

Electroclinical features of benign infantile seizures with mild gastroenteritis

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ABSTRACT – The purpose of this study was to analyze the electroclinical features of patients with benign infantile seizures with mild gastroenteritis and demonstrate the benign nature of this entity. From 30 patients who were included in the study, five were excluded (two with developmental delay, one with microcephaly and two lost during follow-up). Twenty-five patients who fulfilled the diagnostic criteria for benign infantile seizures with mild gastroenteritis were assessed and followed at the Pediatric Department, Armed Forces Hospital, Southern Region, Khamis Mushayt, Saudi Arabia, between January 2004 and January 2009. The median age at presentation was 10.4 months. Of the infants, 14 were females and 11 were males. Seizures were focal tonic or clonic in eight (32%) patients, focal with secondary generalisation in three (12%), generalised tonic-clonic in nine (36%) and consisted of staring only with no motor components in five (20%). Interictal electroencephalograms and brain imaging were normal for all patients. No patient required treatment with antiepileptic drugs. All the patients were found to have normal psychomotor development and neurological examination after a follow-up period of between 15 and 56 months. The limitations of this study are the relatively small sample size, relatively short study period and the fact that the study was conducted in a tertiary referral hospital. The prevalence of this entity may be more common at the level of primary health centres. Increasing the awareness of clinicians regarding the existence of this syndrome and its benign nature in children will limit unnecessary investigations. [*Published with video sequences*]

Key words: infantile seizures, mild gastroenteritis, rotavirus



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Benign infantile seizures (BIS) were first described by Fukuyama more than 45 years ago (1963). The two types (familial and non-familial) are now considered as one syndrome, which is age-related, appearing at 2-24 months of age (Engel, 2006; Caraballo *et al.*, 2003; Saadeldin *et al.*, 2010). Gastroenteritis (GE) is

generally defined as a decrease in the consistency of stools and/or an increase in the frequency of evacuations (≥ 3 in 24 hours), with or without fever or vomiting. Mild GE is defined as diarrhoea and/or vomiting without clinical signs of dehydration or electrolyte disturbance. Benign infantile seizures

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with mild gastroenteritis (BISwG) were described in Japan almost 30 years ago by Morooka (1982). More than 60 reports and 10 series have followed in other parts of Asia and the rest of the world (Caraballo *et al.*, 2003; Contino *et al.*, 1994; Wong, 2001; Lynch *et al.*, 2001), however, to the best of my knowledge, no cases have been reported in Saudi Arabia or other Arab countries.

Benign infantile seizures with mild gastroenteritis is characterized by the following criteria:

- occurrence in infants aged between 4-30 months (average 17 months);
- infants with normal psychomotor development and neurological examination;
- seizures may be focal or generalised;
- seizures are usually brief and repetitive and in clusters but can be isolated;
- associated with mild GE;
- associated with low grade ($<38^{\circ}\text{C}$) or absent fever;
- not associated with electrolyte derangement or hypoglycaemia, normal CSF;
- normal interictal electroencephalography (EEG) and brain imaging;
- benign course (Uemura *et al.*, 2002; Kawano *et al.*, 2007; Komori *et al.*, 1995; Caraballo *et al.*, 2009).

The aim of this study is to discuss electroclinical features and other data with published reports of BISwG, increase the awareness of the benign nature of the disorder amongst paediatricians and paediatric neurologists and demonstrate for the first time the presence of BISwG in Saudi Arabia.

Methods

Patients

A case series was conducted with 25 infants diagnosed with BISwG. The patients were seen at the Armed Forces Hospital, Southern Region (AFHSR), Pediatric Department, Saudi Arabia between January 2004 and January 2009. The AFHSR is a tertiary referral centre in the South of Saudi Arabia with a catchment population of approximately 1,100,000.

Inclusion criteria included: (1) infants between 4-30 months of age; (2) normal psychomotor development and neurological examination before and after the onset of seizures; (3) seizures occurring during a mild course of GE; and (4) normal interictal EEG and brain imaging. Exclusion criteria included: (1) infants younger than four months or older than 30 months; (2) psychomotor delay and abnormal neurological examination before and/or after the onset of seizures; (3) personal history of febrile and/or afebrile seizures; (4) patients with moderate or severe GE and/or associated electrolyte imbalance, fever greater

than 38°C or hypoglycaemia; (5) abnormal CSF study (if performed); (6) positive stool culture for bacteria; and (7) abnormal interictal EEG and brain imaging.

