International Journal of Gastroenterology
2020; 4(1): 27-30
http://www.sciencepublishinggroup.com/j/ijg
doi: 10.11648/j.ijg.20200401.17
ISSN: 2640-1681 (Print); ISSN: 2640-169X (Online)

Case Report

Intractable Vomiting in a Female Child with Gastritis Not Responding to Treatment: Atypical Pseudotumor Cerebri

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To cite this article:
Hadil Samir Darwish, Riham Rabie Issa, Hadeer Said Allam, Sameh Abd Allah Abd el Naby, Muhammad Ahmed Magdy Abdelhamid, Ahmad Mohamed Sira. Intractable Vomiting in a Female Child with Gastritis Not Responding to Treatment: Atypical Pseudotumor Cerebri. *International Journal of Gastroenterology.* Vol. 4, No. 1, 2020, pp. 27-30. doi: 10.11648/j.ijg.20200401.17

Received: April 17, 2020; **Accepted**: May 5, 2020; **Published**: June 4, 2020

Abstract: Intractable vomiting is a challenging complaint in pediatrics that is caused by many gastrointestinal and extragastrointestinal etiologies. Idiopathic intracranial hypertension (IIH) is one of the serious extragastrointestinal causes that could be overlooked due to non-orientation, atypical presentation, or misleading associated pathology. We present here an eleven-year-old female child with intractable vomiting due to IIH. The presence of concomitant *Helicobacter pylori (H pylori)* gastritis, which was not the cause of the vomiting, played a distraction factor away from the real cause. Moreover, the initial absence of the characteristic papilledema led to a delay in the IIH diagnosis. Treatment failure of *H. pylori* gastritis, especially when associated with headache, visual symptoms, and negativity of all other investigations, should direct attention to IIH even with the early absence of its characteristic papilledema.

Keywords: Helicobacter Pylori Gastritis, Idiopathic Intracranial Hypertension, Intractable Vomiting, Papilledema, Pseudotumor Cerebri

1. Introduction

Vomiting is a common symptom of many underlying conditions for which children present for medical care. While gastrointestinal (GI) causes come to mind first, there are many non-GI causes of vomiting. Extragastrointestinal etiologies include a long list as infection, neurologic, metabolic/endocrine, respiratory, psychogenic, drugs/ toxins, foreign bodies, and cyclic vomiting syndrome (CVS). So, vomiting should be systematically approached as it could be due to an underlying non-GI serious condition [1].

Any condition that increases intracranial pressure (ICP) can result in vomiting with or without nausea. Idiopathic intracranial hypertension (IIH), previously known as pseudotumor cerebri (PTC) is a disorder associated with high ICP that causes symptoms and signs of a brain tumor in the absence of evidence of space-occupying lesion with normal CSF composition. The most common presentations are headache, tinnitus, neck stiffness, visual symptoms, vomiting, and walking difficulties. IIH is not common in pediatrics. Besides, most of the symptoms may not be apparent early. So, it may take a long time to reach the diagnosis [2].

It is not uncommon to have a child with severe vomiting due to non-GI cause. Nevertheless, it is the presence of a

significant GI pathology that is not the cause of the vomiting in the presenting child; a condition which could be misleading and causes a more delay in diagnosis of the real etiology of vomiting; especially when the real cause is rare and presenting in an atypical way.

2. Case Report

An eleven-year-old female child presented to our Pediatric Hepatology, Gastroenterology, and Nutrition Department on December 30, 2019 with intractable vomiting lasting for two and a half months. The condition started on October 10, 2019, when the child experienced hematemesis and upper GI endoscopy was performed in our department which revealed *Helicobacter pylori* (*H. pylori*) gastritis (Figure 1A) and nonspecific duodenitis (Figure 1B); no treatment was given for *H. pylori* gastritis because she missed follow up.

One week later she started to experience vomiting that is preceded by nausea and associated with epigastric pain. Vomiting was always related to meal and has no diurnal variation. Parents sought medical advice in another hospital where she was admitted for more than two months and received intravenous (IV) fluid therapy due to her intractable vomiting. She received antiemetic and multiple courses of anti- *H. pylori* triple therapy. She started to experience headaches, mainly occipital. Barium meal follow-through, MRI brain, and fundus examination were all free. The platelet function test showed a low ristocetin-induced platelet aggregation (15%) and fecal calprotectin was high (145 µg/g). She had no improvement with the persistence of her vomiting, and then she was referred to our hospital.

When presented to us, she seemed generally well with average body built (Table 1), vitally stable with no fever and free systemic examination. On the revision of her history, she had no history of previous similar attacks, no drug history and no previous operations. Family history was unrevealing. She had no visual complaint. However, by direct question, she disclosed some blurring of vision.

Investigational workup showed normal liver function tests, renal profile, complete blood count, ammonia, blood gases, thyroid function tests, and other laboratory findings as shown in table 1. Abdominal ultrasound, barium meal follow-through, computed tomography (CT) abdomen with oral contrast, re-fundus examination, CT brain and magnetic resonance imaging (MRI) brain (Figure 1C), and magnetic resonance venography (MRV) (Figure 1D) and magnetic resonance angiography (MRA) of the brain were all normal. A planned re-upper GI endoscopy was postponed till reassessment of her platelet function tests.

