

Unique Method to Manage Ingested Magnets

To the Editor: Swallowing multiple magnets has a high risk for morbidity because of related gastrointestinal injury (1–5). A young child ingested 18 magnetic metal toy balls. x-ray of the abdomen showed 18 balls attracted to each other in the duodenum (Fig. 1). A 9.8-mm endoscope passed into the duodenum revealed the first of 17 spheres strongly adherent to the duodenal wall and attracted by the 18th ball located in another bowel segment. Attempts to remove them with traditional endoscopic devices were unsuccessful. A magnetic ball was put into a glove finger tied as a bag to a 2/0 suture wire and was back-inserted through the endoscope. Unlikely other magnetic tools (6–8), the bag was kept 5 cm away from the tip of the endoscope to allow front view (Figs. 2 and 3). The endoscope was reinserted to the foreign bodies, and the magnetic ball in the bag contacted the other spheres allowing easy retrieval of the long chain. The last, more distal magnet was evacuated 36 hours later. This novel technique proved to be effective for magnet removal.



FIGURE 1. The magnetic balls joined together like necklace beads, presumably in the duodenum.



FIGURE 2. The magnetic spheres attached to the ball in the finger glove bag tied to a long-braided suture wire and inserted in the gastroscope operative channel.

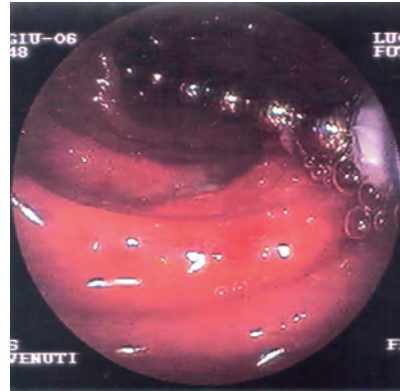


FIGURE 3. The magnetic spheres attached to the ball in the finger glove bag.

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REFERENCES

1. Naji H, Isacson D, Svensson JF, et al. Bowel injuries caused by ingestion of multiple magnets in children: a growing hazard. *Pediatr Surg Int* 2012;28:367–74.
2. Brown DJ. Small bowel perforation caused by multiple magnet ingestion. *J Emerg Med* 2010;39:497–8.
3. Hussain SZ, Bousvaros A, Gilger M, et al. Management of ingested magnets in children. *J Pediatr Gastroenterol Nutr* 2012;55:239–42.
4. Hernández Anselmi E, Gutiérrez San Román C, Barrios Fontoba JE, et al. Intestinal perforation caused by magnetic toys. *J Pediatr Surg* 2007;42:E13–6.
5. Hwang JB, Park MH, Choi SO, et al. How strong construction toy magnets are! A gastro-gastro-duodenal fistula formation. *J Pediatr Gastroenterol Nutr* 2007;44:291–2.
6. Nijhawan S, Kumpawat S, Ashdhir P, et al. Impacted nail in duodenum: endoscopic removal with a novel magnetic foreign body retriever. *Endoscopy* 2009;41 (suppl 2):E62.
7. Nijhawan S, Singh V, Mallikaarjun P, et al. Endoscopic removal of sharp metallic foreign bodies. *Endoscopy* 2007;39 (suppl 1):E331.
8. Nijhawan S, Joshi A, Shende A, et al. Endoscopy-assisted ferromagnetic foreign-body removal with a novel magnetic instrument. *Endoscopy* 2004;36:1130.

Comments on “Diffuse Esophageal Spasm in Children Referred for Manometry”

To the Editor: I read with interest the article by Rosen et al (1); however, I would like to comment on certain aspects of the study, which are questionable and not well elucidated. First is disagreement regarding the criteria used in this study for labeling a given child as having DES with “simultaneous esophageal body contractions associated with >10% of swallows” versus “>20% of swallows,” that is, a criteria used in majority of esophageal

manometry literature (2,3). Hence, it may have led to false higher reporting rate of DES as 13% in this pediatric study, whereas in adult studies, it accounts for 4% to 7% of all esophageal motility disorders (3). Nevertheless, much has changed with advent of high-resolution manometry and esophageal pressure topography followed by the Chicago classification for esophageal motility disorders, wherein the term DES is replaced by distal esophageal spasm defined as “spasm with (CFV >8 cm/s) $\geq 20\%$ of swallows” (4). Many of cases labeled as DES on conventional manometry were later reviewed by high-resolution manometry and found to be lower esophageal sphincter pseudorelaxation secondary to esophageal shortening, leading to erroneous diagnosis of DES instead of achalasia (5,6). Recently 2 new tools have been applied, contractile deceleration point and distal contractile latency that in EPT analysis improve the recognition of spasm (7). Moreover, ineffective esophageal motility in patients with severe eosinophilic esophagitis may mimic DES; therefore, results of esophagogastroduodenoscopy in lieu of unavailable esophageal biopsy results would have given better insight. Additionally, the statement has some disparity of the total number of patients included in the study ($n = 278$) and actual number (total for all subgroups is 277).

