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Case Report

Appearances are Deceptive: Acute Liver Failure After Increased Alcohol Consumption

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Abstract

Our patient was transferred from another hospital to the intermediate care unit. A liver transplantation evaluation was initiated for acute liver failure presumably due to alcohol consumption. The patient presented in a critical condition with impaired vigilance, reduced liver synthesis, liver detoxification disorder, and significant decompensation. Sonographic examination and liver biopsy showed signs of venous congestion. Subsequent echocardiographic examination revealed severe tricuspid regurgitation, which was associated with a ventricular septal defect. Following initiation of diuretic therapy, the patient stabilized and liver function improved. The present case demonstrates the need for thorough work-up of differential diagnoses with interdisciplinary, structured collaboration in treating these patients being critical.

Keywords: Acute Liver Failure; Alcohol-Induced Liver Failure; Ventricular Septal Defect

Introduction

A 65-year-old male patient was admitted to a secondary hospital for reduced vigilance, fatigue, and shortness of breath. The patient reported increased alcohol consumption due to a family stress situation. After admission to the intensive care unit, acute liver failure accompanied by acute renal failure with metabolic acidosis and a serum lactate of 6.5 mmol/L was present. After exclusion of viral and autoimmune hepatitis, alcohol consumption was postulated as cause of acute liver failure (MELD: 32). Due to rapid progression of liver failure, the patient was transferred to the intermediate care ward of our university hospital for evaluation of liver transplantation. Upon admission, the patient was hydropically decompensated and somnolent with oliguric acute renal failure (serum creatinine 171 µmol/L), elevated transaminases (AST 4735 U/L, ALT 4047 U/L) with synthesis disturbance (INR 5.42, Factor II activity 49.2%, Factor V activity 7.2%, Factor XII activity 34.4%) and increased serum lactate 7.8 mmol/l. ECG analysis showed R-wave reductions in leads III, aVF (Figure 1A-B), and

creatinine-kinase was mildly elevated (CK 176 U/l). Sonography of the liver revealed venous congestion and holosystolic flow reversal in the inferior vena cava. A liver biopsy was performed for further evaluation. Histopathological analysis showed no signs of cirrhosis and alcohol-induced necrosis but revealed venous congestion. Liver failure was completely reversed with diuretic therapy (Figure 2). Hemodynamic evaluation (Swan-Ganz catheter) showed evidence of postcapillary pulmonary hypertension and was indicative of an intracardiac shunt (Table 1). Subsequent transthoracic and transesophageal echocardiographic examination revealed severe tricuspid regurgitation with a large ventricular septal defect (VSD) (Figure 1 C-F). Cardiac magnetic resonance imaging (Figure 1G) showed an ischemic scar. Cardiac catheterization revealed an occlusion of the right coronary artery (RCA) (Figure 1H), respectively. The patient was admitted to the cardiac intensive care unit for preparation of surgical VSD closure that was performed later.

