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Case Report

A Case of Irreversible Liver Failure with Cardiogenic Shock

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ABSTRACT

Although liver failure caused by heart failure is mostly reversible, we herein experienced a report presented with irreversible LF and acute liver atrophy in patients with cardiogenic shock. A 72 years old woman was diagnosed with cardiogenic shock with acute liver insufficiency. Management focusing on the underlying acute HF improved heart function. However, liver dysfunction got gradually worse, and she finally died due to not HF, but irreversible liver dysfunction with acute liver atrophy. In cases of severe HF with liver failure, we often devote to treatment of HF with congestive liver or low output liver in mind, but if only liver function does not improve even though HF is being compensated, it is necessary to discuss intervention for the liver, such as artificial liver support and liver transplantation, in consideration of complications of fulminant hepatitis.

Keywords: Cardiogenic shock; Irreversible liver failure; Artificial liver support

INTRODUCTION

Heart Failure (HF) and Liver Failure (LF) often co-exist because of complex cardiohepatic interactions [1]. The most recognized LF in HF includes congestive hepatopathy and hypoxic hepatitis. These types of liver dysfunction need the management of focuses on acute HF [2,3] and improve after HF was compensated. We herein report a woman with HF who develops irreversible LF with acute liver atrophy.

CASE REPORT

A 72 years old woman with a history of paroxysmal Atrial Fibrillation (AF) had suffered from dyspnea and fatigue for two days, and was diagnosed with HF with reduced ejection fraction (HFrEF). She was not frail with a body mass index of 23.4 Kg/m^2 , and had no history of liver disease on admission. She had been admitted to a local hospital and was start on treatment of HF with oxygen, and diuretics (Day 1). The laboratory findings at the local hospital revealed moderate liver and renal dysfunction (Table 1). Blood gas analysis revealed a metabolic acidosis (pH 7.416, base excess-5.1, HCO_3 -18.0 mmol/L, lactate 3.0 mmol/L, and $PaCO_2$ 28.6 mmHg). However, on day 2, she became shock with perspiration, and exacerbation of hepatorenal function at laboratory findings. Then, she was transferred to our hospital on day 2 for further multidisciplinary therapy. On admission to our hospital, a physical examination showed shock with perspiration and pallor, and conscious but non-oriented. Her vital signs showed blood pressure was 92/62 mmHg and her pulse was 140 bpm with irregular narrow QRS rhythm. With 5 L/min nasal oxygen, her oxygen saturation was 95%. A 12-lead Electrocardiogram (ECG) showed AF with rapid ventricular response. A transthoracic echocardiography showed a Left Ventricular Ejection (LVEF) of 20% and moderate mitral valve regurgitation (MR) with left atrial enlargement without left ventricular enlargement. A chest X-ray showed cardiomegaly, pulmonary congestion and pleural effusion (Figure 1). The laboratory findings on our hospital revealed high serum lactate levels, worsening liver dysfunction, and kidney dysfunction (Table 2). It was judged to be multiple organ failure due to cardiogenic shock, and catheter examination was performed after tracheal intubation. Coronary angiography showed no significant stenosis in her coronary arteries, and right heart catheterization showed cardiac output of 1.96 L/min, means pulmonary capillary wedge pressure of 29 mmHg and classified her HF as Forrester class IV. Then, she was diagnosed with cardiogenic shock caused by non-ischemic cardiomyopathy and was placed with an IABP. Tachycardia Induced Cardiomyopathy (TIC) with atrial functional MR was suspected because it was associated with new-onset rapid paroxysmal AF and left ventricular diameter was not enlarged, and no other organic heart disease was noted.

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Table 1: Clinical presentation of demographic data.

	Days					
	1	2	3	5	9	29
Liver function, Heart function, Renal function and lactate level						
T-bil (mg/dL)	1.61	2.83	3.33	6.58	13.6	29.96
AST (IU/L)	220	8779	11557	1270	56	51
ALT (IU/L)	174	2554	3150	1355	234	59
LDH (IU/L)	549	9092	12976	1033	616	748
ALP (IU/L)	549	525	492	410	346	354
GTP (IU/L)	254	193	165	141	85	48
LVEF (%)	20	20	45	55	60	60
Heart rate (/min)	140	136	64	62	97	98
Cre (mg/dL)	2.77	2.68	3.33	1.9	3.92	1.48
Lactate (mmol/L)	3	14.9	3.7	2.9	1.8	8

Abbreviations: ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; Cre: Creatinine; LDH: Lactate Dehydrogenase; LVEF: Left Ventricle Ejection Fraction; T-bil: Total Bilirubin; γGTP: Gamma Glutamiltranspeptidase.

