ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2022 October 26; 10(30): 10823-11213

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

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RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Ying-Yi Yuan*; **Production Department Director:** *Xu Guo*; **Editorial Office Director:** *Jin-Lei Wang*.

NAME OF JOURNAL
World Journal of Clinical Cases

ISSN
ISSN 2307-8960 (online)

LAUNCH DATE
April 16, 2013

FREQUENCY
Thrice Monthly

PUBLICATION DATE
October 26, 2022

World Journal of Clinical Cases

Submit a Manuscript: https://www.f6publishing.com

World J Clin Cases 2022 October 26; 10(30): 11172-11177

DOI: 10.12998/wjcc.v10.i30.11172

ISSN 2307-8960 (online)

CASE REPORT

Spontaneous remission of hepatic myelopathy in a patient with alcoholic cirrhosis: A case report

Chun-Yan Chang, Chen Liu, Fang-Fang Duan, Hang Zhai, Shan-Shan Song, Song Yang

Specialty type: Medicine, research and experimental

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): 0
Grade C (Good): C, C, C, C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Ferrarese A, Italy; Surani S, United States

Received: July 6, 2022
Peer-review started: July 6, 2022
First decision: August 4, 2022
Revised: August 24, 2022
Accepted: September 19, 2022
Article in press: September 19, 2022
Published online: October 26, 2022

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Abstract

BACKGROUND

Hepatic myelopathy (HM) is a rare neurological complication of advanced cirrhosis. Prognosis of patients with HM is generally poor without timely liver transplantation or interventional therapy. Self-resolving HM in patients with alcoholic cirrhosis has never been reported.

CASE SUMMARY

A 53-year-old man with alcoholic cirrhosis and recurrent overt hepatic encephalopathy for 1 year was admitted for lower extremity weakness, slow movement, and stumbling gait. The patient was diagnosed with HM after excluding other causes of spastic paraparesis. The patient refused liver transplantation. However, the patient kept total abstinence and received a multidisciplinary treatment for complications of decompensated cirrhosis. The symptoms of HM resolved gradually after 2 years of treatment. All complications of alcoholic cirrhosis resolved after 4 years of follow-up.

CONCLUSION

The case demonstrates that HM can resolve in patients without liver transplantation after total abstinence and systemic management of complications.

Key Words: Alcoholic cirrhosis; Hepatic myelopathy; Hepatic encephalopathy; Spastic paraparesis; Therapeutics; Case report

Core Tip: Hepatic myelopathy (HM) is a rare neurological complication of advanced cirrhosis. Prompt liver transplantation or interventional therapy may reverse the symptoms of HM. Self-resolving HM in patients with alcoholic cirrhosis has never been reported. Our report presents that self-resolving HM in a patient with alcoholic cirrhosis is possible without any liver transplantation and interventional therapy after promptly controlling the etiology and systemic management of complications. This case provides new insight into the self-remission of patients with HM.

Citation: Chang CY, Liu C, Duan FF, Zhai H, Song SS, Yang S. Spontaneous remission of hepatic myelopathy in a patient with alcoholic cirrhosis: A case report. *World J Clin Cases* 2022; 10(30): 11172-11177
URL: https://www.wjgnet.com/2307-8960/full/v10/i30/11172.htm
DOI: https://dx.doi.org/10.12998/wjcc.v10.i30.11172

INTRODUCTION

Hepatic myelopathy (HM) is a rare neurological complication of advanced cirrhosis. The clinical manifestations of HM are progressive spasmodic paralysis of the limbs and do not involve sensory or sphincter motor symptoms, commonly in patients with recurrent hepatic encephalopathy (HE)[1]. Other causes of spastic paraparesis and partial transverse myelopathy should be ruled out before establishing the diagnosis[2]. Although the first case of HM was reported 30 years ago, the prognosis profiles of patients with HM, especially the rare cases, remain obscure[3]. Limited data have demonstrated that prompt liver transplantation or interventional therapy may reverse the symptoms of HM[4]. In 2017, di Biase *et al*[5] has reported the first case of self-resolving HM in patients with hepatitis C virus (HCV)-related cirrhosis after HCV treatment. Since then, no case of self-resolving HM was reported. To the best of our knowledge, no case of self-resolving HM for alcoholic cirrhosis has been reported. Herein, we report the first case of self-resolving HM from our large cohort of patients with alcoholic cirrhosis[6]. We also reviewed the treatment of patients with HM.

