

Epileptic seizures in nonalcoholic Wernicke's encephalopathy: a case report and literature review

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Abstract Wernicke encephalopathy (WE) is characterized by eye signs, cerebellar dysfunction, and confusion. Epileptic seizures are rare in nonalcoholic WE. We reviewed the clinical, laboratory, radiological, and prognostic characteristics of nonalcoholic WE accompanied by epileptic seizures. We reported 1 case and searched similar cases using PubMed, WoK, Ovid, and Embase. WE was diagnosed according to dietary deficiencies, clinical symptoms and brain magnetic resonance imaging (MRI). We reviewed 13 patients (median age, 27 years; 5 men) with clear histories of thiamine deficiency and symptoms of typical WE. The type of epileptic seizures reported in the 13 cases reviewed was generically reported as seizures or convulsions in 4 patients; 7 patients had generalized tonic-clonic seizures, 1 partial seizure, and 1 generalized convulsive status epileptics. Two patients had epileptic seizures as the first symptom of WE. Laboratory tests mainly indicated metabolic acidosis and electrolyte disturbances. Electroencephalography may present as normal patterns, increased slow waves or epileptic discharge. Six patients had cortical lesions on brain MRI. These lesions were usually diffuse and band-like, and sometimes involved all lobes either

symmetrically or asymmetrically, with the frontal lobe as the most susceptible area. All cortical lesions were accompanied by non-cortical lesions typical of WE. Brain MRI abnormalities, after thiamine treatment, mostly disappeared on follow-up MRIs. The patients had good prognoses. Only 1 patient had repeated seizures, and there were no comas or deaths. Patients with nonalcoholic WE accompanied by seizures are young and generally have good prognoses. Most patients experienced generalized convulsive seizures, which may have been related to abnormal cerebral cortical metabolism due to subacute thiamine deficiency.

Keywords Wernicke encephalopathy · Nonalcoholic · Seizure · Cortical impairment · Prognosis

Introduction

Wernicke encephalopathy (WE) is an acute or subacute neurological disorder due to thiamine deficiency and often occurs in alcoholics (Galvin et al. 2010). It is a severe disease with complex clinical manifestations and is often delayed in diagnosis. Typical lesions involve symmetrical alterations in the medial thalami, mammillary bodies, tectal plate, and periaqueductal gray matter. The classical clinical triad of signs of WE comprise eye signs, cerebellar dysfunction, and confusion (Galvin et al. 2010; Zuccoli et al. 2009; Singh and Kumar 2007; Sechi and Serra 2007; Harper et al. 1986; Lough 2012). Compared to alcoholic WE, nonalcoholic WE leads to a high proportion of atypical brain lesions and a lower proportion of classic triad symptoms. This may result in the high frequency of undiagnosed cases of nonalcoholic WE (Galvin et al. 2010; Zuccoli et al. 2009; Sechi and Serra 2007; Manzo et al. 2014). Epileptic seizure is an exceedingly rare symptom of WE, even

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in patients with WE involving the cortex (Galvin et al. 2010; Wu et al. 2017). However, patients without cortical involvement can also have epileptic seizures (Verma et al. 2014; Hiraki and Kikuchi 2014; Watanabe et al. 2009; Fattal-Valevski et al. 2005; Kesler et al. 2005; Rees et al. 1997). This highlights the complexity of WE and makes its diagnosis more difficult. Given that thiamine administration leads to dramatic improvements in patients with WE, and that the disease may be fatal if left untreated (Galvin et al. 2010; Wu et al. 2017; Vasconcelos et al. 1999), in-depth studies of epileptic seizures in patients with nonalcoholic WE have clinical relevance. Here we report the case of a patient with nonalcoholic WE with seizures and cortical lesions and review the literature on the clinical, radiological, and prognostic features of nonalcoholic WE accompanied by epileptic seizures.

