CARDIAC TAMPONADE

A RARE COMPLICATION OF COXSACKIEVIRUS A PERICARDITIS

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Introduction

Pericardial effusion is a complication seen in 60% of viral pericarditis cases[1]; however, tamponade is rarely seen with only 5% described cases[2].

Although coxsackievirus and other RNA enteroviruses account for 25-40% of acute myocarditis and pericarditis cases in young adults annually, only 4 cases have been reported to cause cardiac tamponade, all which are caused by coxsackievirus type B[1,3].

Coxsackievirus can be divided into 2 groups and 29 subtypes: type A causes meningitis, hand foot mouth disease, hemorrhagic conjunctivitis, and herpangina while type B causes meningitis, pericarditis, pleuritis, pancreatitis, and hepatitis. The virus is primarily transmitted by oral-fecal route, with infection most commonly seen in children and males (77.9% of cases, mostly detected from June to October)[1,4].

Here we present a rare case of a 51-year-old woman who developed cardiac tamponade as a result of coxsackie A pericarditis.

Case

History
A 51-year-old female with a past medical history of hypertension, seasonal allergies, and Meniere's disease presented to the ED with sudden onset severe retrosternal chest pain, nausea, vomiting, dizziness, and diaphoresis. She had flu-like symptoms and a papulovesicular rash on her hands and feet **(Fig. 1)** two-months prior, which was reduced to a dry scaly rash through topical steroids.

Pertinent physical exam findings:
Vital signs were significant for hypotension (88/50 mmHg), tachycardia (130 bpm), elevated jugular venous pressure, and low SpO_2 (86%) on room air.

Imaging:
Chest x-ray showed cardiomegaly with clear lungs. Electrocardiography (**Fig. 2**) revealed low voltage, sinus tachycardia, and non-specific ST changes. CT pulmonary angiogram was performed due to the suspicion of pulmonary embolism (PE). The test was negative for PE however, a large pericardial effusion was seen **(Fig. 3A & B)**. Bedside echocardiography showed collapse of cardiac chambers in diastole and appearance of swinging heart, consistent with tamponade physiology.

Management:
Urgent pericardiocentesis and right heart catheterization was done, draining 800 ml of serosanguinous fluid, exudative with 4,500 white blood cells and 625,000 red blood cells. Her hemodynamics improved after the procedure.

Further workup:
Subsequent lab work showed high antibody titers of Coxsackievirus A2 (1:32), A4(1:32), A9(1:16), and A10(1:16). The patient also had Epstein Barr Virus IgG and positive anti-dsDNA antibodies, although no past medical history of an autoimmune disorder was noted.

Outcome:
The patient was discharged on indomethacin and colchicine. Serial subsequent echocardiograms showed residual 1.3 cm posterior pericardial effusion and stable ejection fraction.

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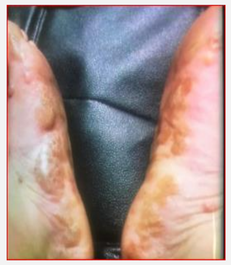


Figure 1: Papulovesicular rash of the feet of our patient, seen two months prior to developing coxsackievirus A pericarditis.

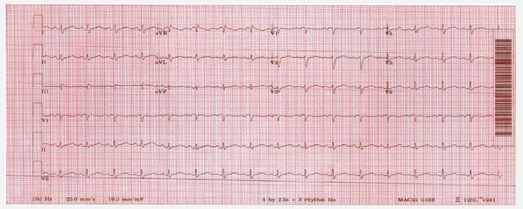


Figure 2: 12-lead electrocardiography of our patient showing low voltage QRS, sinus tachycardia and nonspecific ST changes

