Journal of Biosciences and Medicines, 2023, 11, 303-309
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

A Case of Acute Upper Gastrointestinal Bleeding in Liver Cirrhosis Complicated by Acute Cerebral Infarction and Acute Myelitis

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How to cite this paper: Liao, X.H., Xu, T.T., Zhang, X.H., Mi, X.L., Yang, C.Q. and Wei, Z.B. (2023) A Case of Acute Upper Gastrointestinal Bleeding in Liver Cirrhosis Complicated by Acute Cerebral Infarction and Acute Myelitis. *Journal of Biosciences and Medicines*, **11**, 303-309.
https://doi.org/10.4236/jbm.2023.115022

Received: March 30, 2023
Accepted: May 22, 2023
Published: May 25, 2023

Abstract

Background: Acute upper gastrointestinal bleeding in liver cirrhosis combined with acute cerebral infarction is uncommon in clinical work, and then combined with acute myelitis is even rarer and more complex, which poses a greater challenge to clinical diagnosis and treatment. This paper reports a case of acute upper gastrointestinal bleeding in liver cirrhosis complicated by acute cerebral infarction and acute myelitis, which be hoped to provide a reference for clinical work. **Methods:** We retrospectively evaluated the clinical information of a 68-year-old female admitted to the Digestive Medical Department with acute gastrointestinal bleeding and appeared limb movement disorder on the third day. **Results:** The patient was eventually diagnosed with acute upper gastrointestinal bleeding in liver cirrhosis complicated by acute cerebral infarction and acute myelitis. **Conclusions:** When patients with liver cirrhosis have abnormal neurological symptoms, in addition to liver cirrhosis-related complications, doctors need to consider cerebrovascular diseases and myelitis.

Keywords

Liver Cirrhosis, Upper Gastrointestinal Bleeding, Acute Cerebral Infarction, Acute Myelitis

1. Introduction

Acute upper gastrointestinal bleeding and neurological injury are common complications of advanced liver cirrhosis. When patients with liver cirrhosis present with abnormal neuropsychiatric symptoms, the first clinical cause to consider may be hepatic encephalopathy, hepatic myelopathy or acquired hepa-

tocerebral degeneration. But the possibility of a combination of other neurological diseases should also be considered. Cerebral infarction is the most common type of stroke, but patients with liver cirrhosis are more likely to be complicated with cerebral hemorrhage, which may be related to thrombocytopenia, abnormal coagulation and abnormal fibrinolytic function with liver cirrhosis. However, cases of liver cirrhosis complicated with acute cerebral infarction can still be reported in clinical work. Previous studies have suggested that the causes of acute upper gastrointestinal bleeding in liver cirrhosis complicated by cerebral infarction are related to factors such as reduced effective circulating volume after acute bleeding, compensatory thrombocytosis by bone marrow, and inappropriate application of hemostatic agents [1]. Acute myelitis refers to myelitis caused by allergic reaction induced by infection or other reasons. At present, the etiology is not clear. The clinical characteristics are limb paralysis, sensory loss and autonomic nervous dysfunction below spinal cord lesion [2]. In fact, it is rare to see acute upper gastrointestinal bleeding, acute cerebral infarction and acute myelitis in liver cirrhosis during the short-term course of the disease. Due to the three diseases may present with neurological manifestations including mental changes and impaired limb movement, which may lead to misdiagnosis and omission because of the lack of meticulous questioning and physical examination. In this paper, we report a case of acute upper gastrointestinal bleeding in liver cirrhosis complicated with acute cerebral infarction and acute myelitis, which provides a reference for early prevention, recognition and treatment of multiple neurological diseases in cirrhosis in clinical work.