Patients and methods

We reviewed one patient who was diagnosed in 2016 in our institution, as well as other patients with nonalcoholic WE accompanied by epileptic seizures from the literature. We searched PubMed, WoK, Ovid, and Embase using the terms “Wernicke encephalopathy” AND “seizure OR epilepsy OR epileptic OR convulsion”, and reviewed the references in all of the reports for additional cases. Searches were carried out until January 1, 2017. We reviewed cases that met the following four criteria: 1) consistent with the operational case definition of WE, which requires meeting two of the following four signs: (i) dietary deficiencies, (ii) eye signs, (iii) cerebellar dysfunction, and (iv) either an altered mental state or mild memory impairment; 2) presence of epileptic seizures; 3) availability of brain magnetic resonance imaging (MRI) results; 4) nonalcoholic. Patients with comorbidities such as stroke, cerebral arteriovenous malformation, intracranial infection, or tumor, which may cause epileptic seizures, were excluded. We reviewed information regarding age, sex, past history, causes of thiamine deficiency, clinical manifestations (WE classic triad, type and number of seizures, pyramidal signs, etc.), comorbidities, hematological and cerebrospinal fluid examinations, electroencephalogram (EEG), MRI (location, extent, form, and follow-up of impairment; sequences of MRI), and prognosis for each included patient.

Results

Case 1

A 26-year-old man experienced duodenal obstruction after surgery owing to pancreatic and duodenal injuries caused by an abdominal knife wound. After recurrent vomiting and parenteral nutrition which was deficient of thiamine for 1.5 months,

the patient developed mild memory impairment, ataxia, dizziness, diplopia, bilateral visual and hearing loss, tinnitus, numbness of the whole body, and intractable hiccups. He was admitted to the department of gastrointestinal surgery in our hospital two weeks later. He had a history of hepatitis but no history of alcohol abuse. Blood tests on the day of admission indicated an anion gap of 23.57 mmol/L, amylase levels of 279 U/L, lipase levels of 1950 U/L, glutamic-pyruvic transaminase levels of 69 U/L, glutamic-oxaloacetic transaminase levels of 50 U/L, total bilirubin levels of 26.4 μ mol/L, activated partial thromboplastin time of 21.5 s, and fibrinogen levels of 1.81 g/L. Leukocytes, erythrocytes, hemoglobin, platelets, prothrombin time, thrombin time, international normalized ratio, sodium levels, potassium levels, calcium levels, osmolality, and creatinine levels were normal. Brain MRI showed bilaterally symmetric lesions in the medial thalami, tectal plate, and dorsal medulla, as well as bilaterally extensive and partially symmetric lesions in the cortex (Fig. 1). On the second day of admission, the patient experienced a generalized tonic-clonic seizure (GTCS) which lasted 1 to 2 min. After the seizure, mental status recovered in 1–2 h. Consultation with a neurologist revealed horizontal nystagmus, ocular muscle paralysis, Rinne test left > right, and ataxia. After the seizure, the patient had higher leukocyte count and creatinine levels (18.87×10^9 /L and 123 μ mol/L, respectively), which returned to normal within a few hours. Blood pH as revealed by gas analysis, ammonia levels, and the results of a lumbar puncture and EEG were normal. The patient was treated with thiamine (50–100 mg once per day, intramuscular injection) and Levetiracetam (500 mg twice per day) immediately after the diagnosis of WE was made. His symptoms improved rapidly following treatment. Follow-up brain MRI 2 weeks after admission indicated improvements in most of the lesions, although there were some hyperintense cortical regions on T2WI, FLAIR imaging, and DWI (Fig. 2). Four months later, the patient had persistent nystagmus and ataxic gait.

Literature review

We identified 12 other cases from 11 full-text reports (Wicklund and Knopman 2013; Gregory et al. 2012; Pereira et al. 2011; Doss et al. 2003; Verma et al. 2014; Fattal-Valevski et al. 2005; Kesler et al. 2005; Rees et al. 1997; Kwon 2004; Hiraki and Kikuchi 2014; Watanabe et al. 2009). There were no language restrictions.

Clinical features

The clinical characteristics of all 13 cases are summarized in Table 1. Patients ranged in age from 0.4 to 71 years, with a median age of 27 years. There were 5 men (38.5%, median age of 23 years) and 8 women (61.5%, median age of 30.5 years).

