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Convulsions associated with gastroenteritis in the spectrum of benign focal epilepsies in infancy: 30 cases including four cases with ictal EEG recording

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ABTRACT – Benign convulsions associated with gastroenteritis are now recognized as a clinical entity, characterized by an acute cluster of afebrile seizures during an episode of mild diarrhoea with excellent prognosis. We observed 30 children who each experienced at least two seizures associated with mild gastroenteritis. The inclusion criteria were: afebrile seizures during gastroenteritis, dehydration at ≤ 5%, normal neurological findings, normal psychomotor development and no underlying pathology according to laboratory and neuroimaging studies. Mean age was 21 months (range: 6-38). Familial history for epilepsy was positive in 3/30 (10%) and for febrile convulsions in 11/30 (36.6%). Seizure onset was mainly on the third day of gastroenteritis. Seizures were described as generalised by parents in 16/30 patients (53.3%). We directly observed seizures in 14/30 patients (47.7%), and the semiology was partial with secondary generalisation. Focal onset was confirmed in two patients by EEG and in two patients by video-EEG recording. Twenty of 30 patients (66.6%) received antiepileptic drugs during the acute phase. Ten patients (33.3%) received no treatment. During follow-up (mean duration: 53 months), one patient had an isolated afebrile seizure and two others a febrile seizure. At the end of follow-up, antiepileptic treatment was withdrawn for all but two patients. None developed epilepsy. Although the pathogenesis of this clinical entity is unknown, we hypothesize that mild gastroenteritis may provoke a transient brain dysfunction which in turn provokes seizures in children with genetically determined susceptibility. *[Published with video sequences]*

Key words: epilepsies in infancy, gastroenteritis, rotavirus, seizures, convulsions, seizure susceptibility syndrome

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There have been several reports of benign convulsions with mild gastroenteritis (CwG), mainly from Japan (Abe *et al.*, 2000; Uemura *et al.*, 2002; Okumura *et al.*, 2004; Kawano *et al.*, 2007), Taiwan (Huang *et al.*, 1998), and Hong Kong (Wong, 2001) and also a few reports from USA (DiFazio *et al.*, 2007; Iyadurai *et al.*, 2007) and the United Kingdom (Narchi, 2004). Rotavirus has been shown to be the culprit in more than 60% of cases, and in Japanese studies has been detected in more than 80% of stool samples (Nishimura *et al.*, 1993; Abe *et al.*, 2000). The main features of CwG, originally described by Morooka (1982), are:

– convulsions during mild gastroenteritis (from the first to fifth day of diarrhoea) in previous healthy children aged six months to three years;
– mild dehydration;
– normal laboratory examinations including cerebrospinal fluid serum, electrolytes and blood glucose;
– seizures lasting a few minutes with a tendency to recur over several days in a cluster;
– rotavirus detection in the stool;
– no abnormalities on inter-ictal EEG;
– normal development milestones;
– normal neurological examination;
– good prognosis without developing epilepsy.

Seizures are often described as generalised but also as partial. Ictal EEGs are rarely reported (Capovilla and Vigevano, 2001; Imai *et al.*, 1999). Standard treatment for CwG has not yet been established and usually several antiepileptic drugs are used during the acute phase of seizures. Nevertheless, Ichiyama *et al.* (2005) treated children with CwG with low-dose carbamazepine with a very high percentage of efficacy (93.7%). In this study we describe the data we collected during a ten-year period at our hospital.

Materials and methods

We retrospectively studied 30 children, admitted to the Pediatric Neurology Clinic of the "Bambino Gesù" Hospital in Rome from January 1997 to July 2008, because of convulsions associated with gastroenteritis.

The inclusion criteria were: previously healthy infants/children aged six to 38 months who had experienced two or more afebrile convulsions associated with symptoms of gastroenteritis; normal laboratory examinations, including electrolyte, glucose level and blood cell count; normal psychomotor development and normal neurological examinations and 4) no underlying acute pathology excluded by neuroimaging (CT, MRI). Exclusion criteria were: presence of fever at the time of seizures, acute brain pathologies such as meningitis, encephalitis or encephalopathy and history of previous febrile or afebrile seizures.

