

Tense Ascites Causing Extracardiac Compression: A Case Report and Literature Review

Samuel J Apple¹, Justin Lee², Meagan Freeze³, Dahlia Rizk⁴,
Andrea Trimmingham³, Samy I. McFarlane^{3,*}

¹College of Medicine, State University of New York-Downstate -Health Science University,
Brooklyn, New York, United States- 11203

²Division of Cardiovascular Medicine, State University of New York-Downstate -Health Science University,
Brooklyn, New York, United States- 11203

³Department of Internal Medicine, State University of New York: Downstate Medical Center,
Brooklyn, New York, United States- 11203

⁴Division of Hospital Medicine, Department of Medicine, Mount Sinai Beth Israel, Icahn School of Medicine NY, NY 10003

*Corresponding author: Smcfarlane@downstate.edu

Received March 12, 2021; Revised April 17, 2021; Accepted April 26, 2021

Abstract Constrictive physiology is most commonly caused by pericardial effusions, constrictive pericarditis or large pleural effusions. Rarely, alternative etiologies may cause cardiac compression. In this report, we present a case of a 70-year-old woman with squamous cell carcinoma who was referred by her cardiologist for evaluation of pericardial and pleural effusions found on echocardiography. Initial clinical findings suggesting constrictive pericarditis. However, after further evaluation we determined that tense ascites was the underlying etiology of constrictive physiology in this patient. This diagnosis requires an integrative interpretation of multiple imaging modalities. Our case highlights the interdependent relationship between cardiac and hepatic pathology that needs to be considered on a case by case basis. Additionally, this case underscores the importance of maintaining a high degree of suspicion for extracardiac compression in the setting of cirrhosis, with prompt paracentesis to adequately prevent the life-threatening sequela of cardiac tamponade.

Keywords: ascites, cirrhosis, cardiac tamponade, constrictive physiology

Cite This Article: Samuel J Apple, Justin Lee, Meagan Freeze, Dahlia Rizk, Andrea Trimmingham, and Samy I. McFarlane, "Tense Ascites Causing Extracardiac Compression: A Case Report and Literature Review." *American Journal of Medical Case Reports*, vol. 9, no. 8 (2021): 402-406. doi: 10.12691/ajmcr-9-8-5.

1. Introduction

Constrictive physiology of the heart is defined by impaired diastolic filling due to increased external pressure. In its most extreme form, cardiac tamponade, a large pericardial effusion (or even a small pericardial effusion in the setting of decreased pericardial elasticity) causes bowing of the interventricular septum into the left ventricle, severely compromising cardiac output. Particularly if there is a high rate of increase of intrapericardial pressure [1], hemodynamic instability often ensues, with acute intervention needed to prevent death. The national incidence of cardiac tamponade is estimated at 5 cases per 10,000 admissions, with an overall in-hospital mortality rate of 14.3% [2]. Although there is a dearth of knowledge regarding the prevalence of pericardial effusions, numerous published reports have analyzed the prevalence of different etiologies for pericardial effusions, with different results likely attributable to different populations studied [3,4,5,6,7]. Possible causes of pericardial effusions include infection, uremia, autoimmune, post-myocardial infarction, post-cardiac surgery, trauma, malignancy,

radiation, as well as idiopathic. Less acute causes of constrictive physiology include constrictive pericarditis and large pleural effusions causing external compression. Constrictive pericarditis is a relatively rare phenomenon, although its true incidence is unknown. It has been found present in 0.2-0.4% of patients who have undergone cardiac surgery, as well as less than 1% of cases after idiopathic pericarditis [8]. Although uncommon overall, tuberculosis is the most common cause of constrictive pericarditis in the developing world. Pericarditis is present in 1% to 2% of patients with tuberculosis, while constrictive pericarditis has an incident rate of 31.65 cases for 1000 person-years for tuberculous pericarditis [9].

We report a case of a patient with initial clinical findings suggesting constrictive pericarditis, but later found to have a tense ascites underlying constrictive physiology, the diagnosis of which required an integrative interpretation of multiple imaging modalities.

