

Cyclic Vomiting Syndrome Developed after Stroke

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Cyclic vomiting syndrome is characterized by recurrent episodes of stereotyped vomiting separated by regular symptom-free periods. We describe a case of cyclic vomiting syndrome developed after stroke, which has not been reported to date. A 69-year-old woman experienced recurrent vomiting following left cerebral infarct. The patient's vomiting pattern was consistent with cyclic vomiting syndrome, and the diagnosis of cyclic vomiting syndrome was established by exclusion of other known disorders which could have resulted in vomiting. She was treated with imipramine hydrochloride and her symptom was well controlled.

Key Words Cyclic vomiting syndrome, Stroke

INTRODUCTION

Cyclic vomiting syndrome (CVS) is characterized by recurrent episodes of repetitive and stereotyped vomiting separated by regular symptom-free periods.¹ The diagnosis of CVS is primarily based on history and clinical presentation. CVS is diagnosed if the following conditions are satisfied: stereotypical episodes of vomiting marked by sudden or acute onset with a duration <1 week, 3 or more discrete episodes, no evidence of organic problems, and absence of symptoms between attacks.^{1,2}

Although the pathophysiology of CVS has not been clarified, several hypotheses have been suggested. The

current theory is that CVS is a functional brain-gut disorder involving central neuroendocrine and peripheral gastrointestinal mediation.^{1,3} Anatomical structures responsible for vomiting are neurons within the hypothalamus, the pituitary gland, and the brainstem. If the organic problems related to these structures, such as hydrocephalus, brain tumor, and Budd-Chiari malformation are implicated, CVS should be excluded according to the diagnostic criteria.^{1,4} We present a patient in whom CVS developed following stroke in a region not previously associated with vomiting. To our knowledge, CVS related to a stroke has not been previously reported.

CASE REPORT

A 69-year-old woman was admitted complaining of sudden deterioration of right pre-existing hemiplegia (day 1). She continued to have mild right hemiplegia after 2 previous strokes. Apart from diabetes mellitus, there was no other remarkable medical history. Brain magnetic resonance imaging showed acute infarct in the left corona radiata, which was surrounded by chronic infarct. The

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left ventricle was asymmetrically enlarged due to periventricular tissue loss. There was bilateral leukoaraiosis. No abnormal findings could be seen in structures known to be associated with vomiting (hypothalamus, pituitary gland, and brainstem). After admission, recurrent vomiting developed at intervals of -1 week. The follow-up magnetic resonance imaging was unchanged.

On day 39, she was transferred to our department for rehabilitation. She was alert and her Mini-Mental State Examination score was 19/30. On neurologic examination, she had right hemiparesis with sensory abnormalities, accompanied by right facial palsy and mild dysarthria without aphasia. Deep tendon reflexes were increased in the right limbs. She did not complain of abdominal symptoms, nor was there anorexia, nausea, diarrhea, or constipation.

During rehabilitation, sudden vomiting continued at intervals of -1 week. The vomiting was repetitive and stereotyped. She denied vomiting prior to the recent stroke. The vomiting rapidly developed following nausea and abdominal discomfort, during or after taking medications in the morning. The repeated vomiting occurred 2 or 3 times over a period of 1 hour. After cessation of the vomiting, associated anorexia and anxiety disappeared during the next 24 hours. During intervals of 1 week, the patient was free of associated episodes. Treatment with metoclopramide and mosapride was not effective. Tests for blood sugar, serum glucose, and hemoglobin A1c were consistently normal. To be diagnosed with CVS, various other disorders that can produce cyclic vomiting should be excluded, such as gastrointestinal, renal, neurologic, and endocrinologic disorders.¹ To rule out other conditions associated with cyclic vomiting, she underwent brain magnetic resonance imaging, laboratory tests, simple abdominal x-ray, gastric endoscopy, and abdominal and pelvic ultrasonographies. Results for all diagnostic tests were unremarkable.

