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A Spectrum of Movement Disorders: Asterixis Associated with Chronic Liver Disease: A Case Report

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ABSTRACT

Background: Liver disease is a condition with wide manifestations and can lead to a myriad of complications. The relationship between movement disorder and liver disease should be considered. Movement disorder due to impaired liver function can be precipitated by neurological dysfunction with a wide etiology including abnormally impaired liver detoxification of potentially toxic substances, deranged function of the liver, and/or exposure to exogenous toxins/drugs. ***Case summary:*** A male patient aged 44 years complained of jerking of hands and feet. He also had a history of hepatic cirrhosis and chronic hepatitis B for the past 2 years, HBSAg positive, splenomegaly, and portal hypertension signs on ultrasound. Neurological exam revealed involuntary wrist manus and pedis dextra sinistra movements, wrist rhythmic rhythm, medium amplitude (4-5Hz) speed, suggestibility (-), hands and feet area of, stereotypic (+), worse with rest, improves with rest, resembles involuntary movements in flapping tremor or asterixis. ***Discussion:*** Compromised liver function in patients with cirrhosis can lead to hepatocerebral degeneration through the filtration of neurotoxins such as ammonia, glutamine, and manganese into circulation. They disrupt astrocyte and neuron communication, leading to presynaptic dopaminergic dysfunction and loss of postsynaptic dopaminergic receptors, those manifesting clinically as dementia and movement disorders including tremor, parkinsonism, dystonia, chorea, and ataxia. ***Conclusion:*** There are significant clinical interactions between the liver and central nervous system. Hepatocerebral degeneration should be considered in patients with hepatic cirrhosis who have movement disorders. They comprise movement disorders and intellectual decline, including tremors, dystonia, chorea and ataxia, and parkinsonism.

Keywords: hepatic disease; movement disorder; flapping tremor.

INTRODUCTION

Liver disease is a disease with wide-ranging manifestations and can lead to numerous complications. The association between movement disorders and liver disease needs to be remembered. Liver failure-related movement disorders may result from neurological impairment secondary to multisystem etiologies, such as impaired detoxification of toxic substances by the liver, liver failure, and/or exposure to exogenous toxins or drugs [1]. In some cases, this can be the result of the mutual involvement of both organ systems in a single disease process, secondary neurological damage due to an inability of the liver to clear noxious chemicals, or secondary hepatic and/or neurological damage due to exposure to foreign agents [2].

Symptoms of movement disorders are often underrated and little known to the general population, although they are the second most common reason for visits to neurologists. When patients experience encounters with health professionals, experiences are often invalidated and poor descriptions of symptoms are made, which serves to reinforce patients' concepts about the disease [3,4]. These along with the stigma of physical symptoms from purely psychological causes present a barrier to diagnosis acceptance and resulting treatment [5,6]. The following metabolic disorders can be listed as potentially causing symptomatic myoclonus.

CASE SUMMARY

A 44-year-old male patient presented with jerking of his hands and feet. He was referred due to sudden jerking of his hands and feet that occurred whenever he felt pain in his body but disappeared when he slept. The jerks were akin to repetitive thrusting of limbs. The patient initially came to the emergency room with a decreased level of consciousness a day before admission. He has a history of hepatic cirrhosis and chronic hepatitis B in the past. History of hepatic cirrhosis. He receives chronic tenofovir therapy. No history of the same complaint in the family. He is a barbershop employee.

Physical examination revealed scleral icterus and ascites. Neurological examination induced involuntary rhythmic movements (4–5 Hz) of medium amplitude, with fast velocity, in the upper and lower limbs bilaterally, not suppressible, and stereotyped. Movements were exacerbated by activity and improved with rest. The first laboratory results at Wangaya General Hospital were hemoglobin 8.9 g/dL, hematocrit 27.6%, platelets 324, AST 63, and ALT 34. Abdominal ultrasound showed signs of chronic liver disease with portal hypertension (splenomegaly) and ascites. Non-contrast head CT scan was normal with no evidence of hemorrhage, infarct, or space-occupying lesion. Ascitic tap was positive with xanthochromic fluid of 4000 ml. On the third hospital day, jerking of the limbs was observed.

He was diagnosed with a movement disorder suspected myoclonic jerk hepatic cirrhosis suspected malignant degeneration, and chronic hepatitis B. Treatment included haloperidol 2 × 2.5 mg. Internal medicine management included omeprazole 2 × 1 amp, tranexamic acid 3 × 1 amp, sucralfate syrup 3 × 1, iron supplement 2 × 1, furosemide 3 × 1 amp, and spironolactone 1 × 50 mg.

