



## SHORT REVIEW

# Practical Approach to Constrictive Pericarditis

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## Introduction

Constrictive pericarditis (CP) is characterized by impaired ventricular filling secondary to a scarred pericardium. The scarred pericardium involving both parietal and visceral layers may be thickened or calcified with resultant loss of normal elasticity of the pericardial sac.

The common causes include idiopathic aetiology, post cardiac surgery and systemic diseases affecting the pericardium such as tuberculosis, collagen vascular diseases, malignancy, renal diseases or radiation therapy [1,2]. The risk of developing constrictive pericarditis is however rare post acute viral pericarditis as compared to specific aetiologies such as tubercular [3].

## Clinical Features

After a detailed history taking is done patient is clinically evaluated. The predominant symptoms are dyspnea with peripheral oedema and on clinical evaluation there is evidence of systemic venous congestion with hepatomegaly and ascites.

Clinically the patient will present as a case of predominant right heart failure [4] and CP will be an important differential diagnosis in this setting. Other conditions, which will need to be excluded, would be restrictive cardiomyopathy, pathologies causing failure of right side of heart e.g. Tricuspid stenosis, pulmonary stenosis, Ebstein's anomaly, cor pulmonale and other diseases such as liver cirrhosis, SVC obstruction and portopulmonary stenosis etc.

The clinical findings in vast majority reflect elevated systemic venous congestion such as increased JVP and hepatomegaly. Venous pressure often paradoxically increases with inspiration (Kussmaul sign) due to inability of the right heart to accept increased venous return

with inspiration and a prominent y descent (Freidrich sign) may be discernable. With high atrial pressures the rapid right ventricular filling causes the ventricle to “knock” onto the rigid thickened pericardial shell in very early diastole-the “pericardial knock”.

The important bedside clues, which one should remember, are a prominent “y” descent on JVP, a pericardial knock (to be differentiated from opening snap which is higher pitched, occurring later in diastole and best heard in expiration), absence of a palpable apex beat and a silent heart with no regurgitant murmurs.

## Investigations

The next step should be to confirm the diagnosis and investigations are accordingly ordered i.e. ECG, Pericardial imaging studies, serum NT pro BNP, and lastly cardiac cath study.

ECG may show low voltage complexes. Depolarization abnormalities (such as bundle branch block), ventricular hypertrophy, pathologic Q waves, or impaired atrioventricular conduction would however favor restrictive cardiomyopathy. Thus, there are no characteristic ECG findings in CP [5,6].

Elevation of Serum NT proBNP values would again point against CP. Pericardial imaging may be done by X ray chest, Echocardiography, CT scan or MRI. Radiological presence of calcifications should be looked for in left lateral projections suggestive of CP. An enlarged cardiac silhouette in X-Ray PA view would be suggestive of pericardial effusion or effusive-constrictive pericarditis and the difference would become apparent when there would be persistently elevated right atrial pressures post pericardiocentesis in the later. A pericardial thickness exceeding 4 mm on echocardiography, CT scan or

MRI is highly suggestive of CP with the former being the least sensitive. However, CP can also occur in the setting of a non-thickened pericardium.

Doppler of the hemodynamics is extremely helpful in diagnosing CP in which the pathologically increased pericardial restraint results in an enhanced ventricular interaction or interdependence and prevents transmission of intrathoracic pressures into the cardiac chambers. The respiratory variation in ventricular filling velocity in restrictive cardiomyopathy is usually minimal (less than 10 percent), while patients with CP may have respiratory variations as high as 30 to 40 percent in ventricular filling velocity (similar to that seen in cardiac tamponade). Similar findings may be present in COPD patients but in them additionally a marked increase in inspiratory superior vena cava systolic flow velocity would also be seen. Measurement of hepatic venous flows is also helpful as reversal of forward flow during expiration is seen CP while it happens during inspiration in restrictive cardiomyopathy.

Tissue Doppler imaging records the velocity of the myocardium and has significant diagnostic value. The early diastolic Doppler tissue velocity at the mitral annulus (Ea) is decreased ( $< 8$  cm/sec, normally  $> 10$  cm/sec) in restrictive cardiomyopathy, due to decreased myocardial relaxation in myocardial diseases, but is preserved or even increased in CP. Therefore, if the Ea is more than 8 cm/s in a patient of heart failure one should consider CP. Additionally this preserved or even accentuated Ea value gets reflected in the relationship between Transmitral inflow early velocity (E)/Ea with the LV filling pressures which is usually positive and linear in primary myocardial diseases but gets reversed (annulus paradoxus) in CP [7].

Cardiac cath study findings include increase in right atrial pressures, equalization of diastolic filling pressures i.e.  $< 5$  mm difference between mean Right atrial (RA) pressure, Right ventricular (RV) diastolic pressure, Pulmonary-artery (PA) diastolic pressure, and Pulmonary-capillary wedge pressure (PCWP) and pericardial pressures, dip and plateau configuration of right and left ventricular diastolic wave forms while the simultaneous LV and RV systolic pressure tracings in CP will show discordant changes with respiration i.e. LV systolic will decrease with inspiration while RV systolic will increase.

To summarize patient will present as a case of right heart failure. History taking should aid in identifying systemic disorders or infections. Clinical evaluation followed by logical usage of various investigative modalities should help in arriving at the diagnosis of constrictive pericarditis. Cardiac cath may not be required in every case if the other tests produce a decisive conclusion.

## Management

Medical management has a limited role with diuretics and salt restriction to relieve volume overload,

digoxin for atrial fibrillation with fast ventricular rates, anti-inflammatory agents if there are features of pericardial inflammation and treatment of specific aetiologies e.g. TB. Once the diagnosis of CP is made definitive therapy i.e. surgical complete pericardiectomy should be done in addition to management of comorbidities if any [8].

Pericardiectomy usually leads to rapid hemodynamic and symptomatic improvements in 80-90% of patients who achieve symptomatic improvement to NYHA Class I or II postoperatively.

The surgical procedure itself can be often a technically complex procedure with high mortality [9] requiring extensive surgical experience and aim should be to achieve as extensive pericardial decortication as possible.

If the surgery is performed early in the course of the disease as evidenced by less pericardial calcification and no myocardial dysfunction then the outcomes are excellent [10-12]. In others especially if patients, particularly those with left ventricular systolic dysfunction, advanced New York Heart Association (NYHA) functional class, concomitant myocardial disease and also comorbidities, symptoms may persist after surgery. Patients with end stage CP manifested by cachexia reduced resting cardiac output, hypoalbuminemia or liver dysfunction form a group with markedly increased operative risk and hence pericardiectomy may not be beneficial in such cases [8].

## Sources of Support

Nil.

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