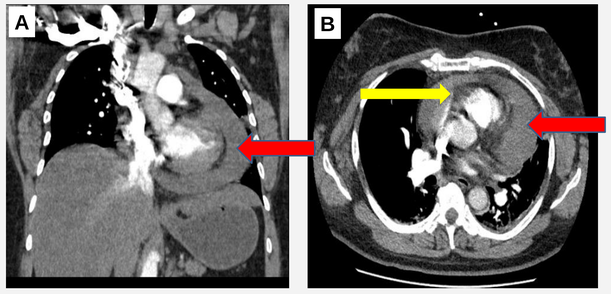
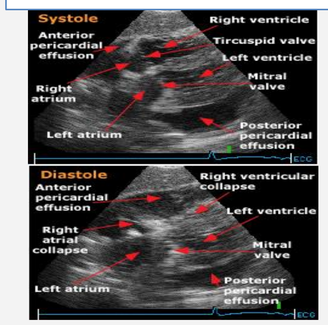


Figure 3: CT pulmonary angiogram **(A)** coronal and (**B**) axial views of the lung showing large pericardial effusion (red arrow) and a collapsed right ventricle (yellow arrow).

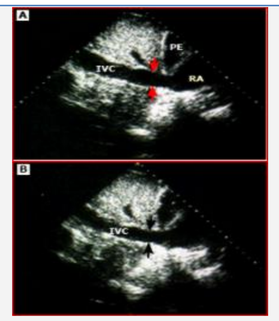
Learning Objective

1- Recognize coxsackievirus type A as a cause of cardiac tamponade in a young, otherwise healthy female.

2- Describe the medical management of cardiac tamponade.



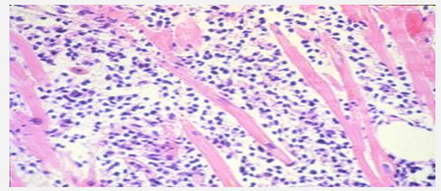
Echocardiogram showing collapse of right atrium and right ventricle during inspiration due to pericardial effusion.
Source: UpToDate



A: The subcostal view of inferior vena cava in a patient with cardiac tamponade. The IVC is plethoric, measuring over 20 mm in diameter.
B: During inspiration, the IVC diameter fails to decrease. There is a large pericardial effusion (PE) surrounding the right atrium (RA).
Source: UpToDate

Discussion

Management of Coxsackievirus Pericarditis:
The exact mechanism of coxsackievirus induced cardiac injury includes immune mediated and direct viral cytotoxicity. Laboratory diagnosis of coxsackievirus is warranted in cases of serious complications, including myopericarditis. RT-PCR is typically used for detection, however endomyocardial biopsy is the gold standard for confirmation of viral infection[5]. Cell culture based methods are only used for isolated serotyping. Serology is not used for diagnosis of acute enterovirus disease. Treatment options are immunosuppressive agents, antivirals and natural medicines. Use of intravenous immunoglobulin is warranted in severe infections[6].



Myocardial muscle in acute myocarditis, with diffuse mononuclear infiltrate and myofiber necrosis.
Source: UpToDate

Management of cardiac tamponade:
The clinical findings of cardiac tamponade are Beck's triad (hypotension, elevated JVP and muffled heart sounds), electrical alternans on ECG, pulsus paradoxus, positive hepatojugular reflux, and a picture of cardiogenic shock.

Standard treatment is drainage of pericardial effusion, which can be performed percutaneously using catheter drainage or surgically. An indwelling catheter is left in the pericardial space, until fluid output is less than 25 ml/day. Patients should be monitored on telemetry for 24-48 hours after the procedure, and echocardiography should be done prior to discharge from the hospital. Inotropes and positive pressure ventilation should be avoided to prevent worsening hemodynamics.

Subsequent treatment includes activity restriction and combination therapy with NSAIDs (tapered dose weekly for 2-4 weeks) and colchicine (3 months). Glucocorticoids are used as second line therapy for refractory cases. Recurrence is seen in 30% patients without preventive therapy. A number of patients with acute viral myocarditis may develop dilated cardiomyopathy as a complication.

Conclusion

Most common cause of viral myopericarditis is coxsackievirus, most common types being B1 through B4. Our case was unique as she had high antibody titers of A4,A9 and A10, and was complicated by cardiac tamponade. Complete resolution was symptoms was achieved with pericardiocentesis and NSAIDs therapy.

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