Problems in the Diagnosis of Cyclic Vomiting Syndrome in Children

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ABSTRACT

The cyclic vomiting syndrome (CVS) is an infrequent condition in pediatric practice, in which recurrent vomiting episodes are followed by asymptomatic periods. The authors report the case of an 8-year and 2-month old child who had been hospitalized on several occasions for persistent vomiting accompanied by nausea, and periumbilical and epigastric abdominal pain. The child’s anamnesis and clinical examination determined the doctors to suspect a CVS, which was later confirmed due to ruling out all of the differential diagnoses: infectious, drug-related or surgical causes. CVS is a severe condition, which causes nutrition disorders, social integration problems, school absenteeism and lower life quality on the whole. This condition requires early detection in order to initiate adequate supportive therapy, to identify the triggering causes and to prevent them.

Keywords: CVS- cyclic vomiting syndrome, children

INTRODUCTION

The cyclic vomiting syndrome (CVS) is an uncommon condition in pediatric practice, in which recurrent vomiting episodes are followed by asymptomatic periods, the diagnosing being more difficult to establish due to the lack of convincing results of the biochemical and medical imaging tests (1-3). Vomiting occurs about 6 times an hour, it consists of cholelemesis (83% of the cases) and is accompanied by nausea (4). Other associated symptoms include paleness, apathy, anorexia, nausea, flatulence, abdominal pain, headache and photophobia, all of which make it difficult for the doctor to distinguish between the CVS and other bilious vomiting (5-7).

Disease etiology and pathogenesis have not been fully elucidated. Diagnosis setting is rather difficult since it is an exclusion diagnosis. There are no specific laboratory tests able to diagnose the CVS, and the positive diagnosis only rules out other vomiting causes (8-10).
CASE REPORT

An 8-year and 2-month old male child who were stunted (BMI = 12.6 Kg/m², p<3DS) came in for food vomiting followed by bilious vomiting also accompanied by periumbilical and epigastric abdominal pain. According to his medical history, there are records of multiple hospitalizations in the pediatric clinic for persistent bilious vomiting accompanied by nausea and periumbilical and epigastric abdominal pain. The disease onset was during his first year of life when the vomiting episodes were initially interpreted as gastroesophageal reflux disease. After the age of one, the patient continued to experience 1-2 vomiting episodes per month, with asymptomatic periods lasting between 1 and 2 months. His vomiting was first thought to have been triggered by recurrent upper airway infections, but it also persisted after the adenotonsillectomy.

The child was admitted to our clinic for influence health status, signs of dehydration, decreased subcutaneous tissue (BMI =12.6Kg/m²). The clinical exam revealed abdominal pain in the epigastrium; bilious vomiting more than 5-6 times per hour; normal intestinal transit time, and no signs of meningeal irritation. The therapy consisted of antiemetics (Metoclopramide, Osetron intravenous administration), and fluid and electrolyte rebalancing perfusions.

Laboratory tests and imaging examination were conducted on the patient in order to determine the etiology of his cyclic vomiting. The biological tests revealed no inflammatory syndrome, normal plasma and urine amino acids levels and non-acidic ketosis. The immunology tests included the IgA and IgG anti-transglutaminase antibodies and Helicobacter Pylori antigen stool, which tested negative, whereas the iontophoresis was normal. The abdomen ultrasonography revealed: supple dysmorphic gallbladder, undiluted main bile ducts. The brain computer tomography with contrast agent did not reveal any pathological changes. The upper digestive endoscopy of the esophagus revealed: congested esophageal mucosa in the lower 1/3 with marked vascular network, the stomach filled with sero-mucous stasis fluid and bile, congested mucosa in the antral area, pale duodenal mucosa. The histopathological examination: moderate active chronic gastritis and duodenitis, intraepithelial lymphocytes (LIE) <30%, normally formed intestinal villi on the examined samples. All these tests ruled out vomiting with organic causes and determined the doctors to set a positive diagnosis of CVS.

The clinical re-evaluation after 6 months revealed a significant weight deficit with a body mass index (BMI) <3%. The carbohydrate metabolism after a 12-hour fasting was normal, the liver and kidney function was normal, and so was the endocrinological function. The abdominal ultrasound revealed gallbladder with thin incomplete infundibular septum, a small free fluid between intestinal loops in ileal region and in the right iliac fossa, and high although still normal portal and splenic veins diameter (note the patient’s age). The X-ray revealed bone age corresponding to the patient’s 6-year age, diffuse osteopenia with thin radio-transparent stripes in the radial distal metaphysis. The 25(OH) vitamin D dosing revealed insufficient vitamin D levels. After 3 months of 25(OH) vitamin D supplementation (1,000 IU/day), the patient’s subsequent evolution was favorable. The ultrasonography performed 3 months later revealed no fluid collection. These tests ruled out organic cause vomiting, and the exclusion diagnosis set by the doctors was CVS.