CASE PRESENTATION

Chief complaints

A 53-year-old man with alcoholic cirrhosis was admitted to Beijing Ditan Hospital of Capital Medical University for lower extremity weakness, slow movement, and stumbling gait that required walking assistance with a crutch in January 2015.

History of present illness

The patient was diagnosed with decompensated alcoholic cirrhosis with ascites in September 2011. In December 2011, he was admitted to the hospital due to gastroesophageal variceal bleeding and received splenectomy combined with a gastroesophageal devascularization surgery. In April 2013, he was hospitalized for comorbid acute hepatitis B and suffered from recurrent overt HE since then. Since January 2015, the patient gradually developed weakness in both lower limbs, slow movement, and hobbling gait.

History of past illness

The patient had no relevant medical history.

Personal and family history

The patient had a history of heavy drinking for 25 years, with an average alcohol intake of 200 g per day.

Physical examination

Abdominal examination suggested hepatomegaly and positive shifting dullness. Neurological system examinations demonstrated slurring speech, normal cranial nerves, increased muscle tension and grade 4/5 power of lower limbs, exaggerated deep tendon reflexes, and no sensory deficit or sphincteric involvement.

Laboratory examinations

Serial results of liver function and whole blood count are presented in Table 1. Blood ammonia concentration fluctuated between 50 and 87 μmol/L during the occurrence of overt HE. In January 2015,

Table 1 Serial results of liver function test and whole blood cell count

	September, 2011	September, 2012	September, 2014	February, 2015	August, 2019	October, 2021
ALT (U/L)	25	18	717	28	42	25
AST (U/L)	42	26	976	46	62	32
ALB (g/L)	30.0	32.0	33.2	32.2	36.1	41.3
TBIL (μmol/L)	53.1	15.7	109.4	19.3	47.0	16.7
Hb (g/L)	108	82	127	139.0	159	153
WBC (109/L)	6.4	8.5	9.4	8.8	7.5	7.6
PLT (109/L)	71	278	159	79	100	121

ALB: Albumin; ALT: Alanine transaminase; AST: Aspartate transaminase; Hb: Hemoglobin; PLT: Platelet; TBIL: Total bilirubin; WBC: White blood cell.

hospitalization, hepatitis B surface antigen, and anti-HCV tests were negative. Additionally, human immunodeficiency virus (HIV), Syphilis, Epstein-Barr virus (EBV), and cytomegalovirus tests were negative. Serum vitamin B-12 level was normal. Cerebrospinal fluid analysis was normal.

Imaging examinations

Contrast abdominal computed tomography revealed liver cirrhosis, esophageal and gastric varices, gastro-left renal shunt, and portal vein thrombosis (Figure 1). Magnetic resonance imaging (MRI) of the brain indicated hyperintensities in the bilateral globus pallidus (Figure 2). Moreover, whole spinal MRI and lumbosacral MRI were performed and revealed normal results. The electromyogram showed normal nerve conduction velocity in the bilateral tibial nerves. Somatosensory evoked potentials of the lower limbs were normal. Motor evoked potential was abnormal in both lower limbs.