We analyzed family history for: febrile seizures as well as for epilepsy, the delay between onset of gastroenteritis and appearance of seizures, the number and duration of seizures, seizure semiology, inter-ictal and ictal EEG when available, anticonvulsive therapy in acute phase, presence of viral antigens in stools and neuroimaging studies. During follow-up, we analyzed seizure relapse, EEGs, antiepileptic treatment and cognitive development.

Results

As shown in *table 1*, 30 children were studied: 18 girls and 12 boys; age at onset of seizures ranged from six to 38 months (mean age: 21 months). A positive family history for epilepsy was found in 3/30 (10%) and in 11/30 (36.6%) for febrile seizures. The mean delay between gastroenteritis onset and appearance of the first seizures was 2.4 days, ranging from the same day to the fifth day. Stool culture was performed for 26 patients: rotavirus was positive for 18 children, adenovirus for two and negative for six. Seizures occurred in 26/30 (86.6%) patients between October and April; in the remaining four children, seizures appeared in May in two cases and in August in the other two. The number of seizures ranged from two to 20 (average: 3.8), with a tendency to repeat in clusters lasting 10 minutes to 40 hours. In 16 children, parents or observers reported generalised tonic-clonic convulsions. We observed the seizures in 14 patients: in two children the seizures were characterized by hemiclonic convulsions, while in the remaining 12, seizures started with staring, lateral eye and head deviation, cyanosis, automatism, hypersalivation, apnoea, loss of consciousness and secondary generalisation with tonic-clonic manifestation *(table 1)*. Clonic jerks were usually asynchronous on both sides of the body.

We obtained ictal recordings in four children; two with EEG and two with video-EEG recording. In all four patients seizures showed a focal onset. In three patients the ictal discharge had a focal onset; one in the left central region and two in the right parieto-occipital regions, which subsequently became bilateral. In the fourth patient ictal discharge started in the left occipital region becoming hemispheric *(figure 1)*. Inter-ictal EEG, performed in all patients, was normal. Antiepileptic drugs were administered to 20 patients (66.6%) during the acute state *(table 1)*: intravenously in 16 children and per os in four with oral CBZ. In the remaining 10 patients no treatment was given in the acute phase. CT scans or MRI performed in 23 patients were negative. Follow-up ranged from nine to 139 months (average: 53 months) *(table 2)*. Nine patients had chronic antiepileptic drug therapy (two with phenobarbital, six with carbamazepine and one with phenytoin), lasting three to 39 months (mean duration: 13 months) and only two children were still on therapy at the last control visit.