2. Report of the Case

A 70-year-old woman with hypertension, type 2 diabetes, dyslipidemia, and squamous cell carcinoma of

the cervix, who underwent radical transabdominal hysterectomy with bilateral salpingo-oophorectomy, chemotherapy and radiation about 7 months prior to presentation. Patient was referred to the Emergency Department from her cardiology clinic for evaluation of pericardial effusion and pleural effusions found on echocardiography. About four months prior, the patient experienced new-onset ascites and anasarca of unclear etiology, requiring two separate therapeutic paracenteses with removal of ~5-6 L each time. Although no definitive cause was ascertained, fluid analysis from the most recent paracentesis showed a serum-ascites-albumin-gradient (SAAG) of 1.8, total protein 3.2 g/dL, negative cytology, negative culture, and amylase <10, suggestive of a cardiac etiology. Prior to this admission, the patient reported mild chest discomfort and dyspnea on exertion with subsequent decreased exercise tolerance. She denied dizziness, chest pain, abdominal pain, headache, nausea, vomiting, fatigue, cough, paroxysmal nocturnal dyspnea, or palpitations. She also denied any tobacco, alcohol or illicit drug use.

On physical exam, her pulse was 109, BP 115/76, RR 18 with 100% O₂ saturation on room air, and was afebrile. She appeared comfortable, obese, and in no acute distress. The patient had jugular venous distension of 15 cm and was tachycardic, but the remainder of the cardiopulmonary exam was benign. Abdominal exam showed profound ascites with shifting dullness. ECG showed sinus tachycardia with low-voltage QRS complexes in all leads other than V1 and V2 (Figure 1). The patient had 3+ edema of the lower extremities and 2+ edema of the upper extremities. Labs were significant for a potassium of 3 mmol/L with a BUN/creatinine of 22/1.6, increased from 20/1.2 the prior month, suggestive of an acute kidney injury secondary to diuretics. Additionally, troponin I was 0.04 (normal cutoff = 0.15) and BNP was 79.

Given the characteristics of the patient's ascitic fluid, differential diagnoses included constrictive pericarditis, Budd-Chiari syndrome, cirrhosis, metastasis and portal vein thrombosis. Ultrasound of the abdomen revealed a coarsened hepatic echotexture with nodular contour, compatible with cirrhosis, as well as splenomegaly. Whether this cirrhosis was the primary etiology or

secondary to another process was unknown, prompting an order for a liver panel with specialized markers for possible causes. Ultrasound also showed portal venous blood flow moving in the appropriate direction, effectively ruling out Budd Chiari syndrome. Transthoracic echocardiogram was significant for a normal ejection fraction, mild pericardial thickening, a small, loculated pericardial effusion, large external fluid collections, and respirophasic changes in transmitral velocities (Figure 2), suggestive of constrictive physiology. Considering the absence of an unclear primary etiology for the cirrhosis, as well as the possibility of a secondary, independent process causing right heart failure in addition to hepatogenic ascites, constrictive pericarditis became a viable diagnosis to explain the clinical features of dyspnea on exertion and jugular venous distension, as well as the echographic findings of constrictive physiology with pericardial thickening. Additionally, as mentioned above, the total protein of ascitic fluid was 3.2 g/dL, which is more consistent with a cardiac etiology than cirrhosis, even though both are associated with an elevated SAAG. Referral to cardiothoracic surgery for possible pericardial stripping was considered but was delayed due to the ongoing investigative workup.

While an initial chest radiograph did not suggest any pulmonary effusion (Figure 3), echocardiographic images were conflictingly suggestive of a large pleural effusion (Figure 4). A computed tomography (CT) scan with contrast was obtained for better visualization, which clearly illustrated tense ascites pushing the diaphragm cranially into the thoracic cavity (Figure 5), thereby compressing the heart externally (Figure 6). After discovering a cardiac diagnosis more likely than constrictive pericarditis or pericardial effusion that could explain the whole clinical picture, therapeutic paracentesis was performed, which drained 8.5 L, with no postoperative complications. After the paracentesis, the patient reported complete resolution of her dyspnea, and follow-up echocardiography showed resolution of the constrictive physiology. The patient was discharged upon clinical stabilization, with outpatient appointment scheduled with gastroenterology to further elucidate the etiology of her cirrhosis.

