Disc batteries, also called button batteries, are small disk-shaped batteries used in watches, calculators, cameras, hearing aids, computer games and toys. Accidental ingestion in children is common. There are different types and sizes of batteries, and depending on the type, these contain varying amounts of heavy metal salts as oxides of mercury, zinc, silver, cadmium or hydroxides of lithium, nickel or manganese dioxide. Usually these consist of a cathode can and an anode cap separated by electrolyte–soaked (potassium hydroxide 45%) fabric (Fig. 1). The diameter varies from 6.8-23mm. The size is an important determinant of esophageal impaction, as batteries larger than 18mm have been reported to result in impaction and perforation. The leakage of metal salts and electrolyte may result in liquefaction necrosis at the site of impaction leading to perforation into the aorta or mediastinum. Rarely, mercury toxicity is reported in case of mercury cell ingestion. However, in approximately 90% of battery ingestion cases, the battery reaches stomach without impaction and passes out through stools uneventfully within 7 days. A review of 2382 cases of battery ingestions (1) showed that in 10% of cases, the battery seal had dissolved leading to corrosive injury and in only two cases fatal trans-esophageal fistula and perforation of the aortic arch had occurred. 9.9% of all battery ingestion cases presented with symptoms of vomiting (2.4%), anorexia, lassitude, mild abdominal pain (2.0%), discolored or bloody stool (1.7%), diarrhea (1.3%), fever (1.3%) and skin rashes mainly in nickel battery ingestion (1.2%). Patients with impacted esophageal batteries presented with fever,
dysphagia, vomiting and tachypnea. Battery impacted in the airway can cause severe respiratory symptoms.

Management of disc battery ingestion

Initial management of the child consists of airway assessment and stabilization followed by antero-posterior and lateral chest X-rays. Batteries lodged in the airway or lower respiratory tract cause respiratory difficulty and require emergent removal via bronchoscopy. Intact battery seen beyond pylorus in an asymptomatic child requires follow-up in home, with serial stool checking for battery passage. Metoclopramide and a cathartic are advocated to hasten passage in stool. H2 antagonist and antacid may be prescribed to reduce GIT irritation. If the child develops pain in abdomen, vomiting and fever and the battery is not passed out, he should be admitted for further management.

In a younger child (<6 years), if the battery is located in stomach and its size is more than 15mm, it is less likely to pass through pylorus. It requires reassessment and a repeat X-ray after 48 hours to check the movement. In case the battery is still in the stomach, it requires endoscopic retrieval after 5-7 days of observation period. The role of whole bowel irrigation is not fully established. If the battery is seen in the esophagus, it must be removed by endoscopy immediately because tissue injury due to esophageal impaction has been reported to occur as early as 4 hours after ingestion. The procedure allows for both removal and tissue inspection. Role of Glucagon (0.05mg/kg IV) to relax esophageal sphincter and increase movement is not yet proven. Do not give emetic or push the disc with catheter as these procedures are ineffective (2).

In mercuric oxide disc ingestion, blood and urine Mercury levels are necessary only when the cell has split in GIT or radio-opaque droplets of Mercury are seen on X-ray. There is no need for chelation therapy unless toxic levels of Mercury are found in the urine (>50µg/L).
Disc battery ingestion

*Localize and confirm by:*
- X-ray - chest and abdomen (PA and lateral view)

**Battery in esophagus**
- Urgent endoscopic removal (Bronchoscopy, if full thickness burn in esophagus)

**Battery in stomach**
- Symptomatic
  - X-ray assessment
  - Surgical removal
  - <15mm diameter
  - Observe
  - >15mm diameter
  - X-ray in 48 hrs.
  - Endoscopic removal after 5-7 days

**Battery beyond pylorus**
- Asymptomatic
  - Cathartic (Bulk agents, Strain stool, Repeat X-ray, 5-7 days)
  - In stomach
  - Observe
  - Cathartic, strain stool
  - Beyond pylorus
References


- Lead poisoning in a two-month old baby: A case report

Poison Control Centre received a blood sample of a two-month old Omani male baby from Rustaq Hospital for lead estimation. The sample analysis revealed alarmingly high blood lead level of 83.3µg/dl (normal limit <10µg/dl).

Going back to the details of the case, it was found that the baby was admitted two days back in pediatric ward with the history of constipation since birth. The baby was full term normal delivery, with history of delayed passage of meconium at birth, followed by passage of stools only after the use of glycerin suppository. The baby was breastfed and weighed 5 Kg. On examination, it looked normal and well. Except for mild distension of abdomen, no abnormality was detected on systemic examination. Per rectum examination showed empty rectum, however, X-ray showed, abdomen loaded with stools and there were scattered radio-opaque speckles, suggestive of lead or metallic mercury ingestion.

Detailed history from the parents revealed that the baby was given a local medicine, bint al dahab, orally, for relieving constipation 2 weeks back.

