

Clinical Characteristics, Treatment, and Outcomes in Children with Benign Convulsions with Mild Gastroenteritis in the Philippine General Hospital: A Retrospective Cohort Study

Karina Terese DJ. Santos, MD, Patricia C. Orduña, MD, and Rhea Angela M. Salonga-Quimpo, MD

Division of Pediatric Neurology, Departments of Pediatrics and Neurosciences, Philippine General Hospital, University of the Philippines Manila

ABSTRACT

Background and Objective. Benign convulsions with mild gastroenteritis (CwG) is common but not readily recognizable to primary care physicians and pediatricians. Most literature comes from East Asia and Western countries. Studies among the Filipino population are lacking. This study aimed to determine the clinical presentation, management, and outcomes, and provide knowledge for accurate diagnosis and appropriate management.

Methods. This is a retrospective cohort study on pediatric patients diagnosed with CwG admitted at a tertiary hospital in the Philippines from January 2020 to December 2023. The study included patients 1-72 months old presenting with seizures accompanied by symptoms of gastroenteritis, without clinical signs of dehydration, electrolyte derangement, and fever (body temperature $<38^{\circ}\text{C}$) during the seizures.

Results. Twenty patients met the criteria for CwG, aged 7-60 months, with a male:female ratio of 1:1. Most seizures were brief, generalized tonic-clonic occurring in clusters, with an average frequency of 3 per day. Laboratory findings, electroencephalogram, and neuroimaging results were mostly normal. Anti-seizure medications (ASMs) were prescribed in 65% ($n=13$), with levetiracetam being the most common. Most seizure clusters did not persist, and none needed additional ASM. Follow-up showed normal neurodevelopmental profiles.

Conclusion. This study highlights that CwG is also encountered among Filipino children. The clinical characteristics align with the known presentation of CwG. Most patients had normal test results and a benign course. Given this self-limiting nature, extensive testing and unnecessary therapy are not recommended, and instead provision of adequate counseling to the caregivers is advocated.

Keywords: *benign convulsions with mild gastroenteritis, seizures, gastroenteritis*

INTRODUCTION

Benign convulsions with mild gastroenteritis (CwG) represent a distinct clinical entity defined by afebrile seizures in healthy patients presenting with acute gastroenteritis symptoms without any evidence of central nervous system infection, electrolyte abnormalities, or clinical features of dehydration.¹ First described in the 1980s by Japanese researchers, CwG was historically more commonly reported in East Asian countries, although cases have also been recently described in Europe and other regions.²⁻⁵ At present, there are no published reports of CwG in the Philippines.

The existing literature on CwG agrees on the following clinical features. It typically affects infants and young children aged one month to six years.⁶ The seizures, usually occur in clusters, commonly after the onset of gastrointestinal



eISSN 2094-9278 (Online)
Published: September 15, 2025
<https://doi.org/10.47895/amp.v59i13.11578>
Copyright: The Author(s) 2025

Corresponding author: Karina Terese DJ. Santos, MD
Division of Pediatric Neurology
Departments of Pediatrics and Neurosciences
Philippine General Hospital
University of the Philippines Manila
Taft Avenue, Ermita, Manila 1000, Philippines
Email: kdsantosmd@gmail.com
ORCID: <https://orcid.org/0000-0003-4108-0329>

symptoms (range 0–6 days), but can occasionally occur before the enteric symptoms.^{3,7} Interictal EEG findings are usually normal, with occasional transient abnormalities.^{3,8} CwG generally has a favorable prognosis, and long-term anti-seizure treatment is not recommended.^{6,9}

Despite growing clinical recognition, several aspects of CwG remain poorly understood. The exact pathophysiological mechanisms, including the potential roles of specific neurovirulent pathogens like Rotavirus, Norovirus, and Shigella, still need to be fully elucidated.^{4,10} Its nosological position, genetic underpinnings, and long-term prognosis also need to be clarified. It is recognized that several features of CwG parallel febrile seizures (FS),^{11,12} and both entities are now classified as age-dependent acute symptomatic seizures (previously called situation-related seizures). They occur in the context of a provoking factor on an immature brain and have normal diagnostic profiles and favorable outcomes.^{13,14} In clinical practice, differentiating between these two entities is challenging as fever is common during infectious gastroenteritis. Still, CwG and FS may have distinct pathogenetic mechanisms.¹⁴ Family history and genetic predisposition are established in FS, but research showed no associations between CwG and some candidate genes like SCN1A and proline-rich transmembrane protein 2 (PRRT2).^{15,16} Limited data on family history of FS and epilepsy in CwG show a small percentage in cohorts, but no conclusion on their association can yet be made.⁹ These factors are the subject of ongoing investigations.

