

Food Allergy Mimicking Pylorus Hypertrophy

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Abstract

Food allergy (FA) is a disease with increasing prevalence and a wide spectrum of clinical manifestations. These include the eosinophilic disorders, which can involve any segment of the gastrointestinal tract (GIT), including the stomach. In this context, three patients with a confirmed diagnosis of food allergy, who were initially diagnosed with pyloric hypertrophy, are presented. All cases showed an adequate response to nutritional management. It is essential to consider food allergy, such as eosinophilic gastroenteritis, as part of the differential diagnosis of gastric outlet obstruction in those patients who present vomiting secondary to pyloric hypertrophy.

Keywords

Food Hypersensitivity, Hypertrophic Pyloric Stenosis, Eosinophilic Gastroenteritis

1. Introduction

Food allergy (FA) is being seen with increasing prevalence and wide spectrum of manifestations. Within its spectrum, manifestations can appear in the respiratory or gastrointestinal tract and the skin. The immunological mechanisms that develop the disease can be immunoglobulin E mediated, cell mediated or both.

Eosinophilic disorders, that are part of this spectrum, cell mediated, of disease presentation, sometimes become a challenge in the diagnosis. Three patients with a confirmed diagnosis of food allergy who were initially misdiagnosed as pyloric hypertrophy are presented. All cases resolved with adequate nutritional management.

2. Case Reports

Case No. 1.

Male infant 2 months and 17 days old with a history of prematurity due to

multiple gestation, cesarean delivery, intrauterine growth restriction (IUGR), neonatal pneumonia, and bronchopulmonary dysplasia. From the first month of life, the patient presented postprandial vomiting. Hypertrophic pyloric stenosis (HPS) was diagnosed through ultrasound. At 2 months old, he underwent pyloromyotomy. After surgery, the frequency of vomiting decreased, without completely disappearing. In addition, he presented irritability for more than three hours daily, food refusal, frequent hiccups, dysphagia, and Sandifer position, symptoms that do not respond to treatment with ranitidine and *Lactobacillus reuteri*. Given the persistence of vomiting and other symptoms, the patient was submitted for evaluation by pediatric gastroenterology. As a background, allergic rhinitis was found in both parents. From birth he received breast-feeding, supplemented with infant formula based on cow's milk protein (CMP); up to the date of the examination, 3 different formulas were tried. The physical examination was within normal limits, and anthropometry for the corrected age showed chronic malnutrition. Because of these symptoms, malnutrition, the type of food received, and a history of allergies in both parents, the diagnosis of FA was considered, possibly eosinophilic esophagitis and/or the enteropathy type. He was given an L-amino acid formula (AAF), and CMP restrictive diet was prescribed for the mother. Despite this, symptoms persisted, so breastfeeding was stopped, and only AAF continued. After this treatment, the vomiting stopped and he gained weight, so the diagnosis of food allergy was confirmed. The initial ultrasound of the pylorus was reviewed, and a pyloric wall thickness of 4mm with a channel length of 17 mm was observed. However, during feeding, passage of gastric contents through the pylorus was seen.

Case No. 2.

23-day-old female, admitted to the neonatal intensive care unit with uncontrollable vomiting and poor weight gain. Laboratory tests were taken in order to rule out an infectious process. Abdominal radiography was requested, on suspicion of enterocolitis, which was discarded. Abdominal ultrasound was performed, resulting in a diagnosis of HPS. The pediatric surgeon considered performing pyloromyotomy. The patient was assessed by the pediatric gastroenterologist, finding that bowel movements exhibited normal characteristics. Cell blood count (CBC) showed peripheral eosinophilia of 6%, and atopy was described in both parents. On physical examination, the pyloric olive was not felt. Because of suspected FA, prior to surgery it was decided to feed the patient with extensively hydrolyzed formula (EHF), with favorable response. There was cessation of vomiting and progressive weight gain, and therefore it was decided to postpone the surgery. The radiologist again reviewed the ultrasound. The diameters were recalculated, finding a thickening of the mucosa rather than the muscle layer (3 mm thick and 17 mm in length in the pyloric channel). After 5 days of nutritional management with EHF, abdominal ultrasound was performed, and result normal (pyloric channel length 17 mm × 1.5 mm thick in the antrum).

Case No 3.