2. Case Presentation

The patient, a 68 year-old female, was admitted to the hospital due to "hematemesis twice". She experienced hematemesis for 2 times without obvious inducement 20 hours ago, with a total volume of about 300 ml, without melena. The auxiliary examinations indicated moderate anemia and thrombocytopenia (see Table 1). Upper abdominal ultrasound results showed liver cirrhosis with ascites (small amount). The patient used to be diagnosed with "liver cirrhosis, ascites and chronic severe hepatitis" in 2016, and was considered that the hepatolenticular degeneration is not excluded after testing immune antibody and liver biopsy. Her gastroscopy rusults indicated esophageal varices (moderate) in July

Table 1. Blood test results of different dates.

	Day 1	Day 2	Day 3	Day 4	Day 7
WBC ($\times 10^9$/L)	7.1	4.2	5.0	6.3	4.3
RBC ($\times 10^{12}$/L)	2.35	2.06	2.58	2.98	2.46
HGB (g/L)	75.0	64.0	83.4	94.0	79.0
PLT ($\times 10^9$/L)	67.0	50.0	50.4	64.0	33.0
D-Dimer (ng/ml)	-	232.00	-	2115	2072

2019. She also had a history of cerebral infarction 20 years ago. Physical examination showed that the patient had a blood pressure of 126/59mmHg, conscious mind, anemic appearance, liver palm (−), spider nevus (−). No abnormalities were detected in cardiopulmonary examination. The patient's whole abdomen was soft, without pressing pain or rebound tenderness. The liver, spleen, and subcostal area were not detected. Shifting dullness was negative, the borborygmi were 5 times/minute, and the lower limbs were not swollen. Therefore, the patient was initially diagnosis with liver cirrhosis with acute upper gastrointestinal bleeding. After admission, the patient was given symptomatic and supportive treatment such as acid suppression, lowering portal pressure, rehydration, prevention and control of electrolyte disorders, blood transfusion, etc. On the third day of admission, the patient carried out gastroscope test and **Figure 1** shows esophagogastric variceal, so she was given sclerotherapy. On that day, the patient complained of right lower limb weakness for 6 hours, and was unable to move. Physical examination showed right lower limb muscle strength was grade 0, the remaining limb muscle strength was grade 5, pathological signs on the right side were positive, but not on the left side, and no obvious positive signs were observed in other limbs. D-Dimer significantly increased compared to before (see **Table 1**). **Figure 2** shows multiple infarcts in the cerebellar hemisphere. Doctors considered to diagnosised cerebral infarction (acute-subacute stage), spinal cord infarction? Subacute combined degeneration of the spinal cord? And we temporary gave lipid regulating and stabilizing treatment to the patient. On the 4th day, the patient's right lower limb muscle strength was grade 1+ and tendon reflex (+). The cranial MRI (stroke 4 series) suggested that her multiple cerebral infarcts in bilateral frontoparieto-occipital cortex, corpus callosum pressure and bilateral cerebellar hemispheres (acute-subacute stage), and more infarct foci than before. Her MRI of cervical + thoracic spine suggested she may combine with acute myelitis. On the basis of providing some treatments, such as suppressing acid, lowering portal pressure, preventing and controling electrolyte disorder, regulating and stabilizing lipid, the patient still had right lower limb mobility disorder. Physical examination suggested her right lower limb muscle strength was grade 2 and bilateral Bartholin's signs were positive. Her brain MRA and Enhanced MRI of the thoracic spine (**Figure 3**) results further indicate the diagnosis for acute cerebral infarction and acute myelitis. The Neurologist considered that hepatic myelopathy, spinal cord embolism and ischemic myelopathy could not be excluded in the general consultation of neurology department.it was suggested to temporarily give guaranteed cerebral perfusion, nerve nutrition and rehabilitation treatment, etc. The patient was given endoscopic sclerotherapy, acid suppression, portal vein pressure reduction, hemostasis, intestinal flora regulation, anti-infection, hematopoietic raw material supplement, cerebral perfusion protection, anti-hepatic encephalopathy, nerve nutrition, lipid-lowering and plaque stabilization, rehabilitation exercise and other treatments, after which the patient's symptoms were improved and discharged.

