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Chronic constrictive pericarditis with peripheral edema

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Background

Constrictive Pericarditis (CP) is a type of diastolic heart failure that occurs because the inelastic pericardium inhibits the filling of the heart. The true population prevalence is unknown, but among those with viral pericarditis, it is estimated to occur in less than 0.5% of cases [1]. Although the causes of CP can be different (idiopathic, post-viral, tuberculosis, post-surgery, radiation-induced, etc). The last end pathological pathway is the development of fibrous thickening or calcification of the pericardium, which leads to pericardial non-compliance [2,3].

A normal pericardium minimally interferes with ventricular expansion at normal cardiac workloads. In CP, pericardial non-compliance creates a stiff ventricular pericardial unit, leading to increased diastolic pressure and a faster rise in ventricular pressure for a given venous return. A non-compliant pericardium limits ventricular relaxation and determines ventricular diastolic pressure, resulting in high and uniform diastolic pressure in all chambers. Clinically, this presents predominantly as right-sided congestion. Increased pulmonary capillary wedge pressure and decreased cardiac output response to exercise lead to dyspnea and exercise intolerance, although overt pulmonary edema is less common than typical systolic heart failure.

The normal pericardium adjusts the coupling of left and right stroke volumes during acute changes in preload, such that a sudden increase in right venous return increases. This in turn reduces LV end-diastolic volume (LV preload) and, therefore, left-sided volume. Due to the constant volume of the pericardium in CP, the pericardial coupling is greatly exaggerated, leading to a dramatic ventricular interdependence. Abnormal movement of the ventricular septum is caused by increased respiratory changes in left and right stroke volume [4].

In CP, because the heart is surrounded by a non-compliant pericardium, the normal inspiratory reduction in intrathoracic pressure is not transmitted to intracardiac pressures. This effect enhances the inspiratory reduction in pulmonary venous pressure, which translates into reduced left inspiratory preload and decreases left inspiratory stroke volume. Multimodal diagnostic evaluation of CP highlights these findings and facilitates diagnosis.

The low prevalence of CP makes identifying key physical examination and historical features an important initial step in the diagnostic process [5]. Laboratory testing in CP is nonspecific.

A high BNP can suggest a greater likelihood of restrictive cardiomyopathy, but studies have shown a great overlap in diagnostic values in this population limiting clinical utility [6-9]. As an initial diagnostic test, echocardiography can confirm the diagnosis of CP in most cases if pre-test probability is sufficiently high [10]. Echocardiography demonstrates features of both exaggerated ventricular interdependence and intrathoracic-intracardiac dissociation. The exaggerated respiratory preload changes are also exemplified by an inspiratory decrease in mitral valve inflow Doppler and an increase in tricuspid valve inflow Doppler. However, these findings are insensitive [11].

In CP, chest x-rays can demonstrate pericardial calcification, a pathognomonic finding in the presence of clinical heart failure, and elevated JVP. Chest CT is more sensitive for pericardial calcification than chest x-ray [12]. Chest CT and MRI allow for precise measurement of pericardial thickness, with MRI, in particular, demonstrating excellent accuracy (93%) in the detection of pericardial thickening >4 mm [13]. Unlike echocardiography, cardiac CT and MRI are not dependent upon patient habitus and can provide better cardiac visualization when echocardiographic imaging is suboptimal. Cardiac catheterization remains the gold standard diagnostic test, if non invasive testing is inconclusive, to assess for the presence of constriction and evaluate hemodynamic significance. While most patients with CP do not require hemodynamic catheterization for diagnosis, one subgroup of particular concern is patients with radiation heart disease, in whom it is often difficult to identify the degree of underlying restrictive cardiomyopathy, even if constrictive features are present [2]. Once CP is identified, medical treatment with diuresis is often only partially effective at palliating symptoms. If there is extensive pericardial inflammation, a trial of anti-inflammatory therapy is warranted to assess for reversibility prior to proceeding with pericardiectomy. Some patients can have an improvement in pericardial compliance if they have only transient constriction from inflammation.14 surgical complete pericardiectomy is indicated to relieve symptoms in patients with CP.

