HEMICHOREA-HEMIBALLISM IN VARIOUS CONDITIONS: SERIAL CASE REPORTS

Neila Raisa¹, Sri Budhi Rianawati¹, Shahdevi Nandar Kurniawan¹, Fahimma¹, Mulika Ade Fitria Nikmahtustsani¹

Correspondence: neilaraisa@ub.ac.id

¹Department of Neurology Faculty of Medicine Brawijaya University, Malang, Indonesia.

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ABSTRACT :

Introduction: Hemichorea-hemiballism (HCHB) is an uncommon movement disorder involved unilateral extremities characterized by irregular, poorly patterned, a continual hyperkinetic involuntary movement disorder in the proximal or distal parts of the body. The acute development of HCHB depends on focal lesions on the contralateral basal ganglia and subthalamic nuclei. Various conditions such as cerebrovascular, neurodegenerative, neoplastic, immunologic, infectious, and metabolic diseases are known as secondary causes of HCHB. This paper aims to compare and discuss the HCHB in various etiologies.

Case Reports: Here, we reported 5 cases of HCHB induced by non-ketotic hyperosmolar hyperglycemia (NKKH), thrombotic stroke, and toxoplasmosis cerebral. We compare the admission data, clinical course, imaging, treatment, and outcome of every case.

Conclusion: Various hypotheses have been proposed to explain the pathophysiology of HCHB due to these conditions. Principally, the main management for these cases is to determine the etiology and correct the underlying disorder.

Keywords: Hemichorea-Hemiballism, Non-Ketotic Hyperosmolar Hyperglycemia, Thrombotic Stroke, Toxoplasmosis Cerebral

Introduction

Chorea-ballism is an uncommon movement disorder characterized by irregular, poorly patterned, a continual hyperkinetic involuntary movement disorder in the proximal or distal parts, mainly involved unilateral extremities and called as hemichorea-hemiballism, but sometimes bilateral. Various conditions such as cerebrovascular, neurodegenerative, neoplastic, immunologic, infectious, and metabolic diseases are known as secondary causes of chorea-ballism. Hemichorea-hemiballism (HCHB) is a rare case, even the prevalence in the population is not known clearly, but most cases have been described in individuals with Asian descent, females, and the elderly.^{1,2,3,4,5} Autoimmune diseases and infections often occur in HCHB at a young age, whereas in the cerebrovascular disease that has a history of hypertension and diabetes mellitus often occurs in older age.^{1,6}

The acute development of HCHB depends on focal lesions on the contralateral basal ganglia and subthalamic nuclei. Hyperkinetic movements caused by disruption of the indirect pathway that promote a loss of inhibition on the pallidum, and excessive dopaminergic activity. It is possible to understand HCHB as the manifestation of various abnormalities in brain.^{5,7} The most common cause of HCHB due to structural lesions is a cerebrovascular disease and the most common cause of metabolic disorders is non-ketotic hyperosmolar hyperglycemia.^{8,9} HCHB in various etiologies and characteristics is not widely reported.

We report 5 cases of HCHB induced by various medical abnormalities.

Case Reports

First Case

A 65-year-old woman with a history of hypertension and uncontrolled diabetes since a year ago came with the left-hand involuntary movement for two weeks before admitted to the hospital. Two weeks after, the involuntary movement progressed to her left leg. Patients felt jerking, writhing, and twisting in her left extremities and could not control the movement. The movements were painless and appear continuously and heavier when they are awake and doing some activities. However, these involuntary movements resolved during sleep.

We found involuntary and arrhythmic movements in her left extremities that appropriate as hemichorea-hemiballism-like-movements. The vital sign was stable and no neurological deficit was found. Laboratory test showed random blood glucose (RBS) level was 500 mg/dL, fasting blood glucose level was 225 mg/dL, 2 hours postprandial blood glucose level was 452 mg/dL, and with HbA1C 10.6%. Electrolyte Serum showed Na 137 Mmol/L and potassium 3.45 Mmol/L with calculated serum osmolality was 305 mOsm/kg. Head CT was performed and showed hyperdense lesions on the right putamen and hypodense lesion in the left anterior periventricular region. Other pertinent admission data can be found in Table 1. We

diagnosed the patient with HCHB due to the hyperglycemic state.

