

A Rare Cause of Abdominal Pain: Abdominal Epilepsy

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Abstract

Abdominal epilepsy (AE) is characterized by paroxysmal gastrointestinal complaints, symptoms of central nervous system disturbance, abnormal electroencephalogram with findings specific for a seizure disorder, and improvement with anticonvulsant drugs. AE is a very rare cause of recurrent abdominal pain in children. In this article, we present the case of a 5.5-year-old patient with gastrointestinal and central nervous system disturbance symptoms who was diagnosed with AE. (JAEM 2015; 14: 44-6)

Key words: Abdominal epilepsy, abdominal pain, childhood

Introduction

Abdominal pain is a common complaint in childhood, although the cause may not always be detected. Recurrent abdominal pain is an important clinical entity that is frequently encountered by pediatricians. Abdominal epilepsy (AE) is a very rare cause of this clinical entity, and it may sometimes be overlooked in the differential diagnosis. AE is characterized by episodic paroxysmal abdominal and periumbilical pain, various abdominal complaints, specific abnormalities on electroencephalography (EEG), and good response to antiepileptic drugs (1). Gastrointestinal signs and symptoms may be the only indication of convulsions. It is believed that painful epileptic seizures stem from the parietal and/or temporal lobe (2). Abdominal complaints including nausea, vomiting, diarrhea, and bloating have been reported before epileptic seizures, particularly in temporal lobe epilepsy (3).

In this report, we describe the case of a 5.5-year-old girl diagnosed with AE who was admitted to the emergency department because of abdominal pain, vomiting, difficulty walking, and urinary incontinence.

Case Presentation

A 5.5-year-old female patient was brought to the emergency department with abdominal pain, vomiting, tremor in the legs, urinary incontinence, and somnolence. Her medical history revealed that she had been admitted to the hospital for recurrent abdominal pain every 3-4 months in the last 2 years. Her parents reported that abdominal pain had persisted around the umbilical region in previous episodes

for approximately 1 h and that the pain was sometimes accompanied by vomiting. The episodes had mostly lasted less than 1 h and had spontaneously resolved, followed by somnolence. The complaints of tremor in the legs, urinary incontinence, and difficulty walking had occurred for the first time. Her medical history did not include seizures, systemic disease, long-term use of medication, or trauma. Investigations, including complete urinalysis, hemogram, biochemical tests, abdominal ultrasonography (USG), and familial Mediterranean fever (FMF) gene mutation analysis, that had been performed previously by other health institutions following these episodes were normal, and the patient had been treated symptomatically.

On physical examination, the patient's weight was 18.5 kg (25-50 p), and her height was 112 cm (50 p). On neurological examination, the patient awoke in response to verbal stimuli, but she was somnolent. The muscle strength in both lower extremities was 3/5. Other system examinations were normal. Routine biochemical tests and blood gas analysis were normal. Her fibrinogen levels were 210 mg/dL, C-reactive protein levels were 0.34 mg/dL, and sedimentation rate was 12 mm/h. Hematological data revealed white blood cell counts of 14.6 k/ μ L, hemoglobin levels of 9.3 g/dL, and platelet counts of 216 k/ μ L. The patient was admitted to the pediatric intensive care unit. The prediagnosis was acute encephalitis, and ceftriaxone (100 mg/kg/day) and acyclovir (30 mg/kg/day) were started. Anti-HAV, HBs Ag, anti-HCV, ANA, and anti-ds-DNA were negative, and anti-HBs was positive. There were no parasites or occult blood on stool examination. Urine and stool cultures were negative. The patient was assessed in the pediatric surgery department using abdominal USG and supine and erect abdominal graphy; however, pathology requiring surgical intervention was not detected. Computerized tomography and magnetic resonance im-



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Received: 10.09.2013 **Accepted:** 04.12.2013

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DOI: 10.5152/jaem.2015.46704



Figure 1. EEG showing prominent epileptiform activity in the frontal region
EEG: electroencephalography



Figure 2. EEG returned to normal after the use of anticonvulsants
EEG: electroencephalography

aging were normal. A lumbar puncture was postponed because the patient's general condition was poor. After 4 h, the patient regained consciousness, the somnolence disappeared, and the powerlessness in her legs began to reduce.