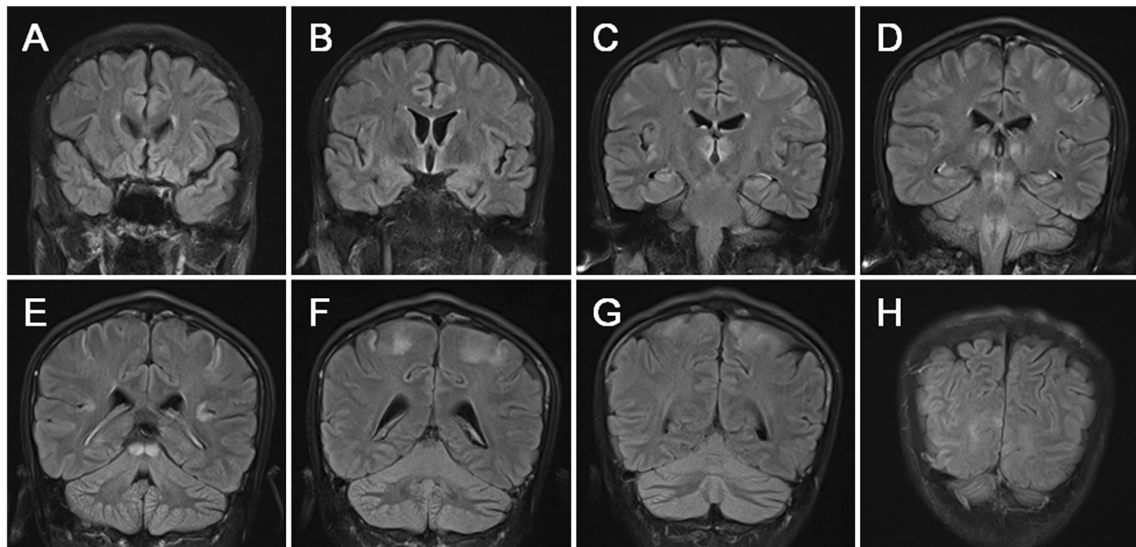


Fig. 1 Fluid-attenuated inversion-recovery (FLAIR) images obtained at onset in Case 1. Hyperintense signals were observed in the bilateral frontal, parietal, and temporal lobes, the right insular and occipital

lobes, the bilateral medial thalamus, tectal plate, and the gray matter around the third and fourth ventricles and the aqueduct

Six patients (46.2%, median age of 30.5 years, 2 men) had cortical lesions. All of the patients had identified causes of thiamine deficiency, which included gastrointestinal disease, persistent vomiting, parenteral nutrition, and cancer. Five of the cases displayed classical WE signs. None of the patients had epileptic seizures as the first symptom of WE. The type of epileptic seizure was specified in 5 of the reports. Four of these cases had generalized seizures (GTCS in 3, partial seizure evolving to generalized seizure in 1), and 1 had simple partial occipital lobe seizures. The prognosis at the end of treatment or at follow-up visits was reported in all 6 cases. Two patients

had an almost normal prognosis, while 4 had neurologic sequelae, although there were no repeated seizures.

Seven patients had no cortical lesions (53.8%, median age of 3 years, 3 male patients). All had identified causes of thiamine deficiency, which included gastrointestinal disease, persistent vomiting, a diet deficient in thiamine, and starvation. These patients displayed classic WE signs. Two patients had epileptic seizures as the first symptom of WE. The types of epileptic seizures were reported in five cases (GTCS in 4 and generalized convulsive status epileptics in 1). The prognosis at the end of treatment or at follow-up visits was reported for all 7 patients. Two patients had