Male patient, 4 months old, since 20 days old presented with 3 hours post-prandial non bilious vomiting, the initial diagnosis was gastroesophageal reflux. During the first month of life he was hospitalized because of persistent vomiting despite pharmacological management, with dehydration. No visible peristalsis nor palpable mass was evident on physical examination. Pylorus ultrasound, which reported 3.3 mm wall thickness and pylorus of 15.6 mm in length, was performed. Given the persistence of emesis, he underwent pyloromyotomy. During the postoperative period, he was fed with lactose-free formula and then was discharged. On the second day after surgery, the patient presented diarrhea, with 12 mucoid stools per day, persistent vomiting, regurgitation, and dysphagia. The formula was changed to EHF, without improvement. Therefore, he was remitted for pediatric gastroenterology consultation. Medical history of importance: cesarean delivery at term with normal weight and height at birth, breastfeeding for a month and parent's urticaria. On physical examination, eczema of the cheeks was found, anthropometry was normal, but at risk of stunting. Suspecting FA, the formula was changed to AAF. Three weeks after the change, the eczema and vomiting disappeared, the frequency of stools decreased, and there was improvement in his length.

3. Discussion

Vomiting is one of the most common symptoms in the first few months of life. The vast majority is of benign origin and self-limited, as is the physiological reflux. However, in newborns and in the first months of life, nausea and recurrent non-bilious vomiting require additional study to rule out malformations of the gastrointestinal tract and gastric outlet obstruction syndrome. In pediatrics, the most common cause of this obstruction syndrome is hypertrophic pyloric stenosis (HPS). The clinical history and a physical examination are essential for diagnosis. However, in order to define it, radiological images are usually needed [1]. Other causes responsible for the symptoms have been described, including antral membrane, gastric volvulus, duplication cysts, polyps, intestinal malrotation, and neoplasms, though these are less frequent [1]. There are also reports in the literature of gastric outlet obstruction secondary to eosinophilic gastroenteritis (EG), consistent with our subject of interest.

HPS is the most common childhood surgical pathology, manifested by non-bilious vomiting. It was described by Hirschsprung in 1888. The first successful surgical correction was performed by Ramstedt in 1912 [2]. The incidence in infants is variable, reported in 2-5:1000 live births in the United States [1]. In Africa and Asia, the incidence is lower, approximately 1:1000 live births [3]. No data exist on the incidence in Colombia. The incidence in older children is unknown. Usually it occurs in infants aged 2-12 weeks with postprandial uncontrollable vomiting. It is characterized by hypertrophy of the circular smooth muscle layer of the pylorus, which causes a thickening and elongation of the pyloric canal, findings that should be corroborated with ultrasound. The following also appear: gastric chamber dilation and an increased gastric peristalsis as com-

compensation mechanisms [2]. These changes may be evident on physical examination through palpation of the “pyloric olive” and visible peristalsis, although none of these findings were present in our cases. The etiology of HPS is unclear. However, the following have been described as triggers: abnormalities in the hormonal control, intrinsic abnormalities of muscle fibers, the cells of Cajal and nerve fibers, infections, and increased insulin-like growth factors, as well as genetic predisposition. To date none of the theories fully explains the development of the disease [2] [4]. On the other hand, Krohg and collaborators listed a number of risk factors for the development of HPS, including: male gender, the use of macrolides, and bottle feeding, without having been able to clarify whether the cause is infant formula per se or technical supply [5] [6]. In addition to the vomiting, the disease may be accompanied by hypochloremic alkalosis and indirect hyperbilirubinemia [7]. These biochemical abnormalities, which were not documented in the cases submitted (see **Table 1**), are useful in the diagnosis and therefore are included in the algorithm (**Figure 1**).

Ultrasound imaging is the method most widely used for the diagnosis of HPS, replacing studies with contrast media used initially. Thickness of the musculature of the antrum ≥ 4 mm, accompanied by an elongation of the pyloric chan-

