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HEMICHOREA-HEMIBALLISMUS AS PRESENTING FEATURE IN ALCOHOL WITHDRAWAL SYNDROME- CASE REPORT

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ABSTRACT-

Basal ganglia are the main control station of controlling movements and fine tuning the act in co-ordination with motor field, cerebellum and thalamus.When choratic movements or ballistic movement present unilaterally it is called as hemichorea-hemiballismus, non ketotic hyperglycaemic hyperosmolar state is the most common cause of this HCHB syndrome. The syndrome is thought to be rare, however the exact epidemiology depends on the cause of HCHB. The syndrome is thought to be caused by a lesion in the basal ganglia, specifically, the subthalamic nucleus and caudate nucleus.

We reported a 58-year-old man with nondiabetes mellitus who presented with chief complaints of involuntary movement of left upper limb and left lower limb which was sudden onset and started about 1 day later when patient stop drinking alcohol, the movement involves both distal and proximal muscle group, distal movements are floating in nature present in wrist elbow and shoulder joint in upper limb, the shoulder joint movements are random jerky type which superimposed on floating movement, the same type of movement involves lt lower limb at ankle knee and hip joint with superimposed random jerky movement at hip joint, these movements are progressive and increased in frequency in 2-4 days.

The patient dramatically responded to tetrabenazine within a day. Subsequent dose reductions lead to a reemergence of symptoms.

Blood, CSF and radiological investigation were normal hence a diagnosis of transient basal ganglia dysfunction was made which was triggered by alcohol withdrawal.

***INTRODUCTION*:-**

Early identification, proper assessment and management of HCHB can lead to complete symptom relief.Chorea is an abnormal involuntary movement of upper and lower limb which are continuous floating from one joint to other. Balissmus is sudden jerky involuntary movement involving proximal muscle group of upper and lower limb. Basal ganglia are the main control station of controlling movements and fine tuning the act in co-ordination with motor field, cerebellum and thalamus.When choratic movements or ballistic movement present unilaterally it is called as hemichorea-hemiballismus, non ketotic hyperglycaemic hyperosmolar state is the most common but rare cause of this HCHB syndrome.

Alcohol is the major neurotoxin present world wide and usually disturb the cerebellar and basal ganglian functions, transient loss of basal ganglian function resulting in parkinsonism or chorea is a rare presentation of alcohol withdrawal syndrome as most of the patient with alcohol withdrawal presents with seizure or altered mental status. The symptoms completely resolved within one week of starting the treatment and the patient was kept on regular home and outpatient follow up for further monitoring. Acute palliative care (APC) approach deals with the management of comorbidities and their complications along with supportive care. Prompt assessment and management of such complications lead to better patient outcomes.

KEYWORDS- *chronic alcoholic, alcohol withdrawal syndrome, hemichorea-hemiballismus,case study.*

Epidemiology

The syndrome is thought to be rare, however the exact epidemiology depends on the cause of HCHB.

Notably, given hemichorea is the clinically milder movement disorder of the two, some patients are seen to transition from hemiballismus to hemichorea as they recover [1].

Pathology

The syndrome is thought to be caused by a lesion in the basal ganglia, specifically, the subthalamic nucleus (corpus Luysi) [1,2]. When this nucleus is damaged, there is reduced excitatory innervation to the internal segment of the globus pallidus, which results in increased inhibitory innervation to the thalamus,

which finally leads to increased excitatory innervation to the primary motor cortex [1]. This cascade of events leads to the development of the involuntary movements exhibited in this syndrome [1].

However, there are many reports, perhaps even a majority of reports, of this syndrome developing in patients with lesions not of the subthalamic nucleus [1,3]. These are thought to occur in regions of the brain with innervation to the subthalamic nucleus, of which there postulated to be many, however their individual exact roles in the development of this syndrome are yet to be elucidated [1,3].