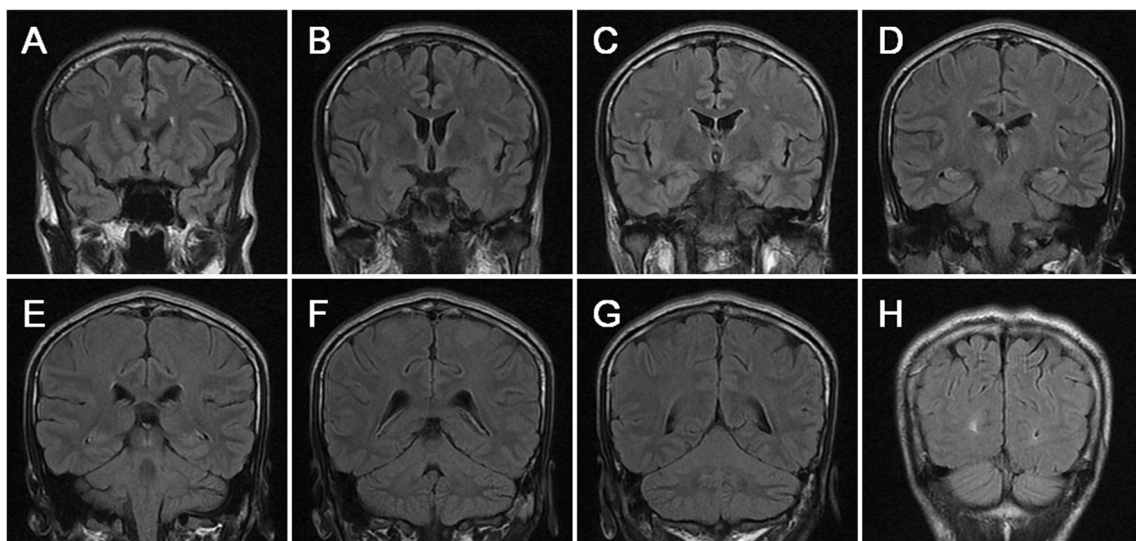


Fig. 2 Follow-up fluid-attenuated inversion-recovery (FLAIR) imaging performed 15 days after thiamine treatment in Case 1. Compared with Fig. 1, the high-intensity alterations have faded significantly

Table 1 Clinical manifestations and prognoses of the 13 cases of WE accompanied by seizures

Author	A(Y)/G	Inducement	Weight loss (kg)	WE triad	Type of seizure	No. of seizures	Pyramidal signs	Other clinical manifestations	Complications	Vitamin B1	Anti-epileptic drugs	Follow-up	
												Duration	Outcome
Cortex-involved													
Case 1	26 M	Duodenal obstruction, frequent vomiting and parenteral nutrition for 1.5 months	light	E,A,C	GTCS	1	(-)	Dizziness, tinnitus and hearing loss, numbness, hiccups	Hepatitis-B	50–100 mg im qd	(+)	4 months	Nystagmus and ataxia sequelae
Wicklund and Knopman 2013	71 F	Myelofibrosis, vomiting and diarrhea after chemotherapy for 2 months	NA	E,C	GTCS	>1	(+)	(-)	(-)	NA	(-)	NA	Severe memory loss
Gregory et al. 2012	51 M	B-cell lymphoma for 6 months	NA	E,C	Simple partial occipital lobe seizures	>1	(-)	Headache	(-)	500 mg iv tid	(+)	NA	Recovered
Pereira et al. 2011	27 F	Crohn's disease, total colectomy and parenteral nutrition for 0.5 months	NA	E,C	NA	NA	(+)	Aphasia, sixth and seventh cranial nerve palsies, upper limb spasticity	(-)	iv	(-)	14 days	Aphasia, tetraparesis and upper limb spasticity sequelae
Kwon 2004	31 F	Pancreatitis, parenteral nutrition	NA	E,A,C	Partial seizure evolving to generalized seizure	2	(-)	Sixth, ninth and tenth cranial nerve palsies, tinnitus	(-)	300 mg iv qd	(+)	25 days	Recovered
Doss et al. 2003	30 F	Self-induced vomiting after meals for 6 months	NA	A,C	GTCS	2	(-)	(-)	(-)	High-dose iv	(-)	NA	Ataxia and mental disturbance sequelae
Non-cortex-involved													
Verma et al. 2014	23 M	Cholecystitis, cholecystectomy and vomiting for 1 months	18.1	E,A,C	GTCS	2	(-)	(-)	Asthma	500 mg iv tid	(-)	Several months	Recovered
Hiraki and Kikuchi 2014	0.7 M	Excessive intake of isotonic drinks for 2 months	Low weight	E,C	Generalized convulsive status epileptics	1	(-)	(-)	(-)	NA	(-)	7 months	Mental retardation
Watanabe et al. 2009	3 M	Infantile autism, severe eating disorder for 0.7 months	3.5	C	GTCS	Cluster	(-)	(-)	(-)	2.1 mg/kg iv qd	(+)	3 months	Recovered
Fattal-Valevski et al. 2005	0.4 F	Thiamine-deficient diet and vomiting for 2–5 months	NA	E,C	NA	NA	NA	Movement disorder	(-)	250 mg iv bid	(-)	5 weeks	Nystagmus and movement disorder sequelae

Table 1 (continued)

Author	A(Y)/G	Inducement	Weight loss (kg)	WE triad	Type of seizure	No. of seizures	Pyramidal signs	Other clinical manifestations	Complications	Vitamin B1	Anti-epileptic drugs	Follow-up	
												Duration	Outcome
Kesler et al. 2005	0.4 F	Thiamine-deficient diet for 4.5 months	NA	E,C	NA	>1	(-)	(-)	(-)	iv	(-)	11 months	Oculomotor and mental disturbance sequelae
Rees et al. 1997	31 F	Hyperemesis gravidarum for 2 months	>13.6	E,A,C	GTCS	1	(-)	(-)	(-)	NA	(+)	3 months	Memory loss
	31 F	Hyperemesis gravidarum for 1.5–2.5 months	>6.4	E,A	GTCS	1	(+)	(-)	(-)	NA	(-)	2 months	Nystagmus and ataxia sequelae

