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SLEEP MEDICINE PEARLS

An Unexpected Abnormality on the EEG

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A 59-year-old male was referred for a sleep study because of observed hypersomnolence during a medical evaluation for kidney transplant.

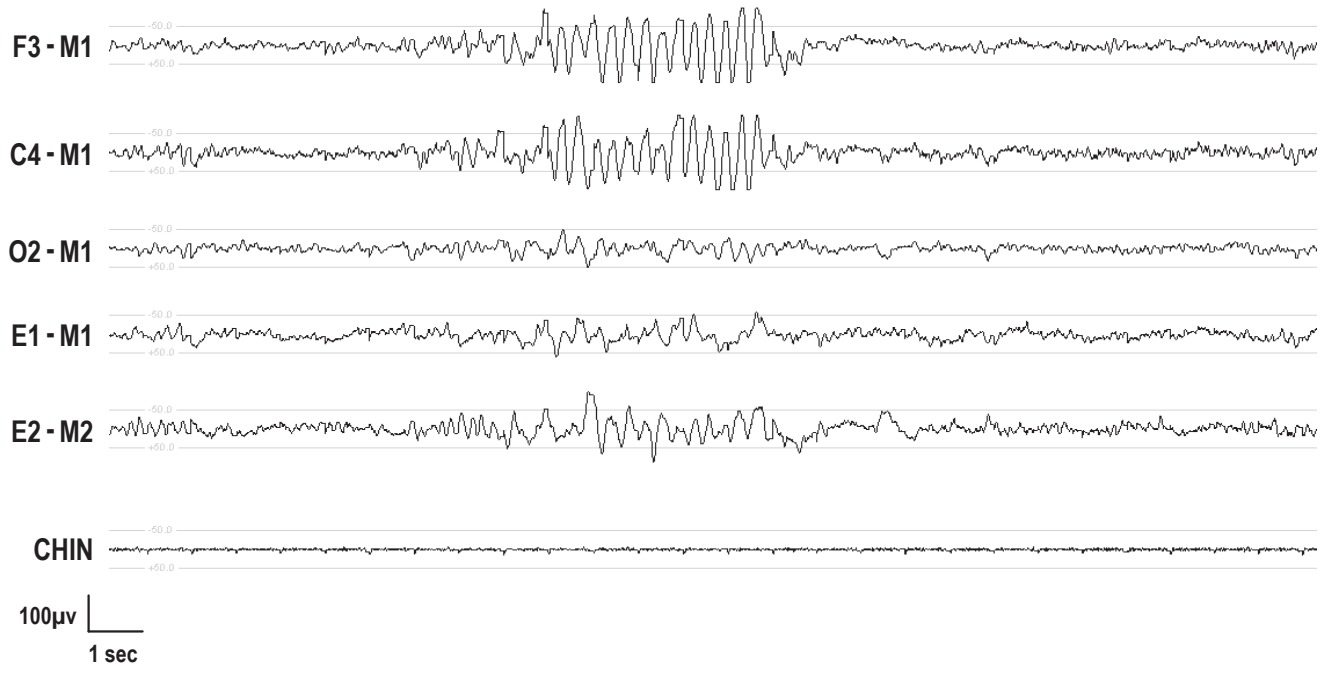
His past medical history was significant for a 30-year history of insulin dependent diabetes. He was known to have many secondary macrovascular and microvascular complications. These included a history of myocardial infarction, microangiopathic disease, symptomatic peripheral vascular disease, and proliferative retinopathy. He also had end-stage renal disease, treated with intermittent hemodialysis 3 times per week for the past 5 years.

He had a 10-year history of obstructive sleep apnea and was on CPAP at 10 cm H_20. However, he had not had a formal reevaluation of the efficacy of his CPAP therapy in over 6 years. There was also a history of daytime hypersomnolence, for which the patient was on modafinil. Other medications included diltiazem, atorvastatin, aspirin, atenolol, ramipril, omeprazole, and insulin.

On physical examination he had a body mass index of 33.6 and Mallampati score of 3 on oropharyngeal examination. Neurological examination was unremarkable.

Q: What does the EEG tracing (Figure 1) show?

Figure 1—A 30-second epoch tracing of polysomnography recorded during wake



The EEG and EOG filter settings are 0.3 Hz to 0.35 Hz. The EMG filter settings are 10 Hz to 100 Hz. F3-M1 refers to Frontal electroencephalogram lead; C4-M1, Central electroencephalogram lead; O2-M1, Occipital electroencephalogram lead; E1-M1, right electrooculogram; E2-M2, left electrooculogram; CHIN, chin electromyogram.

