

## CASE REPORT

# Right Atrial Collapse With Hepatic Hydrothorax in Advanced Liver Disease



David L. Murphy<sup>1,2\*</sup>, Anna E. Condino<sup>1,2</sup>,  
Matthew J. Gittinger<sup>1,2</sup>, Michael E. Vrablik<sup>1,2</sup>

<sup>1</sup> Harborview Medical Center, Division of Emergency Medicine, Seattle, WA, USA, and <sup>2</sup> University of Washington, Division of Emergency Medicine, Seattle, WA, USA

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**Abstract** Cardiac chamber collapse secondary to extrapericardial causes is rare. Focused cardiac ultrasound (FoCUS) in the emergency department can rapidly yield important clinical information and guide management in patients presenting with dyspnea, hypotension, or other cardiopulmonary complaints of uncertain etiology. We report a case of newly-diagnosed cirrhosis with massive ascites and large pleural effusions that distorted normal cardiac anatomy and venous return, in which FoCUS was essential in differentiating underlying pathology of this sick patient and guiding therapy.

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## Introduction

Point of care ultrasound in the emergency department (ED) has become a widespread tool for a variety of diagnostic and procedural applications. Focused cardiac ultrasound can play a role in the evaluation of any patient suspected of having a cardiac origin of their symptoms, from evaluating

for reduced ejection fraction in cardiogenic shock to looking for right heart strain in suspected pulmonary embolism [1,2]. In a patient with abnormal vital signs with suspected cardiopulmonary dysfunction, focused cardiac ultrasound (FoCUS) provides bedside diagnostic information to guide management and direct further diagnostic testing, especially for patients that are unstable and cannot wait for formal imaging studies or laboratory results to help determine the underlying etiology of their disease process [3].

In a patient with suspected advanced liver disease, bedside ultrasound may be used for the diagnosis of ascites, to guide paracentesis [4], to evaluate for pulmonary edema, and to evaluate the gross hepatic morphology and vasculature although liver biopsy remains the gold standard for definitive diagnosis [5]. In the ED, ultrasound guidance for diagnostic

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\* Correspondence to: David L. Murphy, University of Washington, Division of Emergency Medicine, Harborview Medical Center, BOX 359702, 1CT89, 325 Ninth Avenue, Seattle, WA 98104-2499, USA.

E-mail address: [dlmurphy@uw.edu](mailto:dlmurphy@uw.edu) (D.L. Murphy).

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and/or therapeutic paracentesis is strongly suggested where ultrasound is readily available, in order to decrease the likelihood of injury to intraabdominal and abdominal wall structures as a complication of the procedure [6].

Dyspnea is a symptom with a wide differential, with cardiopulmonary causes among the most common and most serious etiologies. A complication of advanced liver disease that may cause dyspnea is hepatic hydrothorax: when pleural effusions accumulate as part of generalized anasarca and compress lung tissue [7]. In the case we report, massive hepatic hydrothorax caused external cardiac compression, compromising pump function which was visualized on bedside ultrasound.