In contrast, epilepsy is an entity where there is an enduring predisposition to generate epileptic seizures. It is diagnosed when the following conditions are met: (1) at least two unprovoked (or reflex) seizures occurring >24 h apart; (2) one unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk after two unprovoked seizures, occurring over the next 10 years; and (3) diagnosis of an epilepsy syndrome.¹⁷ Given these and because epilepsy can also be provoked in states such as fever, diarrhea, and acute infection, distinguishing CwG from an initial seizure of epilepsy is difficult. Efforts made to predict the risk of developing epilepsy often include electroencephalography and, occasionally, neuroimaging. However, unprovoked seizures are still the cornerstone of epilepsy diagnosis and, despite ancillary testing, is still a clinical diagnosis.¹²

Given its benign and self-limiting nature, distinguishing CwG from more severe conditions is paramount to a more conservative approach to management and adequate parental counseling. This study aimed to establish the local burden of illness, define the clinical presentation and outcomes, and ultimately provide knowledge that will help facilitate its recognition and appropriate management, particularly reducing unnecessary diagnostics like cranial imaging and treatment with anti-seizure medications and preventing the wrong diagnosis of epilepsy.

MATERIALS AND METHODS

Study Design and Setting

This was a retrospective descriptive study of pediatric patients diagnosed with benign convulsions with mild gastroenteritis admitted at a tertiary hospital in the Philippines from January 2020 to December 2023.

Case Identification and Sampling

Based on OpenEpi, a minimum of 27 patients were required for this study based on the 1.78% incidence of patients diagnosed with CwG reported in literature¹⁸ with a 95% confidence interval level and 80% power. However, due to the limited number of cases at 2–5 patients annually, all cases from January 2020 and December 2023 were included.

All patients, aged one month to six years old, admitted at the pediatric charity and pay wards, and pediatric emergency room of the University of the Philippines – Philippine General Hospital from January 2020 to December 2023, who were diagnosed with benign convulsions with mild gastroenteritis, were identified from the monthly censuses of the Divisions of Pediatric Neurology and Pediatric Emergency Medicine.

Due to significant under recognition of the condition, the following patients were also included in the initial case identification:

1. diagnosed with acute gastroenteritis, defined as having symptoms of nausea, vomiting, abdominal pain, diarrhea, and having concurrent “acute symptomatic” seizures
2. diagnosed with simple or complex febrile seizures as documented in the patient records

Charts of these patients underwent preliminary review, and those found to be more consistent with benign convulsions with mild gastroenteritis were included in the subject list. The definition for benign convulsions with mild gastroenteritis detailed by Komori was followed in this study.¹ (Figure 1)

Patients with the following characteristics were excluded from the study: patients with an apparent history of epilepsy or with previous neurological deficits; those with bacterial or aseptic meningitis, encephalitis, or virus-related encephalopathy; those with severe dehydration, electrolyte (serum Na <115 mg/dL, serum Ca <5.0 mg/dL, serum Mg <0.8 mg/dL, serum Creatinine >10 mg/dL, BUN <100 mg/dL) and

Benign convulsions with mild gastroenteritis is defined when a patient meets the following conditions:

1. Seizures accompanied the symptoms of gastroenteritis without clinical signs of dehydration or electrolyte derangement
2. The body temperature remained less than 38°C during the seizures
3. No apparent history of epilepsy or previous neurological deficits
4. No clinical evidence of central nervous system infections

Figure 1. Diagnostic criteria for CwG.¹

glucose (RBS <36 mg/dL) derangements presenting with symptomatic seizures or those patients whose medical records could not be fully retrieved or are incomplete.

Data Collection

Data was retrieved from the online and physical medical records of the selected patients via the Medical Records Division as well as the electronic medical records of the institution, whereas pertinent diagnostic results were reviewed from the Central Laboratory of the institution and the online database of the institution.

The data collected was recorded in a Microsoft Excel File using the alphanumeric code as an identifier. These included age, sex, data about the clinical presentation such as the presence or absence of vomiting, diarrhea, abdominal pain, and other symptoms such as passage of worms, decrease in appetite, and decrease in activity; seizure characteristics such as the temporal relationship of onset of seizures and gastroenteritis symptoms; semiology, duration, frequency, recurrence, intervals between seizures, family history of seizures; laboratory investigations and ancillary testing including complete blood count (CBC), serum chemistry and electrolytes, stool studies and culture, Rotavirus antigenicity, vaccination status specifically for Rotavirus, initial blood glucose, cerebrospinal fluid (CSF) study results, electroencephalography (EEG) results, neuroimaging, and patient course including anti-seizure medications, other medications, and length of hospitalization.

Follow-up information after the initial admission was additionally reviewed from the electronic medical records. A minimum follow-up of one week post-discharge was intended, and if these data were available until the end of data collection (April 2024), they were also recorded. These included recurrence of seizures and neurodevelopmental assessments.

In cases wherein specific laboratory tests were not done owing to the clinical working impression and/or non-availability of the test, or follow up information not being available, the data were not imputed, and the cases were excluded from the final analysis of each specific variable.

Statistical Analysis

Descriptive statistics were used to summarize the demographic and clinical characteristics of the patients. Frequency and proportion were used for categorical variables, the median and interquartile range for non-normally distributed continuous variables, and mean and standard deviation for normally distributed continuous variables. Shapiro-Wilk test was used to test the normality of the continuous variables. Fisher’s exact test was used to determine the difference between patients who had seizure persistence versus those who were seizure-free in relation to the patient’s medication status. Missing values were neither replaced nor estimated. Microsoft Excel and STATA 13.1 were used for data management and analysis, respectively.