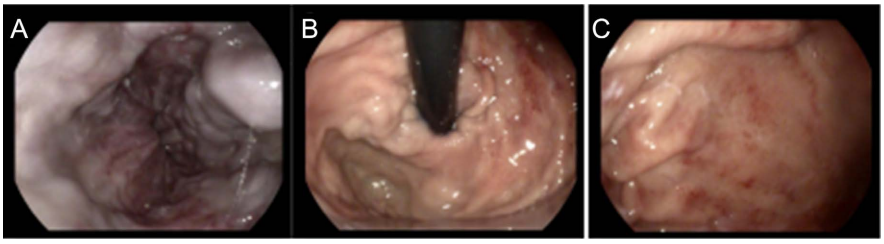

Figure 1. Veins of esophagus and stomach were varicose severly, and the gastric mucosa shows patchy changes, which suggested esophagogastric varices (severe) and portal hypertensive gastropathy.

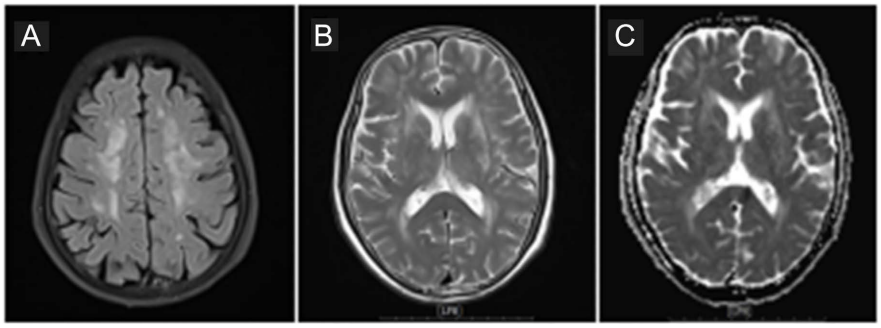

Figure 2. Multiple infarcts in the cerebellar hemisphere on cranial MRI.

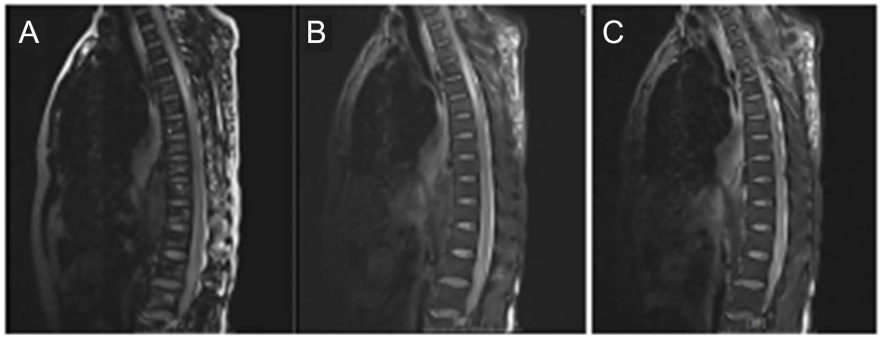

Figure 3. Swelling of the thoracic medulla at the level of thoracic 3 - 6 with mild delayed enhancement suggested acute myelitis.

3. Discussion

The pathogenesis of cerebral infarction mainly includes 3 types of thrombosis, embolism and hypoperfusion. Combined with the pathophysiological mechanisms of liver cirrhosis, the possible causes of acute cerebral infarction complicated by liver cirrhosis are: 1) abnormal coagulation and fibrinolysis. Patients with liver cirrhosis have abnormal coagulation and fibrinolysis, which can progress to a hypercoagulable state when stimulated by certain factors, leading to thrombotic events [3] [4]. Increased platelet aggregation and activation [5] [6], coupled with the use of hemostatic drugs, may increase the risk of thrombosis and induce acute cerebral infarction. 2) Insufficient effective blood volume. Acute upper gastrointestinal bleeding, large amount of ascites and improper use of diuretics can lead to effective blood volume decrease, vasoconstriction, cere-