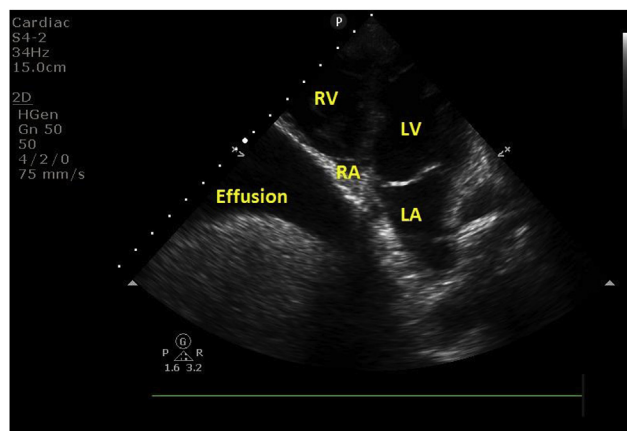
## Case report

A 50-year-old male with long-standing history of heavy alcohol use and wheelchair dependence due to unspecified ataxia presented to the ED after falling out of bed. He was unable to get up and called 911. The patient reported subacute worsening shortness of breath, lower extremity weakness, and increasing edema over the past 6 months. The patient also described progressive abdominal distention over the previous 2 months. He took no medications and had not seen a doctor in 10 years. He denied dizziness, fevers, chills, chest pain, palpitations, cough, wheezing, abdominal pain, changes in urinary or bowel function, or recent illness. He reported drinking 1 L of wine daily for the previous 35 years, and used marijuana and crack cocaine occasionally. His family medical history was unknown.

Physical examination revealed temporal wasting, icteric sclera, distant heart sounds, absent breath sounds in the lung bases, and normal work of breathing. His abdomen was distended and tense, and his skin showed stigmata of liver disease. His lower extremities were notable for weakness, clonus at the ankles, and severe pitting edema to the scrotum. Vital signs were temperature 36.5°C, heart rate 104 beats/min, 114/66 mmHg, respiratory rate 25 breaths/min, SpO<sub>2</sub> 96% on room air. A chest radiograph showed low lung volumes. An electrocardiogram was notable for mild sinus tachycardia and low voltage without evidence of ischemia or electrical alternans. Laboratory values were notable for mild thrombocytopenia, normal aspartate aminotransferase (AST) and alanine aminotransferase (ALT), an international normalized ratio (INR) of 1.3, and albumin of 2.2 g/dL.

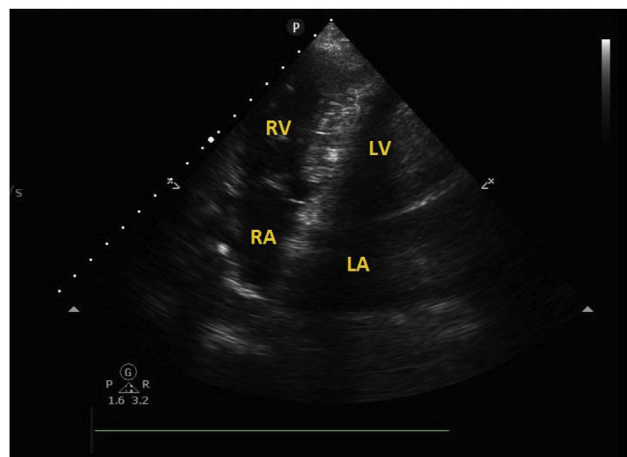
The treating physicians performed bedside FoCUS using a phased-array probe (Phillips, Andover MA, USA). Visualization of the heart was impossible from the usual parasternal view in the third or fourth intercostal spaces or the subxiphoid view; instead, the second intercostal space and left supraclavicular fossa yielded the most complete cardiac visualization. There was no pericardial effusion, and visual estimation of left-ventricular kinesis suggested normal diastolic and systolic function. The apical four-chamber view demonstrated complete collapse of the right atrium and large pleural effusions bilaterally (Figure 1).

A stat, consultative transthoracic echocardiogram showed normal cardiac function, notable only for large pleural and trace pericardial effusions. The patient subsequently had a therapeutic and diagnostic paracentesis draining 3 L of clear, straw colored ascites fluid, with normal



**Figure 1** Complete collapse of the right atrium (RA) and large pleural effusions seen on the apical four-chamber view using FoCUS. FoCUS = focused cardiac ultrasound; LA = left atrium; LV = left ventricle; RV = right ventricle.

