

Epilepsia partialis continua in tick-borne Russian spring-summer encephalitis

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Objectives – Epilepsia partialis continua (EPC) is characterized by localized continuous jerks, from time to time with spreading Jacksonian seizures and, more rarely, secondarily generalized tonic-clonic seizures. EPC has numerous possible etiologies. In this paper we describe EPC in the tick-borne Russian spring-summer encephalitis (TBRSE) and compare it with Rasmussen syndrome. **Materials and methods** – We included patients with EPC in TBRSE (between 2003 and 2010). The diagnosis was verified by immunology (antibodies against TBRSE virus). The patients were followed 1–7 (mean 3.4) years. **Results** – We studied 10 patients (eight males, age 10–21 years) with MRI and video-EEG. Nine developed EPC after acute TBRSE (meningoencephalitic form), and one had a tick bite without clinical symptoms of encephalitis, but with subsequent EPC. All patients came from Ural and Siberia. The onset was at age 4–14 (mean 8.6 years). The interval from onset of TBRSE or the tick bite to seizure onset was 1 day–4 years. We identified three phases of clinical course EPC in TBRSE: (i) acute (meningoencephalitic/encephalitic); (ii) development of EPC; and (iii) chronic EPC. The effect of antiepileptic drugs differed according to seizure types. **Conclusion** – EPC caused by TBRSE is relatively frequent in the Eastern parts of the Russian Federation but not west of the Ural. Unlike Rasmussen encephalitis, EPC with TBRSE does not progress even in the long term. It appears as disabling but not fatal condition with a time course where three phases can be distinguished.

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Key words: epilepsia partialis continua; tick-borne encephalitis; Russian spring-summer encephalitis; non-progressive encephalitis; Rasmussen encephalitis

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Introduction

Epilepsia partialis continua (EPC) is characterized by localized continuous jerks intermingled from time to time with spreading Jacksonian seizures and, more rarely, secondarily generalized tonic-clonic seizures (sGTCS). It was first described, and the term proposed by Kozhevnikov (1) based on four cases where he considered chronic encephalitis as the probable cause, however not ruling out cysticercosis. In recognition of his work, the condition is also eponymed Kozhevnikov's epilepsy.

Epilepsia partialis continua has numerous possible etiologies (2, 3), and Rasmussen syndrome, a chronic progressive encephalitic variety, has been separated out as an own nosological entity (4).

Another encephalitic etiology, tick-borne Russian spring-summer encephalitis (TBRSE), though identified by the work of Omorokov (5), Shubin (6), Propper-Graschenkov (7) and Chumakov (8) has received much less attention, not the least because these authors published in Russian.

In this paper we describe EPC in TBRSE and compare it with Rasmussen syndrome.

Material and methods

We included all patients with EPC who between 2003 and 2010 were seen at Center of Child Neurology and Epilepsy (Moscow), Russian Child Clinical Hospital (Moscow) and outpatient hospital in Perm (endemic zone for TBRSE in Eastern part of Russia (Ural, Siberia, Far East))

The most difficult cases from all Russia were referred to Russian Child Clinical Hospital (Moscow). The diagnosis of TBRSSSE was made on the basis of case history, general and neurological investigation and MRI. The diagnosis had to be verified by immunology (antibodies against the tick-borne Russian spring-summer encephalitis viruses in both – blood serum and CSF. MRI was performed at Russian Child Clinical Hospital on a 1.5-T Sigma Infinity GE System with the study modes T1, T2 and fluid attenuated inversion recovery (FLAIR) in seven patients. In three patients, the MRI was performed in the Outpatient Hospital of Perm on a Siemens workstation with 1.0-T units.

Patients with a diagnosis or a suspicion of Rasmussen encephalitis were excluded.

Twelve to twenty-four hours video-EEG monitoring was performed in all patients on a portable computerized recorder 'ENCEPHALAN-EEGR-19/26' modification 11 (Medicom-mtd) and a computerized portable recorder NeuroScope version 6.1 Rev. 508 (Biola). The international 10–20 system was used for electrode placement.