Ethical Considerations

The study ensured adherence to the 2017 National Ethical Guidelines of Health and Health-related Research. The protocol was approved by the UP Manila Research Ethics Board (UPMREB 2024-0034-01).

RESULTS

Included Patients

Over a four-year period, a total of 64 cases were identified (Figure 2). Of these, 18 patients were excluded due deferred admission or incomplete or unavailable records. Forty-six patients underwent preliminary review of the full admitting history and impression, and the diagnosis was ultimately ruled out in an additional 10 patients due to severe dehydration or an apparent history of epilepsy or neurological disorder. Thirty-six patients were then included for full chart review. Twenty patients with CwG were identified.

The demographic and epidemiological characteristics of the patients are summarized in Table 1. The age at onset ranged from 7 months to 5 years old, with a peak at one year old

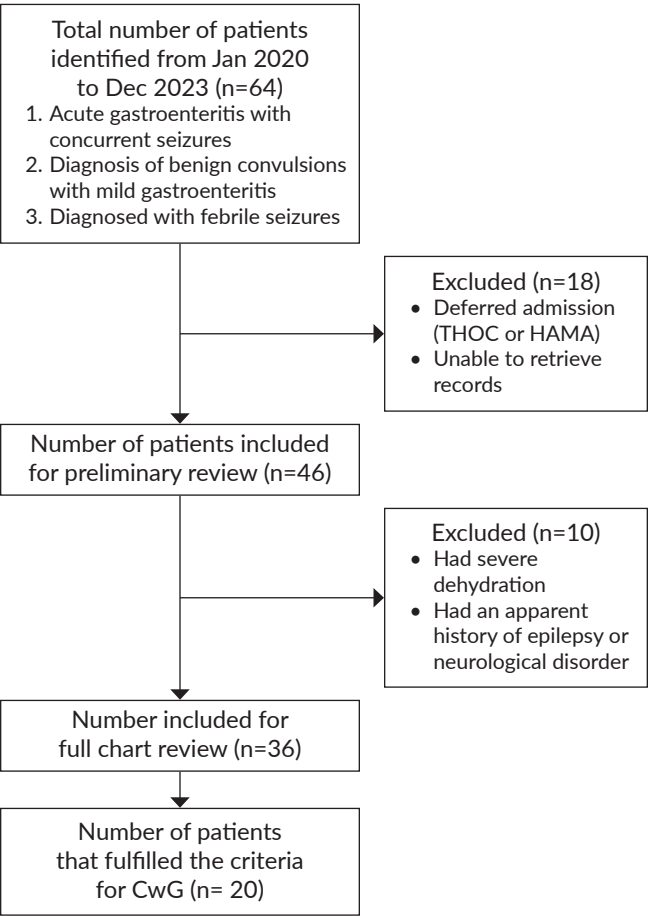


Figure 2. Flow diagram of patient inclusion in the study.

THOC – Transfer to hospital of choice, HAMA – Home against medical advice

(55%). There was an equal distribution of males and females. Most admissions occurred during the dry season (75%), with the highest number of admissions in January (45%).

Personal and Family History of Seizures and Epilepsy

Four children (20%) had suffered from simple febrile seizures on previous occasions. Of the 20 patients, four (20%) had a family history of febrile seizures in first and second-degree relatives, and one (5%) had a family history of epilepsy.

Clinical Profile and Seizure Characteristics

The interval between the onset of gastroenteritis and seizures ranged from 18 to 48 hours (median of 24 hours, IQR of 11.5 hours). The seizure characteristics are shown in Table 2. Most patients presented with both diarrhea and vomiting (70%), and two (10%) had associated abdominal pain. Seven patients had low-grade ($37.5 - 38.3^{\circ}\text{C}$)¹⁹ or undocumented fever either day prior (n=6) to the onset of seizures or postictally (n=1). Among all the patients, Rotavirus vaccination was specifically sought in two and both had none.

Seizures typically occurred repetitively at a median of 3 episodes (range: 1 to 15) in a 24-hour period. Most seizures presented as generalized tonic-clonic seizures lasting

between 10 seconds to 5 minutes (median of 1 minute). The observed seizure types were generalized tonic-clonic seizures in 17 patients, focal-to-bilateral tonic-clonic seizures in three patients, and focal impaired awareness behavior arrest evolving to bilateral clonic seizures in two patients who also had generalized tonic-clonic seizures.

Laboratory Findings

The complete blood counts of all patients were within normal limits for age, with a mean hemoglobin of 119.2 g/dL, mean hematocrit of 0.36, mean white blood cell (WBC) count of $11.28 \times 10^9/\text{L}$, and mean platelet count of $341 \times 10^9/\text{L}$. The sodium levels in two patients were 145 and 150 mmol/L, while the rest were within normal limits for age. Other serum chemistry tests, such as potassium (n=20), chloride (n=19), calcium (n=13), magnesium (n=14), phosphorus (n=2), creatinine (n=16), blood urea nitrogen (BUN) (n=14), aspartate aminotransferase (AST) (n=2), alanine aminotransferase (ALT) (n=2), and albumin (n=2) were all within the normal ranges for age. Random blood sugar (RBS) was normal in all nine tested patients.

