Case Report

Partial Splenic Embolization That Improved Esophageal Varices and Facilitated Antiviral Therapy in a Case of Cirrhosis Due to Hepatitis C

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A 57-year-old man with hepatitis C cirrhosis experienced sudden hematemesis and was brought to the hospital via ambulance. He underwent endoscopic variceal ligation for ruptured esophageal varices (EV). His platelet count was reduced to $6 \times 10^4$ g/dl and splenomegaly was observed. Therefore, partial splenic embolization was performed. However, a complicating portal vein thrombus required thrombolytic therapy with warfarin. His platelet count increased to $15 \times 10^4$ g/dl, the EV did not worsen, and there was no complicating hepatocellular carcinoma (HCC). Thus, a planned 24-week course of combined pegylated-interferon (Peg-IFN)-α2a and ribavirin was initiated. Neutropenia occurred during therapy, which led to a reduction in the dose and frequency of Peg-IFN-α2a administration. A sustained viral response (SVR) was obtained after completion of the treatment. The SVR has persisted up to 6 years since the EV rupture. The patient’s platelet count remains $15 \times 10^4$ g/dl, the liver function remains normal, the EV have improved, and there is no complicating HCC.

Key words: hepatitis C cirrhosis, esophageal varices, partial splenic embolization, portal vein thrombus, antiviral therapy

Introduction

Currently, about 85% of hepatocellular carcinoma (HCC) cases derive from hepatitis B virus (HBV) or hepatitis C virus (HCV). As liver fibrosis advances, the frequency of HCC is said to increase. Moreover, with liver cirrhosis (LC), complications tied to poor prognosis, may occur and lead to liver failure. If the cause is HBV or HCV, it is important to eliminate the virus and stabilize liver functions via antiviral therapy, if possible. In this case, esophageal varices (EV) complicating hepatitis C cirrhosis ruptured twice. After endoscopic therapy, partial splenic embolization (PSE) was performed for splenomegaly, then thrombolytic therapy was performed for a portal vein thrombus (PVT). Then, combined therapy of intermittent low-dose pegylated-interferon (Peg-IFN) α2a and continuous ribavirin (RBV) was given. This obtained a sustained viral response (SVR). At present, 6 years since the first examination, the patient is doing well with improved platelet count and EV, and no HCC.

Case Presentation

The patient here was a 57-year-old man. In April 2008 he experienced sudden hematemesis and was brought to the hospital by emergency transport. An upper gastrointestinal endoscopy showed the bleeding had stopped naturally. He was diagnosed with EV (F2RC2) and endoscopic variceal ligation (EVL) was performed immediately (Fig. 1-A, B). After this he stopped coming to the hospital of his own accord, but in July the EV again hemorrhaged and a second EVL was performed. He was then admitted to the hospital for a PSE to address low platelet count and prevent worsening of the EV. The patient at age 16 years received a blood transfusion after being injured in a traffic accident. At age 43 years a local doctor suspected LC but the patient did not come to the hospital regularly. He had a history of alcohol consumption of 500 ml per day of beer for 35 years. He had no smoking history. Table shows examination findings from the time of admission, which led to a diagnosis of hepatitis C cirrhosis by genotype 1b. The contrast enhancement computed tomography (CE-CT) showed atrophy of the liver, an irregular surface and blunt margin, as well as splenomegaly and collateral circulation, which are compatible with LC. No ascites were found (Fig. 2-A, B). After admission, the low platelet count was determined to be due to hypersplenism accompanying splenomegaly, and the EV to derive from the left and posterior gastric veins. According to the treatment guidelines on Ministry of Health, Labour and Welfare, the adaptation standards of the PSE become less than platelet 50,000. There was 60,000 this example, but judged it with PSE
Fig. 1
A, B: Findings from the esophageal endoscopy at the initial examination.
EV was CBRC2F3Lm.
C, D: Findings from the esophageal endoscopy about 6 years after the initial exam.
EV was CWRC0F1Lm.
EV: esophageal varices, CB: color blue, RC: red color sign, F: form

Fig. 2
A, B: CE-CT at the initial examination.
Atrophy of the liver, splenomegaly, and collateral circulation are observed.
C: CE-CT after PSE.
PVT seen in the right portal vein branch (▶).
There are areas of poor contrast enhancement in the spleen from PSE.
D: CE-CT after warfarin session.
The PVT has disappeared from the right portal vein branch (▶).
Artifacts of coils from PSE are observed in the spleen.

CE-CT: Contrast enhancement-computed tomography, PSE: partial splenic embolization, PVT: portal vein thrombus
adaptation because I thought that I needed enough security (even at least 80,000) of the platelet on using interferon in future. PSE was performed in February 2010. An infarction rate of about 70% was obtained but the platelet count did not rise satisfactorily so a second PSE was done in June 2010, which obtained a

PSE: partial splenic embolization, PVT: portal vein thrombus, Peg-IFN: pegylated-interferon
final infarction rate of about 80%. The platelet count then gradually improved, but a PVT was found in the right portal vein branch. This was thought to be due to PSE (Fig. 2-C), so in October 2010 thrombolytic therapy using warfarin was initiated. This took a long time, but after 10 months, the PVT had completely disappeared (Fig. 2-D). At this point the platelet count had risen to $15 \times 10^3$ g/dl, ALT 13, TB 0.8, there was great improvement of the EV (Fig. 1-C, D), and there is no HCC. In this case, multidisciplinary therapy including PSE was observed, so the Peg-IFN-$\alpha_2a$ dose was reduced to 30 $\mu$g (usually 90 $\mu$g) and it was administered intermittently. Treatment finished in December 2012, obtaining a SVR (Fig. 3). HCV-RNA has not reappeared. Six years after hematemesis occurred (August 2014), the patient’s platelet count was $15 \times 10^3$ g/dl, ALT 13, TB 0.8, there is great improvement of the EV (Fig. 1-C, D), and there is no HCC. In this case, multidisciplinary therapy including PSE was successful for hepatitis C cirrhosis.

**Discussion**

Prognostic factors for cirrhosis include (1) esophageal and gastric varices, (2) uncontrollable hepatic coma, (3) hyponatremia, (4) hepatorenal syndrome, and (5) HCC. In a majority of cases of HCC, there is a background of chronic liver disease caused by HBV or HCV. In particular, patients chronically infected with HCV make up 60%, so onset is often discovered through LC. Antiviral therapy is important for preventing these complications. Ever since interferon was shown to completely eliminate the virus, antiviral therapy has undergone major changes in Japan over the past more than 20 years. Combined Peg-IFN and RBV, which was mainstream at the time, could be administered. Although neutropenia led to the dose being lowered and intermittent administration, treatment was completed without any major complications. The SVR has continued up to the present, about 2 years. As improvements in platelet count and EV were observed more than 6 years after the initial examination in this case, this suggests the importance of close observation during treatment and of not giving up on multidisciplinary therapy. In the future, we would also like to say that even if HCC occurs, treatment can be administered without hindrance.

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