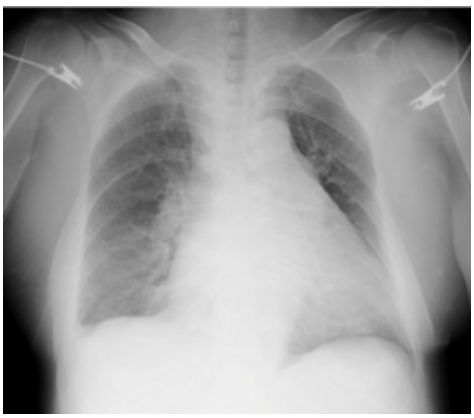

Figure 1: A chest X-ray shows cardiomegaly, pulmonary congestion and pleural effusion.

Table 2: Laboratory data on our hospital admission (day 2).

WBC (/μL)	17600	LDH (IU/L)	9902
RBC (/μL)	316	ALP (IU/L)	525
Hb (g/dL)	9.7	γGTP (IU/L)	193
Ht (%)	30.9	Cre (mg/dL)	2.63
Plt (/μL)	4.6 × 104	PT (%)	10
TP (g/dL)	5.2	APTT (s)	50.6
Alb (g/dL)	3.1	Fibrinogen (mg/dL)	294
T-bil (mg/dL)	2.83	D-dimer (μg/mL)	28
D-bil (mg/dL)	1.98	CRP (mg/dL)	3.62
AST (IU/L)	8779	NH3 (μg/dL)	46
ALT (IU/L)	2554	NT-proBNP (pg/mL)	36661

Abbreviations: Alb: Albumin; ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; APTT: Activated Partial Thromboplastin Time; AST: Aspartate Aminotransferase; Cre: Creatinine; CRP: C-reactive Protein; D-bil: Direct Bilirubin; Hb: Hemoglobin; Ht: Hematocrit; LDH: Lactate Dehydrogenase; NH3: Ammonia; NT-proBNP: N Terminal Pro B Type Natriuretic Peptide; Plt: Platelets; PT: Prothrombin Time; RBC: Red Blood Cell; T-bil: Total Bilirubin; TP: Total Protein; WBC: White Blood Cell; γGTP: Gamma Glutamiltranspeptidase.

Our medical team including intensive care physicians and hepatologists interpreted that liver dysfunction resulted from cardiogenic shock and was expected to improve by the treatment for HF. Therefore, we devoted ourselves to treatment of cardiogenic shock with dobutamine drips for both rhythm and rate control for rapid AF and the IABP for low cardiac output. However, the IABP had to be removed 3 days because of the puncture site oozing-bleeding without difficulty to insert, which was suggested that abnormal coagulation due to LF could not be controlled despite the Fresh Frozen Plasmas (FFP) transfusion every day. We expound the coagulopathy was provoked by Disseminated Intravascular Coagulation (DIC) with unfocused infection disease, and initiated antibiotics (cefepime). After this management, her hemodynamics was stabilized with normal sinus rhythm and re-examined echocardiography on day 3 revealed an improved LVEF of 45%.

However, LF got marked worse despite ameliorated other function such as heart or renal with decreasing serum lactate levels (Table 1). Furthermore, it was difficult to maintain her coagulation system unless FFP was transfused almost every day, suggesting that coagulation factors were not produced from hepatocytes. The CT also showed acute liver atrophy compared with the initial visit on day 11 (Figure 2). Because LF had progressed over time even though HF had been compensated for, it was decided to examine whether there was a cause of fulminant hepatitis. Amiodarone drips were stopped

after AF stopped. Tests for hepatitis viruses, cytomegalovirus and Epstein-Barr virus were all negative. Antibody and drug-induced lymphocyte stimulation for the medicines she had been taking before hospitalization were all negative. We hesitated to perform biopsy for coagulopathy. Despite multidisciplinary treatment and performing further investigation, her liver and coagulation function got worse, and finally she died on day 29 because of liver failure. Autopsy was refused by the family.