The Poison Control Centre, on the basis of such high blood lead level, advised chelation therapy with Disodium Edetate. The baby was referred to The Royal Hospital for antidote therapy. The Centre also asked for the local medication administered to the child, for testing lead concentration. It was found to contain 20192 ppm of lead/gm (20.2% by weight). After one and 3 weeks of chelation therapy, we received the blood samples for repeat lead analysis and the levels detected were 53µg/dl and 49.9µg/dl, respectively. The baby was later referred to the pediatric surgery department for further investigations for Hirschsprung's disease.

Comments

Symptomatic lead intoxication in children generally occurs at blood lead levels above 45-50 µg/dl. Lead encephalopathy, which constitutes a medical emergency usually occurs at levels above 80 µg/dl (4.82µm/l) in children (1). The present case did not have signs and symptoms of lead encephalopathy (vomiting, convulsions or coma) despite blood lead levels of 83.3 µg/dl. Child was having constipation off and on since birth, and therefore was not related to lead poisoning as the local medicine containing lead was administered only 2 weeks back. The diagnosis of lead poisoning, however, was based on abdominal X-ray findings, very high blood lead levels and history of baby being administered orally bint al dahab. The response of the baby to the treatment with Disodium EDTA further confirmed lead poisoning.
'Bint al dahab' is a traditional medication, that is reported to contain 20-90% lead oxide (2) and has been documented as the cause of poisoning in several infants from Saudi Arabia (3) and the United Arab Emirates (4). This local medication is also used in Oman for diarrhoea, colic, constipation, and general neonatal well being (2). A study on 25 Omani infants (1-8 months) admitted in a referral hospital with acute lead encephalopathy, indicated that 80% of these cases had history of 'bint al dahab' been administered. The study suggested that in any infant presenting with an unexplained encephalopathy, the diagnosis of lead poisoning should be considered and possibility of administration of a local medication (bint al dahab) should be explored (5). Though officially its import and sale has been banned in Oman, the lead poisoning in the present case, suggests its continued availability and use.

References

2. EPA, traditional remedies reported to contain lead, compiled by NSW Lead Reference Centre, 1997

Current Concerns

- **Persistent Organic Pollutants and health effects**

Persistent Organic Pollutants (POPs) include polychlorinated bi-phenyls (PCBs), herbicides & pesticides (DDT), dioxins and furans. POPs have been used in various industries (electrical transformer and capacitor, heat transfer system, fluorescent light, hydraulic fluid, lubricating oil, insulating electric wire, plastic, adhesive, textile, paint and paper, etc). Since these compounds resist photolytic, biological and chemical degradation, they persist in the environment for many years. POPs are mostly organic halogenated compounds and have high lipid solubility therefore bio-accumulate in fatty tissues of living organisms and enter into the food chain. Being semi-volatile, these can travel long distances in air to deposit in the soil. POPs are toxic to human health and environment.

Human beings are exposed to POPs mainly through diet, however occupational and environmental exposures may also occur. Acute accidental exposure can occur while production and handling of POPs and during industrial or chemical accidents/fires or burning of garbage in waste dumping sites. Chronic exposure occurs through contaminated food specially milk (including breast milk), edible oils, and animal fats. Exposure from contaminated water, soil, crops is also possible. Acute toxic effects of chlorinated hydrocarbon pesticides have been reported after large accidental or suicidal ingestion of eldrin, dieldrin and include nausea, vomiting, parasthesias, tremors, coma, seizure and respiratory depression. These patients are treated by supportive measures and decontamination by gastric lavage and activated charcoal.

The health effects following acute exposure to hexa-chloro- benzene (HCB) and polychlorinated di benzo furans (PCDF) are reported in the literature. An incident in
the past involving ingestion of HCB treated seed grains led to symptoms including photosensitive skin lesions, hyperpigmentation, hirsutism, colic, severe weakness, porphyria and general debilitation in the exposed population. Placental transfer of HCB and passage through breast milk resulted in pink sore in babies with reported mortality of 95%. (1). Another acute exposure incident known as rice oil disease involving 1291 cases occurred in Japan in 1968. Rice oil accidentally got contaminated with PCBs and PCDF during its manufacture from corroded coolant system. Consumption of the contaminated oil resulted in severe toxicity called ‘Yusho’ disease. A similar episode referred as Yusho disease occurred in Taiwan in 1979. The clinical features included swelling and burning of skin and eyes, white discharge from eyes, skin discolouration, eruptions, hirsutism, chloracne, abdominal pain, nausea, vomiting, anorexia, jaundice, fatigue muscle pain, headache, dizziness, breathing difficulty (decreased forced vital capacity), liver and kidney damage. Prospective and retrospective studies on ‘Yusho’ patients are suggestive of increased risk of carcinogenicity, reproductive impairment, fetal abnormalities and immuno–suppression (2).