Nine patients had stool exam and among these, four had ascaris, one had Giardia and two had *Entamoeba histolytica*. *Clostridium difficile* was detected in one case. All nine patients tested for stool bacterial cultures had negative results. Eight patients were tested for Rotavirus antigen in the stool and only one tested positive.

CSF studies were done in four patients, and all parameters were within normal ranges for age. All CSF samples also tested negative for bacterial antigens and cultures.

EEG and Neuroimaging Features

EEG was done in seven patients, all of which were normal. Four patients underwent neuroimaging with cranial computed tomography scanning. Among these, one had suspicious hypodensities in the white matter regions of the bilateral frontal lobes, while the other three had normal neuroimaging findings.

Anti-seizure Medications and Supportive Medications

A total of 14 patients (70%) received emergency anti-convulsant treatment with a single dose of intravenous Diazepam. Maintenance anti-seizure medications (ASMs) were started in 60% of cases (n=12), with Levetiracetam being the most common (40%), followed by Valproic Acid (15%), and Phenobarbital (5%). ASMs were administered after the initial cluster of seizures, and were given on the first day of admission in most patients. In one patient, intermittent oral Diazepam was prescribed as prophylactic treatment for further episodes. Table 3 presents the clinical profile of the patients for whom maintenance anti-seizure medications were given.

Supportive medications were also given, such as zinc sulfate (n=7), oral rehydration solution (ORS) (n=5),

Table 1. Demographic and Clinical Profile of the Patients (n=20)

	Frequency (%)
Age, years	
0	1 (5)
1	11 (55)
2	4 (20)
3	1 (5)
4	1 (5)
5	2 (10)
Sex	
Male	10 (50)
Female	10 (50)
Month of Admission	
January	9 (45)
February	2 (10)
May	1 (5)
June	1 (5)
September	2 (10)
November	2 (10)
December	3 (15)
Seasonal Distribution	
Dry	15 (75)
Wet	5 (25)

Table 2. Seizure Characteristics (n=20)

	Median (IQR)
Onset of seizures from onset of gastroenteritis symptoms (hours)	24 (12.5 to 24)
Seizure duration (minutes)	1 (0.5 to 3)
Seizure frequency (times per day)	3 (2 to 4)

Table 3. Clinical Profile of Patients Started on Maintenance Anti-seizure Medications (n=12)

Case	Age (yrs)	Seizure Frequency	Personal history of FS	Family History of FS or epilepsy	Recurrence of CwG	CSF studies	EEG	Imaging	ASM	Outcome
1	1	2x	-	-	-	-	-	Normal	LEV	ASM discontinued
2	1	2x	-	-	-	-	-	-	LEV	-
3	2	3x	-	-	-	-	-	-	LEV	-
4	2	4x	-	-	-	-	Normal	Suspicious hypodensities in the white matter regions of the bilateral frontal lobes	LEV	ASM discontinued
5	1	3x	-	-	-	Normal	Normal	-	LEV	-
6	1	3x	-	FS	Yes	Normal	Normal	Normal	LEV	ASM discontinued
7	7 mo	>15x	-	-	-	-	Normal	-	LEV	ASM tapered to discontinue
8	1	2x	-	FS	-	-	-	-	LEV	ASM discontinued
9	1	4x	-	-	-	-	-	-	PB	-
10	2	9x	-	FS	-	-	Normal	-	VPA	ASM discontinued
11	1	8x	Yes	FS	-	Normal	Normal	-	VPA	ASM discontinued
12	2	6x	-	-	-	-	-	-	VPA	-

FS – febrile seizures, CwG – benign convulsions with mild gastroenteritis, CSF – cerebrospinal fluid, EEG – electroencephalogram, ASM – Anti-seizure medication, LEV – Levetiracetam, PB – Phenobarbital, VPA – Valproic Acid

racecadotril (n=2), paracetamol (n=1), ferrous sulfate (n=1), and antiparasitics like Metronidazole (n=2), and Mebendazole (n=2).

Outcomes

All of the patients had a benign course. Hospital stays were brief, ranging from one to four days, with an average of one day. None of the patients needed an additional ASM to achieve seizure cessation. Only one patient continued to have seizure clusters despite initial ASM administration, requiring additional doses to achieve the maximum therapeutic dose for seizure cessation. The medication was eventually tapered off upon follow-up.

Table 4 shows the cross-tabulation of anti-seizure medications and seizure outcomes. In patients wherein medications were administered, only two out of 12 had persistence of seizures, and 10 out of 12 became seizure-free. The eight patients who were not given medications also became seizure-free. No statistical difference was found in

terms of seizure outcomes between those who received ASMs versus those who did not receive ASMs. Thus, no association can be established between seizure freedom or persistence and the use of anti-seizure medication.

