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Frightening Episodes And Possible Brain Stem Inflammatory Lesion In A Child – A Case Of Peduncular Hallucinosis(ID-L3986)

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***Abstract**: A 5 year old female child presented with multiple brief (10-15 sec) frightening episodes 5 months duration without any impairment of consciousness or other stigmata of seizures. Her neurologic and psychiatric examination were normal. EEG was found to be normal. MRI revealed T2 and FLAIR hyperintensities in posterior pons, mid brain, bilateral cerebral peduncles, medial thalami, b/l caudate nuclei and left optic tract. No enhancement on contrast was noted. CSF analysis was found to be normal. The anatomical distribution of lesions allowed us to hypothesise that the frightening episodes are due to unreal visual hallucinations which are due to ARAS dysfunction leading to release phenomena. She was treated with IV steroids and has shown remarkable improvement.*

***Key Words**: Frightening episodes, brain stem hyperintensities, visual hallucinations, ARAS.*

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Visual hallucinations occur due to lesions of cortical visual centres, deafferentation of the visual system, lesions of RAS. Damage to reticular activating system due to lesions in brain stem lead to intrusion of dream like state into wakefulness and characteristically occur in PEDUNCULAR HALLUCINOSIS.

We report a rare case of a girl who developed frightening episodes most probably due to visual hallucinations because of possible inflammatory lesions in brainstem affecting ARAS.

I. Case Report

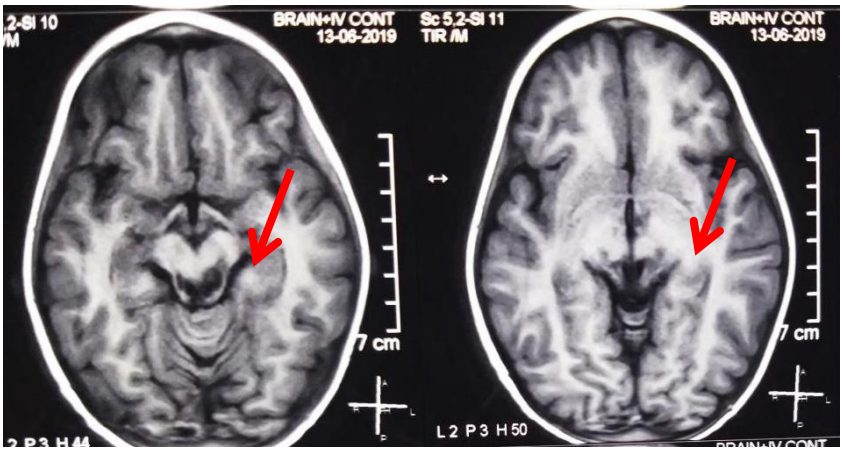
A 5 year old female born out of non consanguineous parentage was brought by her mother with frightening episodes of 5 months duration. Her mother says that she was apparently normal 5 months ago, but started having very brief i.e, 10-15 sec episodes of fearfulness. The child shouts that she is afraid and clings to her parents. There is no history of any clouding of consciousness, uprolling of eye balls, tonic posturing of limbs, tongue bite, autonomic manifestations or hand automatisms during the episodes and no h/o drowsiness after these episodes. Initially 3-4 episodes occurred per day but the no. of episodes have slowly increased to 10-15 / day with 4-5 episodes during night. Child used to sleep normally between the episodes during night. No h/o behavioural disturbances, motor, sensory, and cranial nerve disturbances.

Her general and neurological examination during and in between the episodes was found to be normal. Routine blood investigations were with in normal values. EEG recordings were normal. Brain MRI scans revealed lesions which are hypointense on T1 and hyperintense on T2 and FLAIR in posterior pons, mid brain, bilateral cerebral peduncles, medial thalami, b/l caudate nuclei and left optic tract. Part of the lesion in midbrain is showing facilitated diffusion. There is no enhancement with contrast. There was elevated choline and NAA peaks on MRS. CSF analysis showed 2 lymphocytes with 49mg/dl of glucose and 36 mg/dl of proteins.