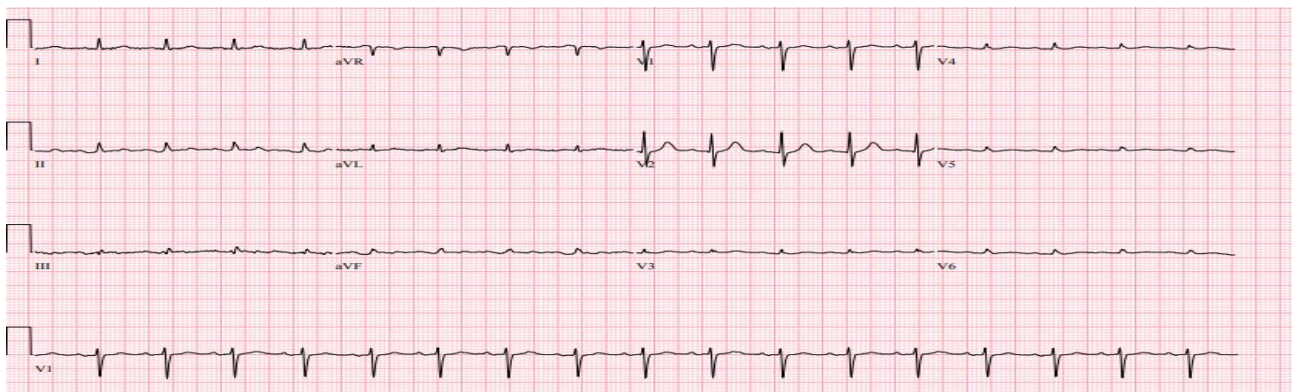


Figure 1. ECG showing sinus tachycardia with low-voltage QRS complexes in all leads other than V1 and V2