Experimental studies provide convincing supportive evidence that PCBs are possibly carcinogenic in humans. PCBs have also been implicated in endocrine disruption, reproductive and immune dysfunction, neurobehavioral and developmental disorders in animal studies. However, low level exposure effects in humans are not yet fully known and concerns are based on animal studies (3). The existing scientific evidence has been judged sufficient to warrant immediate actions to reduce the risks of POPs to human health by eliminating/restricting the production, release and use of these chemicals.

The Stockholm convention on POPs (2001) identified 12 POPs for control and complete elimination, with the aim to protect human health and environment. The three broad categories of these chemicals are as follows:

1) Pesticides – DDT, chlordane, toxaphene, mirex, eldrin, dieldrin, endrin, heptachlor (chlorinated hydrocarbons)
2) Industrial chemicals – Polychlorinated biphenyls (PCBs), hexachlorobenzene (HCB)
3) By-products and contaminants – dioxins and furans.

In accordance with the Convention the production of POPs has been banned in many countries including Sultanate of Oman. However, these compounds persists in the environment from their past use, production and disposition. Also occupational exposure risks continue during waste management and in some of the industrial processes. The Sultanate of Oman is taking necessary steps to implement the Stockholm Convention.

References

1. Stockholm Convention
2. www.foodsafety.gov

► Regulatory issues

- FDA advisory to reduce lead levels in candy products

In the past, regulatory actions against importers and distributors of candy products were taken, if the lead levels exceeded 0.5ppm (FDA 1991). This guideline was based on the permissible lead in the sucrose, the main ingredient of candies. The new specification
of Food Chemical Codex (FCC) for sucrose have been reduced to 0.1ppm. Therefore, FDA in the interest of public health, also decides to publish a new guidance document for further reducing lead in candy products. Apart from sucrose, the other ingredients like tamarind, chili powder, etc. and the methods of preparation and storage would be considered in preparing these guidelines. Such products and candies need to be tested in Oman for lead concentration and national guidelines need to be set-up for reducing exposure of children to lead.

**Source: FDA / Centre for food safety & applied nutrition**

**Poison Control Centre News**

A two day ‘National Symposium on Lead poisoning in Children’ was held in May, 2005 at Al-Bustan Palace, Muscat. It provided a unique opportunity of bringing together the clinicians, public health experts and representatives of regulatory agencies at a common forum to discuss the current situation on lead exposure in children and to plan future strategies concerning lead screening and prevention. Presentations included sources of lead in the environment, routes of possible exposure, monitoring of lead in blood and in the environment. In addition, results of a WHO study conducted by DEH&ME, Poison Control Centre were presented. It was concluded that since prevalence data on lead poisoning in young children below 6 years is lacking, a multi-centric hospital based study is required on blood lead levels in children less than 6 years of age. It was communicated by the Poison Control Centre that the facility for estimation of blood lead level is now available in the toxicology laboratory.

A who study on environmental monitoring and assessment of potential health risks in populations near waste dumping sites was completed and the final report was submitted to WHO, which was approved. Poison Control Centre staff actively participated in the workshop ‘National Priorities and Objectives for POPs Management ’ in June 2005, organized by Ministry of Regional Municipalities, Environment and Water Resources.

- Poison Control Centre staff was invited to participate in the "Third Meeting of users of INTOX Data Management System" organized by International Programme on Chemical Safety (IPCS/WHO) held in UK in July 2005. The usefulness of INTOX DMS in collecting data of Central Registry of poisoning cases was discussed.
• Poison Control Centre staff participated in the follow-up workshop on PCBs in November 2005 and discussed the modalities for PCBs inventory assessment, strategy and action plan priorities.

• Poison Control Centre staff represented Ministry of Health in the Muscat multilateral oil spill drill in December 2005. It was a joint drill, designed to assess the preparedness of the Sultanate of Oman and the member states in the ROPME Sea area, coordinated by Marine Emergency Aid Centre, Bahrain to respond to a security situation as well as a major oil spill. It was one of the largest counter-pollution drill held in ROPME sea area

► Regular features

Brain Teasers

Which of the following is the principal site of toxicity of inorganic mercury compounds?
☐ Kidney ☐ Brain ☐ Heart ☐ Bone

Electric dishwasher detergents are highly
☐ Acidic ☐ Alkaline

A button battery that is lodged in the esophagus for more than 24 hours should be removed.
☐ True ☐ False

Tinnitus is an early warning signal of aspirin and acetaminophen toxicity.
☐ True ☐ False

Petroleum distillates absorbed into the systemic circulation cause a high incidence of pulmonary pathology.
☐ True ☐ False

The antidote for acute methanol intoxication is
☐ Atropine ☐ BAL ☐ Ethanol

► Forthcoming Conferences / Training Courses / Symposia

A workshop on Central Registry of Poisoning Cases in 2006