

2022

Case of Ischemic Hepatitis in the Setting of HFpEF Exacerbated by Persistent Atrial Fibrillation

Christian McClairen

St. Thomas UTHSC, Nashville, TN, USA, christian.mcclairen@ascension-external.org

Tracey Doering

St. Thomas UTHSC, Nashville, TN USA

Follow this and additional works at: <https://scholarlycommons.gbmc.org/jchimp>

Recommended Citation

McClairen, Christian and Doering, Tracey (2022) "Case of Ischemic Hepatitis in the Setting of HFpEF Exacerbated by Persistent Atrial Fibrillation," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 12: Iss. 1, Article 15.

DOI: 10.55729/2000-9666.1014

Available at: <https://scholarlycommons.gbmc.org/jchimp/vol12/iss1/15>

This Case Report is brought to you for free and open access by the Journal at GBMC Healthcare Scholarly Commons. It has been accepted for inclusion in *Journal of Community Hospital Internal Medicine Perspectives* by an authorized editor of GBMC Healthcare Scholarly Commons. For more information, please contact GBMCcommons@gbmc.org.

Case of Ischemic Hepatitis in the Setting of HFpEF Exacerbated by Persistent Atrial Fibrillation

Christian McClairen*, Tracey Doering

St Thomas UTHSC, Nashville, TN, USA

Abstract

Congestive hepatopathy in the setting of chronic heart failure is predominantly cholestatic. Severe hepatocellular injury can be seen in cardiogenic shock, usually in an acute setting with severe reduction in ejection fraction and with significant hypotension. Hepatic ischemia with preserved ejection fraction in the setting of atrial fibrillation has not been widely recognized, although mild elevations of liver enzymes have been seen in such patients in the chronic state. We present a patient with preserved ejection fraction, rapid atrial fibrillation with hypotension who had ischemic hepatitis, with aspartate aminotransferase and alanine aminotransferase over one thousand.

Keywords: HfpEF, Ischemic hepatitis

1. Introduction

The liver enzyme pattern in patients with congestive hepatopathy in the setting of chronic heart failure is predominantly cholestatic.¹ Elevation in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) have been noticed with congestive liver injury that are mild in comparison to alkaline phosphatase (ALP). There is a lower likelihood of severe ALT/AST elevation in chronic heart failure patients, unless it is in the setting of a low-output state.^{1,2} Cardiogenic shock can cause ischemic hepatitis, usually in the setting of severely reduced EF. We present a case report of ischemic hepatitis in the setting of a preserved ejection fraction and atrial fibrillation.

2. Case report

A 90 year old Caucasian female with history of hypertension, grade 2 diastolic heart failure with preserved ejection fraction of 57%, hyperlipidemia, paroxysmal atrial fibrillation on anticoagulation, and sick sinus syndrome post pacemaker, presented to the hospital with chest pain and dyspnea.

On arrival, BP 99/59, pulse 103 irregularly irregular, RR 15 breaths/minute. Her hypotension

resolved after one hour. PE showed no murmurs, clear lungs and 2+ pitting lower extremity edema.

Labs were remarkable for sodium 126, AST 2892 units/L and ALT 1685 units/L, with mild elevation in alkaline phosphatase of 340 units/L and total bilirubin of 1.7 mg/dl.

Her chest x-ray showed moderate bilateral effusions and cardiomegaly.

Her chest CT angiogram did not show evidence of a pulmonary embolism. Additionally, her CT of the abdomen and right upper quadrant ultrasound showed cholelithiasis without ductal dilatation. Echo confirmed ejection fraction 65–70%, and elevated pulmonary artery pressures.

Extensive review of her medications, past history including alcohol use, and over the counter medications was unremarkable.

Patient was started on reduced dose of metoprolol due to her low blood pressure and IV furosemide with adequate diuresis and improvement in her BP to 120–140/60–90. Her transaminases improved over 24 h to AST 1,279, and ALT 1129. Her acute viral panel was negative.

Left pleural effusion was transudative based on Light's criteria.

Interrogation of her pacemaker revealed episodes of atrial fibrillation that spontaneously converted.

Received 13 July 2021; revised 23 September 2021; accepted 29 September 2021.
Available online 31 January 2022

* Corresponding author.
E-mail address: christian.mcclairen@ascension-external.org (C. McClairen).

