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Temporal intermittent delta activity: A marker of juvenile absence epilepsy?

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ABSTRACT

Purpose: To report three cases of juvenile absence epilepsy (JAE) with temporal intermittent, asynchronous delta activity over the temporal regions.
Methods: Long term video-EEG using the international 10/20 system and supplementary anterior-inferior temporal electrodes. Cohort of 1123 patients included in our active file seen at least one time over one year.
Results: Among 23 patients with JAE (2% of our active file), temporal intermittent rhythmic delta activity (TIRDA) was observed in 3 (13%). Moreover, this activity was never observed in 80 patients with juvenile myoclonic epilepsy. None of the three patients had inadequate antiepileptic drug for idiopathic generalized epilepsy. Case 1 had no antiepileptic drug. Case 2 was treated with valproate (1000 mg/day) and case 3 with levetiracetam (1500 mg/day). These delta activities were activated by hyperventilation and drowsiness. They decreased in NREM sleep and reappeared in REM sleep. The frequency was around 3 Hz. These changes were not frequently recorded in any given patient.
Conclusion: The presence of TIRDA in the clinical and EEG context is very suggestive of JAE as posterior delta waves are of childhood absence epilepsy but with a more anterior location over the temporal lobe. This pattern was not described before probably because in this easily diagnosed and treated type of IGE, few patients have long-term video-EEG and also because a wrong diagnosis of focal epilepsy can be made. This pattern must be known to avoid the risk of treating this epilepsy by inappropriate antiepileptic drugs.

1. Introduction

In idiopathic generalized epilepsies (IGE), the EEG shows normal background with fast generalized or diffuse spike-waves or polyspike-waves with anterior predominance. Spike-waves may be regular at 3 Hz, as in childhood absence epilepsies. In addition, some children exhibit posterior delta activity, generally at 3 Hz over the occipital or the parieto-occipital regions.[1] They are activated by hyperventilation. They may be asymmetric. Temporal intermittent rhythmic delta activity (TIRDA) is considered a marker of temporal lobe epilepsy.[2] The aim of our study is to point out the importance and significance of such pattern for JAE in order to avoid misdiagnosis such as of focal epilepsy. In this study we report three cases of JAE patients presenting intermittent, asynchronous delta activity over the temporal regions. This pattern has never been previously described in patients with JAE.

2. Methods

Assessment of patients with JAE who underwent long term video-EEG recordings. An elastic cap fitted with embedded electrodes is used during nap video-EEG. Whereas, electrodes are glued directly onto the scalp during 24-h video-EEG recordings. Supplementary anterior-inferior temporal electrodes are systematically added to the 10–20 system. T1 and T2 are placed 1 cm above and one third of the distance along the line from the external auditory meatus to the lateral canthus of the eye. TA1 and TA2 (temporal-anterior) are placed 1 cm above and two third of the same distance anterior to the auditory canal.[3] Polygraphy with deltoid muscles and chin are systematically used during 24-h EEG recordings. Nap EEGs are performed shortly after lunch following partial sleep deprivation the night before (adults: must sleep 5 h at most). As eye movements are particularly detectable at TA2 and TA1, due to eye proximity, we do not use ocular electrodes in order to determine REM sleep. No sleep inducing medication was used.