Electroencephalography was performed to evaluate the neurological signs of the patient. EEG revealed epileptiform activity characterized by sharp and slow wave complexes over the frontal areas (Figure 1). As FMF gene mutations and blood fibrinogen levels were normal, FMF was not considered. The patient was diagnosed with AE on the basis of the clinical manifestations and EEG findings. Valproic acid was started. After treatment with the drug, the abdominal pain attacks did not recur. Her EEG had returned to normal after 3 months (Figure 2). Given the normal EEG and absence of clinical findings, the valproic acid treatment was stopped at 6 months. Follow-up of painful seizures and EEG checks are ongoing.

Discussion

Abdominal epilepsy is characterized by unexplained paroxysmal gastrointestinal symptoms (which are rare, repetitive, and often self-limiting), clinical signs of central nervous system disorders, abnormal EEG findings, and good response to antiepileptic drugs (4).

The pathophysiology of AE is unknown. The seizures generally arise from the temporal lobe, although parietal or frontal lobe involvement has also been reported. In a study of 150 patients with recurrent abdominal pain, the epileptic focus was generalized in 29.75% of cases; the remaining cases involved the temporal (35.15%), frontotemporal (32.45%), and frontoparietal lobes (2.7%) (5). The epileptic focus was the frontal lobe in the current case.

The disease has a wide spectrum of clinical manifestations. The most common clinical manifestations of AE are abdominal pain, nausea, bloating, diarrhea, nervous system symptoms, headaches, confusion, and syncope (6). Abdominal symptoms may suggest irritable bowel syndrome. However, unlike irritable bowel syndrome, AE is associated with changes in consciousness and abnormal EEG findings during the attacks, followed by fatigue after the attacks (7). Zinkin et al. (8) have reported that abdominal pain (86%), mental status changes (64%), generalized tonic-clonic seizures (36%), lethargy (36%), nausea and/or vomiting (28%), and diarrhea (5%) are common clinical findings in AE. Neurological symptoms, such as convulsions and mental status changes, accompanied by abdominal pain are an important clue in AE. However, every episode may not be accompanied by neurological symptoms. In our patient, there were few complaints of gastrointestinal and neurological systems.

Various drugs, either single or combined, have been used in the management of AE. There are no definite recommendations on the drugs that should be selected. In the study by Kshirsag et al. (5), oxcarbazepine significantly reduced the symptoms in patients with AE. Peppercorn and Herzog (9) used phenobarbital, phenytoin, valproic acid, and carbamazepine in their patients. Anticonvulsant drugs are recommended for all patients (for a maximum of 2 years) until their complaints have disappeared or EEG has returned to normal (5). EEG alone is not decisive in these patients, and it is important to correlate the findings with the clinical status (10). Patients with AE should be followed-up to detect episodes of abdominal pain, and EEG checks should be performed routinely (2). In our case, the patient's symptoms rapidly improved after initiation of valproic acid. EEG returned to normal after 3 months, and the anticonvulsant therapy was stopped at 6 months. Follow-up to detect painful seizures are ongoing, together with EEG checks.

Conclusion

After exclusion of known causes, diagnosis of AE should be considered in children with episodes of paroxysmal abdominal pain, nausea, and/or vomiting accompanied by neurological symptoms. Investigations such as EEG can be helpful in the differential diagnosis. Anticonvulsant therapy should be initiated after a diagnosis of AE.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - Ç.K.; Design - Ç.K., M.T.; Supervision - M.T.; Funding - M.Ç., K.Ö.; Materials - M.Ç., Ç.K.; Data Collection and/or Processing - Ç.K., K.Ö.; Analysis and/or Interpretation - Ç.T., M.T., M.Tu.; Literature Review - Ç.K.; Writer - Ç.K., M.T.; Critical Review - M.T.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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