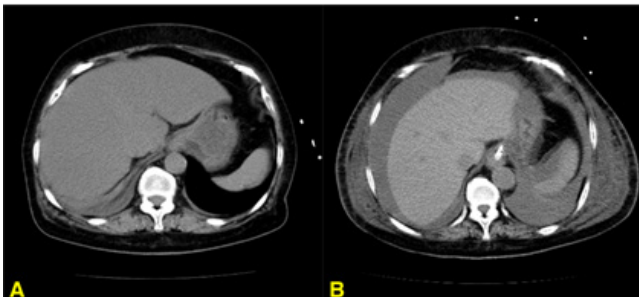

Figure 2: Computed tomography during the initial examination and day 11. Compared with the CT during the (A) Initial visit, there is a rapid atrophy of the liver and increase in ascites on (B) Day 11.

RESULTS AND DISCUSSION

HF is a clinical syndrome caused by the inability sof systemic perfusion and usually present with hepatic dysfunction such as congestive hepatopathy associated with increased central venous pressure transmitted to hepatic sinusoids [4], and hypoxic hepatitis characterized by cellular damage caused by hypoperfusion-induced hypoxia [5]. Both liver diseases are often associated impaired hepatic function characterized by elevation of hepatobiliary enzymes, the normalization of the laboratory tests can equally rapid occur with the liver diseases triggered factor resolves [6,7]. However, the liver dysfunction of the present case was irreversible with acute liver atrophy.

We can assume following two possible mechanisms for this clinical course. One of the probable causes is that LF due to cardiogenic shock might have been prolonged before being transferred to our hospital, which could result in irreversible LF. It has been proposed that in hypoxic hepatitis case, the normalization of the liver functions in laboratory tests can occur within 7-10 days after the triggering factor resolves [8]. On the other hand, little is known about the required time for the factors resolves. In this case, the patient had high liver enzyme levels, and mild metabolic acidosis at the time of admission to the previous hospital, which suggested that cardiogenic shock with LF had already occurred at the time of admission, and the shock might have been prolonged. A slow improvement of lactate levels despite starting treatment including the IABP for cardiogenic shock suggested that treatment response to cardiogenic shock was slower than expected. The second possible mechanism is that LF might have been the cause of fulminant hepatitis whether fulminant hepatitis associated with HF or not. A few studies reported that severe HF is cause of fulminant hepatic failure [9,10] or acute liver atrophy without a history of shock or low cardiac output [11]. The pathological investigation revealed central hepatic necrosis about the central veins in line with past reports [9,10]. In the present case, we cannot completely rule out the possibility that the liver dysfunction of the present case caused by fulminant hepatitis of unknown etiology. Previous study indicated that about 15%-20% of LF occurs with an unknown cause [12].

Although several studies have reported the treatment of liver injury due to HF, most of the treatments were about HF such as inotropic agents, and mechanical circulatory supports [2,6,13]. There were few reports of artificial liver support and liver transplantation for treatment of LF caused by HF [14]. However, this case suggested that this liver replacement therapy should be considered when LF with HF is irreversible. If LF has progressed over time even though HF had been compensated for, we need to discuss the etiology of LF and avoid hesitating to artificial liver support and liver transplantation at appropriate time.

In this case, the unexpected oozing hemorrhage associated with IABP, and the need for daily FFP transfusions to maintain coagulation function could be one of early signs of the irreversible LF associated with HF in the clinical setting. IABP access site-related hemorrhage was noted 7.5% and the risk was IABP duration time and urgently IABP insertion [15,16]. The present case did not have these risks or difficulty to insert. When bleeding event related to IABP occurs in HF patient without these risks, it is necessary to search and intervention for a condition causing a strong coagulopathy

CONCLUSION

From the findings in our case, the LF associated with HF can be occasionally irreversible and fatal factor. If the LF had progressed over time even though HF had been compensated for, we should discuss the possibility of irreversible LF such as fulminent hepatitis, whether or not etiology is HF, and need to discuss the use of aggressive treatment to liver at an early stage. Furthermore, because prolongation of cardiogenic shock can result in irreversible LF, it is important to detect and start to treat before the shock is prolonged.

CONFLICT OF INTEREST

All author report no conflict of interest related to his manuscript.

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