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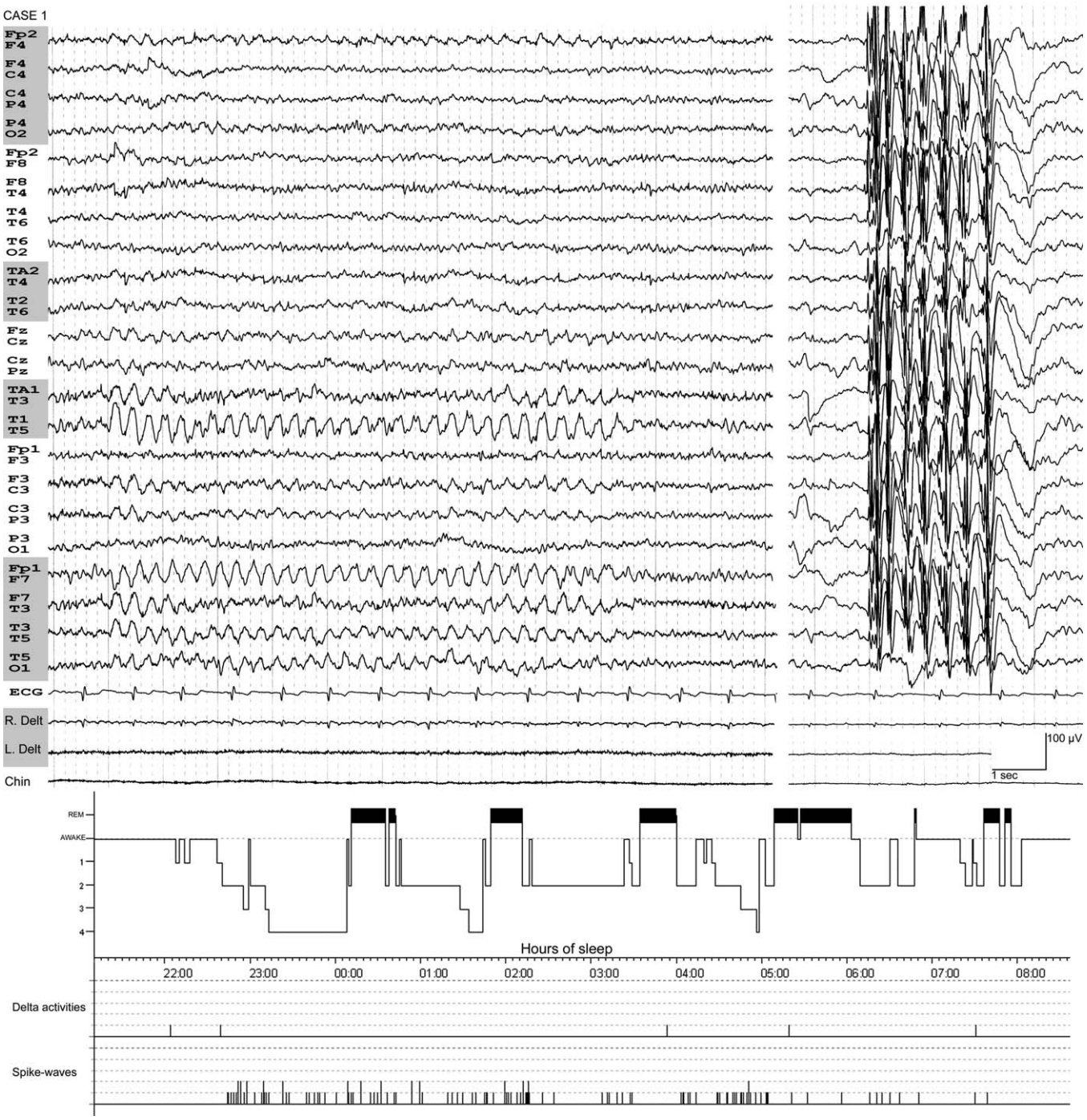

Fig. 1. Case 1: EEG with international 10–20 electrode system with supplementary Anterior/inferior temporal electrodes (TA1, T1, TA2, T2); right deltoid; left deltoid, chin. On the left abstract, delta activities over the left temporal region during drowsiness. On the right abstract, discharge of polyspike-waves during NREM sleep. Hypnogram with delta activities and polyspike waves.

3. Results

In our active file of 1123 patients seen at least one time over one year (April 2009–2010), we identified 23 patients with JAE (2%). In three of them (13%), a peculiar EEG pattern characterized by temporal intermittent delta waves was observed during long term video-EEG. In our centre, specialised in epilepsy, this pattern was never observed in other form of IGE, e.g. juvenile myoclonic epilepsy (80 patients among 1123 of our active file). None of the three patients had inadequate antiepileptic drug for IGE and patient 1 had no treatment. These delta activities were activated by hyperventilation and drowsiness. They decreased in NREM sleep and reappeared in REM sleep. They were not associated with clinical sign on the video and were not frequently recorded in any given patient. Fig. 1 shows that during a 10 h continuous EEG recording, there were only 5 episodes of lateralized temporal slow wave discharges, vs. numerous generalized spike-wave discharges. However, contrary to case 2 and 3, the episodes were quite rare in this patient. The observations of these three patients are presented below.

Case 1 is a 29-year-old man without a significant personal history and with a normal development who experienced his first generalized tonic–clonic seizure after a sleepless night and alcohol drinking. Typical but rare absences started at the adolescence. He