All patients underwent neurological investigation and Wechsler's test.

The patients were followed from 1 to 7 years (mean 3.4 years).

Results

We identified ten patients (eight males and two females), age 10–21 years at report. Nine developed EPC after acute TBRSSSE (meningoencephalitic form), and one had a tick bite without clinical symptoms of encephalitis, but with subsequent EPC. All patients came from Ural or Siberia.

Clinical course

Onset of *TBRSSSE* was at age 4–14 (mean 8.6 years). The interval from onset of TBRSSSE or the tick bite to seizure onset was 1 day–4 years (mean 6.3 months).

Three phases in the clinical course of EPC in TBRSSSE could be distinguished.

1 Acute (meningoencephalitic/encephalitic) phase

All patients but one went through this phase where they were treated in various hospitals of Eastern Russia, and the description is based upon the hospital reports. The onset of encephalitis ranged from 4 days to 2 months after the tick bite (median 17 days). This phase lasted from 3 days to 1 month (median 19 days) and was characterized by fever from 38 to 41°C with

meningeal signs and focal neurological signs (facial asymmetry in four, hemiparesis in three, monoparesis of the hand in two, dysarthria in three, anisocoria in three, and ataxia in one). Three patients were in coma. The clinical picture was not fundamentally different from the acute phase of TBRSSSE in patients who do not later develop EPC. In four patients, epileptic seizures were observed in this phase, and in two of them, EPC started already here.

2 Phase of development of EPC

This phase was observed in all cases, and six of them were first seen by us during this phase which lasted from 6 months to 9 years (average 4.4 years). In eight patients epilepsy started with focal myoclonus (from 3 months up to 10 months from the beginning of the disease, on average after 6 months). In two patients, EPC started before the seizures. In the 2nd stage of the disease focal epileptic myoclonus was observed in all patients in various combinations with sGTCS and focal motor seizures. In this stage neurological findings were present in all cases (Table 1) and comprised severe oculomotor dysfunction in three cases, facial asymmetry with dysarthria in eight, spastic hemiparesis of various degrees in five, spastic monoparesis of the hand in four, slight paresis of the leg in one; nine patients displayed slight cerebellar signs with a positive Romberg phenomenon, and six showed variable cognitive dysfunctions. The neurological symptoms in all patients improved during this phase.

3 Chronic phase

At the time of the study four patients were in this phase. It started after 6 months to 9 years (median 4 years) from the onset, and we have followed them for 5–8 years. There was no further progression of the EPC in extension or severity, and the neurological findings remained stable or improved (three patients with slight monoparesis and one without neurological deficits, none with cognitive dysfunctions). In all patients sGTCS were controlled by pharmacotherapy, whereas EPC resisted to treatment with all antiepileptic drugs. Only one patient continued to have rare focal motor seizures.

Seizures

In the fully developed state (i.e. phases 2 and 3) all patients presented focal myoclonus of variable intensity, asynchronous and arrhythmic, prevailing in face and upper extremities. It was prominent in the flexor muscles of the upper extremities (three cases), face and upper extremities (four cases),