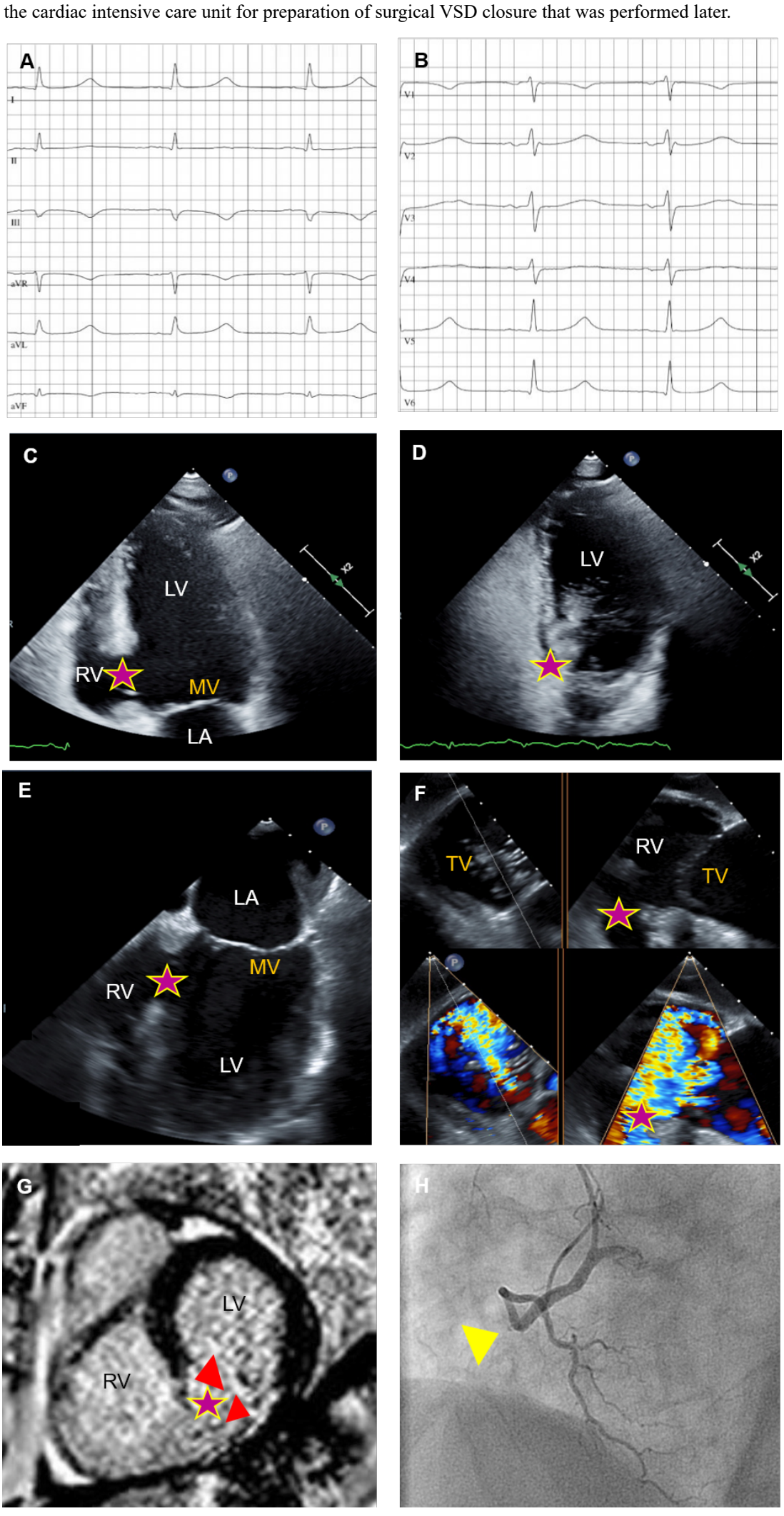

Figure 1: Cardiological work-up A-B: 12-lead ECG without signs of acute myocardial ischemia

C-D: Apical four-chamber view in transthoracic echocardiography displayed a ventricular septal defect (violet asterix). After applying right ventricular contrast media Figure 2D showed a bidirectional shunt with contrast media in the left ventricle. RV: right ventricle. LV: left ventricle. LA: left atrium. MV: mitral valve.

E-F: Transesophageal echocardiography confirmed a ventricular septal defect (violet asterix). In a transgastrical view (F) was the tricuspid valve (TV) regurgitation severe with a ventricular septal defect (violet asterix) close to the tricuspid valve. RV: right ventricle. LV: left ventricle. LA: left atrium. MV: mitral valve. TV: tricuspid valve.

G: In a short axis view in magnet resonance tomography transmural late gadolinium enhancement (red arrow) in the ventricular septal defect (violet asterix) revealed evidence of ischemic origin.

F: In a coronary angiography, there was a chronic total occlusion of the mid right coronary artery (RCA) (yellow arrow).