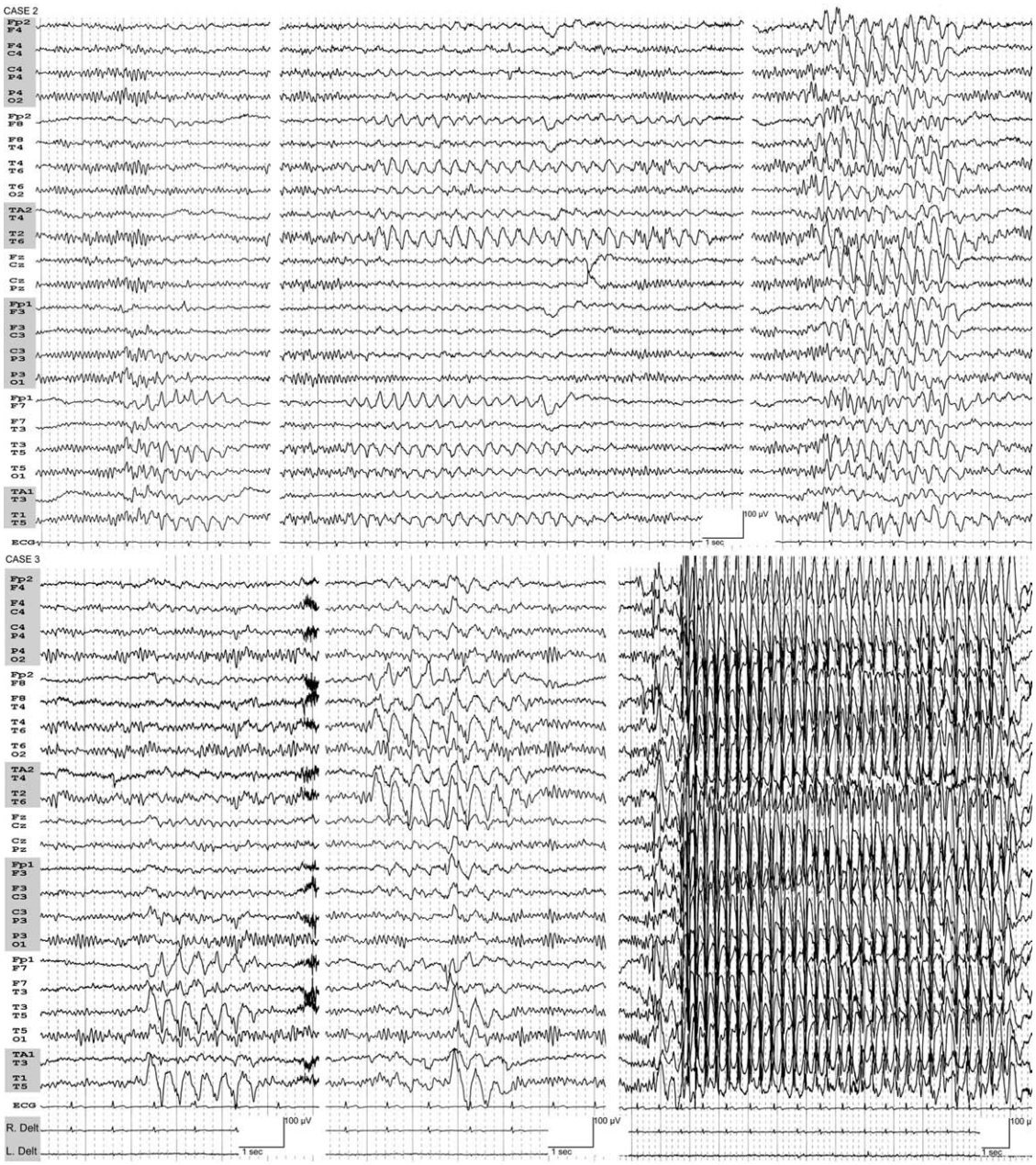

Fig. 2. International 10–20 electrode system with supplementary anterior/inferior temporal electrodes (TA1, T1, TA2, T2); right deltoid; left deltoid. Case 2: On the left, there are slow waves around 3 Hz on the left temporal region. In the middle, independent right and left delta waves. On the right, patient having awakened a few minutes before, there is a 3 Hz generalized spike-wave discharge. Note the end of the discharge with delta waves over the temporal regions with a similar aspect to the right and middle segment. Case 3: Delta waves over the left and right temporal regions. On the right, recording at 10 mm/s and 10 μV/mm, typical absence seizure.

had no myoclonic jerks. When he was evaluated, he had no treatment. The 24-h EEG demonstrated intermittent and asynchronous delta activities predominating over the temporal regions during drowsiness and REM sleep (Fig. 1). They were activated by hyperventilation. The age of onset, the seizure type, the polyspike-waves were suggestive of juvenile absence epilepsy.

