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Convulsion Associated with Gastroenteritis

Maraym Khalesi (MD)[1], Farhad Heydarian (MD)[1], Seyed Javad Sayedi (MD)[1], Shima Badzai (MD)[1], Elahe Heidari (MD)[2]*

[1]Department of Pediatrics, Ghaem Medical Center, Mashhad University of Medical Sciences, Mashhad, Iran.
[2]Department of Pediatrics, Mashhad University of Medical Sciences, Mashhad, Iran.

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ABSTRACT

Convulsion with mild gastroenteritis is an afebrile seizure associated with viral gastroenteritis in a healthy child without fever, dehydration, electrolyte imbalance, meningitis, or encephalitis. Convulsion with mild gastroenteritis is more common in children aged 1 to 2 years. Usually, Convulsions are brief generalized tonic colonic type. Most convulsions occur within first 24 hours of illness onset. Rotaviral gastroenteritis is known as the most common type of gastroenteritis associated with Convulsion. Laboratory investigations are normal. Also EEG and neuroimaging are usually normal. Long term antiepileptic treatment is not necessary. It is usually a benign condition with good prognosis and no risk for developing epilepsy in future. Considering this etiology of seizure could prevent supernumerary evaluations and long-term antiepileptic treatment.

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Introduction

Convulsion is a common pediatric problem that can be caused by multiple etiologies; including: febrile seizure, infections, abnormalities of the brain, epilepsy, metabolic diseases and etc. Convulsion with mild gastroenteritis (CwG) is a new entity in the etiology of convulsion in children triggered by a mild gastroenteritis. CwG were first reported in Japan by Morooka in 1982 (1,2). In recent years, numerous reports have described CwG, mainly in Asian countries, also recently in Western countries (3-8). In this review we describe CwG in children.

Literature Review

Clinical manifestations

CwG is defined as a convulsion following a mild gastroenteritis without coexisting fever (body temperature <38 ^{0}C), dehydration, electrolyte imbalance, meningitis, or encephalitis in a healthy infant or child. The prenatal history and neurodevelopment status are normal. There is no family history of epilepsy (9-12). The interval between the onset of gastroenteritis and seizures ranges from 1 to 6 days (mean 2.3-3.8 days) (10,11). The average interval between the onset of gastroenteritis and seizures is 3.8 days but most convulsions occur within first 24 hours of illness onset (13). However, sometimes seizures occur before the onset of gastroenteritis. The duration of gastroenteritis ranges from 2 to 7 days, and median number of stool defecation is 5 per day. Mean rectal temperature at the moment of seizure is 37.1 ^{0}C. Typical symptoms of gastroenteritis are vomiting and diarrhea. Less vomiting and more diarrheas are noticed after seizure. There is often mild dehydration (5%). Seizure is brief (usually lasting from 30 seconds to 5 minutes), rarely it can last for longer than 30 minutes (4,7,14). Seizure is often

***Corresponding author:** Elahe Heidari.
Department of Pediatrics, Mashhad University of Medical Sciences, Mashhad, Iran.
E-mail: heidarie@mums.ac.ir
Tel: + 985138012496

a generalized tonic colonic convulsion. However, sometimes there are focal seizures such as complex partial seizures (9,12,13). Wang et al. evaluated clinical manifestations of 48 patients with CwG in Japan, 72.92% patients had clustered seizures in their episodes. Most seizures were generalized tonic–colonic (83.33%) and brief (93.75%) (15). In another cohort study out of 14 CwG patients, 12 children had generalized seizures and 2 had absence seizures. The number of seizures per child ranged from 1 to 8. 85.7% of children had seizures lasting lesser than 4 minutes. In 2 children seizures lasted for 10 minutes. Convulsions did not recur after the first day in 10 children, 3 children had recurrences on the second day and one child on the fourth day. No child had seizure recurrence after 4 days (6).

Epidemiology and pathophysiology

The incidence of CwG is higher in Asia suggesting that CwG may be predominant in certain ethnicities (16). CwG is more common in children aged 1 to 2 years with a range between 1 month to 6 years. It is slightly more common in females with a male to female ratio of 1:1.5-1.8. There is a seasonal pattern for CwG so that it is more predominant in winter and early spring, concurrent with the outbreaks of viral gastroenteritis especially rotavirus gastroenteritis. It occurs in 2%-3% of rotavirus gastroenteritis (4,17). Prevalence of stool antigen of rotavirus was 26 to 56% in different studies. Pathophysiology of these convulsions is not yet clear, but effects of virus toxins on the central nervous system have been suggested as the possible pathogenic mechanisms. One hypothesis is that rotavirus may directly invade the CNS via the bloodstream and consequently cause encephalopathy, encephalitis, or seizures. This hypothesis is suggested by detection of reverse transcription polymerase chain reaction of Rotavirus RNA in the throat, blood and cerebrospinal fluid, and IgG anti-rotavirus in the cerebrospinal fluid of only some affected children (4).

Laboratory, EEG and neuroimaging findings

Laboratory investigations such as white blood cell count, serum glucose, serum urea, creatinine, sodium, potassium, calcium and magnesium level are normal. Stool exam is negative for white blood cell, red blood cell and pathogenic bacteria. CSF examinations are normal with sterile cultures for bacteria and viruses and negative polymerase chain reaction for rotavirus. There is often no epileptic discharge in EEGs (11). Tingsong et al. evaluated interictal electroencephalographic findings in 32 patients with CwG. The EEG was normal in 56.25%. Two types of abnormalities were recorded. Slow background activity (37.5%) and suspected sharp wave complexes with predominance in the occipital region (3%) and sharp wave discharge in the frontal parietal region (3%) (18).

There is no indication for neuroimaging in patients with CwG due to normal pattern of imaging in most patients. An interictal decrease in occipital blood flow can be seen in single photon emission computed tomography if it is carried out (11,19). Tingsong Li et al. performed neuroimaging in 28 patients with CwG: cranial CT scan in 15 patients and brain MRI in 13. All CT scans were normal. Enlarged extra cerebral space in the temporal lobe and hypo-intensity in T2WI in centrum semiovale were seen in 3 patients (18).

Treatment, prognosis and prevention

Antiepileptic treatment is not necessary for a single brief seizure, what happens quite often. Treatment is useful only for recurrent seizures. Diazepam is not recommended for the treatment of seizure in CwG and most patients treated with diazepam experience another seizure after administration of diazepam (Because diazepam does not have any effect on it). Several studies have suggested carbamazepine and lidocaine as effective treatments for control of next seizures in CwG (20-22).

CwG is usually a benign condition with good prognosis and no risk for developing epilepsy in future. Hung et al. followed 40 patients with CwG for a period from 5 to 94 months (mean, 39.1 months). The psychomotor development was normal in all of them during the follow-up. None developed epilepsy. Only one patient with normal EEG experienced febrile convulsions 2 months later. All patients had normal psychomotor development without recurrence of convulsion, except for one patient who had a febrile convulsion (17). According to a recent study, a full course of rotavirus vaccination was associated with significant reductions in the risk of seizures during the year following rotavirus vaccination. Therefore, rotavirus vaccination may reduce the risk of CwG by preventing rotavirus infections (23).

Conclusion

CwG is an afebrile seizure associated with viral gastroenteritis. Consideration of this etiology of seizure can prevent supernumerary evaluations and long-term antiepileptic treatment. Prognosis is often favorable, with normal psychomotor development.

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None.

Conflict of Interest

The authors declare no conflict of interest.

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