Case report

A 46-year-old female patient came with a complaint of generalized body swelling. The patient is a young woman who has had swelling of her whole body intermittently every few months for two years ago, which lasts for a few days and resolves by itself, and when she rests and sleeps in bed, the duration and intensity of the swelling increases. While the swelling has been resolved faster with activity, during this period since the onset of the disease, the patient has not visited a doctor until after one year. The period of the patient's generalized swelling became shorter and once a month with a length of It lasted from one week to ten days and gradually symptoms such as heart palpitations, shortness of breath, fatigue, feeling of suffocation, and chest pain was added from time to time. It was not possible to refer to higher centers, and finally, after six months, due to the fact that generalized swelling and the mentioned symptoms were chronic and permanent and without improvement, the patient was referred to Imam Khomeini Hospital in Ardabil. To begin the diagnosis, a Complete Blood Cell (CBC) count, Thyroid Hormone Measurement (TSH), and complete ultrasound of the abdomen and pelvis were performed. And to confirm the diagnosis, echocardiography and CT scan were performed.

Result

A complete blood cell count test showed a decrease in white blood cells, red blood cells, and platelets. In addition, the Coagulation Test (PTT) showed a significant increase. The Thyroid Hormone Test (TSH) showed normal thyroid function. Complete ultrasound of the abdomen and pelvis shows severe dilatation of the intrahepatic veins, which raises the possibility of right heart failure. In addition, a small amount of free fluid was observed in most of the abdominal and pelvic spaces. During echocardiography, numerous radiographs and clinical examinations including severe heart failure and Cliff Pericardial effusions as well as rub friction have been diagnosed in the lung auscultation for the patient with constrictive pericarditis (Figure 1).

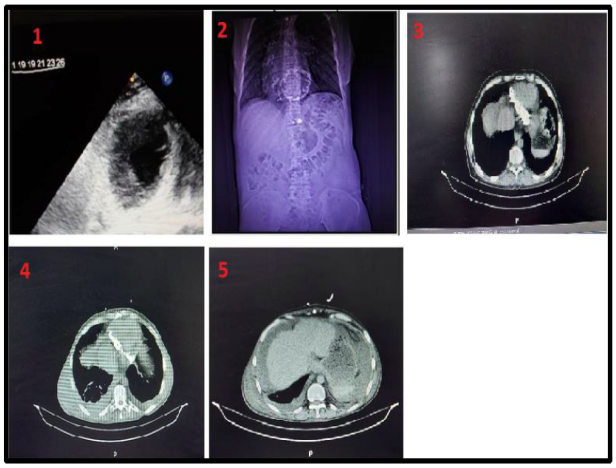

Figure 1: Echocardiography of the patient showing pericardial calcification, 2. Chest radiograph of the patient in PA view showing pericardial calcification, 3. CT scan of the patient; Mediastinal view showing pericardial calcification, 4. CT scan of the patient's chest in the Mediastinal view showing bilateral pleural effusion and pericardial calcification, 5. CT scan of the patient's chest in the Mediastinal view in the end cuts showing pleural effusion of the right lung and abdominal ascites.

It was also reported in the radiographs taken for the patient with pleural effusion and abdominal ascites that during thoracocentesis and palpation of the ascites, the liquid ascites were transudate. It was due to heart failure caused by constrictive pericarditis, and atrial fibrillation rhythm and inverted that was evident in the precordial and inferior leads in the patient's ECG. It should be noted that the patient had no family history, no history of any disease, surgery, or trauma, and no history of taking any type of medicine. It should be noted that the patient was not diagnosed with tuberculosis and hypothyroidism. Finally, the patient was referred to Tehran medical centers and a pericardiectomy was performed for the patient, unfortunately, the patient died one and a half months after the operation.

Conclusions

The diagnosis of constrictive pericarditis remains a challenge because its physical findings and hemodynamics mimic restrictive cardiomyopathy. Various diagnostic advances over the years enable us to differentiate between these two conditions.

Unfortunately, the case mentioned in this study lost her life, but the need to diagnose and follow-up referrals with similar symptoms seems mandatory.

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