Case Report

A case of transient constrictive pericarditis in rural Nova Scotia

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Abstract

This case report describes a 32-year-old female who presented with severe dyspnea and exertional intolerance to an emergency department in a rural area of Nova Scotia. The diagnosis was made by corroborating the value of the erythrocyte sedimentation rate, the level of N-terminal prohormone of brain natriuretic peptide, and a transthoracic echocardiogram, which respectively showed an inflammatory pattern, myocardial strain, and classic sonographic findings consistent with constrictive pericarditis. The patient was treated with oral colchicine, naproxen, and prednisone resulting in complete resolution of laboratory and sonographic abnormalities. Availability of echocardiography in a rural setting can promptly and definitively diagnose and rule out many structural and functional disorders of the heart, including rare pathologies such as constrictive pericarditis.

Case

A 32-year-old woman developed painful, erythematous, swollen feet with a CRP of 170 (mg/L), and no systemic unwellness or other findings. She had otherwise no known allergies or sensitivities. Dermatology assessment included post-viral etiology, erythromelalgia, and chilblains in the differential. She tested negative for COVID-19.

Six months later, she experienced new left-sided pleuritic chest pain, gradually worsening fatigue, dyspnea, and orthopnea over three days. This culminated in a presentation to the emergency department with severe dyspnea. She settled on presentation but remained tachycardic at 124 bpm with other vitals within normal. The jugular venous pulse was elevated. A pericardial knock was audible in early diastole. Initial bloodwork was notable for a normocytic anemia, an elevated CRP, and an elevated NT-proBNP (Table 1). ECG showed sinus tachycardia and CXR showed new cardiomegaly with small bilateral pleural effusions (Figure 1).

The initial echocardiogram showed marked pericardial thickening up to 1.2 cm extending along the inferior and lateral aspects of the left ventricle (Figure 2). There was a leftward ventricular septal motion on inspiration and a marked septal bounce. Mitral and tricuspid inflows showed inspiratory variation: mitral valve 14% and tricuspid valve 30%. The IVC and hepatic veins were plethoric, demonstrating an expiratory diastolic flow reversal. The combination of these findings were diagnostic of constrictive pericarditis[1].

The patient was started on outpatient colchicine 0.6mg daily and naproxen 500mg BID[2], and experienced the resolution of symptoms within the first week. She underwent follow-up echocardiogram at 2 weeks, 6 weeks, and 4 months. Due to an intolerance, colchicine was changed to prednisone 30mg daily after 2 weeks[3]. Subsequent imaging showed resolution of pericardial thickening, respiratory leftward ventricular septal motion, septal bounce, mitral and tricuspid valve inflow variation, and diastolic flow reversal in the IVC (Figure 2), indicating complete reversal of constrictive physiology. Inflammatory markers and the NT-proBNP normalized (Table 1).

Constrictive pericarditis is a member of pericardial syndromes comprising pericarditis, pericardial effusion, and cardiac tamponade[4]. These syndromes typically occur from an inflammatory process of the pericardium. Under normal physiological conditions, the pericardium is a thin elastic sack that stretches to accommodate the dynamic expansion of the ventricles. Whereas in constrictive pericarditis, the pericardium becomes thicker and more rigid, turning into a cage that counters the expansion of the ventricles[1]. This alters normal hemodynamics of the heart, mainly by increasing filling pressures in the right ventricle during inspiration, which leads to congestive findings of elevated jugular venous pulse and hepatic vein flow reversal. The increased pressures in the right ventricle also force the interventricular septum into the left ventricle, which produces the septal bounce and inspiratory septal variation as seen on echocardiography (Figure 2). These hemodynamic changes lead to the clinical presentation of heart failure.

The most common reported causes of constrictive pericarditis are idiopathic and viral (42–49%), cardiac

Table 1. Comparison of select laboratory values at presentation and 1 month following treatment.

Marker		Value at presentation	Value after 1 month
WBC	[x 109/L]	10.1	9.3
Hgb	[g/L]	98	132
Plt	[x 109/L]	457	457
MCV	[mcm3]	86.3	87.6
CRP	[mg/L]	122	10.4
Troponin	[mcg/L]	< 0.012	-
NT-proBNP	[ng/L]	870	105

CRP: C-reactive protein. Hgb: hemoglobin. MCV: mean corpuscular volume. NT-proBNP: N-terminal pro-hormone B-type natriuretic peptide. Plt: platelets. WBC: white blood cells.