Eight out of 20 patients completed the follow-up, with the initial follow-up period ranging from one week to six months. Three of the eight patients were seen at repeated intervals during the whole follow-up period, which extended to ten months post-discharge for one patient. One patient had a recurrence of the CwG episode after two months. Among the twelve patients given anti-seizure medications during the initial CwG episode, anti-seizure medications were weaned off in seven patients. The remaining patients have not completed follow-up. All eight patients assessed had normal neurodevelopmental examinations.

DISCUSSION

This study provided the first account of CwG in the Philippine setting. It described the local characteristics and demonstrated features that were similar to and discordant with those reported previously.

Benign convulsions with mild gastroenteritis typically affects children aged one month to six years, with a peak incidence between 1 and 2 years of age,⁶ as was seen in this cohort. There was an equal number of males and females, contrary to most studies showing a female predisposition.⁹ Most of the admissions clustered during the dry season of November to May, corresponding with the seasonal predisposition of CwG to winter and early spring. Hypothesized to be associated with this is the seasonal circulation of viruses, particularly Rotavirus and Norovirus,^{6,20}

Table 4. Cross-tabulation of Anti-seizure Medication and Seizure Outcome

	Seizure outcome			P-value
	Total (n=20)	Seizure Persist (n=2)	Seizure Free (n=18)	
	Frequency (%)			
Given medication	12 (60)	2 (100)	10 (55.56)	0.495
Not given medication	8 (40)	0	8 (44.44)	

subsequently implicating these enteric pathogens in the pathophysiology of CwG.^{21,22} In the tropics, however, data is still lacking.²³ While studies similarly suggest a higher prevalence of Rotavirus during the colder and drier months,²⁴ our data showed that only one patient was positive for Rotavirus, indicating that additional factors are likely involved in the pathogenesis of CwG.

In this study, 20% had suffered from simple febrile seizures in the past, 20% also had a family history of febrile seizures in first and second-degree relatives, and one out of the 20 patients had a family history of epilepsy. Seven patients also had low-grade or undocumented fever during the illness but not during the seizure episodes. In comparison to its clinical parallel FS, wherein the role of family history and genetic predisposition are recognized, it is still difficult to conclude the same for CwG.⁹ It is clear, however, that the relationship between FS and CwG appears complex, and further studies must be done with larger populations to understand CwG's nosological classification and risk factors better.

The seizures in CwG are brief, usually lasting from 30 seconds to less than five minutes.⁶ They are also classically clustered in 41-80%²⁵ and repetitive within the same cluster up until eight episodes.⁷ The seizure patterns in this study align with this, with most seizures lasting for one minute and clustered on the first day of admission. Some studies have reported a smaller percentage of cases that present with seizures lasting >30 minutes, fulfilling the definition of status epilepticus (SE),^{3,26} but this was not seen in this cohort. In the patients described, seizures occurred at a median time of 24 hours from the onset of gastrointestinal symptoms. Reports show that the range can be from a few hours up to six days later.⁷ They present in a variety of semiologies, including focal seizures (tonic, clonic), focal to bilateral tonic-clonic seizures, generalized tonic-clonic seizures, apnea, hypersalivation, and automatisms.^{3,25,27-30} The characteristics of the seizures in our patients correspond to those published in the literature and have a greater frequency of generalized seizures.

Studies by Imai et al. on video electroencephalography suggest that CwG seizures, despite their often generalized appearance, are likely focal seizures that originate from various regions of the brain, including frontal, temporo-parietal, centro-parietal, and occipital areas.^{28,29} The interictal EEGs were usually normal,³⁰ as is seen in this study. Chen et al. reported that despite an increase in the percentage of abnormalities in the interictal EEGs of recurrent cases, follow-up after one year showed that most have reverted to normal, emphasizing the transient nature of the abnormalities and being non-predictive of epilepsy risk.⁸

Laboratory findings in this study were mostly within normal limits, consistent with the literature. Other reports, however, showed that specific tests may be abnormal. A recent meta-analysis by Miyagi et al. suggests that the laboratory finding most strongly associated with CwG is increased uric acid levels.³¹ Hyponatremia has also been consistently demonstrated in several cohorts, but the evidence is still

inadequate.³²⁻³⁴ In this cohort, hyponatremia was noted in two patients. This finding may still signify mild uncorrected dehydration and, unlike hyponatremia, probably is non-contributory to the seizures.