Based on the typical symptoms and examinations, she was diagnosed as having CVS. On day 86, the patient was treated with imipramine hydrochloride (25 mg) at bedtime. There were no side effects of imipramine hydrochloride. The dose of this medication was gradually increased to 50 mg at bedtime. Eventually, her symptom was controlled.

DISCUSSION

The symptoms of the patient were consistent with CVS. CVS consists of an inter-episodic phase (without any symptoms), a prodromal phase, an emetic phase, and a recovery phase (from subsidence of the vomiting to returning to normal).⁵ The patient had an inter-episodic phase of -1 week in duration. The prodromal symptoms consisted of nausea and abdominal discomfort which started when the patient began to sense the approach of the episodes. These symptoms are the most common prodromal symptoms of CVS.⁵ The duration of the emetic phase was 1 hour, with all of the vomiting occurring in the morning. This is a typical feature of CVS.⁵ This phenomenon is due to a characteristic of the neuroendocrine system, which is based on the theory of brain-gut disorder. The important regions for brain-gut disorder are thought to lie within the hypothalamus and the periaqueductal gray matter of the brainstem. The former controls the release of corticotrophin-releasing hormone in response to stress and the latter controls the autonomic function.³ The interaction between the peripheral afferent stimuli from the gut and these brain regions regulates vomiting. The brain-gut disorder reflects a current hypothesis that in CVS, there is a dysfunction of this interaction.^{1,3-5} The basal secretion of corticotrophin-releasing hormone has a diurnal rhythm. Secretion of the hormone begins to increase at 1 AM and reaches a peak at 6 AM. This may account for the predominance in morning times of the onset of CVS.¹ Many patients with CVS have a triggering factor.^{1,3,5} The triggering factor in our patient was the morning medication. The patient did not have any other symptoms associated with the medication or inter-episodic phase without the episode. Based on these findings, the possibility that cyclic vomiting resulted from the drug was less likely. The patient had a recovery phase that lasted for a day, and there were anorexia and anxiety during the phase.

The diagnosis of CVS requires that various organic disorders associated with a vomiting should be excluded; these disorders include gastrointestinal, renal, neurologic, and endocrinologic disorders.¹ For diagnosis of CVS, our patient underwent brain magnetic resonance imaging, laboratory tests, simple abdominal x-ray, gastric endoscopy, and abdominal and pelvic ultrasonographies. Magnetic resonance imaging excluded neurologic disorder.

ders such as hydrocephalus, brain tumor, and Budd-Chiari malformation. Laboratory tests including complete blood count, urine analysis, and chest x-ray excluded infections. Laboratory hormonal tests excluded endocrinologic disorders. Gastric endoscopy excluded esophageal disorders and peptic ulcer disease. Simple abdominal x-ray excluded bowel obstruction and ileus. Abdominal ultrasonography and laboratory tests that included liver function tests, excluded gastrointestinal disorders such as hepatitis, pancreatitis, cholelithiasis, tumors, and renal disorders. Pelvic ultrasonography excluded gynecologic disorders, such as ovarian cyst. Absence of abdominal symptoms such as diarrhea and abdominal pain excluded motility disorders and inflammatory bowel disease. There was a limitation of this study. The patient was elderly. Functional disorders associated with the delay of emptying of gastric and small bowel should also be excluded. We did not perform functional studies. However, clinical findings of cyclic vomiting and inter-episodic phases without any symptoms supported that there was very little likelihood of such disorders.

It has been well known that neuroendocrine and autonomic dysfunction develop after stroke, although the etiologies have not been definitely demonstrated.⁶⁻⁸ Peripheral afferent stimuli of the gastro-intestine are regulated by vagal afferent signals which are controlled by the autonomic system.⁴

No standard evidence-based regimen currently exists to treat CVS.¹ The treatment is based on the phases of CVS. The best treatment is to prevent the episodes of CVS during inter-episodic phases.^{1,2,5} A tricyclic antidepressant which decreases cholinergic neurotransmission in the medulla is widely used in the phase. Thus, the medication can modify the brain-gut axis and autonomic

dysfunction, a possible pathomechanism for CVS.² Our patient completely recovered with the medication.

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