FINAL DIAGNOSIS

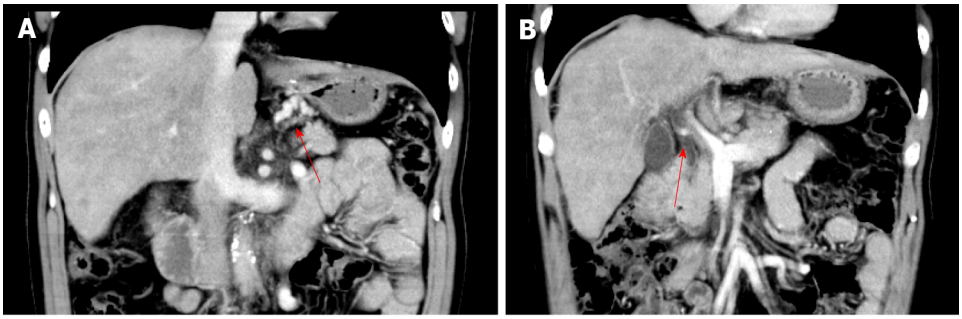
Multidisciplinary expert consultation was performed; this included experts in hepatology, neurology, infectious diseases, and radiology, to find for the cause of the spastic paraparesis. The cranial and spinal MRI showed no intracranial or spinal space occupation. Normal serum vitamin B-12 levels allowed subacute combined degeneration of the spinal cord to be ruled out. Primary lateral sclerosis was not considered since spastic paraparesis in this patient get spontaneously resolved and does not involve the upper limbs. Spinal multiple sclerosis was excluded based on the normal spinal MRI and lack of sensory deficit or sphincteric involvement. Myelopathy related to HIV, EBV or other pathogens infections was ruled out based on the normal infection biomarkers and normal cerebrospinal fluid status. Moreover, hereditary spastic paraplegia, Wilson's disease, radiation myelopathy, vascular spinal cord disease, and other causes of spastic paraparesis were ruled out due to the lack of specific neurological features and lack of characteristic distinguishing abnormalities on neuroimaging. The patient was diagnosed with HM after exclusion of any other potential causes of spastic paraparesis.

TREATMENT

The patient rejected liver transplantation for financial reasons. The patient chose abstinence and took furosemide, spironolactone, lactulose, L-ornithine-L-aspartate for ascites and HE. The patient was followed up every 3-6 mo.

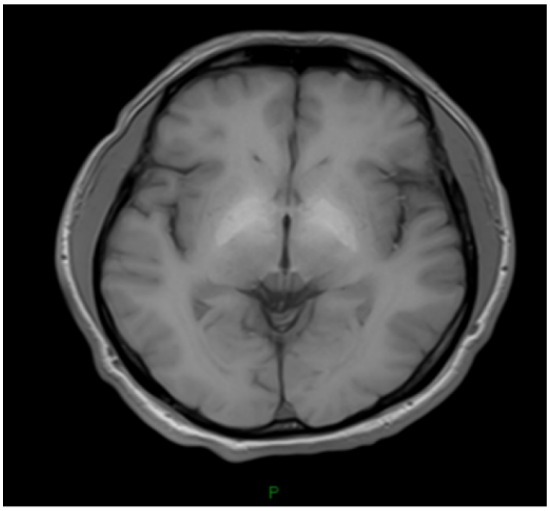
OUTCOME AND FOLLOW-UP

The patient chose abstinence and symptomatic treatment. During follow-up, he had less ascites and overt HE attacks. Since 2018, he had reported gradual improvement of his lower limb weakness and hobbling gait. In August 2019, the patient reported that he could walk without the assistance of a crutch. The liver function test revealed normal alanine transaminase, aspartate transaminase and albumin levels. Abdominal ultrasound revealed no signs of ascites and disappearance of the portal vein thrombosis. The patient was regularly followed up until October 2021, and has since demonstrated normal liver function and regular limb movement.



DOI: 10.12998/wjcc.v10.i30.11172

Figure 1 Contrast abdominal computed tomography revealed cirrhosis, esophageal and gastric fundus varicose veins, fundus-left renal shunt, and portal vein thrombosis. A: Arrow shows portal vein thrombosis; B: Arrow shows esophagogastric varices.



DOI: 10.12998/wjcc.v10.i30.11172

Figure 2 The cranial magnetic resonance imaging revealed increased T1W symmetric signal in the bilateral globus pallidus.