Clinical investigation

Thirty infants with BISwG who were admitted to the paediatric general ward were analyzed for the following characteristics: age at onset, clinical features, frequency and type of seizures associated with GE, and interval between GE and seizures. As most of the seizures occurred during hospitalization, they were witnessed by nurses, paediatricians, and/or a paediatric neurologist (IYS). At the patients' homes, video clips of clusters of seizures were recorded on a mobile telephone by the parents of two patients and examined by a paediatric neurologist (IYS). Three patients developed some of their seizures during their stay in the paediatric ward which were witnessed by expert paediatricians and/or a paediatric neurologist (IYS). A focal seizure was defined when the seizures involved a localized part of the body and generalised seizures when bilateral symmetrical motor manifestations were present. Patients who presented only staring, unresponsiveness and motor arrest were considered as having focal seizures. Personal and family history, consanguinity, developmental history, and physical and neurological examination were obtained. Full blood count, serum electrolytes, serum renal and liver function tests, capillary blood gases, blood sugar, and urinalysis were investigated. Rotavirus antigen was examined from a stool sample for all patients using the following commercially available rapid latex agglutination kits: Remel Rotavirus Latex Test (Remel Europe Ltd, Kent, UK) and Virotect Rota (Omega Diagnostics Ltd, Scotland, UK). All stool specimens were sent for bacterial culture. CSF study was done when clinically indicated. Interictal EEG during sleep and upon awakening (and for one patient an ictal EEG) were performed using the international 10-20 system for all patients. A CT and/or MRI brain study was performed for all patients. The patients were followed at paediatric neurology clinics for up to 56 months by a paediatric neurologist (IYS).

Results

Thirty patients who presented with seizures associated with mild GE between 4-30 months of age were studied (*figure 1*). Three patients were excluded from the beginning (two with developmental delay and one with microcephaly) and another two patients were lost during follow-up. From the remaining 25 patients, 14 (56%) were females and 11 (44%) were males. The median age at presentation was 10.4 months.

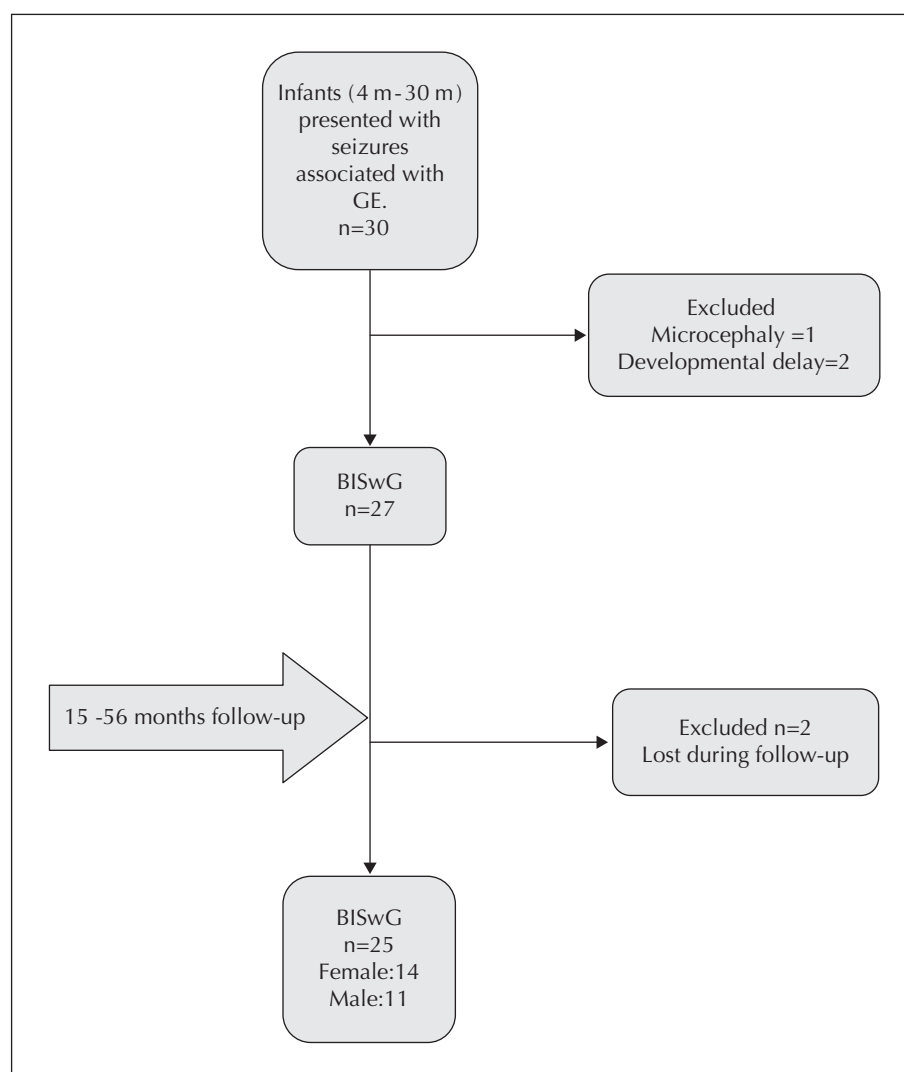


Figure 1. Benign infantile seizures with mild gastroenteritis; follow-up of 25 patients.
BISwG: benign infantile seizures with mild gastroenteritis; m: months; n: number; GE: gastroenteritis.