Etiology

There are numerous etiologies potentially affecting the subthalamic nucleus (or other strategic areas) that have been implicated in this syndrome [1-5]:

- stroke: ischemic or hemorrhagic
- neoplasm
- vascular malformation
- demyelination
- non-ketotic hyperglycemia (when presenting with HCHB, is known as 'non-ketotic hyperglycemic hemichorea', or as 'diabetic striatopathy', or as 'chorea, hyperglycemia, basal ganglia syndrome')
- vasculitis (e.g. due to systemic lupus erythematosus)
- trauma
- infection:
- tuberculoma
- cerebral toxoplasmosis

Treatment and prognosis

The specific treatment and prognosis depend on the underlying etiology . Generally, symptomatic relief should be offered to patients significantly affected and who are unlikely to make a rapid recovery [1]. This symptomatic treatment may include medications such as typical neuroleptics or tetrabenazine, or neurosurgical intervention [1,5,6].

CASE DETAILS-

- A 58yr old non diabetic normotensive chronic alcoholic present with chief complaints of involuntary movement of left upper limb and left lower limb which was sudden onset and started about 1 day later when patient stop drinking alcohol, the movement involves both distal and proximal muscle group, distal movements are floating in nature present in wrist elbow and shoulder

joint in upper limb, the shoulder joint movements are random jerky type which superimposed on floating movement, the same type of movement involves lt lower limb at ankle knee and hip joint with superimposed random jerky movement at hip joint, these movements are progressive and increased in frequency in 2-4 days.

- They are aggravated by anxiety and as per history given by relatives subside during sleeping although pt has reduced sleep in the same period.
- There was no h/o fever/joint pain/palpitation/altered behavior/seizure/loss of consciousness
- O/E-

Thin built old age person,conscious and oriented to time place and person lying supine in bed with constant movement in lt upper and lower limb.

- General condition -fair ,afebrile ,
- pulse is 88/m regular BP-130/80mmhg
- RS- BLAE present with normal vescicular breath sound ,no added sound
- CVS-s1s2 heard no murmur,
- per abdomen is soft no organomegaly.
- CNS- pt is conscious oriented, speech and language normal, cranial nerves are normal
- No atrophy seen in b/l upper limb and lower limb muscle, mild hypotonicity in lt upper and lt lower limb, both UL and LL have normal power and all DTR are present, b/l planters are flexors

Coordination normal in rt side can not be assessed in lt side.

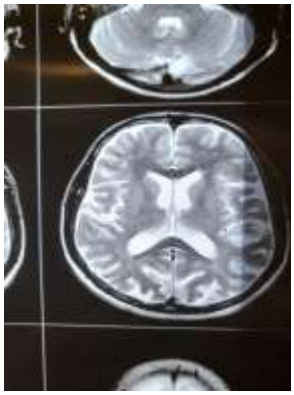
- **INVESTIGATION**-
- routine blood investigations including CBC, RFT,LFT ,
- electrolyte urine analysis was within normal limits,
- CSF- also normal,ecg-s/o left axis deviation with normal sinus rhythm,
- 2D echo- Normal chambers and LV function,
- degenerative aortic valve with mild aortic stenosis, no clot no vegetations
- MRI brain study was also normal.

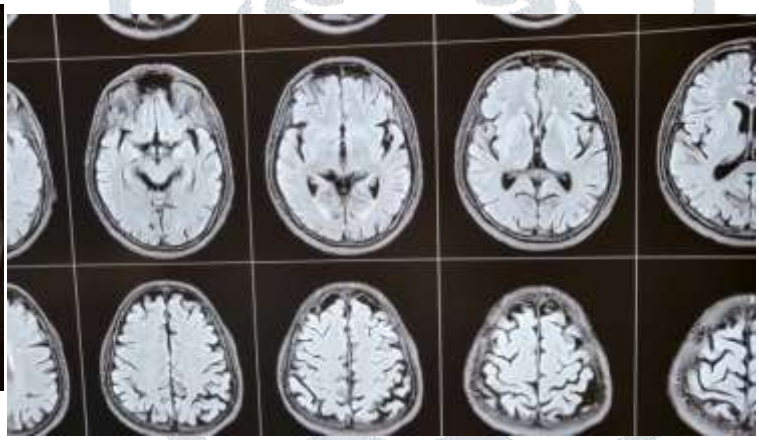
As the all blood, csf and radiological investigations were normal a diagnosis of transient basal ganglia dysfunction was made and patient started symptomatic treatment with-

- Tab valproic acid 500mg bid with
- Tab Tetrabenazine 25mg bid initially f/b 50mg bid,
- he also had one shot of IV haloperidol 5mg.