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REFERENCES

1. Rosen JM, Lavenbarg T, Cocjin J, et al. Diffuse esophageal spasm (DES) in children referred for manometry. *J Pediatr Gastroenterol Nutr* 2013; 56:436–8.
2. Tutuian R, Mainie I, Agrawal A, et al. Symptom and function heterogeneity among patients with distal esophageal spasm: studies using combined impedance-manometry. *Am J Gastroenterol* 2006; 101:464–9.
3. Sperandio M, Tutuian R, Gideon RM, et al. Diffuse esophageal spasm: not diffuse but distal esophageal spasm (DES). *Dig Dis Sci* 2003; 48:1380–4.
4. Kahrilas PJ, Ghosh SK, Pandolfino JE. Esophageal motility disorders in terms of pressure topography the Chicago classification. *J Clin Gastroenterol* 2008; 42:627–35.
5. Pandolfino JE, Fox MR, Bredenoord AJ, et al. High-resolution manometry in clinical practice: utilizing pressure topography to classify oesophageal motility abnormalities. *Neurogastroenterol Motil* 2009; 21:796–806.
6. Roman S, Kahrilas PJ. Distal esophageal spasm. *Dysphagia* 2012; 27: 115–23.
7. Roman S, Kahrilas PJ. Challenges in the swallowing mechanism: non-obstructive dysphagia in the era of high resolution manometry and impedance. *Gastroenterol Clin North Am* 2011; 40:823–35.

Stop Considering Atopic Dermatitis as an Allergic Disease

To the Editor: We read with interest the review by Iyengar and Walker (1) about the implications of breast milk in the development of atopic disease; however, we strongly disagree in considering children's atopic dermatitis (AD) as an allergic disease.

The majority of cases of AD emerge in the absence of signs of immunoglobulin E-mediated sensitization (2) and many

children will not develop it (3). Increasing evidences confirm that AD is the result of a primary epidermal barrier defect, mainly residing in filaggrin abnormalities (4), which increases skin permeability and allows environmental allergens, such as food allergens, to penetrate and stimulate a T_H2 immune response (5); the more the skin is altered, the earlier and the stronger the sensitization (6). Nowadays, because of these facts, we must accept that the traditional way to conceive the cause and effect between allergy and AD has been inverted (7).

We believe that future studies about the effects of breast-feeding or of environmental factors, such as hydrolyzed formulas, on the development and expression of allergy should be based on immunoglobulin E-mediated symptoms and not on the evaluation of AD. If breast-feeding has positive effects on the clinical expression of AD, mechanisms other than allergy must be considered.

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REFERENCES

1. Iyengar SR, Walker WA. Immune factors in breast milk and the development of atopic disease. *J Pediatr Gastroenterol Nutr* 2012; 55:641–7.
2. Illi S, von Mutius E, Lau S, et al. The natural course of atopic dermatitis from birth to age 7 years and the association with asthma. *J Allergy Clin Immunol* 2004; 113:925–31.
3. Novak N, Bieber T. Allergic and non-allergic forms of atopic diseases. *J Allergy Clin Immunol* 2003; 112:252–62.
4. Bieber T. Atopic dermatitis. *N Engl J Med* 2008; 358:1483–94.
5. Dubrac S, Schmuth M, Ebner S. Atopic dermatitis: the role of Langerhans cells in disease pathogenesis. *Immunol Cell Biol* 2010; 88:400–9.
6. Hill DJ, Hosking CS, de Benedictis FM, et al. Confirmation of the association between high levels of immunoglobulin E food sensitization and eczema in infancy: an international study. *Clin Exp Allergy* 2008; 38:161–8.
7. Lack G. Epidemiologic risks for food allergy. *J Allergy Clin Immunol* 2008; 121:1331–6.