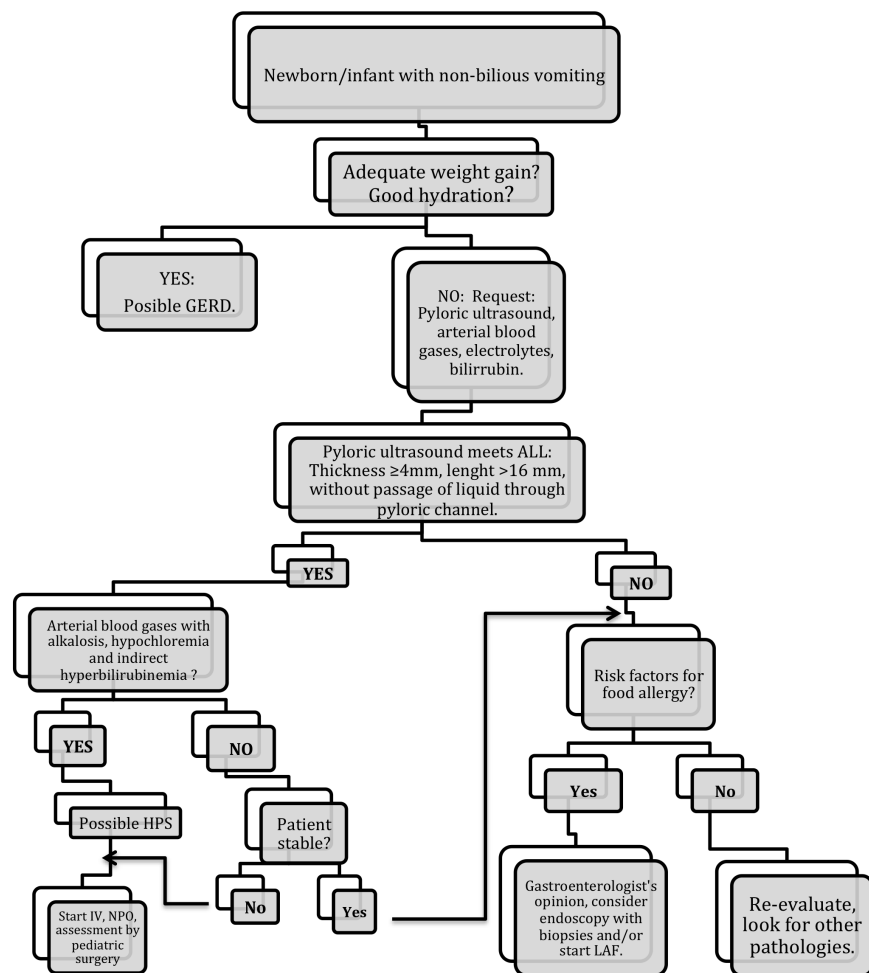


Figure 1. Diagnostic algorithm for neonates/infants with vomiting.

Table 1. Laboratory values in patients submitted (Source: Gastronutriped).

Case	Age	Pyloric Thickness (mm)	Pyloric channel length (mm)	Passage of liquid through pyloric channel	Serum chloride (meq/l)	Bilirubin (me/dl)	Peripheral eosinophilia (%)	PH/HCO ₃
1	2 month	4	17	Yes	Normal	NR	11	NR
2	23 days	3	17	No	Normal	Normal	6	NR
3	1 month	3.3	15.6	No	Normal	TB: 10.12 IB: 9.53	12	7.36/24.2

NR: No Report.

nel >16 mm are the findings needed for the diagnosis. Based on these parameters, the second and the third patient did not meet the diagnostic criteria for HPS.

A thickness of 2 to 3 mm is nonspecific; a value less than 2 mm is considered normal. Pyloric spasms and distension of the gastric antrum with liquid can give the false impression of muscle thickening [8]. For borderline measurements, pyloric channel relaxation after administration of liquid to the stomach excludes the diagnosis of HPS [2]. Although the first case met the ultrasound diagnostic criteria, abnormal measurements could have been due to a “fake” thickening by edema of the gastrointestinal mucosa due to FA; other diagnoses were excluded on the evidence of the passage of gastric contents through the pylorus.

Pyloromyotomy is the surgical treatment of choice for HPS, preferably using the laparoscopic technique, because of the shorter recovery time and lower complication rate. Clinical improvement of HPS has been described with oral administration of atropine; however, treatment failure of 25% and the time it takes for resolution of symptoms make this treatment not routinely recommended [2].

