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Case Report

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Congestive heart failure masquerading as acute abdomen: A case report

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ABSTRACT

Rationale: As an uncommon manifestation of congestive heart failure, congestive hepatopathy requires an early diagnosis in order to render appropriate care. Misdiagnosis as intraabdominal sepsis may lead to erroneous initial intervention, such as fluid boluses, that can potentially tip an already sick patient with poor reserves over into an extreme state.

Patient's Concern: A 65-year-old man was brought to the emergency department for excruciating abdominal pain, vomiting and jaundice. He also had lower limb pitting edema and was hypotensive en route.

Diagnosis: Congestive hepatopathy.

Interventions: Intravenous furosemide and fluid restriction.

Outcomes: The patient declined admission to the cardiology ward and discharged himself against medical advice after his condition was improved in the emergency department.

Lessons: It is important to pay attention to acute abdominal pain induced by extraabdominal pathologies. In this case of acute decompensated congestive heart failure, early recognition of the cause makes a difference to the management.

KEYWORDS: Congestive hepatopathy; Heart failure; Cardiohepatic; Acute abdomen; Liver congestion

1. Introduction

Decompensated congestive heart failure (CHF) is commonly encountered in the emergency department (ED). Typical symptoms include cough, orthopnea and dyspnea, and physical exam typically reveal signs of congestion such as pulmonary rales, raised jugular venous pressures and peripheral edema. In the case of acute pulmonary edema, patients usually present with hypertension and dyspnea[1]. However, end-stage heart failure may present atypical symptoms[2]. We described a case of CHF which presented with signs and symptoms mimicking acute abdomen and later diagnosed as acute cardiogenic liver injury (ACLI) induced by congestive hepatopathy (CH).

2. Case report

The authors' institutions waive the need for ethical board review and consent for case reports involving 1 or 2 patients if patient anonymity can be maintained.

A 65-year old man presented to the ED with epigastric pain, vomiting, and difficult breathing for one day. He denied having any fever, change in bowel habits, or chest pain. He had a significant medical history of ischemic dilated cardiomyopathy with an ejection fraction of 20%, double-vessel coronary artery disease with previous stenting, atrial fibrillation, diabetes mellitus, chronic renal failure, gout, hypertension and hyperlipidemia. He was non-compliant to medications. In addition, he denied regular alcohol intake and smoking.

The patient was brought in by ambulance for hypotension. His initial vital signs were as follows: temperature 34.7℃ (normal: 36-37.5℃), blood pressure 113/84 mmHg (normal: 120/80 mmHg),

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heart rate 125 beats/min (normal 60-100 beats/min), respiratory rate 22 breaths/min (normal 12-16 breaths/min), oxygen saturation 83% (normal >95%) on room air. The patient was alert but in severe distress from unrelenting right hypochondrium pain. He was clinically jaundiced, and his abdomen was guarded and tender on palpation of the right hypochondrium.

Electrocardiogram showed no acute ischemic changes. Point-of-care ultrasound showed dilated ventricles, no pericardial effusion, poor cardiac contractility and a distended inferior vena cava. The gallbladder had thickened walls and calculi without pericholecystic fluid. There was no free fluid in the abdomen and the aorta was normal. Our initial impression was that of hepatobiliary sepsis with type II non-ST elevation myocardial infection and decompensated heart failure.

Blood investigations however, were not suggestive of an infective process. Venous blood gas showed a pH of 7.0 (normal: 7.35-7.45) with lactate of 20.7 mmol/L (normal: 0.5-2.2 mmol/L). Electrolytes were markedly deranged [urea 23.2 mmol/L (normal: 2.7-6.9 mmol/L), sodium 132 mmol/L (normal:136-146 mmol/L), potassium 6.2 mmol/L (normal: 3.5-5.1 mmol/L), chloride 89 mmol/L (normal: 98-107 mmol/L), bicarbonate 5.7 mmol/L (normal: 19-29 mmol/L), glucose 1.4 mmol/L (normal: 3.9-11 mmol/L), creatinine 256 mmol/L (normal: 59-104 mmol/L)]. Troponin-T level was 158 ng/L (normal: <29 ng/L) and NT-proBNP was >70 000 pg/mL (normal: <149 pg/mL). Liver enzymes levels were elevated [bilirubin 99 μmol/L (normal: 7-32 μmol/L), ALP 110 U/L (normal: 39-99 U/L), ALT 228 U/L (normal: 6-66 U/L), AST 616 U/L (normal: 12-42 U/L)]. White cell counts were normal 9.28×10^9/L [normal: $(4\text{-}10)\times10^9$/L] and serum procalcitonin was 1.27 Ug/L (normal: <0.49 Ug/L). Chest radiograph revealed an enlarged heart without focal consolidation or pleural effusion seen.