Neuroimaging, if done, is usually normal and hence is not part of routine work-up for CwG.³ Interestingly, the white matter changes seen in one of the patients have not been recorded previously. It remains difficult to assess the clinical implications of such findings. Still, most patients for whom neuroimaging is obtained have normal results and are not recommended to be part of routine CwG testing.⁸

In this study, thirteen patients (65%) received anti-seizure medication during the acute phase, and most had complete seizure cessation after being given Levetiracetam, Phenobarbital, or Valproic Acid. Only one patient required up-titration to the maximum therapeutic dose, and none had seizures that were refractory to treatment. In cases of seizure clustering, prolongation, and daily recurrences that cause distress to the guardians, the use of anti-seizure medications may be justified.²⁶ Available guidance on preferred medication is limited, and no consensus on a drug of choice has yet been reached.^{35,36} A randomized trial by Zha et al. recommended Phenobarbital at a dose of 10 milligrams per kilogram for the acute phase of CwG.³⁷ In contrast, Okumura et al. reported that Lidocaine was more effective in stopping seizures than Phenobarbital.³⁶ In addition, Diazepam was deemed ineffective and was not advocated. Several other studies investigated the potential of Carbamazepine,^{35,38} Chloral Hydrate,³⁹ and different permutations of anti-seizure regimens.⁶ However, they all still need adequate statistical power to recommend a drug of choice reliably. In this study, an association between seizure cessation and ASM use cannot be made. Prospective studies with larger sample sizes will be needed to assess effectivity. Finally, most seizure clusters of CwG do not persist beyond 24 hours; hence, routine and intensive use of ASMs is not recommended.³ Emphasis is given instead to supportive management of the underlying gastroenteritis and maintaining hydration.

Seven of the twelve patients in our series who were given symptomatic anti-seizure medications were eventually weaned off. Of the patients who completed follow-up, including the patient who had a recurrence of CwG, neurodevelopmental assessment was normal. Most cases of CwG are self-limiting, and neither the recurrence of seizures during subsequent gastroenteritis episodes nor the development of epilepsy is significant.^{1,3,9,40} Maintenance anti-seizure treatment is therefore not recommended.²⁶ Factors that were associated with recurrence include early age of onset (<18 months),⁴¹⁻⁴³ multiple seizures 24 hours apart, a family history of seizures,⁴¹ and the absence of fever.¹² Two of these risk factors were present in our patient. Overall, the long-term outcomes are favorable, and parental counseling should emphasize careful observation and regular follow-up in children diagnosed with CwG, especially those with risk factors for recurrence. Studies designed with a mechanism for adequate follow-up will be

helpful in clarifying the evolution and long-term prognosis of CwG patients in the Philippines.

Our investigations confirm that extensive laboratory testing performed in the acute stage of the illness does not help elucidate the etiologic cause of the gastroenteritis and the seizures, and are therefore assumed as a nonspecific viral infection. The results are mostly normal, with the few deviations attributable to the effects of gastroenteritis itself, or as previously reported in the literature,⁸ are transient and do not have any prognostic significance with regard to the risks of subsequent recurrences or epilepsy. And while we have not studied the effect of ASM administration longitudinally, the course is self-limiting and benign and hence would rarely warrant the additional cost and potential harm of ASM prescription. Future research focusing on the approach to the neurodiagnostic evaluation of patients in this specific population is recommended.

This study is the first to describe the characteristics of CwG in the Philippines. Data from a single center, however, limited the number of patients included in this cohort, in addition to the small sample size. This also limits the generalizability of the findings, and it is therefore suggested that research including more participants possibly from multiple centers be done in the future. The low follow-up rate of 40% could also be a potential source of bias as our cohort may not accurately reflect the CwG population. This may be ameliorated by employing multiple communication methods to ensure adequate follow-up in future studies. The short follow-up period ranging only from one week to 10 months is also a significant limitation as this period may not be enough to conclude on the prognosis of the disease entity. This could be improved by doing longer longitudinal studies. The retrospective and descriptive nature of the survey also precluded any conclusions on the temporal correlation between the variables and outcomes. Because the measures of seizure characteristics, associated signs and symptoms like the presence of fever, and medical history also relied mostly on self-reporting of the patients' guardians, these variables could have been directed towards under- or overestimations. These could be mitigated by employing a prospective design for future studies. Research on CwG exploring the specific regional risk factors for the disease, the long-term consequences, including neurodevelopment and epilepsy risk, is also suggested.

CONCLUSION

This study showed that CwG is an entity also seen in the Philippines, with characteristics that coincide with the known clinical presentation of this condition. Seizure characteristics showed clustering, with predominantly generalized semiology, and close temporal association with the onset of gastroenteritis symptoms. Rotavirus infection was rarely seen. Most ancillary testing was normal. The short-term outcomes were excellent. CwG is a condition that should be recognized in order to avoid extensive testing and unnecessary therapy.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

Funding Source

None.