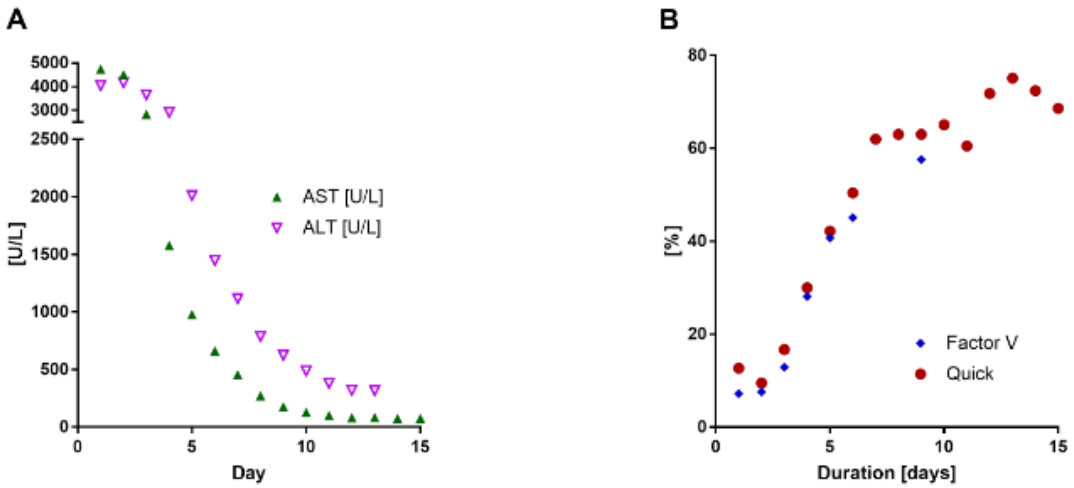

Figure 2: Time course of liver failure A: A) X-axis concentration of liver enzymes (AST and ALT). Y-axis time in days under diuretic therapy. AST: aspartate aminotransferase. ALT: alanine aminotransferase. B: B) X-axis percentage of Quick and Factor V. Y-axis time in days under diuretic therapy.

Parameter	Value
PAP mean [mmHg]	29
PCWP [mmHg]	18
TPG [mmHg]	11
CO [l/min]	5.4
CI [l/min/m²]	2.8
SV [ml]	65
SVR [dyn]	873
PVR [dyn]	163
PA O2 [%]	76
CI- cardiac index, CO- cardiac output, PA- pulmonary artery, PAP- pulmonary arterial pressure, PCWP- pulmonary capillary wedge pressure, PVR- pulmonary vascular resistance, SV- stroke volume, SVR- systemic vascular resistance, TPG- transpulmonary gradient	

Table 1: Pulmonary catheter evaluation.

Acute liver failure is a situation that requires rapid action. There are heterogeneous causes for this, which must be taken into account in the diagnosis. Alcoholic hepatitis has a high mortality, up to 30% over one month [1,2]. Abstinence and nutrition are key therapeutic options. In severe alcoholic hepatitis, the only pharmacological option are steroids [1].

Most transplant centers require a minimum of six months of alcohol abstinence before considering liver transplantation. However, in a meta-analysis of studies examining the risk of relapse to alcohol use after liver transplantation, the pooled risk was 12–30% [3,4]. Based on this, early liver transplantation as a salvage therapy can be considered [1,5]. Patient selection is critical, and the following criteria were postulated [6]: First episode of hepatic decompensation and alcoholic hepatitis; corticosteroid non-responder in severe alcoholic hepatitis; excellent psychosocial status as agreed upon by the whole primary treating team [6].

It is critical to note that the use of alcohol with disease sequelae is subject to prejudice and differential diagnoses should be excluded. Acute liver failure due to venous congestion in tricuspid valve regurgitation was previously described [7]. Importantly, the pathogenesis of tricuspid regurgitation due to an ischemic ventricular septal defect in a subacute disease course is a rare but serious complication. Despite several therapeutic improvements survival in patients with acute VSD remains low [8,9]. Pulmonary artery catheter, echocardiography, cardiac MRI, and coronary angiography in our case revealed a VSD with bidirectional shunt based on a chronic total occlusion of the mid RCA.

The present case highlights the extraordinary role of invasive monitoring, echocardiography, and exclusion of any differential diagnoses in such cases and structured interdisciplinary cooperation. Of note, imaging with echocardiography and invasive hemodynamic monitoring in patients with cryptogenic acute liver failure should be considered, either to search for therapeutic options or to detect severe deterioration in order to adjust therapeutic goals.

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