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# Constrictive Pericarditis: A Rare Cause of Pulmonary Hypertension

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## Introduction

Constrictive chronic pericarditis is a rare form of chronic heart failure representing 0.2-0.4%, and is an even rarer cause of pulmonary hypertension PH type 2. When attempting to differentiate CP from restrictive cardiomyopathy, the presence of severely elevated pulmonary arterial pressure is used as a diagnostic criterion ruling against CP. the case of a child aged 07 who was followed for a long time for pulmonary hypertension is reported. an ultrasound examination with a hemodynmic study allows us to correct the diagnosis of constrictive pericarditis complicated by PH by stenosis aquise of the pulmonary veins.

## Case

A 07-year-old child with no significant medical history who has been followed since the age of 04 for a pulmonary arterial hypertension with a hemodynamic study done in 2014 that concluded that he ftyed on primary pulmonary hypertension after an inquest etiological, including respiratory exposure. He was admitted into the pediatric cardiology department in a right heart failure. On admission, his jugular veins were distended to angle of his jaw when upright. Pitting edema was noted up to the mid thighs bilaterally, hepatomegaly and ascite. Echocardiography on admission showed moderate bi-atrial enlargement (the left atrium was 49 mm * 34 mm, and the right atrium was 47mm*35 mm). The ventricles were deformed; the intermediate part of the left and right ventricles was narrower than the cardiac apex .the E/è ratio was >2 in favor of restrictive filling profile. The left ventricular ejection fraction (LVEF) was 61%. The quantitative assessment of longitudinal mitral annular motion by tissue Doppler imaging was normal. A circumconferenciel pericardic decollement was noted, the pericardium thickness was normal, and calcification was not observed. Finally, pulmonary artery pretension was 60 mmHg. There was a doubt about a stenosis of the left pulmonary veins. CT scan showed epicardic calcification .catheterization was performed two days post admission after the patient had received intravenous diuresis. Pulmonary artery pressures were elevated at 56/22 with a mean of 33 mmHg and stenosis in the mouthing of both left pulmonary veins was objectified (Figure1). Both RV and LV pressures decrease rapidly in early diastole followed by an abrupt plateau ("dip and plateau" or "square root sign"). Classical features which favor CP over restrictive cardiomyopathy include equalization of left and right heart filling pressures to less than 5 mmHg, heightened ventricular interdependence with respiration, and only modestly elevated pulmonary artery systolic pressures (less than 50 mmHg). The diagnosis of constrictive chronic pericarditis was finally accepted according to mayo criters and heamodynamic study. child was proposed for pericardiectomy.

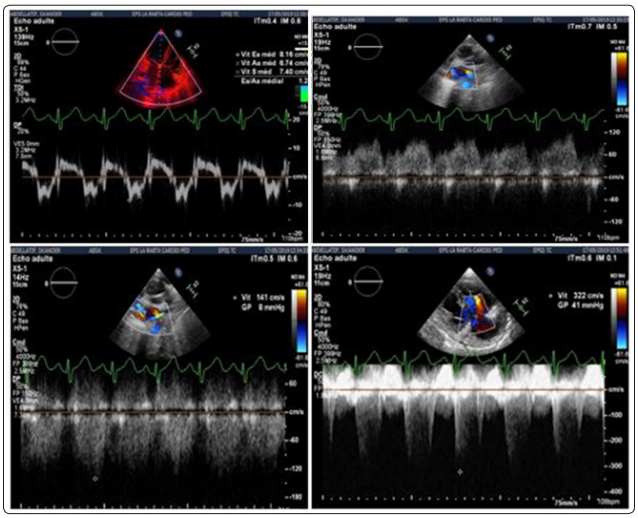

**Figure 1:** Pulmonary stenosis

## Discussion

Constrictive pericarditis (CP) is a form of diastolic heart failure caused by an inelastic pericardium that inhibits cardiac filling. Because its treatment differs markedly from all other forms of heart failure, accurate diagnosis is imperative [1]. Making that diagnosis may be difficult, as CP may mimic other disorders. the main differential diagnosis remains restrictive heart disease, ultrasound criteria (Mayo criteria) and hemodynamics makes the difference [2,3]. The performance of pulmonary artery systolic pressure exceeding 50 mmHg in distinguishing restrictive cardiomyopathy (RCM) from CP has not been ideal when used in isolation. In older series comparing surgically proven CP to RCM, significant overlap in pulmonary arterial systolic pressure (PASP) between the two entities was seen. In a more recent series, there was no statistically significant difference in pulmonary pressures at catheterization between CP and RCM, and PASP < 55 mmHg

had a specificity of only 29% for the diagnosis of CP. The potential for the development of severe pulmonary hypertension due to any cardiac condition resulting in elevated LV filling pressures is well-documented [4].There is no pathophysiologic reason why CP could not lead to pulmonary hypertension. in our case pulmonary arterial hypertension was explained by the stenosis of the left pulmonary veins suspected at the echocardiography and confirmed to the hemodynamic study which reminds us of the mecanism of the type pulmonary arterial hypertension 2.

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