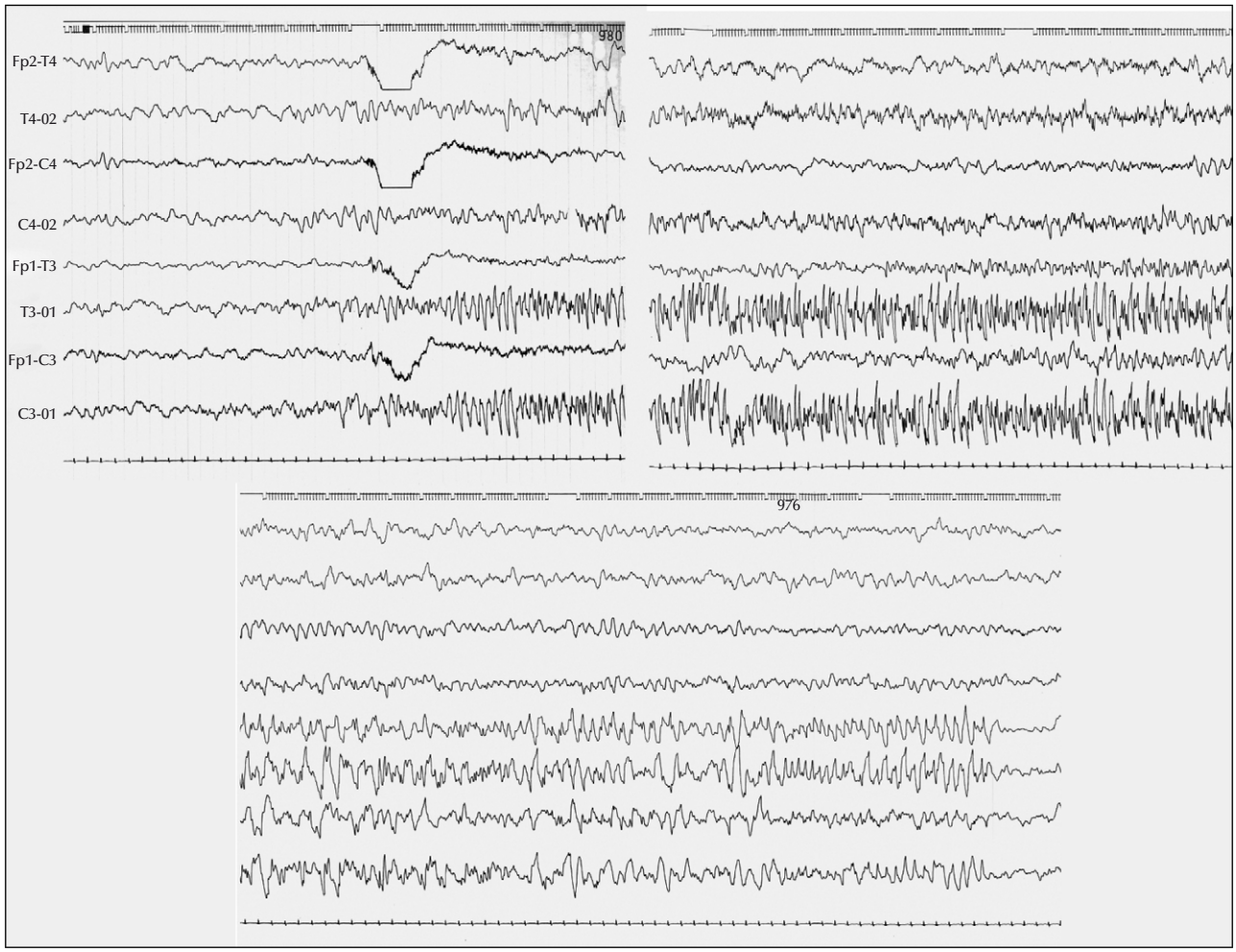

Figure 1. Seizure started in the left occipital region with eyes directed to the right with head deviation followed by tonic-clonic convulsion involving the right side of the body.

During follow-up, two children had a febrile seizure (one at 12 months and one at 24 months of age), and one an isolated unprovoked generalised tonic-clonic seizure at 26 months of life. EEGs, recorded in 21 patients, were abnormal only in two cases. In one case generalised epileptiform discharges were shown during sleep at 24 months of age and disappeared at 48 months. In the other case sleep EEG showed left diphasic temporal spikes at the age of 34 months which disappeared at the last control when the child was six years old. None of our patients developed epilepsy. Twenty-seven patients had normal neurological development; of the remaining three, two patients had mild language delay and one had a mild attention deficit.

Discussion

Rotavirus, like norovirus, sapovirus, and adenovirus, is a well-known cause of mild gastroenteritis in young children (Chung and Wong, 2007; Kawano *et al.*, 2007). The neurotropic characteristic of rotavirus may be an explanation for seizure appearance, however, the mechanism remains unclear. Patients with benign afebrile convulsions during mild gastroenteritis have a normal electrolyte balance and no fever, in spite of the presence of rotavirus infection, suggesting that other mechanisms are involved.

