

### Introduction

Cardiac tamponade is a rare and very serious complication associated with *Mycoplasma pneumoniae*. *M. pneumoniae* usually causes respiratory tract infection in young children and teenagers and rarely causes extrapulmonary manifestations. Pericardial effusion leading to cardiac tamponade is a serious complication and correct diagnosis in a timely manner with appropriate intervention is the key between life and death. Here we present a case of cardiac tamponade in which prompt and efficient management saved the patient's life.

### **Clinical Case**

A 19-year-old male presented to the ER with complaints of pleuritic chest pain that occurred over the last 3 days. The pain was described as dull and substernal in location. The pain was aggravated with inspiration and when supine and relieved by leaning forward. Other associated symptoms included fever, cough, headache, fatigue, and abdominal pain. He had no past medical history, denied consumption of medications, tobacco, illicit drugs, or alcohol.

On examination, the patient was afebrile and tachycardic (HR 116 beats/min). His ECG showed diffuse ST elevation and PR depression (Figure 1) with positive serum *Mycoplasma pneumoniae* IgM. A diagnosis of acute pericarditis secondary to Mycoplasma pneumonia was established and he was placed on doxycycline, ibuprofen, and colchicine.

During admission, rapid response was called for the sudden loss of consciousness, asystole, and hypotension. Telemetry showed pauses of greater than 3 seconds followed by sinus arrhythmia. Cardiac examination revealed muffled heart sounds and jugular venous distention. The patient was placed on transvenous pacing and CT pulmonary angiogram (CTPA) was performed to rule out pulmonary embolism (PE) due to right heart strain seen on ECG. The result of the CTPA (Figure 2) was negative for PE, however, a large pericardial effusion - suspicious for cardiac tamponade, bilateral pleural effusion with patchy infiltrates, and ascites were seen. He was immediately taken for pericardiocentesis where one liter of hemorrhagic fluid was removed. The patients' symptoms immediately improved. The patients' serology was later found to be also positive for Coxsackie type B5, 6. Repeated chest x-ray and transthoracic echocardiogram revealed resolution of pericardial effusion and well-aerated lungs. The pericardial drain was removed on day 4 and the patient was discharged home with oral antibiotics.

# Discussion

Cardiac tamponade is a serious and deadly complication of *M. pneumoniae* and Coxsackie co-infection which should be considered as one of the causes of idiopathic acute pericarditis causing pericardial effusion impending cardiac tamponade. While most cardiac manifestations are thought to be due to direct bacterial infection, through the bloodstream, the bronchial lymphatics or direct seeding but some cases may be immune related. Serology still remains the mainstay test to diagnose *M. pneumoniae* and Coxsackie infections. It can also be postulated that patients with co-infection with *M. pneumoniae* and Coxsackie B could increase the severity of cardiac manifestations as group B Coxsackievirus is known to frequently cause myocarditis.

# Conclusion

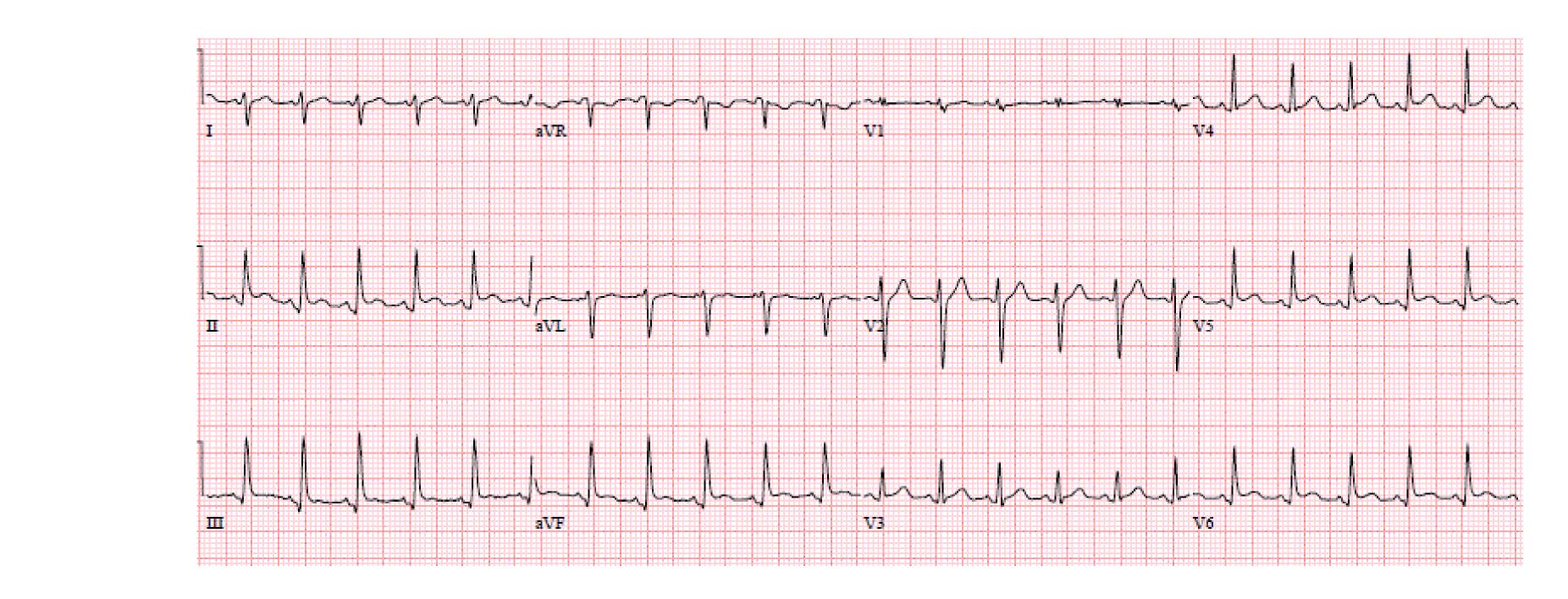
Cardiac manifestations associated with M. pneumoniae are rare presentations but co-infection with Coxsackievirus could increase the severity drastically and hence correct diagnosis with appropriate intervention and treatment is the key.

