Abstract
Introduction: Vomiting is one of the commonest complaints in children. Recently, a child presented to our hospital with an occult cause of vomiting and failure to thrive, for which we decided hospitalization for further investigations. This case report will follow his evolution, the differential diagnosis and therapeutic measures. Aim: To report a case of an infant with recurrent vomiting since early infancy. Case report: Teodor, a 10 months old child, presented to our gastroenterology department for chronic recurrent vomiting since he was 3 months old. At first, he presented 1 episode of vomiting per day and, after a few months, 3-4 episodes per day. We performed a complete assessment. We note 3 previous visits to our hospital during which efforts were made to diagnose the child’s reason for vomiting. At 3 months GERD and Cow’s milk proteins allergy was suspected, which was later excluded. After the second visit the suspicion of an obstruction on the digestive tract was raised, but the parents refused upper digestive endoscopy or barium passage. When he was hospitalized, barium swallow study was performed and it revealed a massive herniation of the stomach and duodenum. Conclusion: Hiatal hernia should be considered as a differential diagnosis in a patient presenting with vomiting episodes with chronic character and failure to thrive.

Keywords: infant, vomiting, hiatal hernia

Introduction
In infants and children vomiting is a very common symptom, which is actually a protective reflex and can be present in a multitude of disorders that can range from mild illnesses to severe, life-threatening conditions. Although vomiting can originate from the gastrointestinal (GI) tract itself, it can also signal more generalized, systemic disorders. The diagnosis should include a focussed history (including characteristics of vomiting and associated symptoms) and physical examination. Also, investigations like serum electrolytes and blood gases, renal and liver functions and radiological studies are required. A common cause of vomiting in children is cow milk’s protein allergy (CMPA), which we considered in our case, as well. Gastroesophageal reflux disease (GERD) and acute viral gastroenteritis can also be found as the cause.

Case report
A 10 months old boy presented in our clinic with complaints of chronic postprandial vomiting within a few hours from the last meal, in the last 5 days acute character (3-4x episodes/ day) and failure to thrive. The child was born to healthy parents (37 years old mother and 41 years old father) and came from an urban setting. Both parents denied any chronic disease in the family or any atopy. The pregnancy was physiological. The child, GII; PI was born at 38 weeks, with a weight of 3.950 g (appropriate for gestational age) and a height of 53 cm, with an Apgar score of 9. He received breastmilk for 3 months and standard formula afterwards. Solid food was introduced correctly at 6 months. His medical history includes various visits in our department for recurrent vomiting.

The onset was at 3 months with 1 episode of vomiting/day, in the second half of the day or nocturnal, usually with food content, rarely mucus, preceded by psycho-motor agitation. After the first ambulatory presentation, we considered GERD and CMPA. We excluded cow’s milk protein from the infants diet and recommended extensively hydrolyzed formula (3x120 ml/day), H2 antihistamines (Ranitidine:10 mg/kg/day), trimebutin (1ml / kg / day). The evolution was favorable for 3 weeks, but afterwards the symptoms have reappeared. At the second ambulatory presentation, we recommended a barium swallow test and upper digestive endoscopy, the parents refused and decided to continue the diet and the above mentioned treatment. At the third ambulatory presentation, he presented with acute symptomatology, 3-4 postprandial vomiting per day. Admission was necessary for establishing the diagnostic, monitoring and treatment.
**Physical assessment.** On examination, the anthropometric measurements revealed a weight of 7.20 kg (Percentile 2) and a height of 75 cm (percentile 75) using WHO growth standards (Figure 1). His general status was altered. He had no fever at the time of examination. He had a diminished appetite. The skin was slightly pale. The examination of the cardiovascular and respiratory system was normal. His abdomen was soft, no tenderness on palpation. His intestinal transit for stool and gas was normal (type 3 on Bristol stool). There were no signs of meningeal or peritoneal irritation.

![Figure 1. Growth chart showing failure to thrive. As shown by the black arrow, after surgery the child’s weight normalized.](image)

**The blood work-up** revealed high white blood cells (WBC) with normal indices and CRP) and polycythemia, with low red blood cells (RBC) indices, low sideremia and a normal ferritin levels. The arterial blood gas analysis revealed a metabolic alkalosis (pH=7.47; B.E.=-10.1 mmol/L) with low chlorine (89 mmol/L). At admission an hydroelectrolytic re-balancing (calculated for his weight) was established, until normalization of the arterial blood gas analysis. At check-up, the blood-work showed normalization of WBC and RBC. Thus we concluded that the values were influenced by the dehydration status of the patient.

**Consultations.** Ophthalmology, pediatric neuropsychiatry and otolaryngology consultations were normal. We decided to perform a sweat test to exclude cystic fibrosis as the cause of the failure to thrive. We also excluded a gastro-enterocolitis, an urinary tract infection and a metabolic disease (the metabolic screening showed normal ranges for ammonia, lactate and blood glucose).