The involuntary movement was improved and completely resolved after she got aggressive fluid rehydration and intravenous insulin therapy. The patient discharged with basal insulin injection therapy 10 IU and prandial insulin injection 6 IU q8hr to control the DM and 1.5mg haloperidol PO q12hr and 2mg clonazepam PO q12hr to control her movement disorder.

Second Case

A 65-year-old woman with a history of hypertension since five years ago and uncontrolled DM since three years ago came to the ER complained involuntary movement of the left limb since seven days before admitted to the hospital. The patient felt uncontrolled dancing when she did an activity and disappears when she was asleep. Previously, consume 5mg Glibenclamide PO qDay but not routinely controlled.

When came to the ER, blood pressure was 160/70 mmHg and no neurological deficit was found. We found involuntary jerking movements in the left extremities. RBS result was 530 mg/dL, fasting blood glucose level was 210 mg/dL, postprandial blood glucose level was 228 mg/dL and HbA1C was 12.3%. Electrolyte Serum showed Na 133 Mmol/L and potassium 3.4 Mmol/L with calculated serum osmolarity was 322 mOsm/kg. We performed head CT and showed a hypodense lesion on left corona radiata, left lentiform nucleus, and left internal capsule posterior limb and hyperdense punctate lesions in the right-left cortical-subcortical frontal, temporal, and parietal lobes and right lentiform nucleus.

The patient was diagnosed with HCHB due to non-ketotic hyperosmolar hyperglycemia. The involuntary movement was decreased after she got fluid rehydration and intravenous continuous insulin therapy. The patient was discharged after 7 days of treatment and got basal insulin therapy 10 IU to control DM, 0.5mg haloperidol PO q12hr, 2mg trihexyphenidyl PO qDay, and 1mg clonazepam PO q12hr to control her movement disorder. Other pertinent admission data can be found in Table 1.

Third Case

A 65-year-old woman came to the ER complained suddenly involuntary movement in her right hand which subsequently extended to her right leg when she woke up since 7 days before. This movement was painless, and no history of weakness in the limbs nor slurred speech. She had no history of the previous disease and was not taking any certain drugs.

Her condition was stable with a normal vital sign, no neurological deficit. We found involuntary movements in the right extremities irregular and continuously. Laboratory tests showed RBS was 476 mg/dL, fasting blood sugar was 240 mg/dL, two hours postprandial blood sugar was 263 mg/dL, and HbA1C 15.20%. Urinalysis showed glucose in the urine was positive, and negative ketones. Serum electrolyte showed sodium was 133 Mmol/L, potassium 4.38 Mmol/L with hyperosmolarity. We performed the head CT scan and showed hyperdense lesions in the left caudate nucleus and left lentiform nucleus which suspected as basal ganglia calcification. We diagnosed this patient with HCHB related to non-ketotic hyperosmolar hyperglycemia.

Her involuntary movement was resolved after we gave fluid rehydration and intravenous continuous insulin therapy and followed by oral drug Glimepiride 2 mg. Her movement disappears completely on day 4 after the treatment. She was discharged with 1mg glimepiride PO qDay, 500mg Metformin PO q8hr, and 2mg clonazepam PO qDay. The summary of the data can be found in Table 1.

Fourth Case

A 64-year-old woman came to the ER complained involuntary movement in her left hand since 8 days ago, her movement became more intense and extended to the left leg and left face. The patient felt likes throwing and stretching irregularly movement which disappeared when she was asleep. She had a history of movement disorder with the same characteristic about 1 month ago when she had a stroke. Movement disorder occurred on the third day of hospitalization. Patients had a history of controlled hypertension with amlodipine 1 x 10 mg and routinely controlled diabetes mellitus with insulin injection.