Convulsions associated with gastroenteritis (CwG) are mostly reported as generalised tonic–clonic convulsions (Abe *et al.*, 2000; Uemura *et al.*, 2002; Narchi, 2004); however, some authors have described partial motor or complex partial seizures (CPS) (Komori *et al.*, 1995; Kawano *et al.*, 2007). Uemura *et al.* (2002) analyzed 82 patients and reported symmetric and generalised seizures in 71 children (87%), while in 11 patients (13%) they were CPS with loss of responsiveness, eye deviation, cyanosis and subtle convulsive movements. Komori *et al.* (1995) reported that focal features such as hemiconvulsions or lateral gaze were observed in ten of 19 CwG episodes. Ictal EEGs are rarely

Table 1. Clinical findings in the 30 reported cases of convulsions with gastroenteritis.

Case	Sex	Age at onset (Months)	Family history	No. of days between GE and seizure onset	No. of seizures in cluster	Duration of seizure (s) (Min)	Seizure type	AEDs in acute state	Interictal EEG	CT/MRI
1	F	38	Negative	4	10	3	G	PB (iv)	Normal	Normal
2	F	17	Negative	2	6	3	G	None	Normal	Normal
3	M	13	Negative	2	5	1	FSG	DZP, PHT (iv)	Normal	Normal
4	F	23	Negative	3	3	5	G	None	Normal	Normal
5	M	8	FS	3	4	2	FSG	PB (iv)	Normal	Normal
6	F	18	FS	4	3	1	G	None	Normal	Normal
7	M	14	FS	1	3	4	G	DZP (iv)	Normal	Not performed
8	F	10	FS	2	2	5	G	DZP (iv)	Normal	Not performed
9	F	27	Negative	2	3	4	FSG	DZP, MZL, PHT (iv)	Normal	Normal
10	M	15	E	0	5	4	FSG	CBZ (os)	Normal	Normal
11	M	31	FS	0	2	3	G	DZP, PB (iv)	Normal	Normal
12	F	29	Negative	3	8	2	FSG	None	Normal	Normal
13	M	17	E	5	2	4	G	None	Normal	Normal
14	F	18	Negative	3	20	1	FSG	DZP (ir, iv)	Normal	Normal
15	M	17	Negative	2	2	2	G	None	Normal	Not performed
16	M	22	FS	5	2	4	FSG	None	Normal	Not performed
17	F	24	FS, E	5	4	2	G	None	Normal	Not performed
18	M	20	Negative	2	5	1	FSG	None	Normal	Not performed
19	F	6	Negative	2	2	2	FSG	CBZ (os)	Normal	Normal
20	M	19	FS	3	7	2	G	TPS, PB (iv)	Normal	Normal
21*	F	19	FS	2	5	2	FSG	PB (iv)	Normal	Normal
22	F	26	Negative	4	2	3	G	None	Normal	Normal
23	F	7	Negative	3	2	3	G	DZP (iv)	Normal	Normal
24	M	31	FS	3	6	2	G	DZP (ir, iv)	Normal	Normal
25	M	31	FS	4	6	1	G	PB (iv)	Normal	Not performed
26	F	23	Negative	2	4	2	FSG	MZL (iv)	Normal	Normal
27	F	34	Negative	4	3	10	G	CBZ (os)	Normal	Normal
28*	F	13	Negative	3	8	4	FSG	DZP,PB (iv)	Normal	Normal
29	F	19	Negative	2	4	4	FSG	CBZ (os)	Normal	Normal
30	F	12	Negative	0	10	2	FSG	PB (iv)	Normal	Normal

AEDs: antiepileptic drugs; FS: febrile seizures; E: epilepsy; G: generalised; F: focal; FSG: focal secondary generalised; PB: phenobarbital; DZP: diazepam; PHT: phenytoin; MZL: midazolam; CBZ: carbamazepine; TPS: sodium thiopental; iv: intravenous; ir: intrarectal; *ictal video-EEG recording attached to the manuscript.

reported. Recently, Maruyama *et al*. (2007) described clinical manifestations and ictal EEGs in six patients, demonstrating that all seizures were focal evolving into secondary generalised manifestations. The partial seizures originated more frequently in the parieto-occipital-central regions than in the frontal regions. Similar features were reported by Okumura *et al*. (2007); the most frequent site of origin being the temporal area for CPS and the parietal or occipital area for secondary generalised seizures. Imai *et al*. (1999) described three seizures in a patient with Benign Infantile Convulsions with Diarrhoea (BICD), all of which were CPS evolving into secondary generalised tonic-clonic seizures (SGTCs). Although the semiology was similar, ictal EEG showed different cortical onsets; the seizures were migratory, beginning in the right occipital, right centro-parieto-temporal and left occipital regions, respectively.

