Athira Mohan S et al/J. Pharm. Sci. & Res. Vol. 14(5), 2022, 746-747

ISSN:0975-1459
Journal of Pharmaceutical Sciences and Research
www.jpsr.pharmainfo.in

A Case Report on Lentiform Nucleus involved Ischemic Hemiballismus

Athira Mohan S[1*], Anoop V Somarajan[1], Dr. Anusreeraj[2]

[1] *Pharm D intern, Ezhuthachan College of Pharmaceutical Sciences, Trivandrum, Kerala.*

[2]*Assistant Professor, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Trivandrum, Kerala.*

Abstract

Hemiballismus is a type of hyperkinetic movement disorder characterized by irregular, involuntary, high amplitude movements. Here we present a case of ischemic hemiballismus presented with acute violent flailing movements. Emergency CT brain revealed chronic infarct on the lentiform nucleus and small vessel ischemic changes. The patient was given escalating doses of haloperidol and underlying acute stroke was treated. The patient was followed up for a period of 2 months and a resolution of ballistic movement was observed.

Keywords: Hemiballismus, haloperidol, lentiform nuclei infarct, small vessel ischemic changes.

INTRODUCTION

Hemiballismus is a type of hyperkinetic movement disorder characterized by irregular, involuntary, high amplitude movements. Most often these movements are unilateral in nature [1]. The term 'Hemiballismus' was first described by Dr. J R Whittier in 1949, while studying the features of subthalamic nucleus of rhesus monkeys. He demonstrated that, hemiballismus would occur contralaterally if there is a minimum of 20% damage to the brain area [2]. This is a less common manifestation of stroke and is reported that it might be due to any lesion or ischemia in subthalamic nucleus and components of basal ganglia [1].

Literatures shows that the incidence of hemiballismus in ischemic stroke is 0.4% - 0.54% and prevalence is 1% [3]. Movement disorders show a higher degree involvement of lesions or infarcts in basal ganglia, internal capsule, thalamus, diencephalon and mesencephalon. Primary, supplementary, premotor cortical areas are cortical areas most commonly linked with movement disorders [4]. Here we present a case of hemiballismus resulting from an infarct in lentiform nucleus and with small vessel ischemic changes.

CASE REPORT

A 75-year-old female patient was admitted with complaints of abnormal movements of left upper and lower limb, with acute onset one day before on 02/07/2021. The patient reported that the abnormal involuntary movements were interfering with her routine activities, aggravated with anxiety and diminished during sleep. The foot, toes, knee of left lower limb, simultaneously with fingers, wrist, elbow and shoulder of left upper limb was affected. The frequency and intensity of the movement persisted the same since the onset. The head, eyes, mouth, face did not show any evidence of abnormal movement or deviation. There were no unusual talk or behavioral change. She was conscious and well responding. She was a known case of Type II diabetes mellitus (HbA1C: 7.6%) and hypertension (130/90 mmHg) on treatment, for 6 years.

On liaison it was understood that she had a similar event of abnormal movement episode in 2016, which was subsequently improved with proper glycemic control. Her regular medications include: T. METFORMIN 500mg OD, T. GLIMIPIRIDE 2 mg OD, T. ATORVASTATIN 10 mg HS, T. AMLODIPINE 5 mg BD, T. PANTOPRAZOLE 40 mg OD, T. RANITIDINE 150 mg BD. Physical examination showed no antecedent fever, trauma, ingestion of toxic drugs or substances. The patient had no social or family history. Her clinical examination revealed slight disorientation, ballistic flailing movement. The pulse rate was recorded as 88 beats/ minute with regular rhythm, BP was normal (130/90 mmHg). Gait was unstable. Higher psychic functions and cranial nerves was normal on monitoring. Reflexes and muscle were normal, sensation prompt and intact. Abdominal examination was unremarkable. Her heart sounds S1 and S2 was heard, and it was normal. Recent and remote memory was intact. Other examination was unremarkable.

TABLE 1: Biochemistry values at the time of hospitalization.

TEST	RESULT
Hemoglobin	10.2 g/dL
WBC	4900 cells/L
Neutrophils	77.6%
Lymphocytes	15.9%
Eosinophils	1.2%
Monocytes	4.9%
ESR	73 mm/hour.

Her hemogram analysis revealed hemoglobin 10.2 g/dL, elevated neutrophil count, elevated ESR, depleted lymphocytes **(TABLE 1)**. Her lipid profile, liver, kidney, thyroid functions and serum electrolytes was within normal limits.

An emergency CT brain was performed. Computed tomography scan was remarkable. It showed following features:

- ✓ Basal ganglia hyperdensity present.
- ✓ Small vessel ischemic changes.
- ✓ Chronic lacunar infarct in lentiform nucleus.
- ✓ Age related atrophic changes in the brain parenchyma.

CT brain revealed a hyperdense foci in the basal ganglia and the chronic infarct on the lentiform nucleus, hence diagnosis of hemiballismus due to chronic lacunar infarct was made **(FIGURE 1)**.

