Constrictive Pericarditis ; Causes, Symptoms, and Treatment

Constrictive pericarditis results when the healing of an acute fibrinous or serofibrinous pericarditis or the resorption of a chronic pericardial effusion is followed by obliteration of the pericardial cavity with the formation of granulation tissue.

The latter gradually contracts and forms a firm scar encasing the heart, which may be calcified.

Causes of constrictive pericarditis

Chronic constrictive pericarditis may follow;

- Acute or relapsing viral or idiopathic pericarditis,
- Trauma with an organized blood clot, or
- Cardiac surgery of any type or result from mediastinal irradiation,
- Purulent infection,
- Histoplasmosis,
- The neoplastic disease especially breast cancer, lung cancer, and lymphoma,
- Rheumatoid arthritis, SLE, or
- Chronic renal failure treated by chronic dialysis.

In many patients, the cause of the pericardial disease is undetermined, and in these patients, and an asymptomatic or forgotten bout of viral pericarditis, acute or idiopathic, may have been the inciting event.

Pathophysiology of constrictive pericarditis

The basic physiologic abnormality in patients with chronic constrictive pericarditis is the inability of the ventricles to fill because of the limitations imposed by the rigid, thickened pericardium.

The pericardium becomes a dense mass of fibrous tissue and this may be calcified. This results in the encasement of the heart within a non-expansile pericardium.

Ventricular filling is unimpeded during early diastole but is reduced abruptly when the elastic limit of the pericardium is reached, whereas, in cardiac tamponade, the ventricular filling is impeded throughout diastole.

In both conditions, ventricular end-diastolic and stroke volumes are reduced and the end-diastolic pressures in both ventricles and

Signs and symptoms of constrictive pericarditis

Symptoms

- Dyspnoea, edema and abdominal swelling due to ascites and hepatomegaly.

Signs

- Small volume pulse and pulsus paradoxus. jugular venous pressure is always elevated, usually very high with prominent ‘x’ and ‘y’ descents.
- A diastolic knock is usually felt at the left sternal border due to the sudden halting of the ventricles during diastolic filling.
- The apex beat may be impalpable.
- Heart sounds are usually soft, and an early third sound coincident with the diastolic knock is usually heard.
- In the pulmonary area, there is a sudden instantaneous widened splitting of the second sound that occurs following the first heartbeat of inspiration (Vogelpoel–Beck sign).
- The liver is commonly grossly enlarged, ascites is marked, and peripheral edema is present.

Diagnosis

An electrocardiogram is abnormal in virtually every case, but changes are non-specific such as a generalized low-voltage QRS complex and widespread flattening and inversion of T waves.

Atrial fibrillation is present in about one-third of patients.

A Chest x-ray shows a normal or near-normal cardiac size in the presence of marked venous distension or heart failure is suggestive of constrictive pericarditis or restrictive cardiomyopathy. Pericardial calcification is diagnostic but its incidence varies from 5% to 70% of cases in different series.

On **echocardiography**, Pericardial thickening may be present. There is a restrictive mitral filling pattern on Doppler, with a respiratory variation of >25% over the AV valves.

Computed tomography (CT) and magnetic resonance imaging (MRI) scan demonstrate pericardial thickening (>5 mm).

Cardiac catheterization demonstrates elevation and equalization of filling pressures. In the typical case, the difference in filling pressures between the right ventricle and left ventricle does not exceed 6 mmHg.

The right atrial waveform shows rapid ‘x’ and ‘y’ descents, and the mean pressure does not decrease normally with inspiration or may show Kussmaul’s sign.

The pulse pressure is normal or reduced. A paradoxical pulse can be detected in about one-third of cases. Congestive hepatomegaly is pronounced and may impair hepatic function and cause jaundice; ascites is common and is usually more prominent than dependent edema. The apical pulse is reduced and may retract in systole (Broadbent’s sign).

The heart sounds may be distant; an early third heart sound (i.e., a pericardial knock, occurring at the cardiac apex 0.09–0.12 s after aortic valve closure) with the abrupt cessation of ventricular filling is often conspicuous.

The **chest roentgenogram** shows a normal or slightly enlarged heart. Pericardial calcification is

most common in tuberculous pericarditis

Treatment

Constrictive pericarditis is treated by surgical removal of the fibrous constrictive tissue, a procedure known as pericardiectomy.

Pericardial resection is the only definitive treatment of constrictive pericarditis and should be as complete as possible.

Dietary sodium restriction and diuretics are useful during preoperative preparation.

Coronary arteriography should be carried out preoperatively in patients older than 50 years to exclude unsuspected accompanying coronary artery disease. Surgical treatment should, if possible, be carried out as early as possible in the course.