Her vital sign showed blood pressure 140/90 mmHg and heart rate 106 bpm. We found sinistra facial and hyoglossus nerve paralysis with upper motor neuron type and sinistra hemiparesis as sequelae. There were involuntary irregular stretching movements on the left part of the body which include face and extremities. On laboratory test, RBS was 117mg/dL, fasting Blood Sugar was 100 mg/dL, and two-hour postprandial blood sugar was 182 mg/dL. Both of serum electrolyte and hemostasis was normal. Head CT scan showed sub-acute cerebral infarct at external capsules dextra and cerebellum dextra, and chronic infarct at thalamus sinistra. We diagnosed this patient with HCHB due to thrombotic stroke. Her involuntary movement resolved completely after 4-day treatment with 1.5mg haloperidol PO q8hr, 2mg clonazepam PO q12hr, levodopa PO q12hr. The summary of the data can be found in Table 1.

Fifth Case

A 26-year-old male came to the emergency room with a gradual decrease in consciousness for 4 days before hospitalization. Previously, he complained of progressive headache, intermittent fever, and intermittent diarrhea for the last 4 months. One month before admitted to the hospital, he complained involuntary movement on his left hand, and 2 weeks later expanded to the left leg. He felt stretching and dancing movements which disappeared when he slept. He felt progressive weakness at his right side of the body. He also complained of cough without shortness of breath and mouth ulcer since 1 week ago. His family said that he never had any history of the previous disease. He consumes alcohol and had tattoos on his chest.

From the physical examination, the patient was unconscious with GCS 8/15, blood pressure was 90/50, HR was 80bpm, respiratory rate was 22tpm, and temperature 37.1 °C. There was a mouth ulcer, the pupillary reflex was reactive with diameter 5mm bilateral, we found rhonchi bilaterally. On the left extremity, there were involuntary movements with large amplitude and irregular writhing movements. We found the right lateralization at the extremities. Laboratory tests showed reactive to HIV. Complete blood count, RBS, and serum electrolyte were normal. Chest X-Ray showed pneumonia and head CT Scan

showed rim enhanced lesion in the bilateral basal ganglia with leptomeningeal enhancement in the bilateral frontal and parietal lobes suggest as cerebral toxoplasmosis with 3 mm subfalcine herniation to the right of and mild communicating hydrocephalus. We diagnosed the patient with cerebral toxoplasmosis in HIV with hemichorea-hemiballism complications. To reduce the involuntary movement, we gave 2mg clonazepam PO qDay, 1mg Haloperidol PO q12hr, 2mg Trihexyphenidyl PO q12hr. We treat the cerebral toxoplasmosis with pyrimethamine and clindamycin. The patient got the septic condition and passed away on the second week of treatment caused by septic encephalopathy. The summary of the data can be found in Table 1.

Discussion

Hemichorea-hemiballism (HCHB) is a hyperkinetic hypotonic movement disorder characterized by non-patterned, jerky, random, irregular, and uncontrolled movements that occur on half parts of the body.^{1,9} This is due to involvement disorders at the basal ganglia, especially in the corpus striatum.^{9,10} The most common cause of HCHB is stroke and the second cause is non-ketotic hyperosmolar hyperglycemia (NKKH).^{6,9} The incidence of HCHB that occurs in the population due to post-stroke patients and NKKH is around 0.008 and 0.001%. Other causes reported are due to infections, neoplasms, drug or toxins, autoimmune, and other metabolic disorders such as hyperglycemia, hypoglycemia, hypokalemia.^{6,9,11} In these present reports, we report 5 newly diagnosed cases HCHB due to NKKH, stroke, and toxoplasmosis cerebral.

The onset of movement disorder developments varies, depend on the cause.^{1,11} HCHB associated with stroke usually will suddenly appear when the paresis in the extremity has improved.^{2,11} HCHB associated with inflammation or infection usually develops slowly, expanding, and gets heavier depends on the development of infection and inflammation.^{6,13} Besides, HCHB associated with cerebrovascular disease and the metabolic disorder usually occurs in older age, while in infection or inflammation occurs at a relatively younger age.^{12,13}

The type and location of structural lesions cause diverse characteristics of movement disorder.^{14,15} In about 20% of cases, structural lesions that occur in HCHB takes place in the contralateral nucleus subthalamic. Besides, bilateral or ipsilateral lesions of the movement were also found in the case report caused by chronic hepatic encephalopathy.^{14,15,16,17}