The concept of random migrating seizures and the appearance in clusters is common in other infantile

Table 2. Follow-up data for the 30 reported cases of convulsions with gastroenteritis.

Case	Follow-up duration (months)	Chronic therapy	Duration of chronic therapy (months)	Withdrawal of AEDs at last visit	Seizure relapse	Age at relapse (months)	EEG at follow-up	Psychomotor development
1	83	CBZ	24	No	None	/	Normal	Hyperactivity
2	67	None	/	/	None	/	Normal	Language Delay
3	84	PHT	20	Yes	1GS	26	Not performed	Normal
4	70	None	/	/	None	/	Not performed	Normal
5	72	None	/	/	1 FS	12	Normal	Language Delay
6	75	CBZ	3	Yes	None	/	Positive: diffuse epileptic discharges during sleep	Normal
7	66	None	/	/	None	/	Normal	Normal
8	61	None	/	/	None	/	Normal	Normal
9	45	None	/	/	None	/	Not performed	Normal
10	11	CBZ	4	Yes	None	/	Normal	Normal
11	23	None	/	/	None	/	Not performed	Normal
12	43	None	/	/	None	/	Not performed	Normal
13	24	None	/	/	None	/	Normal	Normal
14	51	CBZ	8	Yes	None	/	Normal	Normal
15	27	None	/	/	None	/	Normal	Normal
16	29	None	/	/	None	/	Not performed	Normal
17	20	None	/	/	1 FS	24	Normal	Normal
18	43	None	/	/	None	/	Not performed	Normal
19	14	CBZ	14	Yes	None	/	Normal	Normal
20	14	PB	7	No	None	/	Normal	Normal
21*	130	None	/	/	None	/	Normal	Normal
22	103	None	/	/	None	/	Not performed	Normal
23	95	None	/	/	None	/	Normal	Normal
24	9	None	/	/	None	/	Normal	Normal
25	50	PB	39	Yes	None	/	Positive: spikes in left temporal regions	Normal
26	31	None	/	/	None	/	Not performed	Normal
27	13	CBZ	5	Yes	None	/	Normal	Normal
28*	139	None	/	/	None	/	Normal	Normal
29	31	None	/	/	None	/	Normal	Normal
30	72	None	/	/	None	/	Normal	Normal

AEDs: antiepileptic drugs; PHT: phenytoin; CBZ: carbamazepine; PB: phenobarbital; FS: febrile seizure; GS: generalised seizures; *ictal video-EEG recording attached to the manuscript.

benign convulsions as shown by Vigevano (2005) in Benign Infantile Familial Convulsions (BIFC), and by Watanabe (Watanabe *et al.*, 1990) in benign infantile epilepsy with complex partial seizures. In both reports, patients' seizures had a focal onset in the central or parieto-occipital regions and shifted from one region to the other between both hemispheres, even in a cluster occurring in a single patient.

In our study, we directly observed 14 children with homogeneous seizure semiology, suggesting a focal onset. We recorded the ictal EEG in four patients, and found a focal onset involving the right parieto-occipital region in two and the left central region in one, which then became bilateral in all of these children. In the fourth patient discharge involved the occipital left region.