Table 1 Patient overview

Patient no.	Sex	Age of patient (full years)	Age at onset of TBRSE (year)	Interval from onset of TBRSE to seizure and EPC onset	Neurological findings	Concomitant seizure types	EEG findings	MRI findings	Successful drug EPC (worthwhile reduction)	Successful drug seizures
1	Male	14	8	Seizures: day 2 of encephalitis; EPC: day 16	Phase of development of EPC; Oculomotor dysfunction; Right facial hemiparesis; Dysarthria; Right spastic hemiparesis; Cerebellar signs with positive Romberg test; Cognitive dysfunctions	FCS; FTSD	Normal background activity; Intermittent reg. slowing in the left fronto-central region; Continuous reg. S-W.C. in the left fronto-central region; Continuous reg. S-W.C. during sleep in the left fronto-central region	Moderate diffuse cortico-subcortical atrophy and ventricles enlargement	Valproic acid	valproic acid
2	Male	21	14	Seizures: day 6 of encephalitis; EPC: after 6 months	Phase of development of EPC; Facial asymmetry; Dysarthria; Monoparesis of the right hand Chronic phase: Without neurological deficits	FCS; FTSD; Generalized myoclonic seizures	Normal background activity; Intermittent reg. slowing in the left frontal region; Isolated reg. S-W.C. in the left frontal region; Diff. S-W.C.	Normal	None	Valproic acid
3	Female	21	8	Seizures: day 4 of encephalitis; EPC: after 4 months	Phase of development of EPC; Right facial hemiparesis; Dysarthria; Right spastic hemiparesis; Cerebellar signs with positive Romberg test	FCS; FTSD; sGTCS	Low b.a.; Continuous reg. slowing in the left fronto-centro-temporal region; Continuous reg. S-W.C. in the left fronto-centro-temporal region; Continuous reg. S-W.C. during sleep; slowing in the left fronto-centro-temporal region	Moderate diffuse cortico-subcortical atrophy and ventricles enlargement	Benzodiazepine + valproic acid	Benzodiazepine + valproic acid
4	Male	10	5	Seizures: day 1 of encephalitis; EPC: day 4 of encephalitis	Phase of development of EPC; Facial asymmetry; Dysarthria; Monoparesis of the left hand; Cerebellar signs with positive Romberg test; Cognitive dysfunction	FCS; FTSD	Slow b.a.; Continuous reg. slowing; in the right centro-temporo-parietal region; Continuous reg. S-W.C. in the right centro-temporo-parietal region; Continuous reg. S-W.C. during sleep in the right centro-temporo-parietal region	Normal (investigation in dynamics)	Levetiracetam + valproic acid	Levetiracetam + valproic acid
5	Female	17	4	EPC: 4 months after encephalitis; Seizures: after 4 years	Phase of development of EPC; Monoparesis of the right leg; Cerebellar signs with positive Romberg test; Chronic phase: Monoparesis of the right leg	FCS; FTSD	Normal background activity; Intermittent reg. slowing in the left fronto-centro-temporal region; Continuous reg. S-W.C. in the left fronto-centro-temporal region; Continuous reg. S-W.C. during sleep in the left fronto-centro-temporal region	Normal	Valproic acid	Valproic acid

4 **Table 1** (Continued)