HCHB related to NKKH can be an early symptom of unknown diabetes mellitus as happened in our third patient or can appear several days or weeks when the blood glucose level of increase or is not well controlled as in our first and second patients.^{14,15} Several case reports showed that HCHB also can occur a few weeks after the blood glucose levels have been controlled and on treatment which shows delayed reaction to severe hyperglycemia.⁵ All of our NKKH patients are diagnosed as HCHB due to NKKH and were reduced or resolved perfectly after they got therapy to reduce blood glucose levels, which shows the temporal relationship between the restoration of blood

glucose and the improvement of the HCHB.^{5,14,15} In three of our cases, that report HCHB caused by NKKH, the average RBS was above 400 mg/dL and the HbA1C level was above 10%. HCHB has most commonly seen in patients with long-standing, poorly controlled, and often with HbA1C more than 10%.¹⁸ This also consistent with the epidemiological research in India by Nadig, *et al* showed that mean serum glucose levels measured after the onset of NKKH were 439.93 mg% and the mean HbA1C was 10.98%. In the second and third cases, there was mild dilutional hyponatremia that suggests resulting from hyperglycemia. In acute hyperglycemia, dilutional hyponatremia can occur due to fluid transfer from cells to plasma as a result of hypertonic extracellular fluid and osmotic fluid differences. Each increase in blood glucose of 100 mg/dl, serum osmolarity will increase from 1.9 to 2.1 mosm and serum sodium decreases from 1.6 to 1.8 mEq/L. In chronic hyperglycemia, loss of sodium and hyponatremia can occur due to osmotic diuresis.^{14,16}

We concluded the patient experienced HCHB associated with stroke whereas stroke is the leading cause of HCHB.⁶ It was supported by the imaging that showed structural lesions in the basal ganglia region at putamen and thalamus. HCHB associated with cerebrovascular disease or stroke can occur on acute onset after the stroke or maybe several days or months later.^{17,19} In this patient, HCHB first occurred on the 3rd day of the stroke, then resolved with treatment, and reoccurred when the patient did not consume medication to control HCHB during the last 2 weeks. In most cases, HCHB due to stroke can be resolved completely with appropriate medication.^{17,20}

Movement disorders occur due to dysfunction in motor network connectivity. Connection disturbance between internal globus pallidus, subthalamic nucleus, and thalamus promotes the failure of the basal ganglia and cortical circuits.^{6,9} The distinctive characteristics of HCHB with MR and CT modality can exhibits lesions in the basal ganglia.^{21,22}

Hemichorea lesion usually find at contralateral of nucleus caudate whereas in hemiballism at the nucleus subthalamic.^{19,23} Although in some case reports, lesions at nucleus lentiform, thalamus, and cortex also can cause HCHB.^{19,23} Generally, high attenuation changes involving the contralateral basal ganglia are found on the head CT scan, although in some cases of hyperglycemia they can show a normal result.^{15,16,17} Imaging findings in HCHB due to hyperglycemia are caused by acute infarction, hemorrhagic petechiae, myelinolysis, calcium deposits, and a decrease in aminobutyric synthesis and also acetylcholine due to metabolic changes or the presence of secondary injury due to hyperviscosity or vasogenic edema.^{20,24} The hyperviscosity promotes the disruption of the blood-brain barrier and transient ischemia of striatal neurons.^{5,20,24} The synergistic effect of uncontrolled hyperglycemia and vascular insufficiency promote transient striatum dysfunction, which can result in HCHB. Whereas, in case series about thalamic stroke, evidence that strokes which severe sensory deficit and ataxia are more likely to cause movement disorder.^{5,13} This was in line with our fourth case which had a sensory deficit and there was a lesion at thalamus showed by imaging.