DISCUSSION

Definition of Movement Disorders

Movement disorders are a group of conditions characterized by abnormally coordinated, hypokinetic, or hyperkinetic movements. They include tremor, parkinsonism, dystonia, myoclonus, chorea, ballismus, ataxia, tics, dyskinesias, akathisia, restless limbs, etc. The term "movement disorder" can either refer to the abnormal movement itself or the syndromes causing it [7].

Myoclonus

Myoclonus is an event-related motor disorder with shock-like, short jerks occurring suddenly. Positive myoclonus is responsible for the majority of myoclonic jerks and is caused by transient bursts of muscle activity. If caused by transient interruptions in uninterrupted muscle activity, then it is called negative myoclonus. Positive myoclonus manifests as a sudden, transient muscle contraction, while negative myoclonus (e.g., asterixis) manifests as an episodic loss of muscle tone, commonly involving outstretched arms or wrists. Positive myoclonus occurs more often, while negative myoclonus typically occurs in hospitalized patients according to metabolic-toxic etiology [7].

Diagnosis

Electrophysiology can help decide if the etiology is cortical, subcortical, or spinal. Polygraphy is the first step, recording the length, distribution, and sensitivity to the stimulus of the jerks. EEG-EMG recording is useful in further evaluation [7].

Therapy

Treatment is determined by the etiology. Metabolic toxicity states, drug toxicities, or surgically amenable lesions have reversible etiologies. The majority of the patients are symptomatic. Therapy needs to be myoclonus physiology-based: cortical myoclonus responds to valproate, levetiracetam, and piracetam. Clonazepam can be used in all forms of myoclonus [6,8].

Hepatic Cirrhosis

Chronic liver disease with fibrosis and nodular regeneration, producing structural and functional derangement [9]. End-stage disease after extended liver injury. Hepatitis B/C, alcohol liver disease, and non-alcoholic fatty liver disease are frequent etiologies. Portosystemic shunting allows the passage of neurotoxins (e.g., ammonia, glutamine, manganese) bypassing the liver to the brain. Inflammatory disorders increase blood-brain barrier permeability, with secondary toxin accumulation. Ammonia is metabolized to glutamine inside cells, leading to oxidative stress, metabolic derangement, and astrocyte changes [1,2].

Movement Disorders in Hepatic Cirrhosis

Portosystemic shunts allow toxins to bypass hepatic detoxification and enter cerebral circulation. Inflammatory diseases and disruption of the blood-brain barrier lead to parenchymal accumulation. Ammonia disrupts astrocyte-neuron communication, leading to a wide range of neurological manifestations [1]. Hepatic encephalopathy typically presents with fluctuating consciousness (alternating between coma and drowsiness), neuropsychiatric derangement, and movement disorders like asterixis, tremor, hypokinesia, and rigidity [2]. Acquired hepatocerebral degeneration (AHD) is an incurable chronic disorder with basal ganglia manganese deposition, presynaptic dopaminergic dysfunction, and loss of postsynaptic receptors [3]. T1 hyperintensity in globus pallidus is evident on MRI. Cognitive impairment and movement disorders (tremors, parkinsonism, dystonia, chorea, ataxia) are the clinical manifestations. They are mostly irreversible and drug therapy is not effective; liver transplantation is the main treatment.

DISCUSSION (Case)

The jerky limb movement of the patient is characteristic of negative myoclonus, consistent with asterixis. Myoclonus is a feature of hepatic dysfunction, especially in chronic liver disease. The presenting movement symptoms of the patient with chronic liver disease in this case were unusual limb jerks. Liver disease can be graded based on disease severity.

Progressive hepatic insufficiency is the result of chronic liver disease with escalating toxic shunting. Neurological manifestations are likely to occur after the organism crosses a threshold (1). Causes of hepatic encephalopathy include sepsis, electrolyte imbalance, constipation, and GI bleeding (3). This patient presented with electrolyte imbalance, anemia, and cirrhosis. Myoclonus treatment depends on the etiology. While clonazepam is generally effective for all types, in this patient, haloperidol was administered and there was improvement despite going against theoretical guidelines.

CONCLUSION

Cirrhotic hepatic cirrhosis is a chronic liver illness characterized by fibrosis, nodular regeneration, structural abnormality, and dysfunction of the liver. Portosystemic shunt in cirrhosis or non-cirrhotic portal hypertension allows the shunting of neurotoxins such as ammonia, glutamine, and manganese around the liver and into the cerebral circulation. Inflammatory processes impair the integrity of the blood-brain barrier and promote parenchymal accumulation. This case highlights the significant clinical interaction between the central nervous system and the liver. Hepatocerebral degeneration is a disorder that needs to be considered in cirrhotic patients with movement disorders. Mental impairment and movement abnormalities such as tremors, parkinsonism, dystonia, chorea, and ataxia can be present.

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