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Ischemic Hepatitis and Acute Kidney Injury Following Cardioversion

Chelsea Wuthnow, M.D.[1], Aastha Bharwad, M.D.[1], Syed Kamran, M.D.[1], Mona Brake, M.D.[1,2], Kyle Rowe, M.D.[1,2]
[1]University of Kansas School of Medicine-Wichita, Wichita, KS Department of Internal Medicine
[2]Robert J. Dole Veterans Affairs Medical Center, Wichita, KS

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INTRODUCTION

Ischemic hepatitis often is defined as a marked and reversible elevation in serum aminotransferase levels in the absence of other causes.[1] It is thought to be a result of an acute episode of hypoperfusion. This may occur with an acute drop in systolic blood pressure or episodes of transient subclinical falls in perfusion. Patients with chronic heart failure are particularly susceptible to even transient falls in pressure due to underlying hepatic congestion.[1,2] Such a fall in pressure can be seen following cardioversion and may lead to hypoperfusion of the liver.[3] The event is observed in one third of cardioversions and can lead to significant consequences.

The report described a patient with a history of diastolic heart failure and severe tricuspid regurgitation who underwent cardioversion and subsequently developed ischemic hepatitis. A review of the literature led us to believe there was only one other study documenting cardioversion induced ischemic hepatitis and our case was rare with the patient having concurrent tricuspid regurgitation.[4]

CASE REPORT

A 58-year-old male with a past medical history of paroxysmal atrial fibrillation/flutter, diastolic heart failure, severe tricuspid (TR) and mitral (MR) regurgitation, coronary artery disease status post coronary artery bypass graft, and substance abuse presented to the emergency department with shortness of breath, chest tightness, nausea, and lower extremity swelling for one to two weeks. The patient had not been compliant with his medications. His heart rate (HR) was irregular with a documented rate of 95 bpm, blood pressure was 132/81 mmHg, and oxygen saturation was 97% on room air. His weight was 209 pounds, up from a baseline of approximately 185 pounds.

Chest x-ray showed cardiomegaly and pulmonary vascular congestion. B-type Natriuretic Peptide (BNP) and troponin were elevated. Prothrombin time, international normalized ratio, liver enzymes, electrolytes, blood urine nitrogen, and creatine (Cr) were within normal limits.

The urine drug screen was negative. Electrocardiogram (ECG) showed atrial flutter with a 3:1 AV conduction and a rate of 85 bpm. He was admitted for heart failure exacerbation and home medications were resumed including metoprolol, aspirin, apixaban, and atorvastatin. Diuresis was initiated with intravenous furosemide and oral metolazone.

Troponin peaked at 0.185 ng/ mL during the event before trending down back to 0.033 ng/ mL. The trend in troponin was attributed to demand ischemia. The patient tolerated diuresis well and his weight returned to baseline after four days. Heart rate was variable during this time, ranging from 67 to 126 bpm, with most readings being over 100. Blood pressure was stable with systolic at 120-135 mmHg and diastolic at 70-90 mmHg. HR remained elevated despite medical treatment; thus, it was decided to attempt cardioversion. In preparation, a transesophageal echocardiogram (TEE) showed no atrial thrombus, but rather severe mitral and tricuspid insufficiency. The morning of the TEE, vital signs remained stable and labs were unremarkable.

The following morning Cr increased to 1.88 mg/dL and Blood Urea Nitrogen (BUN) 40 mg/dL. The patient underwent successful cardioversion and oral Amiodarone was initiated. Following the procedure blood pressure was 87/61 mmHg. Pressures remained suboptimal throughout the morning: systolic 90-93 mmHg and diastolic 60-73 mmHg. HR was 58-80 bpm. Blood pressure medications were held. Labs later that day showed an increase in serum Cr increased to 2.52 mg/dL, potassium (K+) to 6.4 mEq/L, and bicarbonate decreased from 29 to 19 mEq/L. Aspartate aminotransferase (AST) was 101 U/L and alanine aminotransferase (ALT) was 79 U/L, an increase from AST 31 and ALT 24 one week prior. Total bilirubin (T Bil) also increased from 0.8 to 1.7 mg/dL.