DISCUSSION

The CVS was first described by Heberden in France, in 1806, and then by Samuel Gee in Great Britain, in 1882. It was previously thought that the CVS was a condition that affects mostly children (1,2).

This condition is characterized by severe nausea and vomiting episodes, which last a few days and occur several times a year. The patient experiences asymptomatic periods between these fits. The associated symptoms include paleness, apathy, anorexia, nausea, flatulence, abdominal pains, headache and photophobia, and make it difficult for the doctor to distinguish between the CVS and other conditions. The vomiting occurs during the morning and is associated with a series of psychological factors (birthday, holiday, a school-related event) or other causes such as infections, insufficient sleep (1,2). Its etiology and pathogenesis remain unknown. There seems to be a connection between the CVS and migraines, which is supported by the similarity of the symptoms specific to both conditions.
There is a high migraine prevalence in families with the CVS (8-10). No family history of migraine or other neurological diseases was identified. The mechanism includes the occurrence of several mutations in the mitochondrial DNA, which lead to deficient energy generation in the cell and/or increased hypothalamic stress, which causes an emetic response (10). The precise etiology and pathogenesis of the CVS are still unknown. Nonetheless, a combined management between pharmacology, psychotherapy, psycho-education and lifestyle changes led to good results and to the improvement of the patients’ lifestyle (11).

Consensual diagnosis criteria:
• At least 5 episodes, or a minimum of 3 over a 6-month period
• Episodic attacks of intense nausea and vomiting lasting 1 hour to 10 days, occurring at least 1 week apart
• Stereotypical pattern and symptoms in the individual patient
• Vomiting during episodes occurs at least 4 times an hour for at least 1 hour
• A return to baseline health during episodes
• Symptoms cannot be attributed to another disorder (1).

Differential diagnosis: Medication and toxic agents (antibiotics, high vitamin doses, hormone-based medication, laxatives); infectious causes (enteric agents, hepatitis, Epstein-Barr virus, otitis media, chronic sinustitis); gastrointestinal factors - intestinal obstacles (malrotation with volvulus, hernia cyst doubling), inflammatory lesions (peptic ulcer, gastritis, duodenitis, chronic appendicitis, inflammatory intestinal disease), pancreatitis, liver-gallbladder conditions; neurological and psychiatric conditions (migraine, epilepsy, anxious status, bulimia nervosa); space-occupying lesions (hydrocephalus, posterior fossa tumors, subdural effusion); renal factors (ureteropelvic junction obstruction, renal lithiasis); metabolic and endocrine factors (diabetes mellitus, Addison’s disease, pheochromocytoma, organic aciduria, fatty acid oxidation deficiencies, mitochondrial dysfunctions, urea cycle deficiencies, aminoaciduria).

Treatment:

Prophylactic actions: It is important to identify the causes that trigger the event and to prevent them as much as possible. Therefore, the patient should avoid strong emotions, sleep deprivation, triggering foods (chocolate, cheese), motion sickness (1,9). The avoidance of such triggering factors may reduce the frequency of the vomiting episodes. In case the patient’s anamnesis reveals the occurrence of vomiting induced by the lack of ingested food, the recommendations include eating snacks which are rich in carbohydrates in between the meals, before any physical exertion and before sleeping. Specialists recommend a healthy lifestyle and migraine prevention measures: keeping regular eating and sleeping hours, proper hydration and avoidance of caffeine. Improved their efficiency in the improvement of the course of CVS in children and adolescents (2). Prophylactic drug therapy is recommended if the vomiting episodes are frequent (more than one episode every 1-2 months) (2).

Management of vomiting fits: fluid and electrolyte balance recovery, antiemetics (Osentron 0.3-0.4 mg/Kg every 4-6 hours, maximum 20 mg/day), sedatives (Lorazepam 0.05-0.1 mg/Kg every 6 hours, Chlorpromazine 0.5-1 mg/Kg every 6 hours), analgesics (Ketorol 0.4-1 mg/Kg every 6 hours, maximum 120 mg/day), abdominal pain treatment (Ranitidine, Pantoprazole) (2).

Most published series indicate that CVS lasts an average of 2.5-5.5 years, resolving in late childhood or adolescence (12). Our patient continued to be symptomatic in early adolescence.

CONCLUSION

The CVS is a severe condition, which causes nutrition disorders, social integration problems, marked school absenteeism and lower life quality for both the patient and his or her family.

The early recognition of this condition is vital for an adequate supportive therapy, for the identification of the triggering causes and their prevention, for the changing of the patient’s lifestyle and for his or her psychological support designed to prevent vomiting episodes, and to promote social integration and life quality improvement.

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REFERENCES