WE Wernicke encephalopathy, E eye signs, A ataxia, C confusion, GTCS generalized tonic-clonic seizure, im intramuscular, qd once per day, NA not available, iv intravenous, tid three times per day, bid twice per day

almost normal prognoses, while 5 had neurologic sequelae. One patient had repeated seizures.

Results of laboratory and auxiliary examinations

Details of laboratory and auxiliary tests for all 13 cases are summarized in Table 2.

Of the 6 patients who had cortical involvement, thiamine deficiency was only reported in 1 patient. Results of other blood analyses and lumbar puncture were only reported in 3 and 2 patients respectively and they were normal. One patient had epileptogenic activity over the left occipital region on EEG, which is consistent with simple partial occipital seizures, and 1 case had normal EEG. MRI indicated that 5 patients had cortical lesions involving the frontal lobe, 3 had lesions involving the temporal and occipital lobes, and 1 had parietal lobe and insular lesions. MR images were displayed in 5 articles. Four of the MR images indicated the presence of band-like cortical lesions, while 1 displayed limited band-like cortical lesions. No cortical enhancement was observed in the 3 cases for which MR contrast enhancement was reported. All of these 6 patients had non-cortical lesions, most of which were typical WE lesions. Three patients underwent follow-up MRI, which indicated significant amelioration of the lesions after thiamine treatment.

Of the 7 patients without cortical lesions, thiamine deficiency was reported in 5 patients. 4 patients had metabolic acidosis and 3 had electrolyte disturbance. The results of lumbar puncture were reported for 4 patients, 3 of whom had increased lactic acid, while 1 had increased protein levels. EEG results were reported for 2 patients, wherein merely diffuse slow waves were observed. One patient had normal MRI results and 6 had non-cortical lesions in the typical areas implicated in WE. Follow-up MRI was performed in 3 patients. The lesions had disappeared in 1 patient who had atrophy and were significantly ameliorated in 2 patients.

Discussion

The classic triad of WE symptoms comprises eye signs, ataxia, and confusion (Galvin et al. 2010; Zuccoli et al. 2009; Singh and Kumar 2007; Sechi and Serra 2007; Harper et al. 1986; Lough 2012). Epileptic seizure is an exceedingly rare symptom in nonalcoholic WE. The incidence of epileptic seizure in WE was 0%–3.1% reported by Galvin et al. By reviewing 218 WE patients diagnosed clinically and 256 WE patients diagnosed by autopsy. We reviewed the clinical, radiological, and prognostic features of nonalcoholic WE accompanied by epileptic seizures for the first time. The results revealed that the form of epileptic seizure is various while GTCS is the main type, and epileptic seizure could be the first symptom of WE. The results also showed that about half of

Table 2 Results of the accessory examinations in the 13 cases of WE accompanied by seizures

Author	Thiamine blood level	Other blood tests	Lumbar puncture	EEG	MRI sequences	Non-cortex lesions	Cortex lesions		CE	Follow-up MRI	
							Location	Range		Duration	Cortical lesions
Cortex-involved Case 1	None	Shown in the text	Normal	Normal	T1/T2/FLAIR/CE (DWI on follow-up)	Bilaterally symmetric medial thalamus, tectal plate, and dorsal medulla	Bilaterally symmetric frontal, periaqual, and temporal cortex (including precentral gyrus), and right occipital and insular cortex	Diffuse	Band-like (-)	15 days	Significantly fading
Wicklund and Knopman 2013	None	NA	None	None	T1/T2/DWI/CE	Bilaterally symmetric medial thalamus and tectal plate	Left precentral gyrus	Limited	Band-like	None	/
Gregory et al. 2012	None	NA	None	Epileptogenic activity over the left occipital lobe	NA	Bilateral temporal-occipital leptomeninges and cavernous sinus	Bilateral temporal-occipital cortex	NA	NA	None	/
Percira et al. 2011	None	NA	None	None	T1/T2/FLAIR/DWI	Bilaterally symmetric hypoglossal, abducens and facial nuclei, right medial thalami and tectal plate	Bilaterally symmetric frontal cortex (including precentral gyrus)	Diffuse	Band-like	21 days	Significantly fading
Kwon 2004	None	Almost normal	None	None	T1/T2/DWI	Bilaterally symmetric medial thalamus, tectal plate, and periaqueductal gray matter	Bilaterally symmetric frontal cortex (not including precentral gyrus)	Diffuse	Band-like	25 days	Disappeared
Doss et al. 2003	15 nM/l	Normal	Normal	None	T1/T2/DWI	Bilaterally symmetric medial thalamus and hypothalamus	Bilaterally asymmetric frontal cortex (including precentral gyrus) and hippocampus	Diffuse	Band-like	None	/
Non-cortex-involved Verma et al. 2014	36 nM/l	Almost normal	None	Slow waves	T1/T2/FLAIR	Bilaterally symmetric medial thalamus, periaqueductal	/	/	/	7 days	Significantly fading