REFERENCES

1. Komori H, Wada M, Eto M, Oki H, Aida K, Fujimoto T. Benign convulsions with mild gastroenteritis: a report of 10 recent cases detailing clinical varieties. *Brain Dev.* 1995;17(5):334-337. doi:10.1016/0387-7604(95)00074-L
2. Narchi H. Benign afebrile cluster convulsions with gastroenteritis: an observational study. *BMC Pediatr.* 2004;4(1):2. doi:10.1186/1471-2431-4-2
3. Verrotti A, Nanni G, Agostinelli S, et al. Benign convulsions associated with mild gastroenteritis: A multicenter clinical study. *Epilepsy Res.* 2011;93(2-3):107-114. doi:10.1016/j.eplepsyres.2010.11.004
4. Iflah M, Kassem E, Rubinstein U, et al. Convulsions in children hospitalized for acute gastroenteritis. *Sci Rep.* 2021;11(1). doi:10.1038/s41598-021-95202-4
5. Pajic J, Redzek-Mudrinic T, Kavecian I, Vijatov-Djuric G, Milanovic B, Vorgucin I. Febrile and afebrile seizures associated with mild acute gastroenteritis in childhood. *Med Pregl.* 2022;75(5-6):166-170. doi:10.2298/MPNS2206166P
6. Kang B, Kwon YS. Benign convulsion with mild gastroenteritis. *Korean J Pediatr.* 2014;57(7):304-309. doi:10.3345/kjp.2014.57.7.304
7. Kim YO. Benign convulsions with mild gastroenteritis. *Annals of Child Neurology.* 2020;28(1):2-7. doi:10.26815/acn.2019.00248
8. Chen B, Cheng M, Hong S, et al. Clinical outcome of recurrent afebrile seizures in children with benign convulsions associated with mild gastroenteritis. *Seizure.* 2018;60:110-114. doi:10.1016/j.seizure.2018.05.020
9. Lee YS, Lee GH, Kwon YS. Update on benign convulsions with mild gastroenteritis. *Clin Exp Pediatr.* 2022;65(10):469-475. doi:10.3345/cep.2021.00997
10. Fang C, Fan W, Zhang C, Yang Y. Risk Factors for Benign Convulsions With Mild Gastroenteritis. *Front Pediatr.* 2022;10. doi:10.3389/fped.2022.925896
11. Yu J, Jung K, Kang H. Comparative Study between Febrile Convulsions and Benign Convulsions Associated with Viral Gastroenteritis. *J Epilepsy Res.* 2011;(1(1)):19-26. doi:10.14581/jer.11004
12. Wang D, Peng L, Li T, Xiao N. Benign convulsions with mild gastroenteritis in children: An emerging acute symptomatic seizures. *Pediatric Discovery.* 2024;(2(2)). doi:10.1002/pdi3.59
13. Cappellari AM, Mariani S, Bruschi G. Febrile seizures and convulsions with mild gastroenteritis: age-dependent acute symptomatic seizures. *Front Pediatr.* 2023;11. doi:10.3389/fped.2023.1151770
14. Weng WC, Hirose S, Lee WT. Benign Convulsions With Mild Gastroenteritis: Is It Associated With Sodium Channel Gene SCN1A Mutation? *J Child Neurol.* 2010;25(12):1521-1524. doi:10.1177/0883073810370898
15. Ishii A, Yasumoto S, Ihara Y, et al. Genetic analysis of PRRT2 for benign infantile epilepsy, infantile convulsions with choreoathetosis syndrome, and benign convulsions with mild gastroenteritis. *Brain Dev.* 2013;35(6):524-530. doi:10.1016/j.braindev.2012.09.006
16. Meyer A, Mazzara C, Lava SAG, et al. Neurological complications of rotavirus infection in children: A systematic review and meta-analysis. *Acta Paediatr.* 2023;112(7):1565-1573. doi:10.1111/apa.16775
17. Fisher RS, Acevedo C, Arzimanoglou A, et al. ILAE Official Report: A practical clinical definition of epilepsy. *Epilepsia.* 2014;55(4):475-482. doi:10.1111/epi.12550