The patients were followed for 15-56 months (median: 35.6 months) and at last follow-up visit they all showed normal psychomotor development and neurological examination.

Seizures

As shown in *table 1*, all patients developed seizures during the first five days of GE. No patient developed a seizure before the onset of diarrhoea and/or vomiting. Seizures were isolated or occurred in clusters ranging from two to four clusters during the first five days of GE. Each cluster was composed of two to three episodes. Six patients developed two

clusters (24%), five had three clusters (20%) and two had four clusters (8%). Isolated seizures (one episode only) were observed in 12 patients (48%). The interval between GE onset and appearance of the first seizures ranged from the same day to the fifth day (mean: 2.5 days). Seizures were focal tonic or clonic in eight patients (32%), focal with secondary generalisation in three (12%), generalised tonic-clonic seizures (GTCS) in nine (36%) and consisted of staring only with no motor components (*see video sequence*) in five (20%). The duration of seizures ranged from 25 seconds to two minutes. No patient needed antiepileptic treatment. During follow-up, all infants had normal milestones and there was no recurrence of seizures.

Personal history

Two patients (one girl and one boy) were delivered by Caesarian section and required admission to hospital for a few days. Two infants (Patients 3 and 12) were admitted at the ages of two and five months, respectively, due to viral bronchiolitis. One infant had a history of neonatal jaundice due to glucose-6-phosphate dehydrogenase deficiency and was treated with phototherapy for two days (*table 1*).

Family history

Seven patients (28%) had consanguineous parents and four (16%) had a family history of febrile seizures. Three patients (12%) had parents or uncles with BIS. Three mothers had a history of one abortion and one with one neonatal death with unknown aetiology (*table 1*).

Gastroenteritis/rotavirus study

The frequency of diarrhoea ranged from two to four watery stools per day (*table 1*), and duration of diarrhoea from two to five days. Rotavirus was positive in the stools of 13 patients (52%). Stool specimens sent for bacterial culture showed no bacterial growth. Most of the cases occurred between April and September, peaking between June and July. Ten patients (40%) developed fever between 37.7°C and 38°C. In the remaining patients (60%) the GE was not associated

with fever. No bloody stools were observed in any of the patients. Vomiting occurred in eight (32%) patients (*table 1*).

Investigations

Serum electrolytes, blood urea nitrogen, serum creatinine, liver function tests, urine and stool analysis, CSF study (if performed), and brain imaging were normal for all patients. All interictal EEGs were normal. Patient 5, a male with positive consanguinity, developed a cluster of three apparent GTCS after one day of mild diarrhoea, not associated with fever or vomiting (*table 1*). His ictal EEG showed right-side spike-wave complexes with no secondary generalisation (*figure 2*). None of the neuroimaging performed showed abnormalities.

Discussion

Fukuyama (1963) was the first author to introduce the concept of benign seizures occurring during infancy with a favourable outcome. In the last two decades, the existence of benign partial epilepsies during infancy with a good outcome has been recognized in many countries with different ethnic groups (Echenne *et al.*, 1994; Caraballo *et al.*, 2003; Callenbach *et al.*, 2002; Saadeldin *et al.*, 2010). The existence of benign infantile seizures (familial and non-familial types) in the Saudi paediatric population was confirmed in a previous