Case 2 is a 38-year-old woman with no family history of epilepsy, referred for assessment of controlled epilepsy since 10 years on valproate 1000 mg/day. Her neurological examination and brain MRI were normal. Onset of epilepsy at age 11 with absences, then occurrence of rare myoclonic jerks and a few generalized tonic–clonic seizures during adolescence, in a context of sleep deprivation. Nap video-EEG demonstrated focal delta activity (Fig. 2) with a notched aspect at somnolence, during hyperventilation and at awakening. Analysis of sleep demonstrated rare polyspike-waves during NREM sleep but a discharge of spike-wave several minutes after awakening (Fig. 2). The end of this discharge was quite similar to the delta activities over the temporal regions. REM sleep was not obtained. The EEG and the clinical course were suggestive of juvenile absence epilepsy.

Case 3 is a 17-year-old man referred after two generalized tonic–clonic seizures. Onset of epilepsy at age 14 with typical absences. At this time, he was treated with valproate, quickly stopped after a skin allergy. Tiagabine was introduced and stopped because of a clear worsening of absences. Levetiracetam was started (1500 mg/day) but seizures were not controlled. A nap video-EEG demonstrated intermittent and asynchronous temporal delta activities during drowsiness and awakening (Fig. 2). They were activated by hyperventilation. These activities almost completely disappeared in NREM sleep and reappeared in

intermediate awakenings and in REM sleep. The NREM sleep was characterized by discharges of 3 Hz spike-waves and generalized polyspikes-waves in deeper stages. Typical absence seizure were recorded at somnolence, awakening and during hyperventilation (Fig. 2). Brain MRI was normal. The EEG and the clinical course were typical of JAE. Lamotrigine was introduced with a very slow titration to 50 mg/day with efficacy.

4. Discussion

JAE epilepsy differs from childhood absence epilepsy by a later age at onset (7–17 years, peaking at 10–12), by absences that are typically longer and less frequent, by the frequent association with generalized tonic–clonic seizures and by the possible occurrence of myoclonic jerks.[4] In JAE, contrary to childhood absence epilepsy, there are no 3 Hz posterior delta waves. The intermittent asynchronous temporal delta activity observed in our patients could represent a continuum with the delta activities observed in childhood absence epilepsy but with a temporal location rather than a parieto-occipital location. Concerning the frequency, one must note that it is around 3 Hz. In case 2, the end of the generalized spike-wave discharge is similar to the focal independent right and left temporal changes (Fig. 2). So these changes might constitute aborted absences.

Juvenile myoclonic epilepsy (JME) is close to JAE, as illustrated in case 2 with rare myoclonic jerks. Despite the higher frequency of JME (7% in our active file vs. 2% for JAE), we never detected this type of activity in JME. These changes were not frequently recorded in any given patient and were very rare in case 3. In our experience, the prevalence among JAE patients is 13% but all of our patients underwent long term video-EEG. This activity in JAE was not previously described because in this easily diagnosed and treated type of IGE, few patients have long-term video-EEG recordings and probably because a wrong diagnosis of focal epilepsy can be made. Indeed, TIRDA is an EEG pattern considered to be an indicator of temporal lobe epilepsy.[2] The description is quite similar to our cases with sinusoidal trains, ranging from 1 to 3.5 Hz. Our cases highlight the importance of the clinical background, to which the EEG findings must be correlated. The presence of TIRDA in the clinical and EEG context is very suggestive of JAE as posterior delta waves are of childhood absence epilepsy but with a more anterior location over the temporal lobe. This pattern must be known to avoid the risk of treating this epilepsy by inappropriate antiepileptic drugs.[5]

Conflicts of interest

None of the authors have any conflicts of interest to disclose.

References

1. Hirsch E, Panayiotopoulos CP. Childhood absence epilepsy end related syndromes. In: Roger J, Bureau M, Dravet Ch, Genton P, Tassinari CA, Wolf P, editors. *Epileptic syndromes in infancy, childhood and adolescence*. 4th edition. Montrouge: John Libbey Eurotext; 2005. p. 315–36.
2. Di Gennaro G, Quarato PP, Onorati P, Colazza GB, Mari F, Grammaldo LG, et al. Localizing significance of temporal intermittent rhythmic delta activity (TIRDA) in drug-resistant focal epilepsy. *Clin Neurophysiol* 2003;**114**:70–8.
3. Gelisse P, Crespel A. Slow alpha variant during REM sleep. *Neurophysiol Clin* 2008;**38**:3–8.
4. Wolf P, Inoue Y. Juvenile absence epilepsy. In: Roger J, Bureau M, Dravet Ch, Genton P, Tassinari CA, Wolf P, editors. *Epileptic syndromes in infancy, childhood and adolescence*. 4th edition. Montrouge: John Libbey Eurotext; 2005. p. 363–6.
5. Thomas P, Valton L, Genton P. Absence and myoclonic status epilepticus precipitated by antiepileptic drugs in idiopathic generalized epilepsy. *Brain* 2006;**129**:1281–92.