, Department of Pediatrics, Turkey.

Abstract. ***Objective :*** Intoxication is one of the most common causes of admissions to emergency department in pediatric age group. Incidence of iron poisoning gradually increased because of wide spread use of iron containing drugs. ***Method :*** In this report, we present five cases of iron ingestion who were admitted to our emergency department within a year. ***Result :*** Whole bowel irrigation in addition to gastric lavage with an iron dose of over 50mg/kg as well as deferoxamine treatment for patients in whom clinical and laboratory indications are present. ***Conclusion :*** The prompt recognition and treatment of children with acute iron poisoning is the single and the most critical point for decreasing the morbidity and mortality associated with iron containing products. **[Indian J Pediatr 2002; 69 : (11) : 947-949]**

Key words : *Iron poisoning; Whole bowel irrigation; Deferoxamine treatment.*

Intoxication is one of the most common causes of admissions to emergency department in pediatric age group. Iron poisoning is relatively common in childhood, and has significant morbidity and mortality of about 1%.[1] Incidence of iron poisoning has been gradually increasing because iron containing pills are commonly used in many households.[2] Iron pills or multivitamins containing iron are prescribed for most pregnant women and children. These tablets resemble candies and chewing gums; which make them easier to be ingested by children.

The severity of iron poisoning depends on the ingested dose of elemental iron. Ingestion above 20mg/kg of elemental iron can cause toxic effects. The lethal dose of elemental iron is usually 180mg/kg or greater.[3,4]

The hospitalization rate of iron poisoning in children younger than 4 years has been estimated as 8.7/100 000 according to poisoning records in the USA.[5] In only 1995 more than 22000 children accidentally received iron containing preparations. The exact statistical figures for iron poisoning in Turkey has not been clearly determined. We note that the incidence of iron poisoning has gradually increased in our country due to the widespread use of iron containing products. In this report, we present five cases with acute iron ingestion admitted to our emergency department in one year.

MATERIALS AND METHODS

Five patients with acute iron ingestion were followed in Dr. Sami Ulus Children's Hospital emergency department from January 2000 to January 2001. Iron ingestion over 20mg/kg was regarded as acute iron ingestion. Detailed history of ingestion was obtained from the parents.

Whole physical and neurological examinations were done and the laboratory examinations such as complete blood count, serum iron, serum iron binding capacity and ferritin levels were measured on admission and 6-8 hours after iron ingestion. All cases received an application of lavage with orogastric tube.

Whole bowel irrigation (WBI) was performed and deferoxamine therapy was administered with respect to clinical status and high dose iron ingestion. WBI fluid (ringer lactate without dextrose) was administered through a wide orogastric tube. Dosing schedule for WBI is: 75 cc/kg/h for children between 1 to 3 years of age, 60 cc/kg/h for children older than 3 years. The treatment was carried on at least until the rectal effluent was clear.

RESULTS

Five cases who had ingested over 20mg/kg elemental iron were admitted to our hospital (Table). Physical examination of the cases on admission was normal except case 2, whose mental status had altered. Serum biochemistry of all cases were normal. All cases were applied gastric lavage and case 2 and 3 were applied whole bowel irrigation. All patients were given intravenous fluid and electrolyte treatment; two patients received deferoxamine treatment (15 mg/kg 12 hours IV infusion). On the second day of hospitalization physical examination of all cases were normal.

DISCUSSION

The reasons of drug ingestion in children are multifactorial including inadequate parental knowledge about the toxic potential of individual agents, easy access

Reprint requests : Dr. Tansu Sipahi, Hosdere Caddesi, Fuar Sokak, No : 11/8 Cankaya, Ankara, Turkey TR-06640, Fax : 90 312 4196677, E-mail : tansusipahi@hotmail.com

TABLE. Clinical and Laboratory Characteristics of the Patients

Case no	1	2	3	4	5
Age (months)	36	60	14	20	42
Sex	M	F	M	M	F
Time lapse between ingestion and hospital admission	30 minutes	4 hours	3 hours	2 hours	6 hours
Symptoms	No	Altered mental status	Vomiting	No	No
Ingested dose of elementhal iron (mg/kg)	50	88	100	50	42
Ingested iron preparation	Ferro II glycine sulfate hydroxy	Ferro III hydroxy polymaltose	Ferro II sulfate	Ferro II glycine sulfate	Ferro III polymaltose
Hemoglobin level (g/dl)	11.1	11.3	10.7	9.2	12
Serum iron level (µg/dl)	52	332	302	90	32
Transferrin saturation (%)	16	91	75	23	7
Serum ferritin level (ng/ml)	21	55	62	12	13
Treatment	Gastric lavage	Gastric lavage,			
WBI, deferoxamine	Gastric lavage,				
WBI, deferoxamine	Gastric lavage	Gastric lavage			