Patient no.	Sex	Age of patient (full years)	Age at onset of TBRSE (year)	Interval from onset of TBRSE to seizure and EPC onset	Neurological findings	Concomitant seizure types	EEG findings	MRI findings	Successful drug EPC (worthwhile reduction)	Successful drug seizures
6	Male	17	7	Seizures: 10 months after encephalitis; EPC: after 3 years	Phase of development of EPC: Facial asymmetry; Dysarthria; Monoparesis of the right hand; Cerebellar signs with positive Romberg test; Chronic phase: Monoparesis of the right hand	FCS; FTSD	Normal background activity; Intermittent reg. slowing in the left fronto-central region; Continuous reg. S-W.C. in the left fronto-central region; Diff. S-W.C.; Continuous reg. S-W.C. during sleep in the left fronto-central region	Moderate diffuse cortico-subcortical atrophy and ventricles enlargement	None	Carbamazepine
7	Male	14	7	EPC: 6 months after encephalitis; Seizures: after 8 months	Phase of development of EPC: Left facial hemiparesis; Dysarthria; Left spastic hemiparesis; Cerebellar signs with positive Romberg test; Cognitive dysfunction	FCS; FTSD; Som.aura in the hand; sGTCS	Slow. b.a.; Continuous reg. slowing; in the right fronto-temporal region; Continuous reg. S-W.C. in the right fronto-temporal region; Continuous reg. S-W.C. during sleep in the right fronto-temporal region	Moderate diffuse cortico-subcortical atrophy and ventricles enlargement	Benzodiazepine	None
8	Male	19	11	Seizures: 8 months after encephalitis; EPC: after 14 months	Phase of development of EPC: Monoparesis of the left hand; Cerebellar signs with positive Romberg test; Chronic phase: Monoparesis of the left hand	FCS; FTSD	Normal background activity; Intermittent reg. slowing in the right fronto-central region; Continuous reg. S-W.C. in the right fronto-central region; Continuous reg. S-W.C. during sleep in the right fronto-central region	Moderate diffuse cortico-subcortical atrophy and ventricles enlargement	Valproic acid + phenobarbital	Valproic acid + phenobarbital
9	Male	20	14	EPC: 5 months after encephalitis; Seizures: 6 months after encephalitis	Phase of development of EPC: Oculomotor dysfunction; Right facial hemiparesis; Dysarthria; Right spastic hemiparesis; Cerebellar signs with positive Romberg test; Cognitive dysfunction	FCS; FTSD	Normal background activity; Intermittent reg. slowing in the left fronto-central region; Isolated reg. S-W.C. in the left fronto-central region	Normal	Valproic acid + topiramate; levetiracetam + valproic acid	Valproic acid + topiramate; levetiracetam + valproic acid
10	Male	10	4	Seizures: 3 months after tick bite; EPC: 4 months after tick bite (no acute encephalitic phase)	Phase of development of EPC: Oculomotor dysfunction; Right facial hemiparesis; Dysarthria; Right spastic hemiparesis; Cerebellar signs with positive Romberg test; Cognitive dysfunction	FCS; FTSD; Som.aura in the hand; sGTCS	Slow b.a.; Continuous reg. slowing in the left fronto-central region; Continuous reg. S-W.C. in the left fronto-central region; Continuous reg. S-W.C. during sleep in the left fronto-central region	Moderate cortico-subcortical atrophy and ventricles enlargement on the left	Carbamazepine + levetiracetam	Carbamazepine + levetiracetam

EPC, Epilepsia partialis continua; FCS, Focal clonic seizures; FTSD, Focal tonic seizures with deviation of the head; sGTCS, Secondarily generalized tonic clonic seizures; Som. aura, Somatosensory aura; Slowing b.a., Slowing of background activity with an unilateral absence of alpha-rhythm; Reg. slowing, Regional slowing contralateral to the paresis; Reg. S-W.C., Regional bursts of spike/polyspike and wave complexes; Diff. S-W.C., Diffuse bursts of spike-wave complexes; TBRSE, tick-borne Russian spring-summer encephalitis.

lower extremities and hemibody (two cases each). It was most pronounced in the distal parts of the extremities. In one case, myoclonus spread from the hand to the arm, neck and body. Facial myoclonus involved the angle of the mouth, cheek, and m. orbicularis oculi (Fig. 1), sometimes producing dysarthria. Sensory stimuli and emotional state modified the myoclonus which increased with touch, at movement and after awakening, prevailing in the face and upper extremities. All patients in addition to EPC had focal seizures which are listed in Table 1.

Neurological findings

All patients presented neurological deficits which sometimes improved over time (Table 1). In all, the neurological signs and symptoms were enhanced in periods of increased seizure activity.

MRI findings

The typical finding was moderate diffuse cortico-subcortical atrophy and ventricle enlargement, mainly of the lateral ventricles. In spite of the largely unilateral clinical presentation, the MRI findings were moderately asymmetric in only one case, and MRI was normal in three cases. In three cases, brain MRI was repeated after intervals of



Figure 1. Patient N.S., 8 years. Epilepsia partialis continua after Russian encephalitis. Marked myoclonus of angle of the mouth, cheek, and m. orbicularis oculi.

6 months to 4 years. Only in one case an increase of atrophy was found. In one, the periventricular leukopathy in the temporo-occipital region became less pronounced, only slight subatrophy in the posterior part of lateral ventricles without focal abnormalities was left.

