

Case report

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Allergic enterocolitis and protein-losing enteropathy as the presentations of manganese leak from an ingested disk battery: A case report

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Abstract

Introduction: Disk battery ingestions can lead to serious complications including airway or digestive tract perforation, blood vessel erosions, mediastinitis, and stricture formation.

Case presentation: We report a 20-month-old Caucasian child who developed eosinophilic enterocolitis and subsequent protein-losing enteropathy from manganese that leaked from a lithium disk battery. The disk battery was impacted in her esophagus for 10 days resulting in battery corrosion. We postulate that this patient's symptoms were due to a manganese leak from the 'retained' disk battery; this resulted in an allergic response in her gut and protein-losing enteropathy. Her symptoms improved gradually over the next 2 weeks with conservative management.

Conclusion: This is the first case report to highlight the potential complication of allergic enterocolitis and protein-losing enteropathy secondary to ingested manganese. Clinicians should be vigilant about this rare complication in managing patients with disk battery ingestions.

Introduction

Lithium batteries are used in many portable consumer electronic devices (Fig. 1). The most common type of lithium cell used in consumer applications consists of lithium and manganese (Mn). Disk battery ingestions can lead to serious complications including aerodigestive tract perforation, vessel erosion, mediastinitis, and stricture formation [1]. Mercury batteries have been reported to cause more severe complications including acute poisoning [2], but none of the disk batteries have been reported to cause protein losing-enteropathy. We report a case in which the manganese in a lithium-manganese disk battery impacted in the esophagus presumably led to eosinophilic enterocolitis and severe protein-losing enteropathy.

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Case presentation

A 20-month-old Caucasian child presented with a 10-day history of vomiting and solid food refusal. Her chest X-ray showed a disk battery impaction in the upper esophagus. A corroded lithium-manganese battery was retrieved with a flexible laryngoscope 10 days after ingestion. The patient was transferred to our institution for further monitoring. Her physical examination and laboratory tests on admission were normal, except for an albumin of 2.7 g/dL (normal 3.8 to 5.4 g/dL) which had dropped from 4.3 g/dL on



Figure 1
Lithium disk battery.

the day of battery removal. She had been on a regular diet until 10 days before admission. An esophagogram revealed no perforations.

A week following removal of the battery, she continued to refuse foods. An upper endoscopy was performed that revealed non-circumferential ulceration in the upper esophagus but no biopsies were performed at that time. Her stomach and duodenum were grossly normal at that time. A nasogastric (NG) tube was placed and feeding was started. Subsequently, the protein loss worsened and her serum albumin dropped to 1.1 g/dL (normal 3.8 to 5.4 g/dL). The urinalysis was normal and fecal alpha-1 antitrypsin level was 464 mg/g (normal <2 mg/g dry stool). A computed tomography (CT) scan of the abdomen, chest, and pelvis was performed because of persistent abdominal distention and feeding intolerance; this showed bilateral pleural effusions and moderate ascites. She had a normal echocardiogram and liver function tests. She was diagnosed with protein-losing enteropathy. Albumin 25% was started to maintain albumin at a level of >2 g/dL. A colonoscopy and repeat endoscopy were performed due to her protein-losing enteropathy; this showed complete healing of the previous esophageal ulceration, but with findings of diffuse enteritis and colitis. Small bowel biopsies were taken from the duodenum and terminal ileum. Histology revealed mild eosinophilic esophagitis and moderate eosinophilic enterocolitis (Fig. 2). In addition, the patient developed peripheral eosinophilia of 10.5% compared to 2.9% on admission. Her symptoms

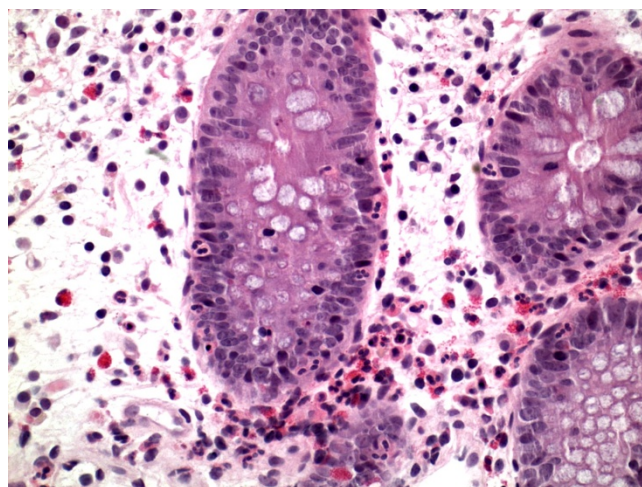


Figure 2
Colonoscopic biopsy showing eosinophilic infiltration of crypts in transverse colon.