A: Frontal intermittent rhythmic delta activity (FIRDA)

Frontal intermittent rhythmic delta activity (FIRDA) was originally described by Cobb in 1945.[1] However, it was Van der Drift and Magnus who coined the term FIRDA in 1959.[2]

FIRDA is a rhythmic 2 to 3 Hz delta frequency activity with an amplitude of 50–100 mv that predominates in the bilateral frontal regions of the waking adult electroencephalogram (EEG). The waves are usually regular with a sinusoidal pattern. FIRDA usually occurs in short bursts lasting 2 to 6 seconds and must be differentiated from slow eye blink artifact **Figure 2**. The electrooculogram (EOG) electrodes can aid in the differentiation of the two. FIRDA, unlike eye blink artifact, may have posterior field extension. It is attenuated by alerting or eye opening and accentuated by eye closure, hyperventilation, drowsiness, and stage N1 sleep. It disappears with the onset of Stage N2 sleep but may reappear during REM sleep. If diagnostic uncertainty is present, a full EEG montage should be used and the EEG recording should include 20 minutes during wakefulness, followed by 3 minutes of hyperventilation and standard intermittent light stimulation to adequately document FIRDA.

The exact neurophysiological basis of the FIRDA rhythm is unclear. When first described, it was attributed to deep midline and posterior fossa pathology.[3,4] Subsequently Fariello et al. demonstrated that hemispheric brain tumors, ischemic brain injury, or metabolic derangements were the most likely culprits.[5]

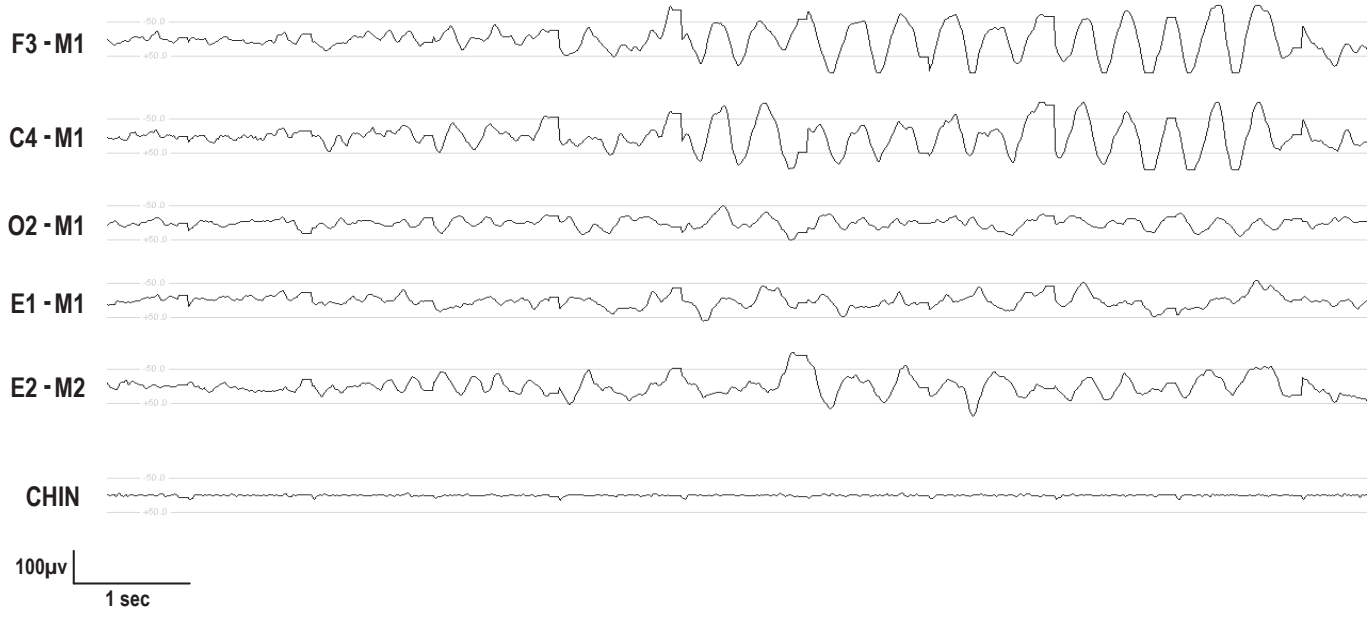
As a variety of pathological processes result in FIRDA, it is a nonspecific finding. In particular, increased intracranial pressure of any cause, tumors, and systemic toxic and metabolic disorders including hyperglycemia, and renal and hepatic failure may be responsible. There is no association between FIRDA and seizures.