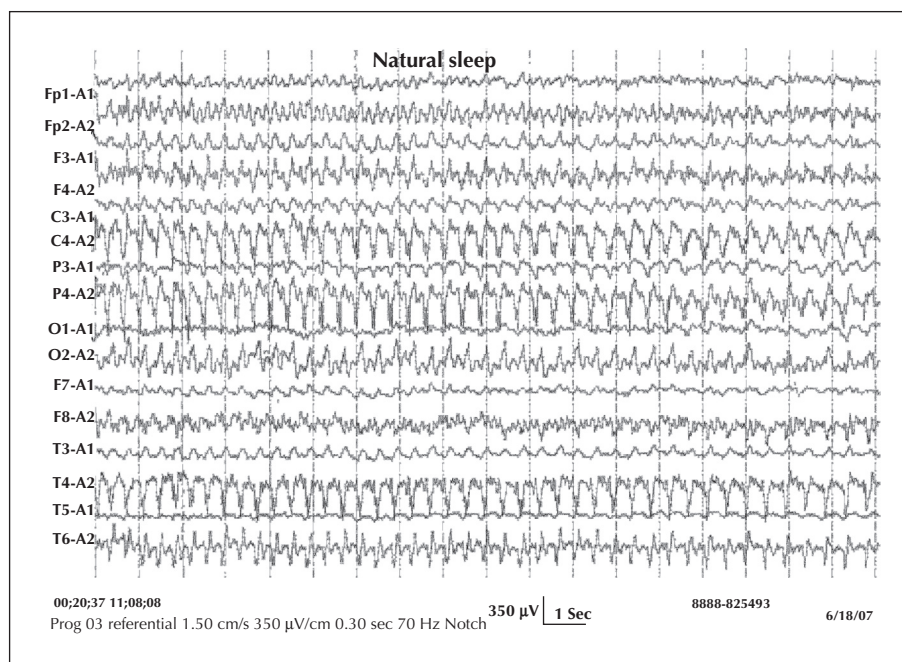


Figure 2. Ictal EEG recording of Patient 5 during natural sleep. Right-sided parieto-occipital paroxysms can be seen. Low-cut filter: 1Hz; high-cut filter: 70Hz.

Table 1. Electroclinical features of 25 infants with benign infantile seizures with mild gastroenteritis.

Patient	Sex	Con- sanguinity	Age- presentation (months)	Personal/ Family History	Age- current (months)	Interval (days) between GE and onset of seizures	Type of seizures	Number of diarrheal stools/ vomiting per 5 days	Number of seizures per 5 days	EEG	Rotavirus stools
1	F	No	4	BIS (parent)	108	1	Apparently focal-tonic	7	Cluster of 2	N (interictal)	Negative
2	F	No	6	1 abortion	77	3	Apparently focal-clonic	7	Isolated	N (interictal)	Positive
3	M	No	20	Viral bron- chiolitis	78	2	Focal then secondarily generalized	10	Isolated	N (interictal)	Negative
4	F	No	10		81	4	Apparently focal-tonic	8/2	Cluster of 2	N (interictal)	Positive
5	M	Yes	7	FS	66	0	Apparently generalized tonic-clonic	7	Cluster of 3	Right side spike-wave complexes (ictal)	Positive
6	F	Yes	16	FS	57	2	Staring, no motor component	12/3	Isolated	N (interictal)	Negative
7	M	Yes	5		38	4	Apparently generalized tonic-clonic	10/4	Isolated	N (interictal)	Negative
8	F	No	9	Delivered by C/S	49	4	Staring, no motor component	6	Cluster of 2	N (interictal)	Negative
9	F	Yes	8		66	1	Apparently generalized tonic-clonic	6	Cluster of 4	N (interictal)	Positive
10	M	No	8		53	2	Apparently generalized tonic-clonic	11/2	Isolated	N (interictal)	Positive
11	F	No	13	FS	59	5	Staring, no motor component	8	Cluster of 4	N (interictal)	Positive
12	M	No	21	Viral bron- chiolitis	77	2	Apparently focal-tonic	7	Cluster of 2	N (interictal)	Negative
13	M	Yes	18		72	3	Apparently generalized tonic-clonic	12	Isolated	N (interictal)	Positive
14	F	Yes	6		90	2	Apparently focal-tonic	8/2	Isolated	N (interictal)	Negative

Table 1. (Continued)

Patient	Sex	Con- sanguinity	Age- presentation (months)	Personal/ Family History	Age- current (months)	Interval (days) between GE and onset of seizures	Type of seizures	Number of diarrheal stools/ vomiting per 5 days	Number of seizures per 5 days	EEG	Rotavirus stools
15	M	No	10	BIS (uncle)	61	4	Apparently generalized tonic-clonic	11	Isolated	N (interictal)	Negative
16	M	No	8	1 abortion	42	0	Staring, no motor component	9	Cluster of 3	N (interictal)	Positive
17	F	No	13		65	3	Apparently focal-clonic	7	Isolated	N (interictal)	Positive
18	M	Yes	11	FS, ND	69	5	Apparently focal-tonic	11/1	Cluster of 3	N (interictal)	Negative
19	M	No	4	G6PD def.	71	2	Apparently generalized tonic-clonic	6	Isolated	N (interictal)	Positive
20	F	No	7		98	4	Focal then secondarily generalized	7/2	Cluster of 2	N (interictal)	Positive
21	F	No	4		84	2	Focal then secondarily generalized	11	Isolated	N (interictal)	Positive
22	M	No	16	Delivered By C/S	70	3	Staring, no motor component	6	Isolated	N (interictal)	Negative
23	F	No	22		80	4	Apparently generalized tonic-clonic	10	Cluster of 2	N (interictal)	Negative
24	F	No	9	1 abortion	65	0	Apparently focal-tonic	12/3	Cluster of 3	N (interictal)	Positive
25	F	No	6	BIS (parent)	89	1	Apparently generalized tonic-clonic	10	Cluster of 3	N (interictal)	Negative