The patient began complaining of right upper quadrant (RUQ) abdominal pain. Abdominal x-ray showed constipation but was otherwise unremarkable. Computed tomography of the abdomen and pelvis without contrast was unremarkable. Repeat labs revealed rising serum Cr up to 3.37 mg/dL, AST of 263 U/L, ALT of 203 U/L, and T bil of 1.9 mg/dL. Lactic acid was 5.0 mmol/L. Electrocardiogram (ECG) showed sinus rhythm with a rate of 60 bpm. Urinalysis was unremarkable. Creatine phosphokinase was within normal limits. The patient's hypotension progressed, and a norepinephrine drip was started. The patient continued to be in sinus rhythm with heart rate ranging between 60-80 bpm. Blood and urine cultures were obtained, and broad-spectrum antibiotics were initiated.

The morning after cardioversion, labs showed AST of 6694 U/L, ALT of 3354 U/L, T bil of 2.1 mg/Dl, and direct bilirubin 1.3 mg/dL. Cr and K also continued to rise, and a plan to start hemodialysis was initiated. Repeat echocardiogram showed similar findings as before, with ejection fraction of 50-55%, severe MR, moderate to severe TR with no evidence of cardiogenic shock. Blood pressures stabilized and the patient was weaned off pressor support, requiring less than 24 hours. Due to evidence of acute renal failure, urgent hemodialysis was started. Amiodarone was held due to elevated liver enzymes; he received a total of 200 mg of oral amiodarone. The next morning, labs were AST 2928 U/L and ALT 2524 U/L, T bil 1.6 mg/dL, and INR 2.3. The viral hepatitis panel was negative. The patient tolerated dialysis well and kidney function and liver function improved. Cultures showed no growth at the 48-hour mark and hence antibiotics were stopped. Metoprolol and furosemide were slowly re-started, and patient tolerated them well.

DISCUSSION

The liver receives 25% of the total cardiac output, two-thirds of which is through portal venous blood, rich in basic nutrients, but lacking in oxygen, and one-third is by the hepatic artery carrying oxygen-rich

blood.[2] The liver's complex vascular supply and high metabolic activity makes it prone to circulatory disturbances. The risk of ischemic injury is increased in patients with preexisting portal hypertension or passive hepatic congestion.[2,5] Passive hepatic congestion can be seen in congestive heart failure, as well as cases of tricuspid regurgitation. Right heart failure in particular leads to increased pressure in the venous system, which affects the hepatic vein indirectly. If the tricuspid valve is also insufficient, then pressures from the right heart are transmitted directly to the hepatic vein and sinusoids.[5]

Cardioversion typically leads to an increase in cardiac output, however, in approximately one third of cases there is a transient decrease in output.[3,6] Upshaw[3] revealed 35% of patients had a reduction in cardiac output at 10 minutes following cardioversion and 39% at 30 minutes. This decrease most often was corrected within 24 hours of cardioversion but lasted up to a week in some patients.[3,6] The reduced cardiac performance post-cardioversion most likely occurred from the combination of heart disease, atrial/ventricular stunning, and cardiac depressant effects of anesthetic drugs used.[3]

This patient had an increase of liver enzymes more than 20 times the upper limit of normal and other causes, such as rhabdomyolysis and viral hepatitis, were ruled out as a cause of injury. The liver enzymes initially increased just hours after cardioversion and prior to sustained low blood pressure readings, leading us to believe there was a transient decrease in cardiac output immediately following cardioversion causing hypoperfusion to the liver and subsequent damage. Progressive hypotension within the 24 hours following cardioversion caused further damage.

To our knowledge, this event is rare and only one case report of cardioversion-induced ischemic hepatitis was found in the literature.[4] Although this may be a rare cause of hepatocellular injury, it is an important risk to consider when a patient undergoes cardioversion, especially in the setting of progressive hypotension.

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