Table 1. *Anthropometric and laboratory data of the child.*

Item	December 30, 2019
Weight (kg)	31
Stature (cm)	137
Total bilirubin (mg/dl)	0.3
Direct bilirubin (mg/dl)	0.0
Total proteins (g/dl)	7.6
Albumin (g/dl)	4.9
ALT (U/L)	21
AST (U/L)	11
ALP (U/L)	213
GGT (U/L)	21
Amylase (U/L)	79
Lipase (U/L)	20
Urea (mg/dl)	20
Creatinine (mg/dl)	0.42
Na (mEq/L)	142
K (mEq/L)	4.4
Ca (mg/dl)	9.5
RBS (mg/dl)	88
Ammonia (µmol/L)	29
Hemoglobin (g/dl)	12.1
WBCs ($\times 10^3$/µl)	6.1
Platelets ($\times 10^3$/µl)	300
ESR	5
CRP (mg/dl)	0.9
Urine culture	Negative
Blood culture	Negative
Stool culture	Negative
Widal test	Negative
Brucella test	Negative

µl, microliter; ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; cm, centimeter; dl, deciliter; g, gram; GGT, γ-glutamyltransferase; Kg, kilogram; mg, milligram; WBCs, white blood cells.

She was maintained on IV fluids, antiemetic, proton pump inhibitors, but with no improvement. A trial of another line of *H. pylori* treatment (levofloxacin, esomeprazole, amoxicillin) also failed to improve vomiting. Despite the non-cyclic pattern of vomiting, propranolol was tried due to negativity of all previous assessments; however all without any response with the persistence of the intractable vomiting immediately after any trial of oral feeding even sips of water. Lastly IV methylprednisolone 1mg/kg/day bid started empirically as a therapeutic test for eosinophilic gastritis. There was an initial improvement of her vomiting for 2 days during which she perceived some oral feeds without vomiting; a relapse occurred on the third day of therapy. Four days later, she had a fainting attack. Her vital signs were all normal. This was followed by successive projectile vomiting, which was for the first time without oral feeding, associated with severe occipital headache and looked very ill.

Urgent CT brain revealed normal imaging. Follow up fundus examination, surprisingly showed bilateral grade I-II papilledema. Follow up MRI, MRV, and MRA brain showed normal findings. A diagnosis of IIH was considered. She started immediately acetazolamide on a dose of 250mg/8 hours together with mannitol. She was planned for a lumbar puncture to measure the CSF opening pressure with CSF analysis. The frequency of vomiting significantly decreased up to no vomiting at all. Follow up fundus examination showed bilateral resolving papilledema. So, lumbar puncture was postponed.

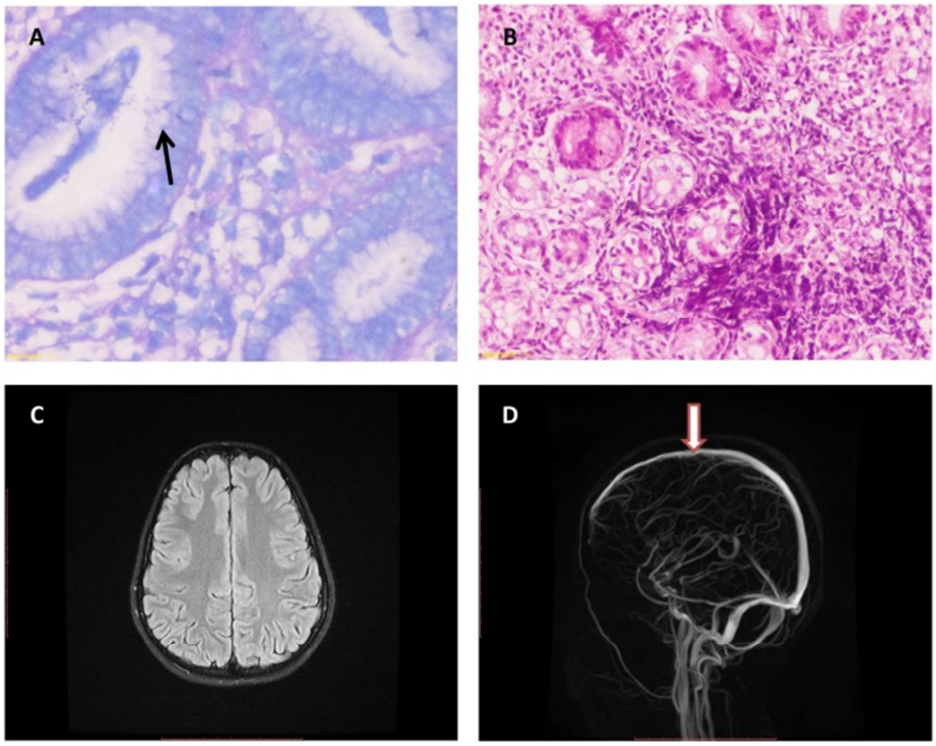

Figure 1. *Gut pathology and brain imaging.*

A: Helicobacter pylori rods at the gastric mucosa (arrow). B: Lamina propria of duodenum shows infiltration by chronic mononuclear inflammatory cells. C: MRI brain (axial cut): it shows no evidence of space-occupying lesions or hydrocephalic changes. D: MRV brain scan: it shows patent superior sagittal sinus (arrow) with no evidence of thrombosis.