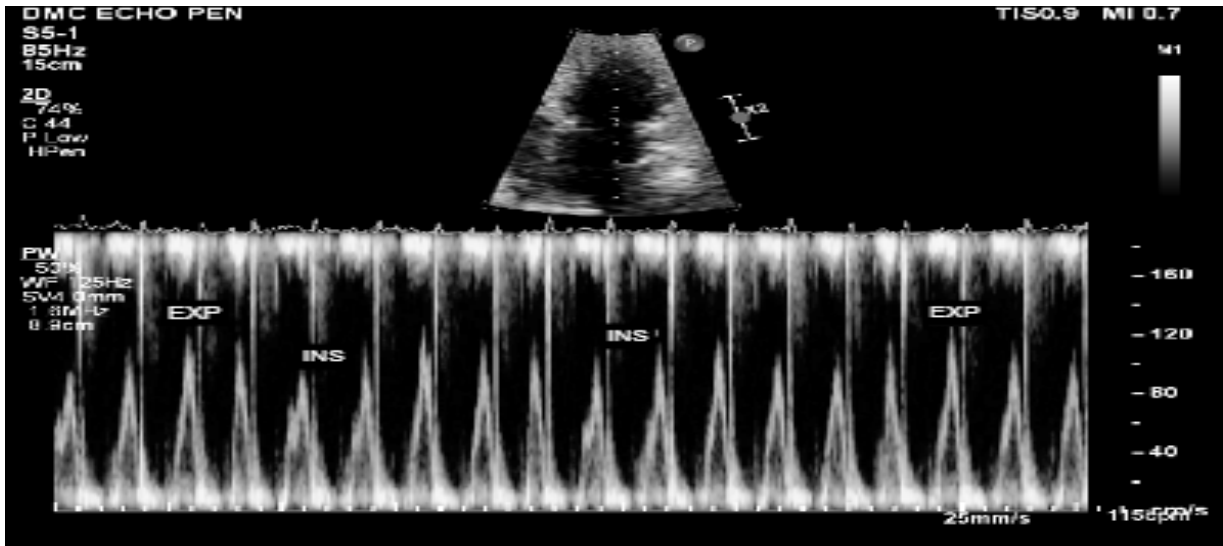


Figure 2. Respirophasic variations in transmitral velocities



Figure 3. Chest radiograph showing no acute cardiopulmonary pathology

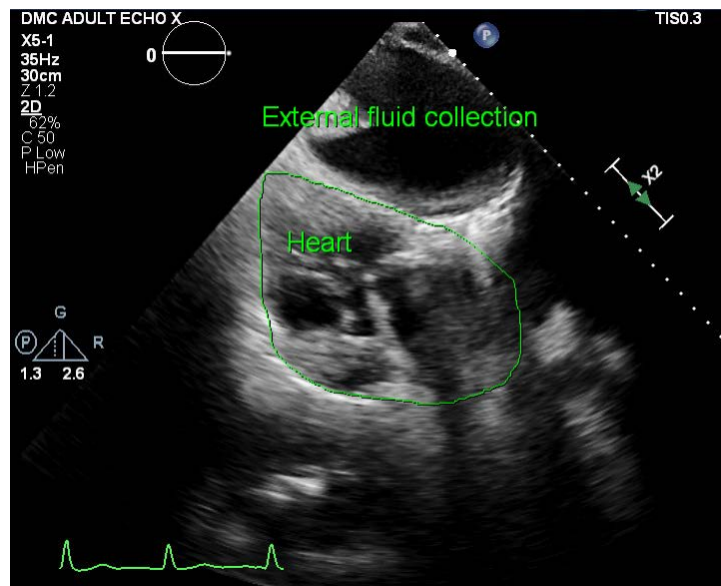


Figure 4. Echocardiographic images showing a large external fluid collection, suggestive of a large pleural effusion



Figure 5. CT scan showing tense ascites bulging into the thoracic cavity, posterior to the heart



Figure 6. CT scan showing bilateral compression of the heart by the diaphragm

3. Discussion

An extensive literature review revealed four similar cases, with notable differences. D'cruz and Kleinman [10] first described paradoxical left ventricular wall motion on echocardiography in a patient with alcoholic cirrhosis and ascites. Swan et al. [11] discussed a case in which tense ascites causing extrapericardial cardiac compression masqueraded as a pulmonary embolism, with hypotension and jugular venous distention, as well as an S1Q3T3 pattern on electrocardiogram (ECG), that resolved with paracentesis. Ahmad et al. [12] analyzed a case of a patient with clinical signs and symptoms of cardiac tamponade, fully satisfying Beck's triad of hypotension, jugular venous distention and muffled heart sounds, whose symptoms and hemodynamic status markedly improved with paracentesis. Lastly, Venketram et al. [13] presented a case of a patient with epigastric pain, nausea, non-bloody vomiting, dizziness and near syncope, as well as echographic findings of tamponade physiology without pericardial effusion, whose symptoms resolved upon

paracentesis. Taken as a whole, together with the case reported here, these cases illustrate a wide spectrum of ostensibly cardiogenic symptoms that may be observed in association with, and resolved with the treatment of, tense ascites. Furthermore, they highlight the need to bear in mind the interdependent relationship between cardiac and hepatic pathology, and to clarify the particular relationship on a case by case basis.

Ascitic fluid analysis is a useful way to determine the etiology of ascites. In our case, with an elevated SAAG as well as an elevated total protein, a cardiac cause was high on the list of differential diagnoses despite the presence of cirrhosis, particularly in light of the constrictive physiology on echocardiography. Careful analysis of each of the different imaging modalities used led to the initial working diagnosis of constrictive pericarditis, which resolved completely following paracentesis, which removed 8.5 L of ascitic fluid. Although echocardiography is certainly warranted in the case of dyspnea in the setting of ascites, our case forewarns that the results of said echocardiography must be assessed holistically. Constrictive physiology, if

observed, can be either a cause or an effect of the ascites, and that determination must involve the integration of various imaging modalities.