DISCUSSION

HM is a rare complication of cirrhosis, which is common in patients with portosystemic shunts and recurrent HE. Its main clinical manifestation is progressive spastic paraparesis. Diagnosis of HM needs to exclude other causes for spastic paraparesis, which include amyotrophic lateral sclerosis, hereditary and toxic myelopathy, multiple sclerosis, paraneoplastic syndromes, radiation myelopathy, infectious causes of myelopathy, and vascular spinal cord disease[7]. Regarding this patient, he had a history of cirrhosis and recurrent HE attacks. Contrast abdominal computed tomography showed portosystemic shunting. In addition, MRI of the brain indicated cirrhosis and HE. The diagnosis of HM was established after exclusion other potential causes of spastic paraparesis by multidisciplinary expert consultation.

Early spinal cord injury in HM is characterized by symmetrical demyelination of corticospinal tracts due to nitrogenous toxins such as ammonia. The demyelination is reversable with prompt management of the underlying liver disease and/or portosystemic shunts. As the disease progresses, axonal loss occurs, which may be irreversible[8,9].

Troisi *et al*[10] reported the first case of a patient with HM in whom myelopathy improved after liver transplantation. Since then, an increasing number of studies have demonstrated that liver transplantation might reverse HM[1,4,11-15], although some studies have reported otherwise[16,17]. When comparing patients in whom HM was reversed after liver transplantation and patients whose HM was not reversed, it is generally recognized that the likelihood of HM reversal may be higher when liver transplantation is performed within 18 mo after the onset of symptomatic HM[4]. This theory was further verified by Koul *et al*'s report, in which two children with acute HM after hepatitis A infection recovered completely after receiving donor liver transplantation[14].

For HM secondary to transjugular intrahepatic portosystemic shunt (TIPS) or surgical splenorenal shunt, reports have revealed that prompt shunt occlusion or shunt limitation may reverse HM[18-21]. Some studys have reported that shunt limitation, not shunt occlusion, is useful for reversing early-onset

HM after TIPS[20,21]. Shunt limiting is preferred, as total shunt occlusion might have a higher risk of adverse events related to the rapid increase of portal hypertension. Moreover, Philips *et al*[22] reported partial splenic artery embolization (PSAE) for a patient with HM. Neurological function improved rapidly and constantly after PSAE. The authors concluded that PSAE may improve liver function, decrease PHT, and lower portosystemic shunting in this way to ameliorate neurological symptoms. Intestinal microbiota is closely related to HE, and some studies have reported that fecal microbiota transplantations (FMT) might improve HE[23]. Based on this, Sun *et al*[24] reported a case of HM in a patient who received FMT, and neurological function improved after three repetitions of FMT. More studies have revealed that repairing gut microbiota may decrease portal hypertension and repair the blood-brain barrier[25,26]. Further, there is increasing data to demonstrate the usefulness of FMT for improving HE[27,28]. Considering the shared pathogenesis of HM and HE, FMT for HM seems promising and is worth further investigation.

In 2017, di Biase *et al*[5] reported an interesting case of self-resolving HM. This patient with HCV-related cirrhosis was treated with sofosbuvir plus ribavirin. HM improved 6 mo after HCV treatment. The case demonstrates that self-resolving HM might be possible after relief of the underlying liver disease. As in our case, HM was relieved with total abstinence, and liver function was restored. Additionally, the 6-year follow-up demonstrated sustained re-compensation of liver cirrhosis in this case.

CONCLUSION

As the first reported case of self-resolving HM in a patient with alcoholic cirrhosis, the case demonstrates that self-remission of HM is possible even without liver transplantation after total abstinence and systemic management of complications.

FOOTNOTES

Author contributions: Chang CY designed and contributed to the manuscript draft; Liu C and Duan FF analyzed and interpreted the imaging data; Zhai H and Song SS collected the patient's clinical data; Yang S reviewed this paper and approved the final version of this manuscript.

Supported by Chinese foundation for hepatitis prevention and control, Tianqing liver disease research fund subject, No. TQGB20210050; and Beijing Municipal Administration of Hospitals Incubating Program, No. PX2022071.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country/Territory of origin: China

ORCID number: Chun-Yan Chang 0000-0002-4717-4361; Song Yang 0000-0001-6046-5756.

S-Editor: Wang JJ
L-Editor: A
P-Editor: Wang JJ

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