Similar to other benign epileptic childhood syndromes, CwG can be partial, suggesting the neurophysiopathological hypothesis of an age-related benign seizure susceptibility syndrome, involving different brain areas in the same cluster (Guerrini *et al.*, 1997). Sakai *et al.* (2006) described CwG in siblings whose father and paternal grandfather had had infantile convulsions, pointing to a genetically determined susceptibility for seizures during mild gastroenteritis. The dramatic appearance of seizures

in clusters, lasting two to four days, occurring *ex abrupto* in healthy children is characteristic of other benign childhood epileptic syndromes as well as those described by Vigevano (2005) and Watanabe *et al.* (1990).
Anticonvulsant therapy is not necessary when the patient has a few seizures, even in clusters, because such seizures are generally very brief (Komori *et al.*, 1995; Narchi, 2004). However, CwG often occur in prolonged clusters and are reported to be refractory to antiepileptic drugs. Okumura *et al.* (2004) reported administering two or more drugs to more than half of their patients who had seizures in clusters. In 58% of the episodes, the first-line anticonvulsants failed to stop the seizures. Diazepam (DZP) is not recommended for the treatment of CwG and Omata *et al.* (2002) reported that seven of 11 patients treated with DZP had a recurrence of seizures. Ichiyama *et al.* (2005) observed a good response using low-dose CBZ (5 mg/kg/day). Okumura *et al.* (2004) proposed lidocaine (LD) as the drug of choice for patients with CwG. So far, there is no consensus concerning therapy in the acute phase and different authors, using the same treatment, have reported different results in homogeneous series of children with a good prognosis.
In our patients, when the diagnosis was made and we were able to observe the seizures directly, treatment was withheld during the interval between one seizure and another because the children were clinically healthy. This could suggest that seizures which appear and then disappear are self-limiting and that their occurrence may be independent of the treatment with antiepileptic drugs. The inter-ictal EEG abnormalities detected during the follow-up in two children, both with family history of febrile convulsions, suggests a genetic EEG trait limited to a specific period of life.
Without a clear pathophysiology to explain the association between the uncommon afebrile seizures and the much more common gastroenteritis in young children, it remains possible that chance alone may lead some children who develop seizures to have a concomitant viral illness. Current results support a direct role of rotavirus in afebrile benign convulsions. Rotavirus antigen has been detected in some patients with this condition, and rotaviral RNA has been found in spinal fluid specimens from patients with benign convulsions and mild gastroenteritis (Nishimura *et al.*, 1993). The presence of benign afebrile seizures during mild gastroenteritis is now being increasingly recognized as a clinical entity, thus raising the question whether underlying genetic factors might not only cause the infantile seizures but also confer the susceptibility to CwG (Maruyama *et al.*, 2007). In this report, a genetic study was not performed and genetic predisposition may be a contributing factor in the genesis of CwG.
Although CwG are likely to be categorized as situation-related seizures, they have some features that are similar to those of idiopathic benign partial epilepsies in infancy and early childhood, such as benign partial epilepsy in infancy with secondary generalised seizures.
Finally, some clinical considerations such as age, the dramatic onset in clusters, and the ictal EEG features reported in the literature and in our Italian series, suggest an acute, random and transient brain dysfunction, involving different cortical areas simultaneously in a group of children genetically predisposed to seizures. □

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

Legends for video sequences

Video sequence 1

Seizure discharges originated in left central region and became bilateral. The ictal clinical manifestations began with eye and head deviation, cyanosis and evolved to asynchronous clonic movement involving the face.
EEG montage of first case:
Fp2-T4; T4-O2; Fp2-C4; C4-O2;
Fp1-T3; T3-O1; Fp1-C3; C3-O1;
T4-C4; C4-C3; C3-T3;

Video sequence 2

Seizure discharges originated in the right parieto-occipital region and became bilateral. The ictal clinical manifestations began with eye deviation to the left side followed by asynchronous clonic generalised movements.
EEG montage of second case:
Fp2-F4; F4-C4; C4-P4; P4-O2;
Fp2-F8; F8-T4; T4-T6; T6-O2;
Fp1-F3; F3-C3; C3-P3; P3-O1;
Fp1-F7; F7-T3; T3-T5; T5-O1;
Fz-Cz; Cz-Pz;

References

Abe T, Kobayashi M, Araki K, Kodama H, Fujita Y, Shinozaki T, *et al.* Infantile convulsions with mild gastroenteritis. *Brain Dev* 2000; 22: 301-6.