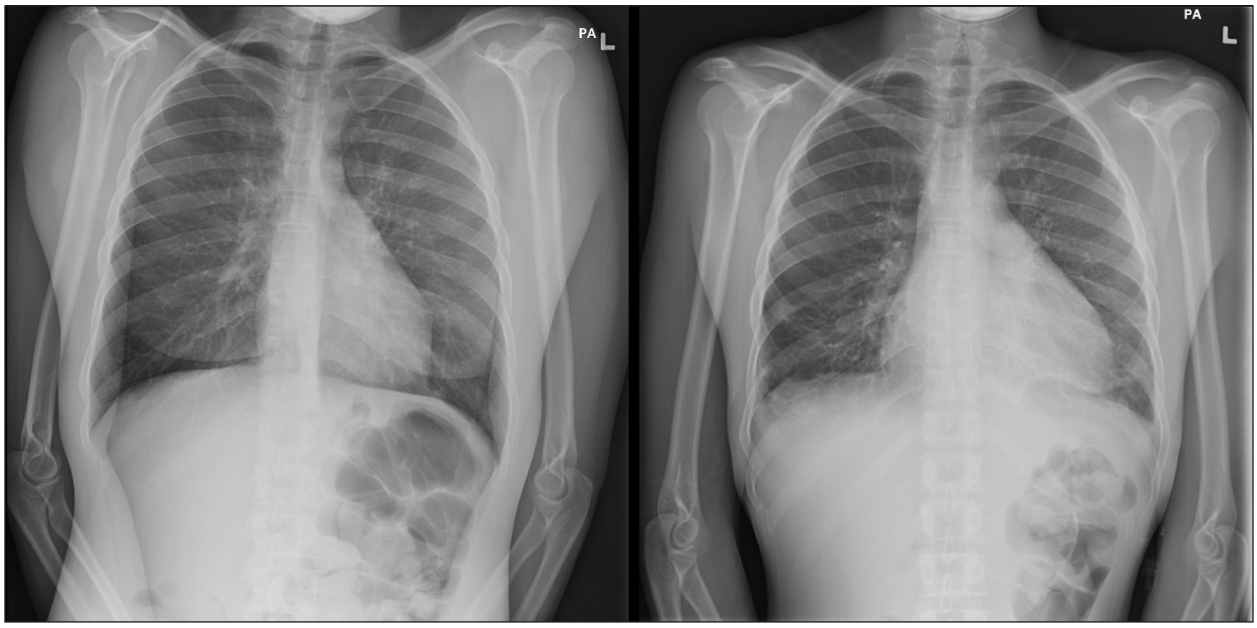

Figure 1. Comparison of diagnostic x-ray images of the chest in the posteroanterior view 6 months prior to onset of symptoms (left) and at clinical presentation (right) showing new and significant cardiomegaly, small pleural effusions bilaterally, linear opacities in the lung bases, and noticeable weight loss.

surgery (11–37%), radiation therapy (9–31%) (especially for Hodgkin's lymphoma and breast cancer), and connective tissue disorders (3–7%)[4]. The mainstay of diagnostic work up is a chest x-ray and a transthoracic echocardiogram. If echocardiographic evidence is inconclusive, a multi-modal approach is recommended that involves cardiac MRI to further ascertain the presence structural and inflammatory pericardial abnormalities[4,5].

First-line treatment is medical therapy with anti-inflammatory medications that includes dual therapy with colchicine and an NSAID, and triple therapy that adds steroids for more severe or refractory cases4. The constrictive pericarditis is labeled as transient when pathology resolves without residual disease[4,5], and is often attributed to viral or idiopathic cause. This was the case with our patient, and so, no further investigations into etiology were performed. In other cases, diagnosis is often delayed with average work up duration of up to 2 years. Longer chronicity tends to be more refractory to medical treatment due to pericardial fibrosis and calcification[5]. Failure of symptoms resolution with medical therapy after 3 months necessitates further investigations for underlying causes (e.g. rheumatological work up, cardiac MRI), addressing any new findings, and an assessment for a pericardiectomy[4].

Availability of echocardiography in a rural setting can definitively diagnose and rule out many structural and functional disorders of the heart. This exemplary case of a rare pericardial pathology was discovered with echocardiography and completely reversed with anti-inflammatory agents, which possibly prevented progression to pericardial fibrosis and irreversible constriction[4].