M: Male, F: Female

to medications, and lack of parental attention[3]. Iron containing drugs have become more available in many households, leading to an increase in iron poisoning during childhood. Iron related injuries increased 150% in the USA from an annual average of 1200 in 1980 to 3000 in 1996. About one third of injuries from 1980 through 1996 involved infants under 2 years and one third involved of 3 to 4 years. Pediatric iron related fatalities increased in 1986 making its peak at 10-year-old children, then the peak declined to 2-year-old children by 1995.[3]

The measurement of free iron in the serum is the best way to determine the potential of toxicity. This could be done by assesing levels of serum iron and total iron binding capacity. If serum iron exceeds total iron binding capacity, free iron plays a toxic role on the tissues. Ingestion above 20mg/kg of elemental iron can cause toxic effects. Ferrous sulfate is the leading cause of accidental pediatric iron poisoning due to its more frequent prescription. Ferrous fumarate, carbonyl iron, ferrous gluconate and iron containing multivitamins are the other causes. Iron toxicity is related to the generation of free radicals. Free iron reacts with $O2^-$ and H2O2 to produce more reactive and toxic-free radicals such as hydroxyl radical. The hydroxyl radicals depolymerize polysaccaharides, lead to breakage of DNA strands, inactivate enzymes, and initiate lipid peroxidation, which is a self-amplifying process particularly damaging cellular and subcellular membranes.[4,5]

The clinical progression of iron poisoning is divided into five stages. Stage I is related to gastrointestinal toxicity in which nausea, vomiting, and diarrhea are expected. The direct effect of iron on the gastric and intestinal mucosa is corrosive and occur early, usually within a few hours. Stage II is an asymtomatic phase, that is resolution of gastrointestinal symptoms with apparent clinical improvement. Stage III represents systemic toxicity which defines true iron poisoning. Systemic toxicity is clinically manifested as shock with associated signs of hypoperfusion. Stage IV is the period of clinical recovery that will begin soon after the initiation of fluid and chelation therapy. Stage V is the late onset of gastrointestinal complications such as gastric and pyloric strictures which may occur 2-8 weeks after the initial injury.[3]

In the present cases, all patients ingested more than 20mg/kg of elemental iron but only case 2 and 3 presented with clinical symptoms and thus could accordingly be considered as iron poisoning. However, the others having no symptoms were considered as acute iron ingestion (Table).

Therapy of iron poisoning requires immediate attention to airway management and fluid resuscitation ipecac induced emesis should be followed by chelation therapy when necessary. Gastric lavage can also be used for gastric discharging. Iron pills tend to conglomerate, forming sticky mass which may not pass through the lavage tube. Therefore WBI has been advocated as the ideal procedure for gastrointestinal lavage. WBI for the management of poisoning is the enteral administration of large volumes of osmotically balanced solutions such as polyethylene glycol electrolite solution (PEG-ES), dextrose free ringer lactate or normal saline solution. WBI is contraindicated in patients with bowel obstruction, perforation, ileus, chronic renal and liver insufficiency and in patients with hemodynamic instability.[6-10] Activated charcoal does not have a major role in the treatment of iron poisoning because metallic ions such as iron isn't adsorbed by it.

For chelation therapy deferoxamine is recommended.[3,6] Deferoxamine should be initiated if the clinical examination, laboratory evaluation, or radiographical data suggest the possibility of significant exposure or when they confirm severe toxicity. Any clinical signs of shock, lethargy, coma, altered mental status, persistent vomiting-diarrhea, gastrointestinal bleeding may be an indication for chelation therapy. In addition, a serum iron level of greater than 500µg/dl or an estimated ingestion dose of greater than 60mg Fe^{+2}/kg

are the laboratory indications of deferoxamine treatment.There are some cases in whom a small number of pills were ingested and severe toxicity developed; this suggests that toxicity may not be related to the dose of ingestion. Therefore, deferoxamine should be administered if the clinical examination, laboratory evaluation, or radiographic data suggests the possibility of significant exposure or if they confirm significant toxicity. The current recommended dose of deferoxamine in iron poisoning is 15mg/kg per hour intravenously. The most common complication seen with intravenous administration of deferoxamine is hypotension. Severe adverse effects due to its application such as adult respiratory distress syndrome were also reported by Ioannides and Panisella.[5]

In the present cases, gastric lavage was initially done. WBI was performed and deferoxamine therapy was administered in case 2 and 3 because of clinical status and ingestion of high dose.

It is implied that iron related poisoning has been increasing in our country due to widespread use of iron pills but we don't know the real statistical information about iron poisoning in our country. In the present study period, 34500 patients were admitted to the emergency department of the hospital, 177 of these patients had intoxication, and 5 of them were admitted due to acute iron ingestion. The hospitalization rate of acute iron ingestion in children is 14.5/100 000 according to the records of our hospital.

In conclusion, we propose whole bowel irrigation (WBI) in addition to gastric lavage with an iron ingestion dose of over 50mg/kg as well as deferoxamine treatment for patients in whom clinical and laboratory indications are present. Unit dose packaging of iron supplements is proposed to reduce the frequency of severe pediatric iron poisoning incidents. The prompt recognition and treatment of children with acute iron poisoning is the single and critical point in decreasing the morbidity and mortality associated with iron containing products.

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