Prolonged video-EEG monitoring

Slowing of background activity and unilateral absence of alpha-rhythm (contralateral to the paresis) was found in four cases (with an early onset of disease). Six patients had normal background activity. All patients had epileptiform activity: continued regional bursts of spike/poly-spike and waves in eight, isolated sharp waves and spike-and-wave and polyspike-and-wave complexes in another two cases. Continuous regional epileptiform discharges during sleep were revealed in eight cases. In sleep epileptiform activity was less prominent. Combination of diffuse and regional epileptiform activity was found in two of 10 patients (with an early onset), presented by poorly synchronized short bursts of spike-and-wave complexes at a frequency of 3 Hz indicating secondary bilateral synchronization (Table 1).

Typical localization of regional epileptiform discharges (eight cases) was in the fronto-central or fronto-centro-temporal region (Fig. 2). In one case epileptiform discharges were registered in the frontal region and in another one in the centro-temporo-parietal region with phase reversal under the parietal electrode. Focal (or regional) slow activity also was ascertained in all cases: Periodic regional theta-slowing contralateral to the paresis in six and continuous slowing contralateral to the paresis in four. Regional slowing with epileptiform activity in the fronto-centro-temporal areas presented the most typical EEG pattern in our cases.

In all cases EMG has been registered during seizures synchronously with video-EEG. Myoclonus was found in all 10 cases, focal motor seizures revealed in two cases. Direct correlation between focal myoclonus and epileptiform activity was not found. In three cases, the myoclonic jerks did not coincide with epileptiform activity on EEG at all and in four they incidentally correlated with epileptiform activity on EEG. From those, in two cases massive focal myoclonus evolved to focal motor clonic-tonic seizures. This evolution correlated on the EEG with rhythmical regional polyspike and wave activity or groups of sharp waves. Likewise in the interictal period, the initiation of the burst was in the fronto-centro-temporal regions with subsequent spread to the entire hemisphere (Fig. 3).

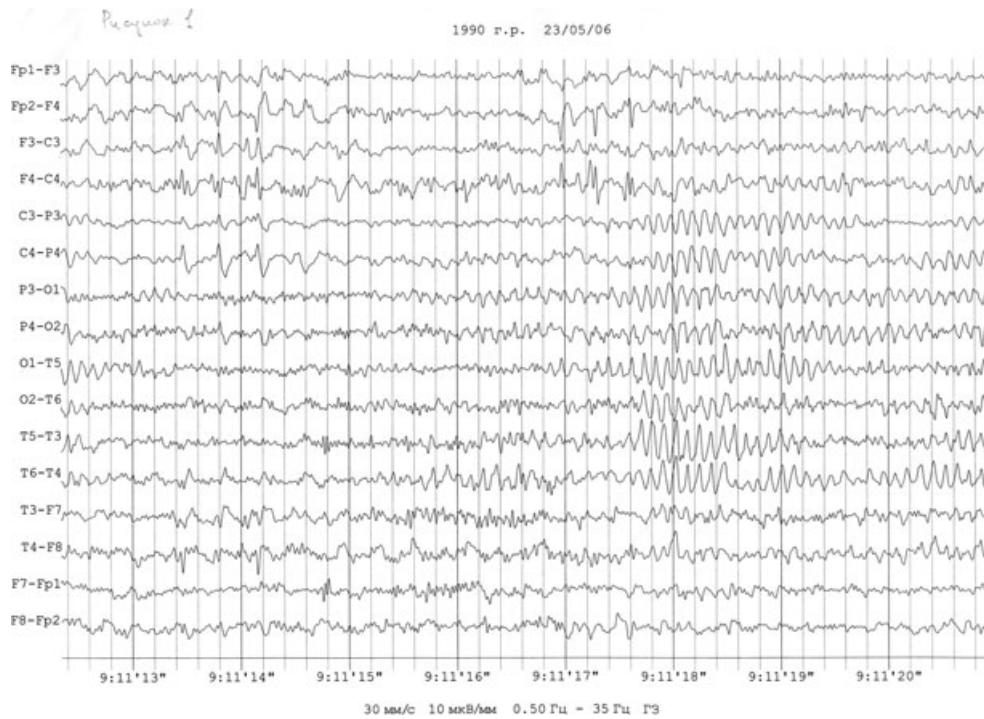


Figure 2. Patient O.A., 16 years. Epilepsia partialis continua after Russian encephalitis. Interictal continuous regional epileptiform discharges over the right hemisphere, maximally involving the right fronto-central regions.