F: female; M: male; N: normal; GE: gastroenteritis; BIS: benign infantile seizures; FS: febrile seizures; C/S: caesarian section; G6PD def.: glucose 6 phosphate dehydrogenase deficiency; ND: neonatal death; EEG: electroencephalography; CT: computerized tomography; MRI: magnetic resonance imaging.

study, in which two patients were found to have BISwG (Saadeldin *et al.*, 2010). The authors demonstrated that benign infantile seizures with favourable outcome are not uncommon in Saudi Arabia and are present in about 5% of the total patients with epileptic seizures during childhood (two months to 13 years) and about 12% of epilepsies during infancy (2-24 months) (Saadeldin *et al.*, 2010). In 1982, Morooka was the first to report a clinical condition with an excellent outcome in which mild GE was associated with benign seizures during infancy. From various reports and series in the last three decades, it is clear that this entity, which is now referred to as BISwG, is common in Japan and Asia. Later, patients from other countries were reported with similar electroclinical features and outcome (Contino *et al.*, 1994; Lynch *et al.*, 2001; Caraballo *et al.*, 2009; Narchi, 2004; Gómez-Lado *et al.*, 2005). Currently, BISwG is considered by many as a benign situation-related seizure (Morooka, 1982; Uemura *et al.*, 2002). To the best of the author's knowledge, no reports of BISwG have been published in Saudi Arabia or other Arab countries. The present study demonstrates electroclinical features of BISwG similar to those of other studies (Kawano *et al.*, 2007; Abe *et al.*, 2000; Maruyama *et al.*, 2007).

The age at seizure onset associated with GE in this series was 4-22 months. Some authors reported a higher age at onset of up to 60 months (Uemura *et al.*, 2002; Gómez-Lado *et al.*, 2005). Forty-four percent of the patients developed focal seizures, of which 27.2% developed focal and subsequent generalised seizures, 36% developed GTCS and in 20%, the seizures were not associated with a motor component. The recognition of focality in patients with BISwG is sometimes difficult for parents and even doctors (Caraballo *et al.*, 2003). Focal features were described as hemiconvulsions, lateral gaze, or lateral posturing (Caraballo *et al.*, 2003; Morooka, 1982; Uemura *et al.*, 2002). For BISwG, many investigators have described the seizures to be generalised, based only on clinical features (Caraballo *et al.*, 2003; Morooka, 1982; Uemura *et al.*, 2002).

Nordli *et al.* (1997) analyzed 39 seizures in 20 infants recorded with simultaneous closed-circuit television and EEG. These recordings were independently, blindly reviewed by two epileptologists who concluded that clinical manifestations of seizures during infancy did not reliably indicate the laterality of a seizure or even whether a seizure was focal or generalised. Five patients in our study developed seizures in the form of staring and unresponsiveness with no apparent motor activity, which could not be confidently classified as either focal or generalised. Home video clips recorded for two patients were examined by a paediatric neurologist (IYS), who ascertained that these episodes

were true seizures (*see video sequence*). Patient 5 developed generalised seizures, but his ictal EEG showed focal epileptiform discharges in the right parieto-occipital area with extension to the central area with no secondary generalisation (*figure 2*). This demonstrated that clinical manifestations of infantile seizures are variable and the distinction between partial and generalised seizures in infants can only be confidently made routinely by analyzing ictal EEG recordings preferably with the use of video-EEG monitoring (Nordli *et al.*, 1997). Reports of ictal EEGs are rare in BISwG and all are reported as partial seizures with or without secondary generalisation (Uemura *et al.*, 2002; Maruyama *et al.*, 2007; Tsurui *et al.*, 1989). The site of seizure origin varies among patients with BISwG and many have reported the seizure origin in frontal, temporo-parietal, centro-parietal and occipital areas and occurring mostly with secondary generalisation (Nordli *et al.*, 1997; Imai *et al.*, 1999), unlike that of the present patient. The site of seizure origin could not be confirmed in two cases of BISwG reported by Tsurui *et al.* (1989).