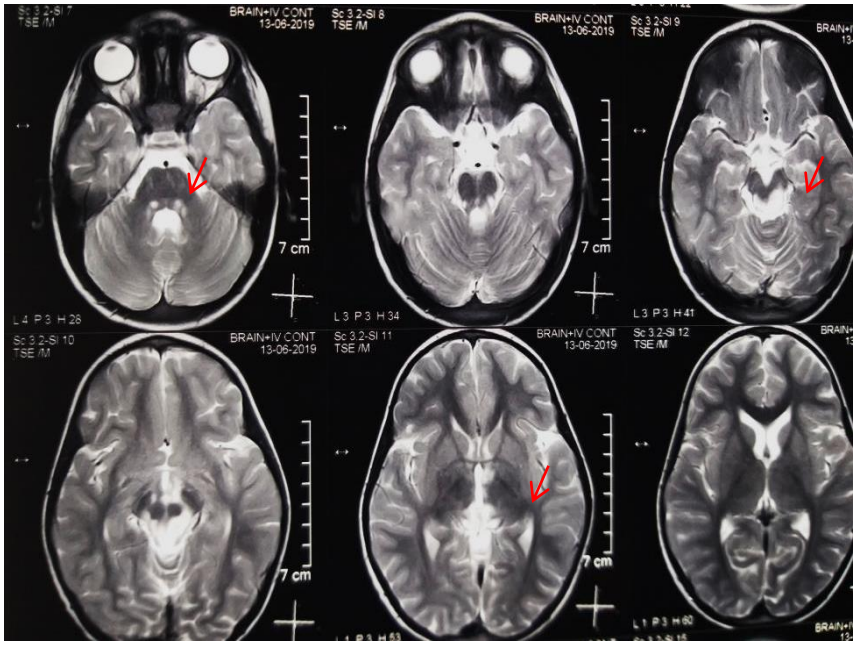
With this history, examination and investigations, we attributed the frightening to visual hallucinations which the child couldn't describe because of her younger age. These visual hallucinations were thought to arise from dysfunctional ascending reticular system and represents a release phenomena and is termed PEDUNCULAR HALLUCINOSIS.

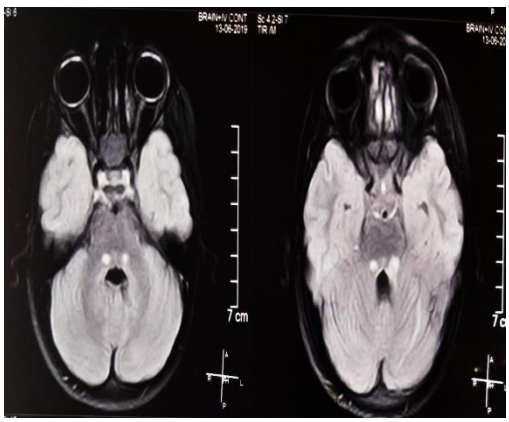
A definite diagnostic conclusion of the brain stem lesion was not possible. The differential diagnosis included demyelinating disorder, early granulomatous lesion disease, metabolic disease. Metabolic disease has been ruled out because of normal serum lactate , pyruvate and ammonia levels and normal wilsons disease profile. Normal CSF analysis rules out granulomatous lesions. Among these ,the most likely diagnosis was a transient demyelinating syndrome, definable as *clinically isolated syndrome* .