improved, and her albumin and eosinophilia normalized gradually over the next 2 weeks with conservative support and no steroids.

At 6 months follow-up, the patient has remained well, with normal albumin levels and no symptoms of protein-losing enteropathy. She developed an upper esophageal stricture that required recurrent dilatation and steroid injections. Her most recent endoscopic biopsies showed moderate eosinophils in the esophageal mucosa. We postulate that this patient's symptoms were due to a manganese leak from the 'retained' corroded disk battery before or during the process of removal from the esophagus; this caused an allergic response in her gut resulting in a protein-losing enteropathy.

Discussion

The best known manifestations of chronic Mn exposure are neurological symptoms such as hypokinesia, rigidity and tremor that resemble Parkinson's disease [3]. Rarely, allergic responses have been described as well. Metal allergy to stainless steel wire containing Mn has been reported after coronary artery bypass grafting. A refractory pruritic erythematous wheal over the body with positive Mn patch testing and peripheral eosinophilia proved this to be a systemic allergic reaction to Mn [4]. Mn used in the manufacture of dental prosthesis has also been reported to cause contact dermatitis, manifested by diffuse oral edema, erythema and ulcerations; this was confirmed by positive patch testing [5,6].

Epidemiological studies have reported an acute impact of particulate Mn on the pulmonary system, including reversible decrement of pulmonary functions and increase

in bronchial hyperreactivity [7,8]. In children, peak expiratory flow was decreased with a high concentration of Mn in the air, suggesting an obstructive allergic response rather than restrictive airway disease [9]. Exposure to high inhaled Mn concentrations has demonstrated an increased incidence of cough, rhinitis, bronchitis, and pneumonitis [10]. A study in rhesus monkeys documented subacute bronchiolitis and alveolar duct inflammation with lymphocytes, neutrophils, and a few eosinophils following inhalational exposure to Mn [11]. In general, serum or blood Mn does not serve as a reliable indicator of the total body burden of Mn because of its intracellular distribution and relatively short half-life [12].

We postulate that our patient developed an allergic enterocolitis and protein losing enteropathy in response to the Mn exposure in the gastrointestinal (GI) tract. The ingested battery was composed of lithium perchlorate and manganese dioxide. The possibility that some other component of the battery could have contributed to the pathogenesis cannot be ruled out, but in the literature, Mn is the only constituent that has been attributed to the allergic responses. This is supported by the previously suggested evidence that Mn can cause rhinitis, pneumonitis, and bronchial hyperreactivity. Manganese exposure from a cardiac stenting wire and dental prosthesis has also caused allergic symptoms with peripheral eosinophilia. Our patient's most recent esophageal biopsies suggest that she either had a baseline mild asymptomatic eosinophilic esophagitis that acutely worsened with exposure to Mn or the Mn was a trigger to her eosinophilic esophagitis.

Conclusion

This case shows strong circumstantial evidence that the eosinophilic enterocolitis and protein-losing enteropathy were caused by the Mn leak from a retained disk battery; she was completely asymptomatic before battery ingestion with normal albumin levels and eosinophil counts before battery removal. Additionally, there was complete resolution without any treatment aside from removal of the Mn-containing disk battery. Clinicians should be vigilant about this rare complication while managing children with ingested disk batteries as symptoms might not appear immediately after battery removal.

Abbreviations

CT: Computed Tomography; GI: Gastrointestinal; Mn: Manganese; NG: Nasogastric.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

All authors (MAA, PSG, GT) contributed in the management of the patient, writing of the manuscript and reviewing of the literature. All authors read and approved the final manuscript.

Consent

Written informed consent was obtained from the parent for publication of this case report, as the child was a minor. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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