

• CASE REPORT •

Successful rescuing a pregnant woman with severe hepatitis E infection and postpartum massive hemorrhage

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Abstract

AIM: To sum up the experience of the successful therapy for the severe hepatitis of pregnant woman with postpartum massive hemorrhage.

METHODS: The advanced therapeutic methods including the bilateral uterine artery embolism, hemodialysis and artificial liver support therapy were performed with comprehensive medical treatments and the course of the successful rescuing the patient was analyzed.

RESULTS: Through the hospitalization of about two months the patient and her neonatus had gotten the best of care in our department and pediatric department separately. Both of them were discharged in good condition.

CONCLUSION: The key points for a successful therapy of the pregnant woman with severe hepatitis are termination of the pregnancy and the control of their various complications. It was suggested that the proper combination of these measures of modern therapy would race against time for renewing of hepatic and renal functions.

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INTRODUCTION

Hepatitis E infection is the most common cause of severe hepatitis in pregnancy, being seriously dangerous for both maternal and fetal life with a mortality rate of 58 %, even up to 100 % if a postpartum massive hemorrhage occurs^[1-4]. A pregnant woman with severe hepatitis E infection complicated with postpartum massive hemorrhage and fulminant hepatic and acute renal failure is presented in this report. With the comprehensive medical treatments in an intensive care setting and three advanced therapeutic measures including bilateral

uterine artery embolism, hemodialysis and artificial liver support therapy, a satisfactory outcome of both the mother and child was achieved.

CASE REPORT

A 24-year-old pregnant woman was admitted to our hospital in May 11th 2002 and presented with a 31-week pregnancy and an 11-day history of asthenia, abdominal distention with deep jaundice. On examination she had a blood pressure of 20/12 kPa, peripheral and facial puffiness, icteric sclera and xanthochromia. The fetal heart rate was 140 beats/min. Laboratory findings showed a normal white blood cell count and a decreased platelet counts ($76 \times 10^9/L$) with a hemoglobin level of 102 g/L. Liver function tests were obvious abnormal (albumin 25.9 g/L, bilirubin 579 $\mu\text{mol/L}$, alanine transaminase 158 IU/L). Prothrombin time activity (PTA) was 21.5 %. The markers of hepatitis virus showed negative for hepatitis A, C and D, positive for anti-HB surface and anti-HE (IgM). So a diagnosis of fulminant hepatic failure of pregnancy (FHFP) with hepatitis E infection was made.

A further clinical course gave a risk signal for the maternal and fetal life. After admission, the gravid woman in the intensive care unit was treated by catharsis with lactulose in order to terminate the pregnancy. With the regular uterine contraction and colporrhagia that appeared as parturient signals, she was immediately sent to the childbearing ward for parturition. After the childbirth of a 1 300 g-weight male baby who was sent to the pediatric care ward, the patient developed postpartum massive hemorrhage. Her blood pressure dropped to 10.7-6.67/6.67-4.0 kPa, at a minimum falling to 0/0 kPa. With the drug hemostasis, such as uterotonic, thromboplasin, and p-amino-merthyl bezoic acid, her colporrhagia could not be stopped. The total of her hemorrhage was about 3 500 ml, and she was on the verge of death. Therefore, it was decided to perform the bilateral uterine artery embolism by the method of intervention. As a result, her colporrhagia was effectively controlled. At the same time fresh blood was transfused together with albumin, and her blood pressure rose up to 15.5/11.5 kPa with the normal vital signs.