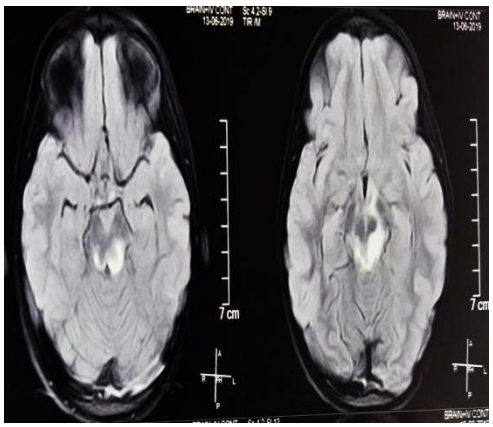
Child has been treated with IV steroids for 1 week followed by tapering doses of oral steroids. Resperidone was also started as advised by psychiatrist. There was remarkable improvement without any frightening episodes within 3 weeks after completion of treatment.

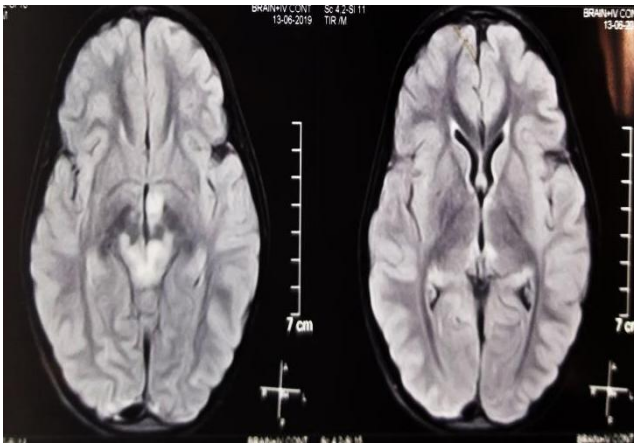


T1 image showing hypointense lesion in midbrain tectum and tegmentum and medial thalami.