Table 2 (continued)

Author	Thiamine blood level	Other blood tests	Lumbar puncture	EEG	MRI sequences	Non-cortex lesions	Cortex lesions		CE	Follow-up MRI	
							Location	Range		Duration	Non-cortical lesions
Hiraki and Kikuchi 2014	8 ng/ml	pH 7.45; BE, -6.2 mmol/l;	None	None	T1/T2/DWI	gray matter, and hypothalamus	/	/	/	2 months	Disappeared and atrophy
Watanabe et al. 2009	<5 ng/ml	Lac, 7.2 mmol/l; Glu, 22.2 mmol/l; hyponatremia	Lac, 4.8 mmol/l	Slow waves	T1/T2/FLAIR	symmetric caudatum and putamina	/	/	/	None	/
Fattal-Valevski et al. 2005	TPPE>15%	Lac, 5.9 mmol/l	Lac, 6.9 mmol/l	None	T1/T2	symmetric basal ganglia, mammillary bodies, and periaqueductal gray matter	/	/	/	5 weeks	Significantly fading
Kesler et al. 2005	TPPE >15%	Lac, 6.6 mmol/l	Lac, 4.7 mmol/l	None	T1/T2/DWI	gray matter	/	/	/	None	/
Rees et al. 1997	None	Hypocalcemia, hypomagnesemia	Protein, 1.3 g/dl	None	T1/T2	symmetric basal ganglia and periaqueductal gray matter	/	/	/	None	/
	None	Hyponatremia, hypokalemia	None	None	NA	Gray matter around the third ventricle	/	/	/	None	/

WE Wernicke encephalopathy, EEG electroencephalogram, MRI magnetic resonance imaging, CE contrast enhancement, NA not available, T1 T1-weighted imaging, T2 T2-weighted imaging, FLAIR fluid attenuated inversion recovery, DWI diffusion-weighted imaging, BE base excess, Lac lactic acid, Glu glucose, TPPE thiamine pyrophosphate effect, normal values range from 0% to 15%, a value of >15% indicates thiamine deficiency

these patients didn't have cortical lesions on brain MRI, and their prognoses were good.

Our review indicates that the main type of epileptic seizures observed in nonalcoholic WE is generalized seizure, and especially GTCS. However, the exact cause of seizures in nonalcoholic WE has not been clarified yet. Our review included 6 patients with cortical alterations. Five of these patients had bilateral lesions, while Case 1 had bilateral cortical lesions on brain MRI before the seizure. Among the 7 patients without cortical alterations, 3 had electrolyte disturbances, which might evoke epileptic seizures (Rees et al. 1997), and 4 had metabolic acidosis, which might inhibit epileptic seizures (Ziemann et al. 2008). The EEG results were variable, and included normal, epileptic discharge, and slow waves. The last pattern may also be found in the later periods of WE in the absence of epileptic seizures (Sechi and Serra 2007). Meanwhile, epileptic seizures stopped as the WE symptoms improved. Thus, we hypothesized that epileptic seizures might be associated with cortical damage and pathophysiological changes after thiamine deficiency. These changes include impaired energy metabolism, glutamate-mediated excitotoxicity, impaired synthesis of glucose-derived neurotransmitter gamma-aminobutyric acid, oxidative stress, and inflammation (Manzo et al. 2014; Jhala and Hazell 2011; Jung et al. 2012; Sechi et al. 2016).