Kaijser first described EG in 1937 [9]. It can occur at any age, with male predominance, as happens with HPS [10]. It is characterized by a diffuse infiltration of eosinophils, which can affect different layers of the GIT, from the esophagus to the anus [11]. There is no consensus for diagnosis; however, Talley proposed three criteria: the presence of gastrointestinal symptoms, biopsies showing eosinophil infiltration into one or more areas of the GIT, and exclusion of parasitic infections and/or extraintestinal disease [12]. Although peripheral eosinophilia is not a diagnostic criterion, its presence increases suspicion [13], as happened in case N.2. EG consists of three histologic types depending on the layer involved. The most common compromise is in the mucosa, in 60% of cases, followed by the muscle (30%) and to a lesser extent (10%) the involvement of the subserosa [14]. The predominant clinical expression is secondary to the infiltration of eosinophils in the mucosa, which causes malabsorption and failure to thrive, the latter observed in cases 1 and 3. Also, the involvement of the muscular layer can predispose the patient to obstructive symptoms. If this involvement is antral, it can be confused with HPS [11], a fact which we assume happened in the three

patients described. Already in 2007, Liacouras suggested that EG should be suspected in an infant with vomiting, dehydration, electrolyte imbalance, and thickening of the gastric outlet, if there are also symptoms of atopy [11]. This is very similar to that described in our three patients and is consistent with the favorable response to nutritional management with AAF. Also, there have been reports of older children and adults with similar symptoms, with suspected HPS, where the symptoms resolved with medical treatment [10] [15]. Since eosinophil infiltration into tissues is segmental, histologic confirmation is difficult, and therefore multiple biopsies should be taken [9]. In EG, thickened mucosa and submucosa can coexist, in contrast to HPS, where the condition is manifested exclusively in the muscle layer. To be reliable, the ultrasound should be performed with a high-frequency linear transducer (7-MHz) [9]. The ultrasound findings help to clarify the diagnosis; however, they are operator dependent and require a high index of suspicion and expertise to avoid confusion, as in case N.2. If in doubt, the opinion of the pediatric gastroenterologist is important, in order to resolve the diagnosis by performing an upper endoscopy with segmental biopsies.

Medical treatment is the mainstay for the management of GE. As part of this treatment, EHF, AAF, and/or allergen elimination diets may be used, which have been found to show clinical and histological improvement. However, there is no consensus with respect to this recommendation [13]. Also, the use of systemic steroids has demonstrated remission of symptoms and normalization of endoscopic, ultrasonographic, and histological findings [11]. In our three patients, symptoms improved with nutritional intervention, two with AAF and one with EHF. Some medications, such as cromolyn sodium, montelukast, anti-IgE, and anti interleukin 5, have some immunomodulatory effect; however, there is insufficient evidence to suggest their routine use [10] [11] [14]. Surgical treatment is reserved for patients with symptoms of obstruction that do not respond to medical treatment [14] [16]. In general, the prognosis of the disease is favorable, as evidenced in the follow-up of the three patients, who continue to be symptom free and with adequate growth and development.

4. Conclusion

HPS is a condition to be considered in the diagnosis of infants with recurrent emesis and symptoms suggestive of gastric outlet obstruction. However, food allergy (FA) should be included as a differential diagnosis, particularly when there are risk factors for allergic sensitization such as c-section, lack of or short duration of breastfeeding, early use of antibiotics, and first-degree relatives with atopy, as we described in the cases presented. While pyloric ultrasound is the most widely used diagnostic method for suspected HPS, it should be remembered that ultrasound findings may be present in both conditions. When the diagnosis of HPS is doubtful, performing a medical therapy trial and/or endoscopic biopsies should be considered as part of the differential diagnosis algorithm (see **Figure 1**).

Conflict-of-Interest Statement

Authors declare that there are no conflicts of interest to disclose.

Authors declare that the parents/guardians of the patients were informed and agreed to the publication of these cases for strictly academic purposes.