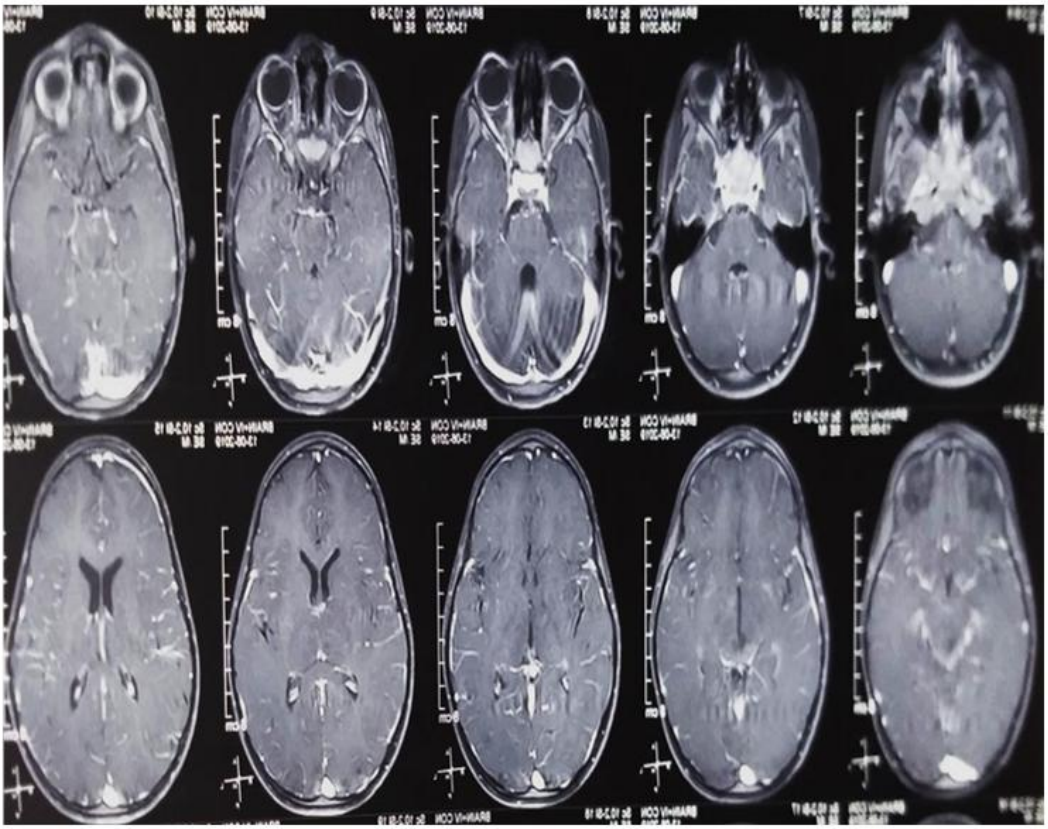








T2 and FLAIR images showing hyperintensities in pontine tectum and midbrain tectum and tegmentum, medial thalami.



Contrast images showing no contrast enhancement.

II. Discussion

Peduncular hallucinosis is characterized by formed, vivid, colorful, mostly visual, but also combined visual-acoustic or visual-tactile hallucinations due to lesions in brainstem and thalamus. In 1922, L'hermitte described the clinical syndrome of visual hallucinations in a 72 year old woman who developed headache, vomiting and vertigo, followed by left sided ptosis, opthalmoplegia and pyramidal and cerebellar signs, suggesting a lesion of the pons and midbrain.Von Bogaert coined the term 'peduncular hallucinosis'. These hallucinations may be highly coloured, mobile and are composed of concrete, often animate objects, sometimes distorted images of animals and people, scenes, lilliputian hallucinations. These are not stereotyped and vary from one occasion to the next, are more pronounced nocturnally and are often associated with disordered sleep. This condition has primarily been reported in single case reports, so information about associated symptoms is inconsistent.

The exact lesion and pathogenesis of peduncular hallucinosis are still unknown, although many cases involve vascular lesions in the midbrain or thalamus.

- Two common mechanisms have been suggested for pathogenesis:
- ✓ Imbalance between neurotransmitters in the reticular activating system (RAS),
- ✓ Disruption of the basal ganglia- temporal lobe loop

The RAS is composed of neuronal circuits connecting the brainstem to the cortex and is responsible for regulating arousal and sleep-wake cycles. Increased cholinergic transmission leads to REM sleep where as serotonergic firing inhibits REM sleep. Whenever there is RAS lesion, interruption to serotonergic inhibitory afferents in the dorsal raphe nucleus is suspended, resulting in an increase of PGO waves and thus an increase in REM sleep. Also it causes transient imbalance between REM-on and REM-off pontine circuitry. This could result in the intrusion of a dream-like state into wakefulness leading to hallucinations.

Another possible mechanism for peduncular hallucinosis involves a closed loop between the basal ganglia and the inferotemporal lobe. Middleton and Strick suggested that the inferotemporal lobe, which is responsible for recognition and discrimination of visual objects, may also be an output target for the basal ganglia. lesions of the substantia nigra and brainstem compression may result in visual hallucinations by blocking the stimulatory signal from the subthalamic nucleus to the substantia nigra, in turn decreasing the inhibitory signal to the thalamus and resulting in overactivity of the thalamus and the inferotemporal lobe.

Etiology of peduncular hallucinosis:

- Vascular stroke,
- Encephalitis,
- Intoxication,
- Following vertebral angiography
- Transient brain stem compression.
- Demyelination

✓ Other causes of visual hallucinations include schizophrenia, delirium, dementia with lewybody disease, occipital lobe and temporal lobe seizures, migrane, Charles bonnet syndrome, drugs like phencyclidine (PCP), ecstasy, atropine, and dopamine agonists ,mescaline, psilocybin, and lysergic acid diethylamide, cocaine and amphetamine, tumors that lie along, or compress, the optic path may cause visual hallucinations, Temporal lobe tumors, Occipital lobe tumors,inborn errors of metabolism kike Homocysteine remethylation defects,Urea cycle defects, GM_2 gangliosidosis, Neimann-Pick disease type C, α-mannosidosis, heidenhain variant of CJD.

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