In the present study, 52% of patients had clusters of seizures and 48% had isolated seizures. Similar results have been reported (Komori *et al.*, 1995; Caraballo *et al.*, 2009; Maruyama *et al.*, 2007). Uemura *et al.* (2002) reported a greater number of seizure clusters with two or more seizures occurring in 75% of patients. Others have reported multiple seizures to occur in 40-80% of patients with BISwG (Morooka, 1982; Komori *et al.*, 1995). In the present study, the seizures occurred during the first five days of the onset of GE, and the duration of the seizures was brief and ranged from 25 seconds to two minutes; similar to previously reported cases (Uemura *et al.*, 2002; Kawano *et al.*, 2007; Abe *et al.*, 2000). The diagnosis of BIS, in general, including BISwG, is determined by exclusion of symptomatic seizures. In this study, No patients showed any evidence of electrolyte or metabolic impairment which could be responsible for the seizures. Neuroimaging did not show any abnormalities.

BISwG should be differentiated from febrile seizures (FS) (*table 2*). As the name implies, FS are precipitated by fever which is usually more than 38°C, unlike BISwG where the fever is absent or less than 38°C (Verrotti *et al.*, 2009). By definition, the age at seizure onset in FS is between six months and five years (peak at 18-22 months) (AAP, 2008) while the agreed age at onset in patients with BISwG is 4-30 months (Morooka, 1982; Uemura *et al.*, 2002; Narchi, 2004). Other researchers limited the age at onset to less than two years (Abe *et al.*, 2000). In patients with BISwG, clusters of seizures are common (Uemura *et al.*, 2002; Komori *et al.*, 1995; Caraballo *et al.*, 2009; Narchi, 2004), unlike patients with simple FS. In the present series, although there is

Table 2. Clinical characteristics and comparison between benign infantile seizures with mild gastroenteritis, febrile seizures, and nonfebrile illness seizures (Morooka, 1982; Uemura *et al.*, 2002; Kawano *et al.*, 2007; Verrotti *et al.*, 2009; Zerr *et al.*, 2005; Lee and Ong, 2004).

	Benign infantile seizures with mild gastroenteritis	Febrile seizures (simple)	Nonfebrile illness seizures (Afebrile seizures associated with minor infections)
Age	4-30 months	6-60 months	< 3 years
Psychomotor development	Normal	Normal	Normal
Neurological examination	Normal	Normal	Normal
Fever	<38°C or absent	Usually >38°C	Afebrile
Type of seizures	Focal, focal with secondary generalization, and/or generalized	Generalized	Generalized, focal, and/or unresponsive
Clusters of seizures	Characteristic (multiple)	Isolated	Isolated
Gastroenteritis	Present (by definition)	Can be absent	Present (may be associated with rhinorrhea and cough)
Family history of febrile seizures	Rare	Common, familial with genetic predisposition	Rare
Recurrence rate	Infrequent	Common	Infrequent
Prognosis	Excellent (later epilepsy not documented)	Very good (twofold excess of later epilepsy)	Very good

high incidence of consanguinity, genetic factors do not apparently play a major role in BISwG. Febrile seizures are often familial with a genetic predisposition (Hirose *et al.*, 2003). Although the prognosis is excellent for both conditions, the recurrence rate appears to be low in patients with BISwG as shown in this series; a longer follow-up period may be required to confirm this. The differences between BISwG and FS suggest they are two separate conditions, but share some similarities (Uemura *et al.*, 2002). It seems that there are seasonal differences in the prevalence of cases; while 80% of cases occurred during December to March in some studies, reflecting a winter prevalence of GE (Narchi, 2004), in this series the majority of cases occurred between April and September, peaking between June and July.

Another clinical entity with similarities to BISwG was considered by Lee and Ong (2004) to be a distinct type of situation-related seizure, which they referred to as “afebrile seizures associated with minor infections”. Later, Zerr *et al.* (2005) reported 36 children with first-time afebrile seizures occurring in the context of mild illness, characterized mainly by diarrhoea, vomiting, and rhinorrhoea, which they referred to as

“non-febrile illness seizures.” The authors compared the electroclinical features of these children with those of children with FS and unprovoked seizures (Zerr *et al.*, 2005). Their data demonstrated an association between diarrhoeal illness and “non-febrile illness seizures”, and the condition appeared to have occurred as often as unprovoked seizures sharing characteristics with both FS and unprovoked seizures (table 2). The author’s opinion is that this entity could be a form of BISwG, and further viral analyses of stool specimens are warranted to establish the causal relationship.

Many studies have stressed that the presence of multiple seizures in clusters in patients with BISwG are rather refractory to antiepileptic drugs (Uemura *et al.*, 2002; Kawano *et al.*, 2007; Okumura *et al.*, 2004). The clusters in the present series were brief and ceased spontaneously, and two patients only developed four seizures (16%).