bral hypoperfusion and finally cause cerebral infarction in patients with liver cirrhosis. 3) Anemia. Hemorrhage, abnormal hematopoiesis, hemolysis, ascites formation, these reasons lead to anemia. Therefore, blood oxygen carrying capacity is decreased, which resulting in decrease of oxygen supply of cerebral terminal artery, vasoconstriction and decrease of cerebral blood supply, and cause cerebral infarction possibly. 4) Infection. Infection is a common complication of liver cirrhosis. Pathogens such as bacteria, fungi and viruses can increase the susceptibility to cerebral infarction by promoting atherosclerosis, inflammation and local thrombosis.

The most common clinical type of acute myelitis is acute transverse myelitis, which may be associated with direct infection, post-infection autoimmune response and autoimmune disease. The neurological symptoms of acute myelitis appear rapidly and may manifest as single or bilateral lower limb weakness and gradually progresses upward. White blood cell count usually is normal or mildly elevated and protein content is normal or mildly elevated in the cerebrospinal fluid examination of acute myelitis. MRI of the spinal cord shows thickening of the spinal cord segments and T2-weighted intra-medullary lamellar or abnormal signal changes, mainly in the upper thoracic and lower cervical segments, often centered on T3-4 and continuing up and down several segments. The acute phase of treatment can be treated with high-dose glucocorticoids. The etiology of acute myelitis is not clear, even though cases of viral hepatitis and primary biliary cirrhosis complicated by acute myelitis have been reported at home and abroad [7] [8] [9] [10] [11], but cirrhosis complicated by acute myelitis is rare in clinical practice and there are few related studies. Combined with previous studies, the possible causes of cirrhosis complicated by acute myelitis are considered: 1) direct cytotoxic effects of hepatitis virus or immune effects secondary to infection; 2) autoimmune antibody effects. Several autoimmune diseases, including primary biliary cirrhosis and dry syndrome, are associated with acute myelitis [12], which may be related to the anti-neuronal effect of autoimmune antibodies [13]. 3) Infections. Infection-induced metaplasia by hepatitis B virus or other pathogenic bacteria is a possible cause of acute myelitis.

The etiology of cirrhosis in this patient is unknown. This patient started with acute upper gastrointestinal bleeding, followed by sudden onset of right lower extremity weakness, and was diagnosed with concomitant cerebral infarction and acute myelitis after examination, which was considered to be mainly related to acute blood loss followed by a decrease in effective blood volume and a hypercoagulable state of blood, which induced acute cerebral infarction. Because the patient developed multisystem infections of the respiratory, urinary and digestive systems at a later stage, the development of acute myelitis was considered to be probably related to the antecedent infection. In conclusion, the patient's condition was severe and complex, and the various measures to improve cerebral blood circulation (such as thrombolysis, antiplatelet, anticoagulation, fibrin lowering, etc.) required for the treatment of acute cerebral infarction and the

high-dose glucocorticoid shock required for the treatment of acute myelitis were in conflict with the hemostatic treatment of upper gastrointestinal bleeding in cirrhosis, so the patient's condition needed to be integrated and individualized. For this patient, the clinician gave therapeutic measures such as acid suppression, lowering portal pressure, appropriate hemostasis, regulating intestinal flora, replenishing hematopoietic materials, protecting cerebral perfusion, anti-hepatic encephalopathy, nourishing nerves, lowering lipid and stabilizing plaque, and rehabilitation exercises.

4. Conclusion

In clinical work, when patients with cirrhosis develop neurological symptoms, in addition to considering complications of cirrhosis such as hepatic encephalopathy, hepatic myelopathy, acquired hepatocerebral degeneration. It is also necessary to identify the presence of cerebrovascular disease, acute myelitis and other neurological diseases. Through check the Physical examination meticulously and improve the relevant auxiliary examinations to make a clear diagnosis, and achieve early prevention, early detection, early treatment, improving the survival rate of patients.

Funding

National Natural Science Foundation of China (81600422).

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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