Generally, epileptic seizures are not the first symptoms of nonalcoholic WE. However, among patients in our review, 1 patient had ocular movement and mental status disorder after status epilepticus (Hiraki and Kikuchi 2014), and 1 patient had epileptic seizure after 6 weeks of hyperemesis gravidarum, which was followed by ocular movement disorders and ataxia 4 weeks later (Rees et al. 1997). In addition, one study revealed that only 16% of patients had the classical clinical triad and that 19% had no documented clinical signs (Harper et al. 1986). This suggests that the presentations and onset of nonalcoholic WE may be more complex than the current cognition. Epileptic seizures may be the first symptoms of nonalcoholic WE, although usually one or more symptoms may appear later in the course of WE (Sechi and Serra 2007).

Brain MRI impairments of nonalcoholic WE with epileptic seizure could involve the cortex or not. The non-cortical impairments were located in typical areas of WE (Zuccoli et al. 2009; Zuccoli et al. 2010; Zuccoli and Pipitone 2009; Manzo et al. 2014). And we found some features of the cortical impairments. First, all of the brain lobes could be involved, with the frontal lobe (usually including the precentral gyrus) the most susceptible area, followed by the temporal and occipital lobes. Second, cortical impairments were usually diffuse and band-like. Third, in contrast to typical lesions of WE, cortical impairments might be asymmetric or unilateral. Fourth, all cortical impairments were accompanied by non-cortical impairments. The cortical impairments of nonalcoholic WE without epileptic seizure described by Wu et al. (2017) are

band-like and asymmetric in some cases, which was consistent with our observation. However, in Wu's review, all lesions were bilateral with the frontal and parietal lobes as the most susceptible areas.

The median age of nonalcoholic WE patients with epileptic seizure and cortical impairments was 31 years (range, 26–71 years), which was significantly younger than that of nonalcoholic WE patients with cortical impairments but without epileptic seizure reviewed by Wu et al. (2017) (median age, 53 years; range, 13–81 years). The nonalcoholic WE patients with epileptic seizure but without cortical impairments was even younger (median age, 3 years; range, 0.4–31 years) and 3 of them were infants. Therefore, young patients and infants may be more sensitive to these changes induced by thiamine deficiency, which would in turn makes them more prone to epileptic seizures, especially in the absence of cortical lesions.

Patients in our review had better prognoses than other WE patients reported in previous studies. Among the 13 patients, 4 had almost normal outcomes, 5 had persistent mental or memory disturbances, 6 had persistent eye signs and/or ataxia, and only 1 had repeated seizures. There was no cases of coma or death. The mortality rate of WE has been reported to be 14.3–20% in previous studies (Sechi and Serra 2007; Lough 2012; Manzo et al. 2014), and may be as high as 26.1% in those with cortical lesions (Wu et al. 2017). About 80% of patients who survived from WE developed Korsakoff's syndrome, which is a severe memory disorder (Sechi and Serra 2007; Victor et al. 1971). Our results indicate that these patients with atypical WE respond well to thiamine and antiepileptic drugs, and that epileptic seizures do not lead to poor prognosis in WE.

Our review has following limitations. First, some data was insufficient in those cases, and we couldn't make a stricter description and explanation on some results like blood tests, lumbar puncture and electroencephalogram. Second, the effectiveness and route of administration of antiepileptic drugs for these patients were not clear. Antiepileptic drug use was only reported in 4 cases, and we were unable to determine whether antiepileptic drugs were used in the other 10 cases. Only 1 case report that did not mention the use of antiepileptic drugs indicated repeated seizures in follow-up. Therefore, the method required to control epileptic seizures (thiamine vs. antiepileptic drugs), and how long anti-epileptic drugs should be used require further study.

Conclusions

Epileptic seizures are very rare in nonalcoholic WE. Patients with nonalcoholic WE accompanied by epileptic seizures are usually younger than those reported in previous studies. Epileptic seizures may be the first symptoms of WE, and the most common type of epileptic seizures in WE is GTCS. The mechanisms underlying these epileptic seizures are as yet not

clear, but may be related to cortical lesions and abnormal metabolism owing to thiamine deficiency. The cortical lesions on brain MRI are mostly diffuse and band-like, and may involve all lobes either symmetrically or asymmetrically. The frontal lobe is the most susceptible area, followed by the temporal and occipital lobes. In addition to cortical lesions, brain MRI lesions typical for WE are usually present. The prognosis for nonalcoholic WE is good and seizures rarely recur.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Informed consent Informed consent was obtained from the one case included in the study.

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