The mechanism that causes seizures associated with mild GE is not fully understood. Viruses or viral fragments invading the central nervous system is one of the suggested mechanisms (Contino *et al.*, 1994; Abe *et al.*, 2000). Lynch *et al.* (2001) detected rotavirus

RNA in the CSF of two patients who developed encephalopathy. Brain dysfunction, caused indirectly by viruses, may be the effect of defects in neurotransmission through calcium channel abnormalities (Tian *et al.*, 1996). High levels of nitric oxide (NO) in the serum may produce deleterious toxic effects to the brain (Kawashima *et al.*, 2004). The authors found high levels of NO in these patients due to increased production of NO which may play a role in the pathogenesis of rotavirus-associated seizures (Kawashima *et al.*, 2004). Moderate to severe GE causing significant dehydration and/or electrolyte imbalance and haemolytic uraemic syndrome may cause afebrile seizures. Although rotavirus disease is the single most important cause of GE in children in Saudi Arabia and throughout the world (Mohammed *et al.*, 1994; El Assouli *et al.*, 1992; Abe *et al.*, 2000), seizures have been observed more commonly in children with GE caused by norovirus than by rotavirus infection (Chen *et al.*, 2009). Kawano *et al.* (2007) studied 39 patients with BISwG and found that 30 patients were positive for rotavirus, nine patients were positive for norovirus, two patients were positive for sapovirus, two patients were positive for adenovirus, and one patient was positive for coxsackievirus A4. In this study, rotavirus antigen was positive in stools in 52% of the patients. The incidence of BISwG seems to be high in many Asian countries like Japan (Kawano *et al.*, 2007; Abe *et al.*, 2000) and Hong Kong (Wong, 2001), whereas it is uncommon in Europe and the United States (Contino *et al.*, 1994). Seizures due to bacterial enteritis causing an encephalopathy-like picture may be specifically due to shigella (Ashkenazi, 2004), campylobacter (Solomon *et al.*, 1982), and non-typhoidal salmonellosis (Arii *et al.*, 2001).

The limitations of this study are the relatively small sample size, relatively short study period and the fact that the study was conducted in a tertiary referral hospital. The major strength of this study is the convincing confirmation that BISwG is not uncommon in Saudi Arabia. The prevalence of BISwG may be more common at the level of primary health centres, and it is hoped that such case series will increase the awareness of clinicians regarding the existence of this syndrome and its benign nature in children. The author stresses the importance of recognition of this benign condition in order to avoid unnecessary laboratory and neuroimaging investigations, EEG recordings and antiepileptic treatment. □

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Disclosure.

None of the authors has any conflict of interest or financial support to disclose.

Legend for video sequence

Patient 16. Video clip recorded at home using a mobile telephone of a nine-month-old boy showing staring with deviation of eyes to one side and no motor component, of about 40 seconds.

References

- Abe T, Kobayashi M, Araki K, *et al.* Infantile convulsion with mild gastroenteritis. *Brain Dev* 2000; 22: 301-6.
- American Academy of Pediatrics (AAP). Febrile seizures: clinical practice guideline for the long-term management of the child with simple febrile seizures. *Pediatrics* 2008; 121: 1281-6.
- Arii J, Tanabe Y, Miyake M, *et al.* Acute encephalopathy associated with nontyphoidal salmonellosis. *J Child Neurol* 2001; 16: 539-40.
- Ashkenazi S. Shigella infections in children: new insights. *Semin Ped Infect Dis* 2004; 15: 246-52.
- Callenbach PM, De Coe RFM, Vein AA, *et al.* Benign familial infantile convulsions: a study of seven Dutch families. *Eur J Paediatr Neurol* 2002; 6: 269-83.
- Caraballo R, Cersósimo R, Espeche A, *et al.* Benign familial and non-familial infantile seizures: a study of 64 patients. *Epileptic Disord* 2003; 5: 45-9.
- Caraballo R, Gañez L, De los Santos C, *et al.* Benign infantile seizures with mild gastroenteritis: study of 22 patients. *Seizure* 2009; 18: 686-9.
- Chen SY, Tsai CN, Lai MW, *et al.* Norovirus infection as a cause of diarrhea-associated benign infantile seizures. *Clin Infect Dis* 2009; 48: 849-55.
- Contino MF, Lebby T, Arcinue EL. Rotavirus gastrointestinal infection causing afebrile seizures in infancy and childhood. *Am J Emerg Med* 1994; 12: 94-5.
- Echenne B, Humbertclaude V, Rivier F, *et al.* Benign infantile epilepsy with autosomal dominant inheritance. *Brain Dev* 1994; 16: 108-11.
- El Assouli SM, Banjar ZM, Mohammed KA, *et al.* Rotavirus infection in children in Saudi Arabia. *Am J Trop Med Hy* 1992; 46: 272-7.
- Engel J. Report of the ILAE Classification Core Group. *Epilepsia* 2006; 47: 1558-68.
- Fukuyama Y. Borderland of epilepsy with special reference to febrile convulsions and so-called infantile convulsions (in Japanese). *Seishin Igaku (Clin Psychiatry)* 1963; 5: 211-23.
- Gómez-Lado C, García-Reboredo M, Monasterio-Corral L, *et al.* Benign seizures associated with mild gastroenteritis: apropos of two cases. *Ann Pediatr* 2005; 63: 558-60.