A recent admission for similar complaints was discovered in his electronic health records during which he was treated presumptively for cholangitis in the ED and admitted to General Surgery. Inpatient computed tomography of the abdomen and pelvis showed right pleural effusion, ascites, and cardiomegaly while magnetic resonance cholangiopancreatography showed non-dilated bile ducts, and absence of gallbladder/biliary calculi and gallbladder wall thickening likely due to third spacing. The reviewing cardiologist attributed his presentation to CH. During this last admission, the patient's symptoms resolved with treatment directed at volume reduction including fluid restriction and *i.v.* diuretic.

During the current presentation, he was treated with *i.v.* sodium bicarbonate, *i.v.* furosemide, *i.v.* omeprazole, and given empirical antibiotics cover with Terazosin. Cardiologist's review concurred with the diagnosis of ACLI with underlying CH and accepted him for admission.

The patient responded well to the treatment rendered in the ED. With significant pain alleviation, he requested discharge against medical advice. He was informed also to comply with fluid restriction and medications. The patient was then lost to follow-up.

3. Discussion

Being one of the forms of cardiac hepatopathy, CH occurs when chronic right heart failure results in increased hepatic venous pressures induced by impaired outflow[3]. The incidence of CH is 15% to 65% in severe CHF[4]. Any cause of right-sided heart failure (*e.g.* valvular rheumatic heart disease, congenital heart disease, or end-stage cardiomyopathies) can lead to CH[3]. The advancement of medical and surgical treatments such as heart transplantationshas significantly changed the profile of patients with CH. Ischemic cardiomyopathy is now the leading cause of CHF having surpassed rheumatic heart disease, and CH following Fontan surgery is more common[2].

Congestion predisposes to hepatic injury through three major mechanisms: decreased hepatic blood flow, decreased arterial oxygen saturation, and increased hepatic venous pressures. Elevated central venous pressure transmits to the hepatic veins and sinusoids, and thereby decreases portal venous inflow. A "nutmeg liver" is a characteristic anatomical finding of CH. Dark centrilobular zones reflecting sinusoidal congestion alternate with periportal zone which are pale[5].

Many patients with CH may present in a spectrum. Some have asymptomatic liver disease and are identified only through abnormalities in routine laboratory evaluations. Some patients may experience dull right hypochondrium pain and rarely jaundice. In acute decompensated heart failure states, patients may present with ACLI–a sudden excruciating pain related to stretching of the liver capsule resulting in abrupt onset of jaundice and marked elevation of liver enzymes and bilirubin[2]. The liver panel often reveals a cholestatic picture with predominance of bilirubin and alkaline phosphatase. Often renal impairment also co-exists due to hepatorenal reflex[4].

Other than blood investigations, work-up includes imaging to exclude surgical causes of abdominal pain and jaundice as well as cardiac investigations to ascertain the extent and etiology of heart failure are needed if not already known[5]. While imaging with ultrasound or computed tomography can help diagnose liver disease in CHF patients, liver biopsy remains a gold standard[6].

The cornerstone of treatments for CH consists of management of the underlying cardiac condition with optimization of cardiac output. Diuresis can improve hepatic congestion but requires caution to avoid precipitating hepatic ischemia from overdiuresis in ACLI. CH itself uncommonly leads to serious sequelae of chronic liver disease such as varices and has a degree of reversibility with good management of cardiac failure. Of note, in patients who require anticoagulation for their cardiac condition, acute decompensation with concomitant CH may predispose patients to coagulopathy[5,6].

In an acute setting such as the ED or primary care, CH should be suspected in patients presenting with right-sided abdominal pain and/or jaundice with underlying known or suspected cardiac failure. It is important to have a high index of suspicion to initiate timely and appropriate care for the patient.

Conflict of interest statement

The authors report no conflict of interest.

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Authors' contributions

All authors have made significant contributions to the manuscript. Y.K.P involved in the management of the case, provided the case details and case write-up. E.O. performed the literature search and wrote the manuscript. Both authors reviewed and approved the final version of the manuscript.

References

[1] Martindale JL, Wakai A, Collins SP, Levy PD, Diercks D, Hiestand BC, et al. Diagnosing acute heart failure in the Emergency Department: A systematic review and meta-analysis. *Acad Emerg Med* 2016; **23**(3): 223-242.

[2] Fortea JI, Puente Á, Cuadrado A, Huelin P, Pellón R, González Sánchez FJ, et al. Congestive hepatopathy. *Int J Mol Sci* 2020; **21**(24): 9420.

[3] El Hadi H, Di Vincenzo A, Vettor R, Rossato M. Relationship between heart disease and liver disease: A two-way street. *Cells* 2020; **9**(3): 567.

[4] Xanthopoulos A, Starling RC, Kitai T, Triposkiadis F. Heart failure and liver disease: Cardiohepatic interactions. *JACC Hear Fail* 2019; **7**(2): 87-97.

[5] Wells ML, Venkatesh SK. Congestive hepatopathy. *Abdom Radiol* 2018; **43**(8): 2037-2051.

[6] Pendyal A, Gelow JM. Cardiohepatic interactions: implications for management in advanced heart failure. *Heart Fail Clin* 2016; **12**(3): 349-361.

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