Possible Benign Partial Epilepsy

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I read with interest the recent case series in *Indian Pediatrics* [1]. I appreciate the efforts of the authors for publishing this under-reported epileptic syndrome in India. Through this communication, I wish to seek certain clarifications:

1. Seizure semiology of these patients was not included in the study. Semiology may be helpful to further classify these patients as benign partial epilepsy in infancy with complex partial seizures *versus* benign partial epilepsy in infancy with secondary generalized seizures (SGS). Though, proposal was made to combine these syndromes in the past, some subtle differences exist such as predominant seizures during wakefulness, and temporal ictal onset with the first entity, but mostly extratemporal seizure onset was noted with the latter.
2. Did they have a normal magnetic resonance imaging of brain? Focal cortical dysplasia is the most common cause of symptomatic focal epilepsy in infants and should be ruled out in these patients.
3. If any metabolic or genetic work-up was performed? Caution should be exercised to rule out inborn error of metabolism and chromosomal disorders, especially if differentiation between prolonged post-ictal state and underlying encephalopathy is difficult.
4. If any of these patients have gastroenteritis? The other entity with similar presentation is 'Benign convulsion with mild gastroenteritis', first described in Japan [2]; though rare in other countries, directed history of diarrhea should to taken in infants with clusters of seizures.
5. Though I agree with the authors' finding that recognition of this syndrome helps in avoiding long term anti-epileptic therapy and treatment with antiepileptic medication is not mandatory, benign nature of the condition is extremely difficult to ascertain during initial presentation; and rather than non-initiation of antiepileptic drugs, treatment for shorter time period may be justifiable. Though suspicion of this entity is possible to some extent, definite diagnosis can only be possible at age 5 years in presence of normal psychomotor development [3].

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Possible Benign Partial Epilepsy: Authors' Reply

We thank the author for his comments. An abbreviated description of these cases had been provided because these cases were part of a larger study, which is under publication. The purpose of the report was to highlight this relatively under-reported entity [1]. Our responses follow:

1. We agree with the author's contention. Two of the cases had secondary generalization, as mentioned in the original article also [1].
2. Three infants had a normal MRI brain (1.5T), whereas, due to financial constraints, one child underwent a non-contrast CT head. Normal neuroimaging had been mentioned in the original article [1].
3. We are unclear about what the authors mean by a metabolic and a genetic work-up. There is no single metabolic/genetic panel that may be ordered in all children with seizures. We followed standard guidelines for evaluation; metabolic profile to rule out inborn errors of metabolism was done only when indicated on the basis of history or examination findings, or results of other investigations. Genetic testing was only done, if there was a suspicion of a disorder on the basis of dysmorphology, seizure semiology, family history, and associated clinical findings. Otherwise, genetic testing is likely to be a low-yield strategy.
4. We agree that we should take a directed history of diarrhea in infants with clusters of seizures. None of these had such a history. In fact, we have previously

reported one patient with Benign infantile seizures with mild gastroenteritis, who was diagnosed during the same study [2].

5. We agree that a definite diagnosis may only be possible later; however, most of the literature is still of the view that treatment with anti-epileptic drugs is not mandatory [3]. In fact, in the study referred to by the author [4], the definite diagnosis could be made for more than three-fourth of those initially diagnosed as having 'possible' Benign partial epilepsy of infancy. The 'possible' terminology; however, has no scientific sanction.

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Horizontal Gaze Palsy: Additional Issues

I read with interest the article by Gautam, *et al*. [1]. I agree that brainstem tuberculoma can cause nuclear gaze palsy and fascinating neuro-ophthalmological findings. However, I think further clarification regarding pathophysiological mechanism of the gaze palsy may be interesting for the readers. Particularly, two points I want to raise are: difficulty in differentiating pathological lesions located in VI nerve nucleus and parapontine reticular formation (PPRF), and less usefulness of vestibule-ocular reflex to differentiate between lesions present in these two anatomical regions.

Gaze palsies – limited movement of two eyes in one direction – are caused by malfunction of one of the gaze centers located either in the cortical (premotor frontal cortex) or in PPRF located in the brainstem. Nuclear gaze palsy is caused by a lesion in the brainstem gaze center, whereas supranuclear gaze palsy is caused by a lesion in the cortical gaze center. Horizontal eye movements are initiated by the stimulation of PPRF from the contralateral premotor frontal cortex. PPRF, then, activates the ipsilateral lateral rectus muscle via VI nucleus and contralateral medial rectus muscle via contralateral medial longitudinal fasciculus (MLF). PPRF doesn't have a defined anatomical location but located anterior and lateral to the MLF and anterior to the VI nucleus. Excitatory burst neurons (EBNs) in the PPRF generate the "pulse" movement that initiates a horizontal saccade by providing input to the VI nucleus via axonal fibers.

Though lesions of the VI nucleus can produce paralysis of both the ipsilateral lateral rectus and contralateral medial rectus for all conjugate eye movements, clinical lesions that affect only the nucleus are rare, and there is usually involvement of adjacent structures such as PPRF as well. Because of proximity of location, right VI nucleus lesion likely associated with right PPRF lesion in this patient, causing right lateral rectus and left medial rectus weakness, appreciated as right lateral gaze palsy.

Doll's eye maneuver has been suggested as useful in differentiating among different types of horizontal gaze palsy as passive horizontal rotation of the head directly stimulates the sixth nerve nucleus via the vestibule-ocular reflex. In all practical purpose, it is more helpful to characterize gaze palsy between frontal lobe lesion versus nuclear and infranuclear lesion: gaze palsies induced by frontal lobe lesions will be corrected but gaze palsies caused by pontine nuclear and infranuclear lesions will persist during the maneuver. Though theoretically sixth nerve nucleus and PPRF lesion can be differentiated with this maneuver, controversy exists regarding clinical utility in this scenario [2], and in most situations combined lesions are seen due to close association of these areas with no defined boundary described for PPRF.

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