The patient experienced more headaches and blurring of vision with subsidence of vomiting. Perimetry visual field testing reported bilateral central scotoma. She was transferred to the Pediatric Neurology Unit and continued on acetazolamide and received high dose methylprednisolone with a dramatic improvement of all symptoms.

3. Discussion

Intractable vomiting in children is one of the challenging presentations to the treating physician. Despite some cases could be treated by the general pediatrician, a magnitude of cases are still challenging even to the experienced pediatric gastroenterologist [1].

It is not surprising to have a non-GI cause of intractable vomiting that lasts for a long time. Nevertheless, the atypical presentation of non-GI causes with the negativity of all the specific investigations for the system affected poses a great difficulty. This difficulty is more and more when it is associated with a frank GI pathology which is not the real cause of the presenting complaint; as in our case with the presence of *H. pylori* gastritis. Nonresponse to the usual treatment of this associated GI pathology should alert the pediatric gastroenterologist to the presence of another underlying cause.

Reporting *H. pylori* infection in children is increasing nowadays with many controversies regarding their management. Many children are infected without having *H. pylori*-induced gastritis. Moreover, despite *H. pylori* gastritis could manifest with abdominal pain, nausea, and vomiting, in others it could be asymptomatic [3]. In our case, gastric biopsy showed a chronic inactive *H. Pylori* gastritis. No response to multiple treatment regimens occurred, as it seems not to be the cause of the underlying symptoms. Recent guidelines recommend the determination of the underlying cause of symptoms in those with *H. pylori* infection. Thus, the "test and treat" strategy is not recommended for children with *H. pylori* infection [4].

In the present case, thorough investigations for all possible extragastrointestinal causes showed negative study; namely infections, endocrine/ metabolic, renal, hepatic, pancreatic, and neurologic causes. At which time CVS, despite non-cyclic pattern in our case, was a possibility. More than one-third of CVS patients can show vomiting symptoms even during the well phase between the vomiting episodes [5]. Besides the given PPI and the antiemetic ondansetron, a trial of propranolol together with ondansetron failed to have any impact on the course of vomiting.

Despite the absence of described pathological picture of eosinophilic gastritis in gastric biopsies of the child, it has been reported that gastric eosinophilia could be limited to the fundus [6]. So, a therapeutic test with steroids was tried. Interestingly, there was a dramatic response with improvement of vomiting for two days, then relapsed again.

While IIH is more commonly recognized as a disorder of adults, it affects children of all ages with increased incidence around 12- 15 years of age [7]. Despite papilledema is a hallmark for its diagnosis; it shouldn't be missed even with initial negative assessment [2, 8]. Etiopathogenesis of IIH is not completely clear, so multiple theories were suggested. Some drugs can put the patient at risk for developing IIH, like steroids [9]. Our reported child had her frank presentation of IIH with projectile vomiting and bilateral papilledema after steroid therapy for one week. It could be the factor that triggered the typical presentation of a previously atypical disease without papilledema.

Presentation of IIH may be asymptomatic [10], or in the great sector, with visual symptoms like blurring of vision or transient vision changes that can be explained by transient ischemia of the optic nerve and rapid loss of vision, which is the most feared sequel of IIH. The second most common presentation is a headache that is accompanied by nausea and vomiting. In our case headache was not severe and blurring of vision was masked by the severity of vomiting. Vomiting as a main complaint as in our case hasn't been reported before.

IIH is a diagnosis of exclusion. Papilledema which is the main presenting sign may be absent at least early as reported in some cases [2]. Despite measuring CSF pressure hasn't been performed in our case, the dramatic improvement after starting brain dehydrating measures was in favor of IIH diagnosis; especially with the presence of all other modified Dandy criteria [7] which are; 1) increased ICP symptoms and signs, 2) no localizing findings on neurological examination, 3) normal MRI/CT brain scans with no evidence of central venous sinus thrombosis, 4) no other identified cause of increased ICP.

4. Conclusion

In conclusion, IIH should be considered in children with intractable vomiting when there are associated headache and visual symptoms, even in the absence of the characteristic papilledema. Moreover, the presence of *H. pylori* infection and even related gastritis should be approached cautiously as it couldn't be the cause of the presenting symptoms.

Acknowledgements

We would like to thank the residents and nursing staff of the Pediatric Hepatology, Gastroenterology, and Nutrition Department and all physicians and working staff of the Radiology Department, and Pediatric Neurology Unit for their contribution.

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