**Imaging studies.** The ECG and the heart ultrasound were normal, as well as the abdominal ultrasound. The abdominal x-rays described digestive lumen images, located above and below the median line and right. The boy underwent a barium swallow study that completed the diagnostic, revealing a massive herniation of the stomach in the chest (type III paraesophageal hernia), with a gastric volvulus (Figure 2).
Management. During admission he received nasogastric tube feeds with an elemental formula (6x120 ml/day), which he tolerated well, with no gastric residue. Six days after hospitalization, we progressively let him feed by mouth, but the vomiting reappeared. The treatment consisted of hydroelectrolytic and acid-base rebalancing and symptoms’ management (proton pump inhibitors and prokinetics). He was referred to the Pediatric Surgery Clinic, where the herniation was reduced and Nissen fundoplication was performed. The evolution was favorable, with no complications. The boy is presently asymptomatic for hiatus hernia with no symptoms of heartburn, nausea, vomiting and is on a regular follow-up.

Discussions.

Back in the 16th century there were descriptions of hiatal hernia, but until the first half of the 20th century it wasn’t accepted as a clinical entity [2]. The incidence in the pediatric population is low, therefore there is a lack of data concerning the diagnostic and management of hiatal hernia in children. The etiology in this population is mostly correlated with genetic factors, such as familial inheritance, Marfan syndrome, but, most of the cases are congenital [3].

In the literature there are two major types of hiatal hernia described: sliding hiatal hernia and para-esophageal hiatal hernia. A more comprehensive classification divides the paraesophageal hernia in 3 types (II,III and IV- Figure 1). Type I (concentric or axial hiatal hernia), represents more than 95% of all hiatal hernias and is characterized by a widening of the esophageal hiatus, plus laxity of the phrenoesophageal ligament. The clinical significance of this type is in association with GERD [4,5].

Type II, classical form in which the gastroesophageal junction stays below the diaphragm and only the gastric fundus herniates. This type of hernia progressively enlarges and it can cause volvulus or incarceration, which is why it is indicated surgical repair as treatment. Type III actually represents a mixture of type I and II of hiatal hernias, meaning that both the GEJ and the gastric fundus herniated. This type was present in our patient. When type IV of hiatal hernia is described, it means that other organs (such as spleen, colon, pancreas) are found in the thoracic cavity [2].

Other types of hernia, such as congenital diaphragmatic defects, traumatic diaphragmatic hernias, iatrogenic hernias (misguided chest tubes), exist, but they are rare findings.

Hiatal hernia may be asymptomatic, discovered incidentally on routine chest x-rays or CT scans. If it is symptomatic they usually present with gastro-esophageal reflux symptoms (epigastric pain, regurgitations, heartburn), nausea, vomiting, anemia, failure to thrive, melena. They can
complicate- volvulate, strangulate, bleed, giant hernia can give mechanical complications (chest pain, respiratory distress) [4].

To confirm the diagnosis of hiatal hernia barium swallow test, upper gastro-intestinal endoscopy or CT scan need to be used. Barium swallow test is helpful to determine the size of the hernia, orientation of the stomach and to localize precisely the gastroesophageal junction in relation to the esophageal hiatus. Over the past few years, upper digestive endoscopy spread and is now used as a way to diagnose hiatal hernia, the criteria being: the proximal dislocation of GEJ of >2 cm above the diaphragmatic indentation (Z-line). CT-scan can be useful in an urgent situation, when having the suspicion of volvulized PEH, in most cases being able to distinct clearly any herniated organs in the chest cavity [3].

The presence of symptoms of gastroesophageal reflux (since GERD is the most common clinical manifestation) indicate that therapy is needed. Medical treatment consists of antacids, H2 receptor antagonists and PPIs (proton pump inhibitors) [3,6]. Drugs such as prokinetics (Metoclopramide- careful to potential extra-piramidal effects) or 5 Ht3 receptors antagonist (Ondasetron) can be used for the symptoms like vomiting. Patients who are refractory or don’t answer to the treatment are considered for surgical repair. In case of a type I of hiatal hernia with no reflux disease antireflux surgery is not recommended. If GERD is present, the indication of an antireflux procedure (fundoplication) is mandatory. In a prospective trial it was noticed that hernia and symptomatic gastroesophageal reflux managed conservative had high failure rates. Thus, they recommend surgical repair in this population. The transabdominal laparoscopic repair is preferred by most pediatric surgeons. Of course, the morbidity of an open approach being much higher that the laparoscopic approach [3].

Postoperative management consists of attention to the caloric and nutritional intake, because postoperative dysphagia is common. In patients who are asymptomatic after surgery there are no recommandations for routine contrast studies [3].

Though not so frequent in pediatric population, through this paper we hope to bring more data on hiatal hernia in pediatric population and its forms of presentation. The limitations that we, as clinicians have is the fact that the parents refuse what they consider invasive procedures, thus this kind of pathology are diagnosed late. The particularity of this case was the form of presentation with recurrent vomiting and failure to thrive.

Conclusion

Hiatal hernia should be considered as a differential diagnosis in a patient presenting with vomiting episodes with chronic character and failure to thrive...

References


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