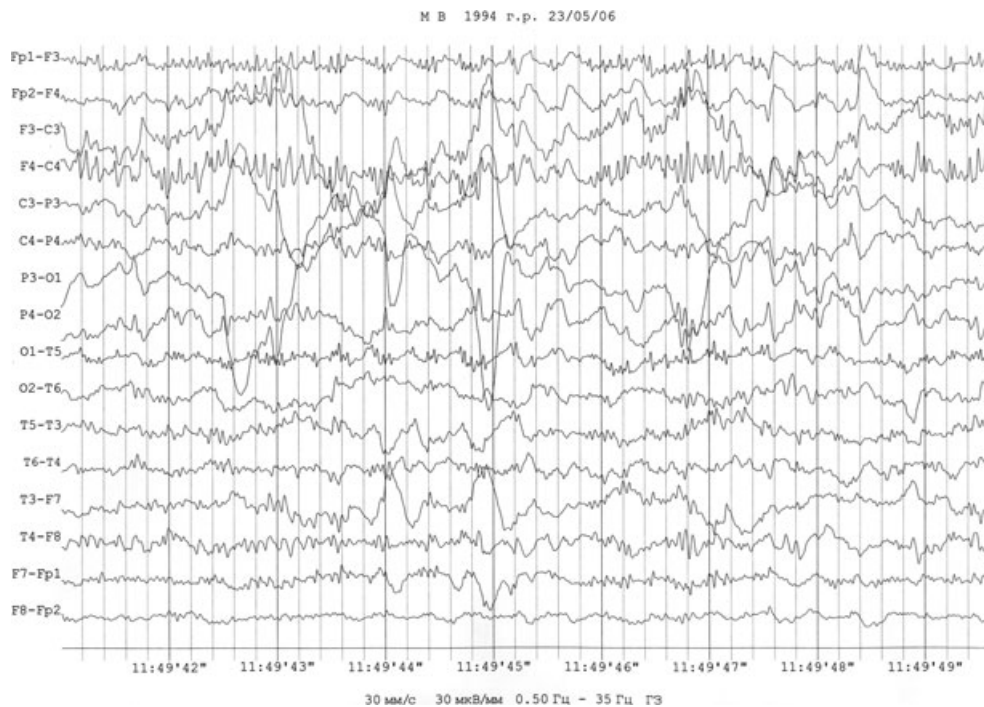


Figure 3. Patient O.A., 13 years. Epilepsia partialis continua after Russian encephalitis. A focal myoclonus was transformed to focal motor clonic-tonic seizures. during video-EEG monitoring: myoclonus (in the left arm), then rhythmic clonic movements, then tonic contraction. Ictal EEG-pattern: rhythmic regional polyspike-wave complexes, groups of sharp waves maximally involving the right fronto-central regions.

Therapy

The effect of antiepileptic drugs (see Table 1) differed according to seizure types. The sGTCS in all cases were controlled by monotherapy with phenobarbital, carbamazepine or topiramate. Focal motor seizures were completely stopped in nine of 10 patients with mono- and duotherapy of phenobarbital, carbamazepine, topiramate, levetiracetam and valproic acid. Drug treatment improved the EPC in all cases but never fully controlled it. The best effect was obtained with benzodiazepines and valproic acid. Levetiracetam and topiramate were less effective. Aggravation of the myoclonus was observed in one patient with phenobarbital and in another with lamotrigine.

Discussion

Omorokov in 1922 (5) reported 84 cases of EPC from rural parts of Western Siberia, noting seasonal prevalence in the spring and summer. High fever, headache and vomiting often preceded the neurological symptoms.

Shubin in 1938 (6) first proposed a correlation of EPC with TBRSSSE, Propper-Graschenkov in 1941 (7) started virological investigation of EPC, and Chumakov in 1944 proved that EPC develops in patients with TBRSSSE. He infected laboratory mice with this virus from brain tissue of patients with EPC (8).