After a dangerous delivery the patient underwent a clinical course of exacerbation with hepatic encephalopathy, hepatic renal syndrome, bacterial infection of puerperium and disseminated intravascular coagulation (DIC). The parturient presented fever (to 39.8 °C), oliguria (<400 ml/d) and confusion. The physical signs included severe edema of the whole body, intensive stasis of blood under subcutaneous layer of the abdominal wall and bilateral groin, and huge amount of hemorrhagic ascites. Laboratory study showed that the white blood cell count was $28.8 \times 10^9/L$, N 0.89, Hb $49 \times 10^9/L$, serum creatinine 583.3 $\mu\text{mol/L}$, blood urea nitrogen 24.2 $\mu\text{mol/L}$, blood NH_3 164 $\mu\text{mol/L}$. The tests for DIC revealed thrombocytopenia ($42 \times 10^9/L$), prolonged prothrombin time (PT 32.8 seconds, normal range from 11 to 14 seconds) and active partial thromboplastin time (APTT 88.5 seconds, normal range from 25 to 36 seconds), and hypofibrinogenemia (0.23 g/L).

It was the key time to rescue her hepatic and renal function. The comprehensive medical treatments including diuresis,

hemostasis (antifibrinolytic agents and prothrombin complex) was used with antibiotics for controlling infection and a large number of albumin for improving hypoproteinemia, and in the meanwhile twice hemodialysis for getting rid of the toxic metabolite and the much abundance of her body water were performed. And so the renal and coagulative functions were effectively improved with the increase of her urine volume and the reduction of her whole body edema. However, the improvement of her hepatic function was not significant, total bilirubin remaining at 468 $\mu\text{mol/L}$. Therefore, it was decided that the artificial liver support therapy, an advanced therapeutic approach, was carried out and then the patient got better in her liver function and clinical syndromes with a good appetite. Through the hospitalization of about two mouths the patient and her neonatus had gotten the best of care in our department and pediatric department separately. Both of them were discharged in good condition.

DISCUSSION

Viral hepatitis during pregnancy is common in China^[5-13]. It is one of the most dangerous conditions with a high mortality both for the mother and her fetus, particularly when fulminant hepatic failure occurred^[2,3]. Severe hepatitis is the main cause of FHF in terms of the trimester of gestation^[4]. The pregnant woman with severe hepatitis presents frequently hypodynamia, deep jaundice, coma, hypoproteinemia and sometime acute renal failure^[5]. The patient in this report had a 31-week pregnancy developed severe hepatic dysfunction with deep jaundice (TB 579.9 $\mu\text{mol/L}$, PTA 21.5%), and a positive anti-HE-IgM in her serum, therefore severe hepatitis of pregnancy with HEV infection was diagnosed. It was reported that there were many viruses causing severe hepatitis of pregnancy, but HEV and HBV were frequent, especially HEV infection. The mortality rate was highest (56 %) among HEV infected FHF cases during the third trimester of pregnancy^[4, 14].

Intensive care facilities and an early diagnosis are essential for the management of pregnant patient with severe liver disease^[1,2,14]. The key points for a successful therapy of them are termination of the pregnancy on the proper time and the control of their various complications. It was reported that the severe hepatitis of pregnant woman was with a high mortality rate, even up to 100 % if a postpartum massive hemorrhage and hepatic renal syndrome occur. In this case she presented the very severe hepatic dysfunction and coagulative dysfunctions, and complicated with postpartum massive hemorrhage, bacterial infection, hepatic encephalopathy and hepatic renal syndrome meanwhile or continuously. Therefore the treatments of her were usually very difficult and contradictory.

We considered that the key points of the successful rescuing the patient were on the proper time to adopt three important therapeutic measures. The first was to perform decisively on proper time the bilateral uterine artery embolism by the intervention that is an advanced method with a smaller damage in comparison of the complete hysterectomy and effectively stopped the postpartum massive hemorrhage. The second was to use twice hemodialysis by which removed the toxic metabolite and the abundance of water and the proper comprehensive medical treatments for the improvement of her

renal and coagulative function. The third was to carry out in time the artificial liver support therapy that provides a temporary liver support and promotes the spontaneous liver regeneration in the patient with the remove of toxic metabolic products and the decrease of total bilirubin^[15-18]. It was suggested that the proper combination of these measures of modern therapy would race against time for renewing of hepatic and renal functions, and therefore a better outcome of treatment was anticipated.

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