In patients with chronic renal disease, FIRDA was initially described in those with progressive dialytic encephalopathy. Due to the elimination of aluminium-containing dialysate, dialysate encephalopathy is now rare. However, cognitive impairment occurs in up to 70% of individuals over the age of 55 years with chronic renal failure. This is frequently associated with cerebrovascular disease.[6] Furthermore, encephalopathy due to uremia, fluid and electrolyte disturbances, hypertension, or drug toxicity is common.[7] Presentations may vary from mild sensorial clouding to delirium and coma. Radiological studies of correlates of FIRDA have demonstrated that periventricular white matter disease and cortical atrophy were the most prevalent findings.[8] Furthermore, a recent retrospective study of patients in whom FIRDA was found on the EEG tracings showed that 50% had acute renal failure on admission to hospital and 33% had hyperglycemia.[8]

Although, usually described in adults, Watenberg et al. have shown FIRDA to be present in 1.3% of pediatric EEGs.[9] In contrast to the adult patients, no acute encephalopathy was evident clinically in these subjects. However, 50% of the children were cognitively impaired and 50% had epilepsy. Their EEGs had concomitant epileptiform discharges in 55%.

Less commonly, intermittent rhythmic delta activity may also occur in other EEG locations, namely occipital and temporal. Occipital intermittent rhythmic delta activity (OIRDA) was

Figure 2—A 10-second epoch tracing of polysomnography recorded during wake demonstrates intermittent rhythmic delta activity



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originally considered an equivalent of FIRDA in children. The difference in location was considered a result of maturational and developmental factors. However, more recent reviews suggest that unlike FIRDA, OIRDA is strongly associated with seizures in children and not with encephalopathy.[10]

Temporal intermittent rhythmic delta activity (TIRDA) raises concern for temporal lobe epilepsy and is usually unilateral. It differs markedly from both OIRDA and FIRDA in that it necessarily implies a focal rather than global lesion.[11]

Although the exact pathophysiological significance of FIRDA is uncertain, in otherwise normal individuals a search for underlying pathology should be undertaken.

In this patient, the presence of FIRDA was probably multifactorial. He had known microangiopathic disease, renal failure, and inadequately controlled diabetes mellitus and obstructive sleep apnea at the time of the study. On a subsequent titration study with CPAP no further episodes of FIRDA were observed. There are no cases of FIRDA in association with OSA in the literature; in view of this patient's multiple comorbidities, it is unlikely that the FIRDA is a result of this.

CLINICAL PEARLS FOR FIRDA

- It is a rhythmic high voltage delta activity at 2–3 Hz
- The rhythm is intermittent and occurs in bursts lasting 2 to 6 seconds
- Occurs in the waking and drowsy adult EEG in the frontal regions
- May occur with
  - Metabolic encephalopathies
  - Toxic encephalopathies
  - Increased intracranial pressure
  - Deep structural abnormalities
- Pathophysiological significance uncertain

REFERENCES

1 Cobb W. Rhythmic slow discharges in the electroencephalogram. *J Neurol Neurosurg Psychiatry* 1945;8:65
2 Van der Drift J, Magnus O. The value of the EEG in the differential diagnosis of cases with cerebral lesion. *Electroencephalogr Clin Neurophysiol* 1959;11:733-46
3 Daly D, Whelan JL, Bickford RG, et al. The electroencephalogram in cases of tumors of the posterior fossa and third ventricle. *Electroencephalogr Clin Neurophysiol* 1953;5:203-16
4 Bagchi BK, Kooi KA. Electroencephalography and brain tumors. *Med Bull (Ann Arbor)* 1961;27:50-60
5 Fariello RG, Orrison W, Blanco G, et al. Neuroradiological correlates of frontally predominant intermittent rhythmic delta activity (FIRDA). *Electroencephalogr Clin Neurophysiol* 1982;54:194-202
6 Murray AM. Cognitive impairment in the aging dialysis and chronic kidney disease populations: an occult burden. *Adv Chronic Kidney Dis* 2008;15:123-32
7 Brouns R, De Deyn PP. Neurological complications in renal failure: a review. *Clin Neurol Neurosurg* 2004;107:1-16
8 Watemberg N, Alehan F, Dabby R, et al. Clinical and radiologic correlates of frontal intermittent rhythmic delta activity. *J Clin Neurophysiol* 2002;19:535-9
9 Watemberg N, Gandelman R, Neufeld MY, et al. Clinical correlates of frontal intermittent rhythmic delta activity in children. *J Child Neurol* 2003;18:525-9
10 Gullapalli D, Fountain NB. Clinical correlation of occipital intermittent rhythmic delta activity. *J Clin Neurophysiol* 2003;20:35-41
11 Gambardella A, Gotman J, Cendes F, et al. Focal intermittent delta activity in patients with mesiotemporal atrophy: a reliable marker of the epileptogenic focus. *Epilepsia* 1995;36:122-9

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DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.