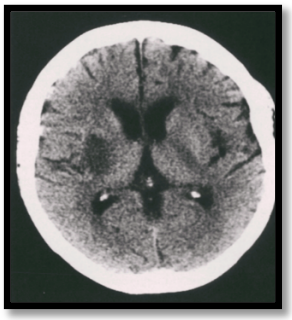

FIGURE 1: Hyperdense foci in the basal ganglia

The patient was given Inj. HALOPERIDOL IV stat 5 mg/mL at the time of admission. She was given IV fluids. Shifted to Tab. HALOPERIDOL 0.25 mg 2-2-2 for 2 days, followed by Tab. HALOPERIDOL 1.5 mg 1-1-2 next day, then dose escalated to 1.5 mg 2-2-2 for next 4 days. Since she had ischemic- infarct CNS features and her involuntary movement were thought to be secondary to an ischemic stroke, she was given T. CLOPIDOGREL 75 mg 0-1-0, T. GABAPENTIN 100 mg 1-0-1, T. SODIUM VALPROATE 200 mg 1-0-1 along with her own medications. T. RANITIDINE 150 mg was discontinued on the first day, since she was already taking PANTOPRAZOLE. Her blood glucose level was controlled with intake of T. GLIMIPIRIDE 2 mg 1-0-0, T. METFORMIN 500 mg 1-0-0 and administration of Inj. HUMAN ACTRAPID depending on blood glucose level. Her involuntary movements were decreased, and discharged home on 08/07/2021 continuing the medicines. Follow up was recorded for 2 months **(TABLE 2)**.

TABLE 2: Follow up and progression of patient.

FOLLOW UP	PROGRESS NOTES
After 10 days	Ballistic movements reduced.
After 2 months	No ballismus present, blood glucose controlled.
After 6 months	No further evidence of ballistic movements.

DISCUSSION

Hemiballismus is a rare kind of hyperkinetic movement disorder characterized by irregular, involuntary, high amplitude movements. This involves one side of the body, mostly the upper limb and lower limb [1,2,3,4]. The movement reduces upon rest and resolves upon rest. Lesions or infarcts in the subthalamic nucleus (STN) is a hallmark feature. These alterations in STN causes decreased activation of globus pallidus interna, leading to disinhibition of thalamus, causing violent flailing movements.

The risk factors associated are hyperglycemia, dyslipidemia, early stroke. Stroke remains the most common etiological factor. Others include recent head trauma, nonketotic hyperglycemia, ALS (Amyotrophic Lateral Sclerosis), neoplasms, tuberculomas, vasculitis, other infections like HIV, toxoplasmosis. Management includes managing hyperglycemia, lysing lesions, managing hypertension, ruling out other infections. Provision of good hydration enables to prevent complications like rhabdomyolysis and dehydration. Initiation of thrombolytic therapy for underlying acute stroke with hemiballismus is recommended. However, it is mandatory to rule out stroke mimics and give attention to inter-patient factors [6].

The differential diagnosis includes Huntington's disease, drug toxicity, hemorrhagic stroke, head trauma. This patient had no recent trauma to head, ho family history of Huntington's disease, no history of intake of medications like antipsychotics or antiepileptics. Here chronic lacunar infarct on lentiform nucleus, ischemic changes, intermittent hyperglycemia are probable causes of hemiballismus. Certain case reports say that hyperkinetic movements reversibly resolve with normalization of blood glucose level, reducing the size of infarct and has a good prognosis. Most cases of hemiballismus have a good outcome of movement resolution within days to weeks. Hemiballismus at initial stages can be injurious and thus requires prompt medical treatment. Neuroleptics, gabapentin, topiramate, valproic acid are medications to treat hemiballismus. Refractory patients who do not respond to existing medications require surgical methods like pallidotomy or thalamotomy or deep brain stimulation. Our patient was given haloperidol 0.5 mg given three times a day then titrated to 3 mg given three times a day during a period of one-week treatment. This led to a drastic recovery in ballistic movements.

The treatment with haloperidol was continued for next 2 months. Other case reports of hemiballismus with infarcts or lesions had a good prognosis. None of the cases reported were refractory to existing medical treatment.

CONCLUSION

We report a patient with chronic lacunar infarct in lentiform nucleus with small vessel ischemic changes that resolved with haloperidol treatment and blood glucose correction.

REFERENCES

1. Dewey RB, Jankovic J. Hemiballism-hemichorea: clinical and pharmacologic findings in 21 patients. Archives of Neurology. 1989 Aug 1;46(8):862-7.
2. Kötter R. Postsynaptic integration of glutamatergic and dopaminergic signals in the striatum. Progress in neurobiology. 1994 Oct 1;44(2):163-96.
3. Patel AR, Patel AR, Desai S. Acute hemiballismus as the presenting feature of parietal lobe infarction. Cureus. 2019 May;11(5).
4. Kwon DY. Movement disorders following cerebrovascular lesions: Etiology, treatment options and prognosis. Journal of movement disorders. 2016 May;9(2):63.
5. Padmanabhan S, Zagami AS, Poynten AM. A case of hemichorea-hemiballismus due to nonketotic hyperglycemia. Diabetes Care. 2013 Apr 1;36(4): e55-6.
6. Salins N. Time for change: Integrating palliative medicine to mainstream medicine. Indian journal of palliative care. 2014 May;20(2):97.
7. Lin JJ, Chang MK. Hemiballism-hemichorea and non-ketotic hyperglycaemia. Journal of Neurology, Neurosurgery & Psychiatry. 1994 Jun 1;57(6):748-50.