<https://doi.org/10.55729/2000-9666.1014>

2000-9666/© 2021 Greater Baltimore Medical Center. This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).

With slow diuresis, patient's dyspnea resolved, minimal residual left sided effusion. After decongestion via diuresis, her transaminases improved to AST 125 units/L and ALT 298 units/L.

3. Discussion

The evaluation of severely elevated transaminases usually involves medication toxicity or overdose, acute viral infection, and ischemic hepatitis. Congestive hepatopathy originates from elevated filling pressure in the right heart leading to increased pressure in hepatic veins, decreased blood flow in hepatic arteries, and decreased oxygen saturation in the hepatic arteries.³ Perisinusoidal edema, secondary to congestion, impairs diffusion of nutrition and oxygen to the hepatocytes.¹ This effect is superimposed by the liver's hypermetabolic state, with increased oxygen consumption in the setting of inadequate perfusion.¹ These mechanisms lead to ischemic hepatocellular injury, with marked elevation in transaminases, that peak in 1–3 days and resolve after treatment of underlying cause.¹

While congestive hepatopathy is seen in many cases of heart failure, the liver function pattern depends on the classification of heart failure. The difference in hemodynamics and liver function pattern has been established between the two different subtypes of heart failure.^{4,5} Most cases of ischemic hepatitis are in the setting of cardiogenic shock in critically ill ICU patients or with significantly decreased ejection fraction.¹ Individuals classified with right sided heart failure, cardiogenic shock, or ejection fraction less than 45% are more likely to have a hepatocellular pattern than those with hypertensive heart failure or preserved ejection fraction heart failure (HFpEF).¹ Those with HFpEF, as is the case with our patient, tend to demonstrate cholestatic pattern since there is lower likelihood of hypotension or hypoperfusion in these patients to cause hepatic injury.⁶ In patients with HFpEF, and gradually burdening atrial fibrillation, the cardiac output lessens, creating elevated filling pressures that cause pulmonary vascular disease leading to

right heart failure.⁴ This passive congestion through increased filling pressures causes mildly elevated transaminases, which can be markedly elevated in the setting of hypotension and hepatic ischemia.² In situations where both a low output state and passive congestion are present, hypoxic induced hepatitis can occur.

4. Conclusion

In summary, when transaminases are greater than 1000, without a hepatic origin as the cause, ischemic hepatitis is present. This case highlights that ischemic hepatitis can occur in heart failure patients with preserved ejection fraction. This is a result of worsening diastolic capacity leading to low cardiac output, elevated filling pressures and/or right sided congestive heart failure.

Conflict of interest

None.

References

1. Alvarez AM, Mukherjee D. Liver abnormalities in cardiac diseases and heart failure. *Int J Angiol.* 2011 Sep;20(3):135–142. <https://doi.org/10.1055/s-0031-1284434>. PMID: 22942628; PMCID: PMC3331650.
2. Henrion J, Descamps O, Luwaert R, Schapira M, Parfonry A, Heller F. Hypoxic hepatitis in patients with cardiac failure: incidence in a coronary care unit and measurement of hepatic blood flow. *J Hepatol.* 1994 Nov;21(5):696–703. [https://doi.org/10.1016/s0168-8278\(94\)80226-2](https://doi.org/10.1016/s0168-8278(94)80226-2). PMID: 7890882.
3. Hilscher M, Sanchez W. Congestive hepatopathy. *Clin Liver Dis (Hoboken).* 2016 Oct 2;8(3):68–71. <https://doi.org/10.1002/cld.573>. PMID: 31041066; PMCID: PMC6490201.
4. Atrial dysfunction in patients with heart failure with preserved ejection fraction and atrial fibrillation. *J Am Coll Cardiol.* 2020; 76:1051–1064.
5. Abramov D, Cohen RS, Katz SD, Mancini D, Maurer MS. Comparison of blood volume characteristics in anemic patients with low versus preserved left ventricular ejection fractions. *Am J Cardiol.* 2008;102:1069–1072. <https://doi.org/10.1016/j.amjcard.2008.05.058>.
6. Liang W, He X, Wu D, et al. Prognostic implication of liver function tests in heart failure with preserved ejection fraction without chronic hepatic diseases: insight from TOPCAT trial. *Front Cardiovasc Med.* 2021;8:618816. <https://doi.org/10.3389/fcvm.2021.618816>.