Given the lack of large pericardial effusion or significant pericardial thickening on echocardiography, cardiogenic ascites seemed unlikely. With insights gleaned from the chest radiography and CT scan, the suspected pleural effusion seen on initial echocardiography was determined to most likely be ascitic fluid intruding into the thoracic cavity with elevation of the diaphragm. This understanding suggested that the most efficient way to proceed would be to perform a therapeutic paracentesis followed by a repeat echocardiography, which helped avoid any unnecessary and/or invasive diagnostic procedure, such as pericardial stripping or pericardiocentesis, were the constrictive physiology to be misattributed to constrictive pericarditis or pericardial effusion, respectively. Additionally, paracentesis effectively halted the progression of constrictive physiology on echocardiogram into a more critical condition, such as partial or total ventricular wall collapse. Our case highlights the unique relationship between ascites and pericardium and, similarly, the need to maintain a high degree of suspicion for extracardiac compression, with prompt paracentesis to adequately prevent the life-threatening sequela of cardiac tamponade.

Acknowledgements

This work is supported, in part, by the efforts of Dr. Moro O. Salifu M.D., M.P.H., M.B.A., M.A.C.P., Professor and Chairman of Medicine through NIH Grant number S21MD012474.

References

- [1] Appleton C, Gillam L, Koulogiannis K. Cardiac Tamponade. *Cardiol Clin*. 2017 Nov; 35(4): 525-537.
- [2] Al-Ogaili A, Ayoub A, Fugar S, et al. Cardiac tamponade incidence, demographics and in-hospital outcomes: analysis of the National Inpatient Sample database. *J Am Coll Cardiol*. 2018; 71(11 suppl): A1155.
- [3] Sagrista-Sauleda J, Merce J, Permanyer-Maralda G, et al. Clinical clues to the causes of large pericardial effusions. *Am J Med* 2000; 109:95.
- [4] Corey GR, Campbell PT, VanTrigt P, et al. Etiology of large pericardial effusion. *Am J Med* 1993; 95: 209.
- [5] Levy PY, Corey R, Berger P, et al. Etiologic diagnosis of 204 pericardial effusion. *Medicine (Baltimore)* 2003; 82: 385.
- [6] Ma W, Liu J, Zeng Y, et al. Causes of moderate to large pericardial effusion requiring pericardiocentesis in 140 Han Chinese patients. *Herz* 2012; 37: 183.
- [7] Strobbe A, Adriaenssens T, Bennett J, et al. Etiology and long-term outcome of patients undergoing pericardiocentesis. *J Am Heart Assoc* 2017; 6: e007598.
- [8] Kyriakakis C, Herbst P and Doubell A. (2017): Constrictive pericarditis – prevalence, causes and clinical presentation. *European Society of Cardiology*, 15(22): 1-8.
- [9] Imazio M, Brucato A, Maestroni S, Cumetti D, Belli R, Trincherio R, Adler Y. Risk of constrictive pericarditis after acute pericarditis. *Circulation* 2011; 124: 1270-1275.
- [10] D'Cruz IA, Kleinman D. Extracardiac causes of paradoxical motion of the left ventricular wall. *American Heart Journal*. 1988 Feb; 115(2): 473-475.
- [11] Swan P, Wu F, Dahle TG, Duprez DA. Extrapericardial Cardiac Compression Syndrome. *International Journal of Cardiology*. 113.2 (2006): 285-87.
- [12] Ahmad S, Jamali H, K, Waqar F, Harris D, M: Cardiac Tamponade Physiology Secondary to Tense Ascites. *Cardiology* 2016; 134: 423-425.
- [13] Venkatram S, Kassis G, Gardezi A, Mallapu S, Diaz-Fuentes G: An unusual case of tense ascites causing tamponade physiology. *American Thoracic Society Meeting Abstracts*. *Am J Resp Crit Care Med* 2010; 181: A6068.



© The Author(s) 2021. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<http://creativecommons.org/licenses/by/4.0/>).