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References

- [1] Otjen, J.P., Iyer, R.S., Phillips, G.S. and Parisi, M.T. (2012) Usual and Unusual Causes of Pediatric Gastric Outlet Obstruction. *Pediatric Radiology*, **42**, 728-737. <https://doi.org/10.1007/s00247-012-2375-5>
- [2] Ranells, J.D., Carver, J.D. and Kirby, R.S. (2011) Infantile Hypertrophic Pyloric Stenosis: Epidemiology, Genetics, and Clinical Update. *Advances in Pediatrics*, **58**, 195-206. <https://doi.org/10.1016/j.yapd.2011.03.005>
- [3] Saula, P.W. and Hadley, G.P. (2011) Hypertrophic Pyloric Stenosis in the Third World. *Tropical Doctor*, **41**, 204-210. <https://doi.org/10.1258/td.2011.110145>
- [4] Modarressi, T. (2014) Question of an Infectious Etiology or Contribution to the Pathogenesis of Infantile Hypertrophic Pyloric Stenosis. *Journal of Pediatric Gastroenterology & Nutrition*, **58**, 546-548. <https://doi.org/10.1097/MPG.0000000000000261>
- [5] Krogh, C., Biggar, R.J., Fischer, T.K., Lindholm, M., Wohlfahrt, J. and Melbye, M. (2012) Bottle-Feeding and the Risk of Pyloric Stenosis. *Pediatrics*, **130**, e943-e949. <https://doi.org/10.1542/peds.2011-2785>
- [6] Krogh, C., Gørtz, S., Wohlfahrt, J., Biggar, R.J., Melbye, M. and Fischer, T.K. (2012) Pre- and Perinatal Risk Factors for Pyloric Stenosis and Their Influence on the Male Predominance. *American Journal of Epidemiology*, **176**, 24-31. <https://doi.org/10.1093/aje/kwr493>
- [7] Kleinman, R., *et al.* (2008) Walker's Pediatric Gastrointestinal Disease. PMPH-USA, 852 p.
- [8] Maheshwari, P., Abograra, A. and Shamam, O. (2009) Sonographic Evaluation of Gastrointestinal Obstruction in Infants: A Pictorial Essay. *Journal of Pediatric Surgery*, **44**, 2037-2042. <https://doi.org/10.1016/j.jpedsurg.2009.05.019>
- [9] Hümmer-Ehret, B.H., Rohrschneider, W.K., Oleszczuk-Raschke, K., Darge, K., Nützenadel, W. and Tröger, J. (1998) Eosinophilic Gastroenteritis Mimicking Idiopathic Hypertrophic Pyloric Stenosis. *Pediatric Radiology*, **28**, 711-713. <https://doi.org/10.1007/s002470050448>
- [10] Sheikh, R.A., Prindiville, T.P., Pecha, R.E. and Ruebner, B.H. (2009) Unusual Presentations of Eosinophilic Gastroenteritis: Case Series and Review of Literature. *World Journal of Gastroenterology*, **15**, 2156-2161.
- [11] Liacouras, C.A. (2007) Eosinophilic Gastrointestinal Disorders. *Practical Gastroenterology*, **31**, 53-66.
- [12] Choi, S.-J., Jang, Y.-J., Choe, B.-H., Cho, S.H., Ryeom, H., Hong, S.J., *et al.* (2014) Eosinophilic Gastritis with Gastric Outlet Obstruction Mimicking Infantile Hypertrophic Pyloric Stenosis. *Journal of Pediatric Gastroenterology & Nutrition*, **59**, e9-e11. <https://doi.org/10.1097/MPG.0b013e31828ee524>

- [13] Lucendo, A.J., Serrano-Montalbán, B., Arias, Á., Redondo, O. and Tenias, J.M. (2015) Efficacy of Dietary Treatment for Inducing Disease Remission in Eosinophilic Gastroenteritis. *Journal of Pediatric Gastroenterology and Nutrition*, **61**, 56-64.
- [14] Holroyd, D.J., Banerjee, S., Chaudhary, K.S., Reshamwalla, S., Vijay, V. and Ward, M.W. (2010) Transmural Eosinophilic Gastritis with Gastric Outlet Obstruction: Case Report and Review of the Literature. *Annals of the Royal College of Surgeons of England*, **92**, e18-e20. <https://doi.org/10.1308/147870810X12659688851915>
- [15] Kellermayer, R., Tatevian, N., Klish, W. and Shulman, R.-J. (2008) Steroid Responsive Eosinophilic Gastric Outlet Obstruction in a Child. *World Journal of Gastroenterology*, **14**, 2270-2271. <https://doi.org/10.3748/wjg.14.2270>
- [16] Leal, R., Fayad, L., Vieira, D., Figueiredo, T., Lopes, A., Carvalho, R., et al. (2014) Unusual Presentations of Eosinophilic Gastroenteritis: Two Case Reports. *The Turkish Journal of Gastroenterology*, **25**, 323-329. <https://doi.org/10.5152/tjg.2014.6735>



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