Table 1. Summary of the cases admission data and clinical courses

	Case 1	Case 2	Case 3	Case 4	Case 5
Sex, Age	Female, 65	Female, 65	Female, 65	Female, 64	Male, 26
Blood Pressure	140/70	160/90	120/70	140/90	90/50
Temperature	36.1	36.3	36.4	36.3	37.1
Haemoglobin	14.4	13.4	13.70	10.90	11
WBC	13.860	8.600	9.720	7.600	4.880
Platelet	312.000	366.000	406.000	317.000	181.000
Glucose	500	530	476	117	83
GD1/2PP	225/ 452	210/ 228	240/ 263	100/ 182	n/a
HbA1C	10.6%	12.3%	15.20%	n/a	n/a
Keton Urine	Negative	Negative	Negative	n/a	n/a
Sodium	137	133	133	136	133
Potassium	3.45	3.4	4.38	4.36	3.8
Ureum/ Creatinin	39.6/ 0.81	40/ 0.6	16.5/ 0.48	39.0/ 1.02	25/ 0.9
Osmolality	305	322	347.8	287.8	292.8
HIV Test	Negative	Negative	Negative	Negative	Positive
Duration emerge of HCHB after disease onset	2 weeks	7 days	7 days	3 days	1 month
Head CT Scan	Hyperdense lesions on the right putamen and hypodense lesion in the left anterior periventricular region.	Hypodense lesion on left corona radiata, left lentiform nucleus, and left internal capsule posterior limb and hyperdense punctate lesions in the right-left cortical subcortical frontal, temporal, and parietal lobes and right lentiform nucleus.	Hyperdense lesions in the left caudate nucleus and left lentiform nucleus which suspected as basal ganglia calcification.	Sub-acute cerebral infarct at external capsules dextra, and cerebellum dextra, and chronic infarct at thalamus sinistra.	Rim enhanced lesion in the bilateral basal ganglia with leptomeningeal enhancement in the bilateral frontal and parietal lobes.
Therapy	Insulin Basal and Prandial, Haloperidol 2x1.5mg, Clonazepam 2x2mg	Insulin Basal, Haloperidol 2x0.5mg, Trihexyphenidyl 1x2mg, Clonazepam 2x1mg	Glimepiride 1x2mg, Metformin 3x500mg, Clonazepam 1x2mg	Haloperidol 3x1.5mg, Clonazepam 2x2mg, Levodopa 2x1mg.	Haloperidol 2x1mg, Trihexyphenidil 2x2mg, Clonazepam 1x2mg
Glucose when HCHB resolved	267	170	116	n/a	n/a
Time until HCHB resolved after start the therapy	9 days	7 days	4 days	4 days	Not resolved

Figure 1. Head CT Scan of cases (A) Hyperdense lesions on the right putamen (Case 1) (B) Hypodense lesion on left corona radiata, left lentiform nucleus (Case 2), (C) Hyperdense lesions in the left caudate nucleus and left lentiform nucleus (Case 3) (D) Hypodense lesion at external capsules dextra, (Case 4) (E) Rim enhanced lesion in the bilateral basal ganglia (Case 5).

Various hypotheses have been proposed to explain HCHB due to hyperglycemia. Hyperglycemia promotes cerebral autoregulation failure, hypoperfusion and activation of anaerobic metabolism, and depletion of GABA in the basal ganglia. Generally, GABA has a role as a main inhibitory neurotransmitter, glutamate as activator neurotransmitter,

and acetylcholine as a modulator neurotransmitter.^{5,17,19} Hyperglycemia inactivates the tricarboxylic acid (Krebs) cycle due to decreased regional cerebral blood flow and failure of glucose metabolism, and the brain's metabolism shifts to an anaerobic pathway.^{16,17,20,21} In ketotic condition, acetoacetate can be used to synthesize GABA, whereas, in

non-ketotic hyperglycemic, changes to anaerobic metabolism promote the brain to use aminobutyric acid, which is synthesized from acetoacetate that causes the decrease of GABA levels rapidly, reduce GABAergic activity, and reduce inhibition of the thalamus by the medial globus pallidus. This condition causes a decrease in inhibitory signals to the thalamus and results in hyperkinetic movements.^{5,16,17,18,19,21}