18. Hao X, Liang J, Wu X, Hao Y, Wang J, Liu S. Clinical characteristics, treatment, and long-term outcomes in children suffering from benign convulsions with mild gastroenteritis: a retrospective study. *BMC Pediatr.* 2020;20(1). doi:10.1186/s12887-020-02406-0
19. Castellazzi L, Principi N, Agostoni C, Esposito S. Benign convulsions in children with mild gastroenteritis. *Eur J Paediatr Neurol.* 2016;20(5):690-695. doi:10.1016/j.ejpn.2016.05.014
20. Sugata K, Taniguchi K, Yui A, et al. Analysis of Rotavirus Antigenemia and Extraintestinal Manifestations in Children With Rotavirus Gastroenteritis. *Pediatrics.* 2008;122(2):392-397. doi:10.1542/peds.2007-2290
21. Liu B, Fujita Y, Arakawa C, et al. Detection of rotavirus RNA and antigens in serum and cerebrospinal fluid samples from diarrheic children with seizures. *Jpn J Infect Dis.* 2009;62(4):279-283.
22. Ng RL. Benign afebrile convulsion with gastroenteritis – A differential to consider for afebrile seizure in children. *Med J Malaysia.* 2018;(73(5)):328-329.
23. Levy K, Hubbard AE, Eisenberg JN. Seasonality of rotavirus disease in the tropics: a systematic review and meta-analysis. *Int J Epidemiol.* 2009;38(6):1487-1496. doi:10.1093/ije/dyn260
24. Wu YZ, Liu YH, Tseng CM, Tseng YH, Chen TH. Comparison of Clinical Characteristics Between Febrile and Afebrile Seizures Associated With Acute Gastroenteritis in Childhood. *Front Pediatr.* 2020;8. doi:10.3389/fped.2020.00167
25. Cusmai R, Jovic-Jakubi B, Cantonetti L, Japaridze N, Vigeveno F. Convulsions associated with gastroenteritis in the spectrum of benign focal epilepsies in infancy: 30 cases including four cases with ictal EEG recording. *Epileptic Disorders.* 2010;12(4):255-261. doi:10.1684/epd.2010.0340
26. Uemura N, Okumura A, Negoro T, Watanabe K. Clinical features of benign convulsions with mild gastroenteritis. *Brain Dev.* 2002;24(8):745-749. doi:10.1016/s0387-7604(02)00097-9
27. Saadeldin IY. Electroclinical features of benign infantile seizures with mild gastroenteritis. *Epileptic Disorders.* 2011;13(1):8-17. doi:10.1684/epd.2011.0418
28. Khosroshahi N, Rahbarmanesh A, Boroujeni FA, Eskandarizadeh Z, Zoham MH. Afebrile Benign Convulsion Associated With Mild Gastroenteritis. *Child Neurol Open.* 2018;5:2329048X1877349. doi:10.1177/2329048X18773498
29. Imai K, Otani K, Yanagihara K, et al. Ictal video-EEG recording of three partial seizures in a patient with the benign infantile convulsions associated with mild gastroenteritis. *Epilepsia.* 1999;40(10):1455-1458. doi:10.1111/j.1528-1157.1999.tb02020.x
30. Verrotti A, Moavero R, Vigeveno F, et al. Long-term follow-up in children with benign convulsions associated with gastroenteritis. *Eur J Paediatr Neurol.* 2014;18(5):572-577. doi:10.1016/j.ejpn.2014.04.006
31. Miyagi Y, Sasano T, Kin K. Laboratory Findings of Benign Convulsions With Mild Gastroenteritis: A Meta-Analysis. *Cureus.* 2023;(15(3):e36784). doi:10.7759/cureus.36784
32. Motoyama M, Ichiyama T, Matsushige T, Kajimoto M, Shiraishi M, Furukawa S. Clinical Characteristics of Benign Convulsions With Rotavirus Gastroenteritis. *J Child Neurol.* 2009;24(5):557-561. doi:10.1177/0883073808327829
33. Ma X, Luan S, Zhao Y, Lv X, Zhang R. Clinical characteristics and follow-up of benign convulsions with mild gastroenteritis among children. *Medicine (United States).* 2019;98(2):E14082. doi:10.1097/MD.00000000000014082
34. Heydarian F, Bakhtiari E, Badzaee S, Heidarian M. Gastroenteritis Related Seizure with or without Fever: Comparison Clinical Features and Serum Sodium Level. *Iran J Child Neurol.* 2019;13(2):47-52.
35. Kawano G, Oshige K, Syutou S, et al. Benign infantile convulsions associated with mild gastroenteritis: A retrospective study of 39 cases including virological tests and efficacy of anticonvulsants. *Brain Dev.* 2007;29(10):617-622. doi:10.1016/j.braindev.2007.03.012
36. Okumura A, Uemura N, Negoro T, Watanabe K. Efficacy of antiepileptic drugs in patients with benign convulsions with mild gastroenteritis. *Brain Dev.* 2004;26(3):164-167. doi:10.1016/S0387-7604(03)00121-9
37. Zha J, Chen Y, Yu X, et al. Efficacy and safety of phenobarbital for benign convulsions with mild gastroenteritis: A prospective randomized controlled study. *Medicine.* 2022;101(50):e31495. doi:10.1097/MD.00000000000031495
38. Tanabe T, Okumura A, Komatsu M, Kubota T, Nakajima M, Shimakawa S. Clinical trial of minimal treatment for clustering seizures in cases of convulsions with mild gastroenteritis. *Brain Dev.* 2011;33(2):120-124. doi:10.1016/j.braindev.2010.02.007
39. Enoki H, Yokota T, Nagasaki R, et al. Single-Dose Chloral Hydrate for Benign Convulsions with Mild Gastroenteritis. *Epilepsia.* 2007;48(5):1026-1028. doi:10.1111/j.1528-1167.2007.01027.x
40. Durá-Travé T, Yoldi-Petri ME, Gallinas-Victoriano F, Molins-Castiella T. Infantile convulsions with mild gastroenteritis: a retrospective study of 25 patients. *Eur J Neurol.* 2011;18(2):273-278. doi:10.1111/j.1468-1331.2010.03120.x
41. Zaki SA, Rashid A. Risk factors for recurrence of benign convulsions with mild gastroenteritis in children: A prospective cohort study. *J Paediatr Child Health.* 2023;59(2):404-406. doi:10.1111/jpc.16320
42. You SJ. Older Patients May Have More Frequent Seizures among Children Diagnosed as Benign Convulsions with Mild Gastroenteritis. *Neuropediatrics.* 2020;51(05):354-358. doi:10.1055/s-0040-1701442
43. Shi K, Yang J, Wu Y, Han H, Guo J, Chen W. Risk factors for the recurrence of convulsions with mild gastroenteritis in children. *Seizure.* 2020;80:192-195. doi:10.1016/j.seizure.2020.06.016