Capovilla G, Vigevano F. Benign idiopathic partial epilepsies in infancy. *J Child Neurol* 2001; 16: 874-81.

Chung B, Wong V. Relationship between five common viruses and febrile seizure in children. *Arch Dis Child* 2007; 92: 589-93.

DiFazio MP, Braun L, Freedman S, Hickey P. Rotavirus-Induced Seizures in Childhood. *J Child Neurol* 2007; 22: 1367-70.

Guerrini R, Bonanni P, Parmeggiani L, Belmonte A. Adolescent onset of idiopathic photosensitive occipital epilepsy after remission of benign rolandic epilepsy. *Epilepsia* 1997; 38: 777-81.

Huang CC, Chang YC, Wang ST. Acute symptomatic seizure disorders in young children--a population study in southern Taiwan. *Epilepsia* 1998; 39: 960-4.

Ichiyama T, Matsufuji H, Suenaga N, Nishikawa M, Hayashi T, Furukawa S. Low-dose therapy with carbamazepine for convulsions associated with mild gastroenteritis. *No To Hattatsu* 2005; 37: 493-7.

Imai K, Otani K, Yanagihara K, Li Z, Futagi Y, Ono J, *et al.* Ictal video-EEG recording of three partial seizures in a patient with benign infantile convulsions associated with mild gastroenteritis. *Epilepsia* 1999; 40: 1455-8.

Iyadurai S, Troester M, Harmala J, Bodensteiner J. Benign Afebrile Seizures in Acute gastroenteritis: Is Rotavirus the culprit. *J Child Neurol* 2007; 22: 1367-70.

Kawano G, Oshige K, Syutou S, Koteda Y, Yokoyama T, Kim BG, *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.

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: 334-47.

Maruyama K, Okumura A, Sofue A, Ishihara N, Watanabe K. Ictal EEG in patients with convulsions with mild gastroenteritis. *Brain Dev* 2007; 29: 43-6.

Morooka K. Convulsions and mild diarrhea. *Shonika Rinsho* 1982; 23: 131-7.

Narchi H. Benign afebrile cluster convulsions with gastroenteritis: an observational study. *BMC Pediatrics* 2004; 4: 2.

Nishimura S, Ushijima H, Nishimura S, Shiraishi H, Kanazawa C, Abe T, *et al.* Detection of rotavirus in cerebrospinal fluid and blood of patients with convulsions and gastroenteritis by means of the reverse transcription polymerase chain reaction. *Brain Dev* 1993; 15: 457-9.

Okumura A, Watanabe K, Negoro T, Hayakawa F, Kato T, Natsume J. Ictal EEG in benign partial epilepsy in infancy. *Pediatr Neurol* 2007; 36: 8-12.

Okumura A, Uemura N, Negoro T, Watanabe K. Efficacy of antiepileptic drugs in patients with benign convulsions with mild gastroenteritis. *Brain Dev* 2004; 26: 164-7.

Omata T, Tamai K, Kurosaki T, Nakada S, Furusima W, Motoyoshi Y, *et al.* Clinical study of convulsions with mild gastroenteritis. *Nihon Shonika Gakkai Zasshi (Tokio)* 2002; 106: 368-71.

Sakai Y, Kira R, Torisu H, Yasumoto S, Saito M, Kusuhara K, *et al.* Benign convulsion with mild gastroenteritis and benign familial infantile seizure. *Epilepsy Res* 2006; 68: 269-71.

Uemura N, Okumura A, Negoro T, Watanabe K. Clinical features of benign convulsions with mild gastroenteritis. *Brain Dev* 2002; 24: 745-9.

Vigevano F. Benign familial infantile seizures. *Brain Dev* 2005; 27: 172-7.

Watanabe K, Yamamoto N, Negoro T, Takahashi I, Aso K, Maehara M. Benign infantile epilepsy with complex partial seizures. *J Clin Neurophysiol* 1990; 7: 409-16.

Wong V. Acute gastroenteritis-related encephalopathy. *J Child Neurol* 2001; 16: 906-10.