Dopaminergic activity changes in the striatum caused by hyperglycemia are also other mechanisms that can occur, especially in postmenopausal women who lack estrogen.^{3,21} After menopause, hyperglycemia induces an increase in the sensitivity of dopamine receptors in the striata. Patients HCHB that related to hyperglycemia commonly older women with uncontrolled diabetes and non-ketotic hyperglycemia condition.^{3,15,21}

Cerebrovascular insufficiency mechanisms also have a role in HCHB pathophysiology due to hyperglycemia.^{3,21} This condition is similar to the changes that occur in diabetic retinopathy. Hyperglycemia promotes insulin resistance, oxidative stress, and loss of nitric oxide in the endothelium, resulting in endothelial dysfunction and removing endothelial vasoactivators that lead to an increase of free fatty acids, prothrombotic state and dysfunction in smooth vascular muscle and decreased regulation of microRNAs. Vasoconstriction promotes tissue hypoperfusion and neuronal loss or damage.^{15,17,22}

Pathophysiology of HCHB due to infection, in this case, cerebral toxoplasmosis, is a tumor-like lesion that causes an increase of intracranial pressure, confusional state, and appearance of focal deficits on the pyramidal pathway.^{25,26} The most common movement disorder in cerebral toxoplasmosis is HCHB or hemichorea-athetosis. Cerebral structures generally affected in the basal ganglia area include the subthalamic nucleus, thalamus, and head of the caudate, putamen, globus pallidus, midbrain, and internal capsule, which can promote contralateral lesions or bilateral movements.^{9,26} HCHB believed to be associated with the subthalamic nucleus damage or its efferent pathways, which eliminates the excitation of the globus pallidus, thereby eliminating the ventral lateral and ventral anterior nuclei that receive projections from the globus pallidus.^{1,4,9,25} In our fifth case, there was rim enhanced lesion on basal ganglia which is believed to be the cause of HCHB on the patient.

HCHB in AIDS patients is commonly associated with the presence of cerebral toxoplasmosis. This is found in 7.4% of case reports and is generally caused by a subthalamic toxoplasmosis abscess.^{25,26} Some researchers mention that HCHB that occurs in AIDS patients is a pathognomonic symptom to cerebral toxoplasmosis.²⁵ This is interesting as in patients with non-reactive HIV with toxoplasmosis and abscesses in subthalamic, movement disorders are not found.^{25,27}

The management principle of HCHB is to determine the etiology and then correct the underlying disorder.^{6,28,29} Pharmacological therapy is not essential in all cases as many patients have a mild and self-limited disorder. HCHB due to hyperglycemia must treat with aggressive glycemic control. The movements will slowly improve after glucose correction.^{10,27,29,30} Patient with acute stroke and HCHB

recommends to reduced uncomfortable feeling and distress.^{22,23} Antidopaminergic therapy has the main role of pharmacological treatment, especially a group of typical and atypical neuroleptics and catecholamine-depleting agents. Typical neuroleptic agents including haloperidol, pimozide, perphenazine, and fluphenazine are the first-line drug treatments for HCHB and work by blocking dopamine receptors. The atypical neuroleptic drugs, such as olanzapine, risperidone, clozapine, quetiapine are less to cause drug-induced parkinsonism and tardive dyskinesia. Clozapine has been successful in refractory cases but can cause agranulocytosis.³¹ Benzodiazepine drugs including clonazepam and gabapentin often used for additional treatment. Clonazepam suggests using in the short term because of addictive side effects. Levodopa can be considered as a second-line drug of HCHB. Long-term use of levodopa can cause dyskinesia, so the treatment process must be controlled carefully.^{29,30} Management in HCHB due to HIV with toxoplasmosis cerebral includes opportunistic infection treatment, symptomatic treatment of the movement disorder, and the use of antiretroviral agents.^{25,26,29} It has been reported that response to therapy was poor and ineffective in controlling the involuntary movements in the generalized chorea seen with HIV encephalitis.²⁵

Conclusion

Hemichorea-Hemiballism is a rare case of movement disorder. The natural history of hemichorea-hemiballism varies depend on etiology. Characteristic features depend on contralateral disorder within the putamen, caudate nucleus, and globus pallidus. The main management strategy is to determine the etiology and correct the underlying disorder. Prognosis is usually benign in most cases.

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