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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