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Abstract Form

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Project Title:	A Case of Severe Pulmonary Hypertension Exacerbated by Compression of the Inferior Vena Cava

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction
Pulmonary hypertension (PH) is a disease defined by increased pressure in the pulmonary vasculature. It is usually accompanied by various pathophysiologic mechanisms including vascular remodeling and hypoxic pulmonary vasoconstriction and classified into five groups based on etiology. Symptoms are typically nonspecific, with most common complaint being dyspnea on exertion, and easily attributable to other conditions. Physical examination may provide some context, however usually the condition has progressed and is associated with increased mortality by then. Severe pulmonary hypertension accompanied by physical compression of the inferior vena cava is an uncommon finding. Here we present a patient receiving work-up for atrial fibrillation and found to have severe pulmonary hypertension. Incidentally, he was noted to have an extra-hepatic mass compressing the inferior vena cava. When followed up outpatient for further evaluation, repeat Echo showed no evidence of IVC compression. This was accompanied by drastic improvement in his condition.
Methods: IRB approval was obtained; single patient case report was conducted.
Case Presentation: A 53-year-old Caucasian male with a 20-pack year smoking history, renal cell carcinoma, and HTN s/p left nephrectomy presented to our institution for elective hernia repair. During the operative procedure, the patient was observed to be in atrial fibrillation with occasional fast ventricular response and pharmacologically converted to sinus rhythm. Post-operatively the patient remained in sinus tachycardia and was admitted for further evaluation.
A cardiology consult was placed and further history revealed patient was diagnosed with an “unknown murmur” during adolescence and endorsed intermittent episodes of “fluttering” in his chest, typically associated with stress or increased physical work. He otherwise denied any fatigue, shortness of breath, episodes of exertional syncope, chest pain, weight gain, and swelling. Physical exam was unremarkable. A transthoracic echocardiogram (TTE) was ordered to evaluate for a left atrial appendage thrombus. Upon imaging, no apparent thrombus was visualized but instead he was found to have moderate tricuspid valve insufficiency with a PAP of 78 mmHg and maximum tricuspid velocity of 4.4 m/s, consistent with severe pulmonary hypertension. Incidentally, he was seen to have an extrahepatic cystic structure measuring 6 x 8 cm compressing to the IVC. Further imaging was advised to primary team to evaluate mass however patient was discharged prior to further workup. Six-months later patient was seen in cardiology clinic for follow up. During his visit he reported no new complaints. He continued to endorse intermittent fluttering in his chest but denied any exertional syncope, fatigue, dyspnea, chest pain, swelling or weight gain. Repeat Echo was ordered to reevaluate his pulmonary hypertension. Results continued to show tricuspid insufficiency; however, PAP and maximum velocity had significantly decreased to 34 mmHg and 2.8 m/s, respectively. These findings were consistent with borderline pulmonary hypertension. Remarkably there was no longer any evidence of IVC compression/mass.

Conclusion
It can be concluded our patient has pre-existing pulmonary hypertension which was exacerbated by extra-hepatic compression on the inferior vena cava. He currently exhibits good functional capacity with no clinical symptoms. Echocardiogram provides evidence of residual pulmonary hypertension. While confirmatory testing with RHC remains to be performed it is imperative for more frequent follow up visits to assess symptoms of right heart failure, exercise tolerance, and resting and ambulatory oximetry.