Epilepsia partialis continua caused by TBRSSSE is a rather frequent condition in the Eastern parts of the Russian Federation (Ural, Siberia and Far East) where, according to Gulyaeva (9, 10), it is observed in ca. 1% of cases of TBRSSSE. West of

the Ural no such cases seem to have been reported. It may develop in both adults and children, and the clinical picture is not fundamentally different in the age groups. In the Russian literature, 98 cases of EPC and TBRSSSE were reported by Gulyaeva (9, 10), and seven cases of EPC and TBRSSSE by Mukhin et al. (11). The present paper reports a selection of these cases which could be studied with high resolution MRI and video-EEG monitoring. From these studies, EPC with TBRSSSE appears as a disabling but not fatal condition with a time course where three phases can be distinguished. In the first phase of acute meningoencephalitis which usually ends with minor neurological deficits, symptomatic epileptic seizures may be observed and rarely EPC. In the second phase EPC develops and individual seizures may continue. The neurological deficits remain unchanged or improve. In the third phase of chronic EPC no further progression is observed, concomitant focal seizures are often well-controlled, and neurological deficits remain stable or further improve.

It has been suggested by Omorokov (12) that the patients in Kozhevnikov's original description of 1894 also were cases of TBRSSSE. However, it clearly appears from Kozhevnikov's publication (1) that the clinical course in at least three of them was chronic progressive, Rasmussen syndrome thus being the more likely diagnosis. The clinical features and EEG characteristics of EPC in both conditions are identical. Otherwise these two kinds of encephalitis differ in many respects as summarized in Table 2, the clearest distinctors being the longterm clinical course and prognosis. The typical neuroimaging finding with Rasmussen encephalitis is progressive local atrophy, whereas TBRSSSE

Table 2 Comparison of EPC in TBRSSSE and in Rasmussen's encephalitis

Disease/symptoms	TBRSSSE	Rasmussen
Geography region	Endemic in Ural, Siberia, Far East Russia	Ubiquitous
Typical onset	Some months after acute encephalitis or after tick bite	Some weeks after intercurrent infectious disease or no apparent event
Age of onset	Any, usually up to 16 years	Usually 1–14 (peak is 3–10). Rarely in adulthood
Epileptic seizures	Myoclonus, focal motor and secondarily GTC	Myoclonus, focal motor and secondary generalized, complex partial, isolated aura; high frequency of status epilepticus
Neurologic symptoms	Static: Paresis, atrophy of muscles of limbs, symptom 'a hanging down neck'	Progressive: Hemiparesis, cognitive disorders: hemianopsia, visual agnosia, astereognosis, dysphasia. Neuroendocrinologic disorders
Hyperkinesias	Myoclonus, athetosis, tremor, torsion dyskinesia	Not typical
MRI	Cortical-subcortical atrophy with secondary (asymmetric) ventricle dilatation, non-progressive.	Localized atrophy of the cortical perisylvian area with tendency to progress and extend on the nearby areas
Laboratory Studies (CSF)	High virus antibody titers for TBRSSSE	Possible high antibody titers for glutamate receptors
Course and prognosis	Initial increase of symptoms over some years, then stabilization, then incomplete regression of symptoms. Severe disability. Lethal outcome extremely rare	Steady slow progress leading to severe disability. Possible lethal outcome several years after onset

EPC, epilepsia partialis continua; TBRSSSE, tick-borne Russian spring-summer encephalitis.

typically shows non-progressing local atrophy mainly in the perisylvian region together with diffuse atrophic changes which as a rule are asymmetric, but stable. This was also confirmed by Gulyaeva (9, 10) who followed 10 cases with CT\MRI for periods up to 40 years and found no progressive cortical atrophy.

Like with other etiologies (3), EPC in TBRSE is rather resistant to antiepileptic drugs whereas the concomitant individual seizures are much more responsive. No drug stands out as particularly useful with this etiology.

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Conflict of interest

None of the authors has a conflict of interest to declare.

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