- Hirose S, Mohny RP, Okada M, *et al.* The genetics of febrile seizures and related epilepsy syndromes. *Brain Dev* 2003; 24: 304-12.
- Imai K, Otani K, Yanagihara K, *et al.* Ictal video-EEG recording of three partial seizures with the benign infantile convulsion associated with mild gastroenteritis. *Epilepsia* 1999; 40: 1455-8.
- 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: 617-22.
- Kawashima H, Inage Y, Ogihara M, *et al.* Serum and cerebrospinal nitrite/nitrate levels in patients with rotavirus gastroenteritis induced convulsion. *Life Sci* 2004; 74: 1397-405.
- Komori H, Wada M, Eto M, *et al.* Benign convulsions with mild gastroenteritis: a report with 10 recent cases detailing clinical varieties. *Brain Dev* 1995; 17: 334-7.
- Lee WL, Ong HT. Afebrile seizures associated with minor infections: comparison with febrile seizures and unprovoked seizures. *Pediatr Neurol* 2004; 31: 157-64.
- Lynch M, Lee B, Azimi P, *et al.* Rotavirus and central nervous systems symptoms: cause or concomitant? Case report and review. *Clin Infect Dis* 2001; 33: 932-8.
- Maruyama K, Okumura A, Sofue A, *et al.* Ictal EEG in patients with convulsion with mild gastroenteritis. *Brain Dev* 2007; 29: 43-6.
- Mohammed KA, El Assouli SM, Banjar ZM. Human rotavirus subgroups and serotypes in children with acute gastroenteritis in Saudi Arabia from 1988 to 1992. *J Med Virol* 1994; 44: 237-42.
- Morooka K. Convulsions and mild diarrhea (in Japanese). *Shonika (Tokyo)* 1982; 23: 131-7.
- Narchi H. Benign afebrile cluster convulsion with gastroenteritis: an observational study. *BMC Pediatr* 2004; 4: 2.
- Nordli DR, Bazil CW, Scheuer ML, *et al.* Recognition and classification of seizures in infants. *Epilepsia* 1997; 38: 553-60.
- Okumura A, Uemura N, Negoro T, *et al.* Efficacy of antiepileptic drugs in patients with benign convulsions with mild gastroenteritis. *Brain Dev* 2004; 26: 164-7.
- Saadeldin IY, Housawi Y, Al Nemri A, *et al.* Benign familial and non-familial infantile seizures (Fukuyama-Watanabe-Vigevano syndrome): a study of 14 cases from Saudi Arabia. *Brain Dev* 2010; 32: 378-84.
- Solomon NH, Lavie S, Tenney BL, *et al.* Campylobacter enteritis presenting with convulsions. *Clin Pediatr (Phila)* 1982; 21: 118-9.
- Tian P, Ball JM, Zeng CQ, *et al.* The rotavirus nonstructural glycoprotein NSP4 possesses membrane destabilization activity. *J Virol* 1996; 70: 6973-81.
- Tsurui S, Oguni H, Fukuyama T. Analysis of ictal EEG in benign infantile convulsions (in Japanese, with English abstract). *J Jpn Epil Soc* 1989; 7: 160-8.
- Uemura N, Okumura A, Negoro T, *et al.* Clinical features of benign convulsions with mild gastroenteritis. *Brain Dev* 2002; 24: 745-59.
- Verrotti A, Tocco AM, Coppola GG, *et al.* Afebrile benign convulsions with mild gastroenteritis: a new entity? *Acta Neurol Scand* 2009; 120: 73-9.
- Wong V. Acute gastroenteritis-related encephalopathy. *J Child Neurol* 2001; 16: 906-10.
- Zerr DM, Blume HK, Berg AT, *et al.* Nonfebrile illness seizures: a unique seizure category? *Epilepsia* 2005; 46: 952-5.