Patient responded well to treatment and his movements reduced to almost none in 3 days of therapy and was discharged with same treatment.

- DISCUSSION- Hemiballismus is characterized by high amplitude, violent, flinging and flailing movements confined to one side of body and hemichorea is characterized by involuntary random-appearing irregular movements that are rapid and non-patterned confined to one side of body.
    - As the investigations of the said patients were within normal limits, no organic cause was found hence a diagnosis of transient basal ganglian dysfunction with HCHB was made and patient given symptomatic medical management.
    - As the symptoms were started after patient stops drinking alcohol the correlation with alcohol withdrawal was made and as it is very rare presenting feature of alcohol withdrawal syndrome.
    - Hence a normoglycaemic patient with HCHB as a presenting complaints with normal radiological report a strong association of the symptoms with alcohol withdrawal can be thought and treated symptomatically.





SUMMARY: one chronic alcoholic patient developed hemichorea-hemiballismus involving the face, lips, tongue and, in one case, all limbs; 1 patient for the first time, 1 day after alcohol withdrawal. The patient dramatically responded to tetrabenazine within a day.These abnormalities improved spontaneously with maintained abstinence from alcohol for 1 to2 weeks. None had a family history of movement disorder, there was no history of other psychoactive drug use or abuse, and there was no evidence of portal-systemic encephalopathy. The symptoms completely resolved within one week of starting the treatment and the patient was kept on regular home and outpatient follow up for further monitoring. Prompt assessment and management of such complications lead to better patient outcomes.

REFERENCES-

May 01, 1990; 40 (5) **VIEWS & REVIEWS**

1]Movement disorders in alcoholism, A review

Jack Neiman, Anthony E. Lang, Luis Fornazzari, Peter L. Carlen

https://www.sciencedirect.com/science/article/abs/pii/S0733861918301439

https://tremorjournal.org/articles/10.5334/tohm.560/

2] Postuma RB, Lang AE. Hemiballism: revisiting a classic disorder. (2003) The Lancet. Neurology. 2 (11): 661-8. Pubmed

3] Compston A. Hemichorea resulting from a local lesion of the brain. (The syndrome of the body of Luys.) By James Purdon Martin, MD (London). Brain 1927: 50; 637–651; Hemichorea associated with a lesion of the corpus Luysii. By James Purdon Martin and N.S. Alcock. Brain 1934: 57; 504–516; and Hemichorea (hemiballismus) without lesions in the corpus Luysii. By J. Purdon Martin (From the National Hospital, Queen Square, W.C.1) Brain 1957: 80; 1–10. (2006) Brain. 129 (7): 1633. doi:10.1093/brain/awl168

4] Damani A, Ghoshal A, Salins N, Deodhar J, Muckaden MA. Management of hemichorea hemiballismus syndrome in an acute palliative care setting. (2015) Indian journal of palliative care. 21 (1): 72-5. doi:10.4103/0973-1075.150193 - Pubmed

5]Sanchez-Ramos JR, Factor SA, Weiner WJ, Marquez J. Hemichorea-hemiballismus associated with acquired immune deficiency syndrome and cerebral toxoplasmosis. (1989) Movement Disorders. 4 (3): 266. doi:10.1002/mds.870040308 - Pubmed

6]Umeh CC, Nichols P, Rosenthal LS, Mari Z. Dual treatment of hemichorea-hemiballismus syndrome with tetrabenazine and chemodenervation. (2012) Tremor and other hyperkinetic movements (New York, N.Y.). doi:10.7916/D86Q1VZD - Pubmed

7] Sitburana O, Ondo WG. Tetrabenazine for hyperglycemic-induced hemichorea–hemiballismus. (2006) Movement Disorders. 21 (11): 2023. doi:10.1002/mds.21100 - Pubmed