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CASE REPORT

Reversal of Ischemic Hepatitis after Cardiac Surgery Under Cardiopulmonary Bypass: A Case Report

Lokesh Shekher Jaiswal[1] • **Narendra Pandit**[1] • **Jagat Narayan Prasad**[2]

Abstract

Acute hepatic failure due to ischemic hepatitis is associated with high mortality. The safety of cardiopulmonary bypass in this setting is not fully described. Here we report a case of a 21-year-old female who developed an acute fulminant hepatic failure due to ischemic hepatitis following a cardiogenic shock. She underwent subsequent successful mitral valve replacement under cardiopulmonary bypass, thus providing an evidence of its safety in acute fulminant hepatic failure.

Keywords: acute fulminant hepatic failure, cardiopulmonary bypass, ischemic hepatitis

Ischemic hepatitis (IH) is a rare cause of acute liver injury (ALI).[1] Severe liver dysfunction due to ischemia can occur in patients of congestive cardiac failure with associated hypotension. The possible pathophysiologic mechanism is a sudden and profound reduction in systemic blood pressure which leads to a reduction in hepatic blood flow with subsequent hypoxia of hepatocytes and IH. Hepatic congestion due to severe tricuspid regurgitation and right heart failure are additional contributors for IH.[2] The presence of an acute elevation of liver enzymes to at least 20 times the upper limit of normal in the appropriate clinical setting has been accepted as criteria for IH.[3] Postoperative morbidity and mortality after cardiopulmonary bypass (CPB) are reported to be high in patients with advanced liver cirrhosis.[4] However, the safety of CPB in ALI of ischemic cause is not fully described. Here we describe a case of an acute fulminant hepatic failure due to ischemic hepatitis following cardiogenic shock, who underwent successful cardiac surgical intervention requiring CPB. The patient gave her written consent to use case information and images for educational and publication purposes.

CASE

A 21-year-old female presented with shortness of breath and easy fatigability for one year. She was evaluated with an echocardiogram which revealed severe mitral stenosis and severe tricuspid regurgitation with hepatomegaly. She underwent percutaneous transmitral commissurotomy (PTMC), following which she developed acute cardiac failure with hypotension. The echocardiogram showed prolapsed anterior mitral leaflet due to ruptured chordae and papillary muscle causing severe mitral regurgitation. She was not responsive to medical management of maximum inotropes (dopamine 30 µg/kg/min and dobutamine 30 µg/kg/min) and vasopressors (noradrenaline 0.3 µg/kg/min, vasopressin 0.1 units/minute) support and therefore was referred to us for emergency mitral valve replacement after forty hours. Her pre-PTMC liver function test was within the normal range. Her blood investigations before surgery showed severely deranged liver functions (total bilirubin 12.1 mg/dl, aspartate transaminase (AST) 7,373 U/L, alanine transaminase (ALT) 2,140 U/L), coagulopathy (INR > 5) with thrombocytopenia (platelet count 34,000 cell/mm^3), hypoglycemia and impaired renal function. She developed ALI due to low cardiac output. Hence, she was scheduled for emergency surgical correction. Total intravenous anesthesia consisting of midazolam, propofol, fentanyl, and vecuronium was used for general anesthesia. A median sternotomy was performed followed by routine aortic and bi-caval cannulation. CPB was established using membrane oxygenation, roller pump perfusion, and moderate systemic hypothermia. During CPB, hematocrit was maintained at 22 to 25%, perfusion flow at 2.6 to 2.8 L/min/m^2, mean arterial pressure between 60 and 70 mmHg, and systemic temperature around 32°C. There was a rupture of the anterolateral papillary muscle leading to severe mitral regurgitation. Mitral valve replacement, tricuspid valve repair and closure of atrial septal defect was performed. The total bypass time and cross-clamp time were 60 and

Lokesh Shekher Jaiswal
lokesh.jaiswal@bpkihs.edu
[1] Department of Surgery
[2] Department of Anaesthesiology and Critical Care
BP Koirala Institute of Health Sciences, Nepal

40 minutes respectively. Postoperatively, she was kept on mechanical ventilation and inotropic support to maintain cardiac output. From the third postoperative day (POD), her liver enzymes (AST 2,560 U/L, ALT 1,984 U/L) and renal function started improving. Her coagulation profile also improved from the fourth POD (INR 2.3). However, her bilirubin showed a progressive increase till the eighth POD (total bilirubin 22 mg/dl). On the sixth POD, ascites was noticed that was managed with diuretics. Her sensorium gradually improved and inotropes were progressively tapered off. She was extubated successfully on the tenth POD. She had complete normalization of her liver function (total bilirubin 1 mg/dl, AST 40 U/L, ALT 50 U/L). She was discharged in satisfactory condition on the sixteenth POD with an INR of 2.4 on warfarin.

At one year of follow-up, she was doing well with New York Heart Association functional class I. She had a normal left ventricle and normal prosthetic valve function on echocardiogram and normal liver function test.

DISCUSSION

ALI due to a cause other than acetaminophen toxicity tends to have a worse prognosis and needs a liver transplant.[1] Natural history of ALI due to ischemia in the previously normal liver is not fully described in the literature. Liver has very high regenerative capacity and timely removal of ischemia can lead to return of normal function. Some amount of derangement in liver enzymes can occur after CPB. A few patients with a low physiologic reserve and chronic cardiac failure can develop severe liver dysfunction with very high mortality.[5,6] However, the outcome and safety of CPB in patients with preoperative ALI of ischemic cause is not well established. In a case report by Lim et al, acute cardiac failure with liver dysfunction following infective endocarditis was successfully managed surgically with the use of cardiopulmonary bypass.[7] In our case, the cause of ALI was low cardiac output due to severe mitral regurgitation after failed PTMC on pre-existing severe tricuspid regurgitation and hepatic congestion, which lead to IH. Cardiac surgical intervention under CPB can theoretically lead to a worsening of liver dysfunction and may require a liver transplant. However, if bypass time is kept minimal and the cause of ischemia is corrected on time, reversal of liver injury is expected, as in our case. In present case, once the cause of ischemia was removed and cardiac output maintained, liver function resumed normally. Further studies are required to demonstrate the safety and feasibility of CPB in acute liver failure.

CONCLUSION

We safely performed surgical intervention under CPB in a patient with acute fulminant hepatic failure due to ischemic hepatitis of correctable cardiac cause. The liver function returned to normal after treatment of ischemia.

DECLARATIONS

Ethics approval and consent to participate: Not applicable

Consent for publication: Obtained from the patient

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request. All relevant data are within the manuscript and its supporting information files.

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