Authors' Response

To the Editor: The response from Bramuzzo et al to our review article examining the complex relation between breast-feeding and allergic disease development underscores several important issues. As highlighted in the response, many infants initially develop atopic dermatitis (AD) because of deficits in their skin barrier that lead to a predisposition to food sensitization. These events can, in turn, serve to initiate the atopic march (1,2). Indeed, approximately 15% to 20% of patients with AD have mutations in filaggrin, a protein that is important in skin barrier function. Filaggrin-deficient mice have demonstrated permissiveness to epicutaneous sensitization to allergenic proteins and exhibit T_H17 -dominated skin inflammation, which is characteristic of atopic skin lesions (3). Although we agree with this concept outlined in the response from Bramuzzo et al, to state that “atopic” dermatitis is not an allergic disease would be to deny the interplay of this skin condition with multiple exacerbating factors, such as food allergens that have been proven to result directly in AD skin lesions. Approximately 40% of patients with AD have concomitant food

allergies (4). Regardless of whether the initial food sensitization occurred through the skin or by another route, studies have also demonstrated the ingestion of implicated allergenic foods in these patients triggers immunologic changes characteristic of allergic responses in the skin that result in the formation of AD skin lesions or “flares” (5). This established concept is supported by the European Academy of Allergy and Clinical Immunology and the consortium of European research centers specialized in allergic diseases (6). In addition, a recent study in 92 exclusively breast-fed infants with established AD demonstrated that 73% of infants showed improvement of skin lesions when their mothers avoided highly allergenic foods such as dairy, tree nuts, and soy (7). In addition, nearly 40% of people with filaggrin mutations do not develop AD (8). Similarly, patients with ichthyosis vulgaris, an inherited dry, scaly skin disorder, who also have filaggrin mutations do not have clinical skin inflammation that is characteristic of AD (9). Therefore, additional factors are necessary to interact with filaggrin in the pathogenesis of AD.

We agree with the authors that future studies of the effects of breast-feeding on the development and expression of allergy should be based on immunoglobulin E–mediated symptoms and not solely on the evaluation of AD. In our review, we stated that future studies examining the association between breast-feeding and allergy development should evaluate for immunoglobulin E–confirmed allergic disease (by examining both clinical symptoms and IgE testing simultaneously) and not just the presence of allergic sensitization. In addition, we recommend that each type of allergic disease be examined separately, rather than being grouped into the same category. This would serve to elucidate more precisely the complex relation between specific breast milk factors and the development of different types of allergic disease.

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REFERENCES

1. Hogan MB, Peele K, Wilson NW, et al. Skin barrier function and its importance at the start of the atopic march. *J Allergy (Cairo)* 2012;2012:901940.
2. Zheng T, Yu J, Oh MH, et al. The atopic march: progression from atopic dermatitis to allergic rhinitis and asthma. *Allergy Asthma Immunol Res* 2011;3:67–73.
3. Oyoshi MK, Murphy GF, Geha RS. Filaggrin-deficient mice exhibit TH17-dominated skin inflammation and permissiveness to epicutaneous sensitization with protein antigen. *J Allergy Clin Immunol* 2009;124:485–93. 493.e1.
4. Eigenmann PA, Sicherer SH, Borkowski TA, et al. Prevalence of IgE-mediated food allergy among children with atopic dermatitis. *Pediatrics* 1998;101:E8.
5. Caubet JC, Eigenmann PA. Allergic triggers in atopic dermatitis. *Immunol Allergy Clin North Am* 2010;30:289–307.
6. Werfel T, Ballmer-Weber B, Eigenmann PA, et al. Eczematous reactions to food in atopic eczema: position paper of the EAACI and GA2LEN. *Allergy* 2007;62:723–8.
7. Uenishi T, Sugiura H, Tanaka T, et al. Aggravation of atopic dermatitis in breast-fed infants by tree nut-related foods and fermented foods in breast milk. *J Dermatol* 2011;38:140–5.
8. Henderson J, Northstone K, Lee SP, et al. The burden of disease associated with filaggrin mutations: a population-based, longitudinal birth cohort study. *J Allergy Clin Immunol* 2008;121:872–7. e9.
9. Smith FJ, Irvine AD, Terron-Kwiatkowski A, et al. Loss-of-function mutations in the gene encoding filaggrin cause ichthyosis vulgaris. *Nat Genet* 2006;38:337–42.