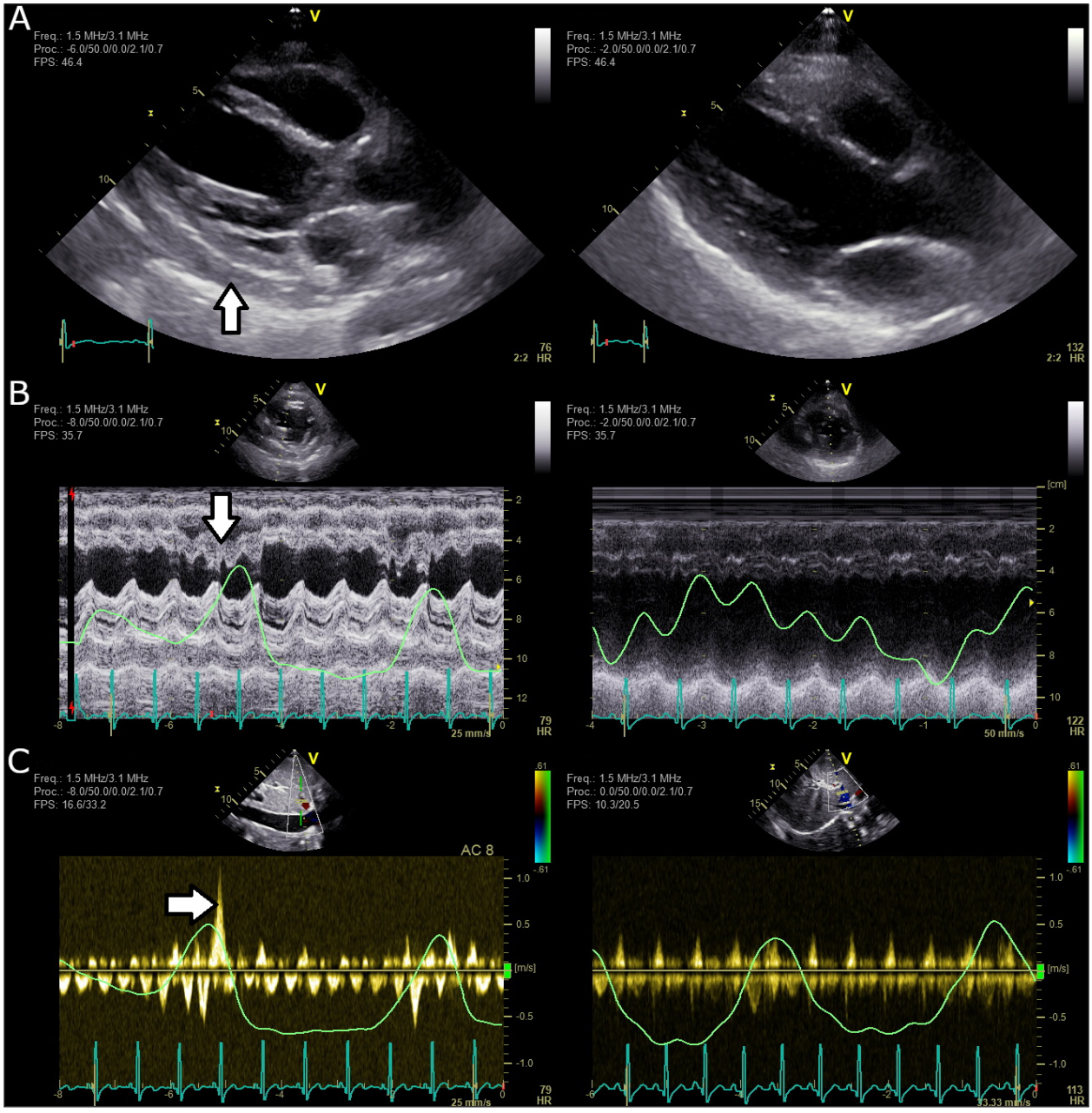

Figure 1. Comparison of echocardiographic images of the pericardium in the transthoracic long view (A), the septal motion in the M mode of transthoracic transverse view (B), and blood flow through the hepatic vein in the subcostal view (C). The cardiac rhythm and respiratory strips are present where appropriate. At clinical presentation (left), the images showed thickened pericardium of 1.2 cm (arrow), leftward inspiratory ventricular septal motion (arrow), and retrograde inspiratory hepatic vein flow (arrow), in keeping with constrictive pericarditis, whereas these features resolved at 4 months following treatment (right).

References

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