cell count. His dyspnea improved, heart rate and respiratory rate normalized, and systolic blood pressure decreased to 102/66. A postparacentesis bedside echocardiogram showed resolved right atrial collapse (Figure 2). He was admitted to the medicine ward with a diagnosis of probable cirrhosis with dyspnea, failure to thrive, and volume overload. While as an inpatient, a formal abdominal ultrasound identified a shrunken, nodular liver contour compatible with patent vasculature and appropriate directional flow. A computed tomography (CT) scan of the abdomen and pelvis identified cirrhosis with portal hypertension. Viral hepatitis panel, ferritin, and ceruloplasmin levels were normal, suggesting underlying alcohol use as the cause of his liver disease, which was classified as Child–Pugh Class B with a Model for End-Stage Liver Disease (MELD) score of 9. The patient was started on spironolactone, furosemide, and ciprofloxacin for prophylaxis against spontaneous bacterial peritonitis. He was discharged with outpatient follow up for esophageal varices screening.



**Figure 2** Normalization of the right atrial size following large volume bedside paracentesis, as seen on the apical four-chamber view postprocedure using FoCUS. FoCUS = focused cardiac ultrasound; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

## Discussion

Advanced liver disease manifests in multiple physiologic abnormalities that can compromise the cardiopulmonary systems of the patient, and dyspnea is a common presenting symptom [6]. Large pleural effusions complicate advanced liver disease in approximately 5% of patients and are referred to as hepatic hydrothorax when there is no other attributable etiology (e.g., intrinsic cardiopulmonary pathologies such as coexisting heart failure, pulmonary hypertension, etc.) [7]. Typically, effusions associated with hepatic hydrothorax have a right sided preponderance and are unilateral, although 2% of patients develop bilateral effusions. The physiology of hepatic hydrothorax is thought to be direct translocation of ascitic fluid across the diaphragm, with the negative intrathoracic pressure causing a steep gradient that favors accumulation of the fluid in the pleural cavity [8]. Although thoracentesis may be pursued, pleural effusions often recur and thus the treatment for hepatic hydrothorax is generally large volume paracentesis, which was performed in the case presented. Hepatic hydrothorax was favored over malignant, parapneumonic, or cardiogenic etiologies of the effusions in our patient due to the bilateral, symmetric nature, the finding of massive anasarca, and the lack of infectious symptoms, focal lung infiltrate or mass, and grossly normal intrinsic cardiac function noted on FoCUS.

Patients presenting with dyspnea have a wide range of possible etiologies that are suitable for investigation with point of care ultrasound, both cardiac and pulmonary [9,10]. Besides cardiac tamponade or effusion, pulmonary embolism can be suspected based on right atrial and right ventricular enlargement and reverse blood flow into the inferior vena cava; sepsis or intravascular volume depletion can be suggested by a flat inferior vena cava and a hyperdynamic precordium; myocardial infarction, cardiogenic shock, and congestive heart failure with reduced ejection fraction can be suggested by poor global or regional left ventricular function with or without wall motion abnormalities. Pulmonary edema is diagnosed with an excess ( $\geq 3$ ) of bilateral B-lines seen on pulmonary ultrasound; pneumothorax can be diagnosed with the loss of normal lung sliding at the lung apices; larger pleural effusions can often be seen as fluid filled areas surrounding the edge of the lung bases [9,11].

In our patient, it was clear from the history and physical examination that he was exhibiting stigmata of liver disease with profound anasarca, and that his tense abdominal ascites were either the entire cause or a significant contributor to his dyspnea. However, on cardiac examination, his heart sounds were noted to be distant and not obtainable at the usual auscultation locations, and his electrocardiogram revealed low voltage sinus tachycardia, which raised the

question of a possible cardiac effusion, tamponade, or external compression; or lung pathology such as pulmonary edema, pulmonary embolism, pleural effusion, or infiltrate as contributing to his symptoms. FoCUS was integral in making an expedient diagnosis of right atrial compression from his large pleural effusions and ascites due to undiagnosed liver disease, and helped guide timely therapy. The case presented above demonstrates a potential cardiorespiratory consequence of excessive abdominal pressures, and the resultant relief provided from paracentesis by improving diaphragmatic excursion leading to increased thoracic space, decreased thoracic pressures, and reexpansion of the right atrium.

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