#### **References:**

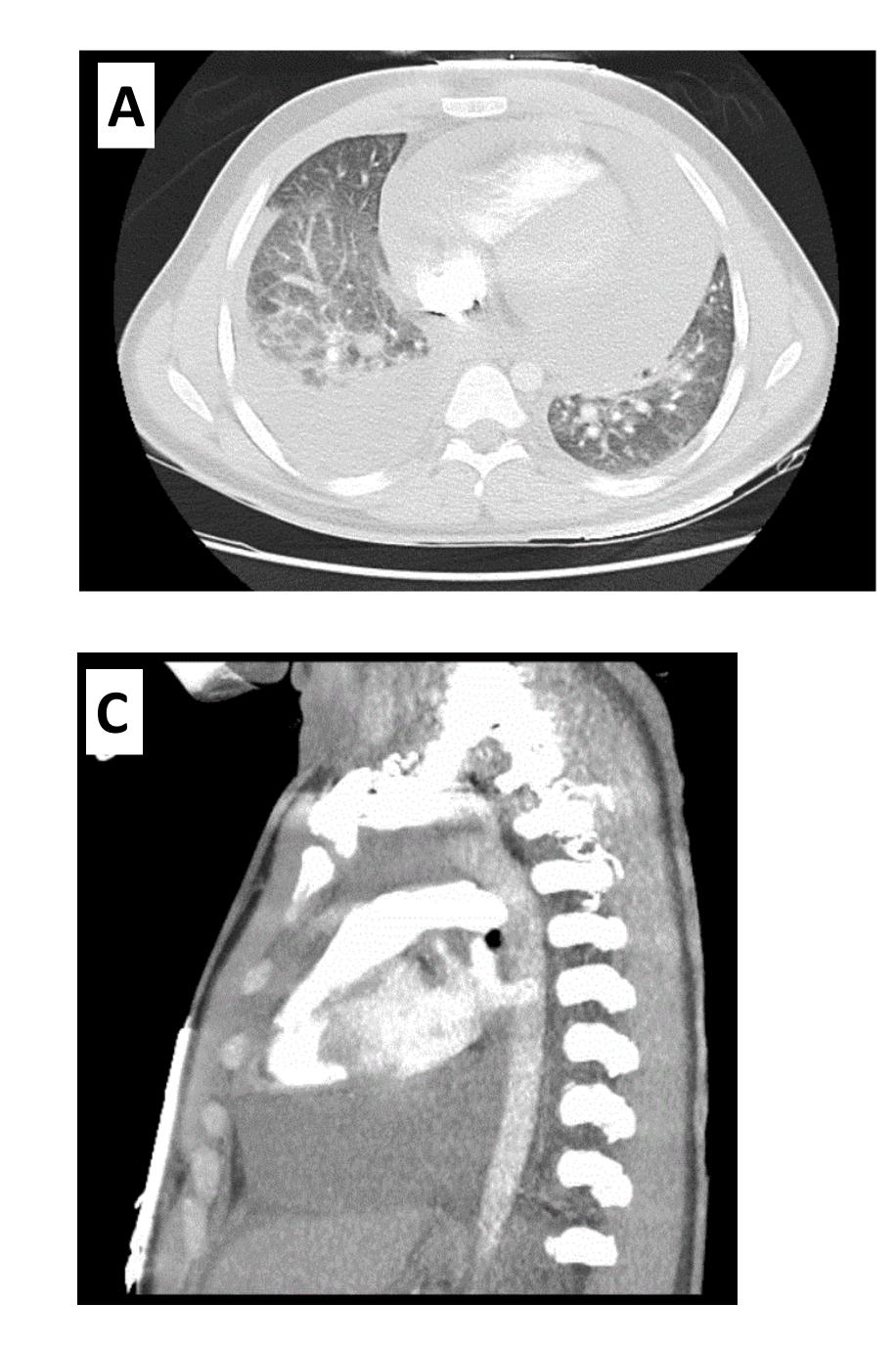
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**Figure 1.** Electrocardiogram showing sinus tachycardia, rightward axis, and ST segment elevation with PR depression, classically seen in acute pericarditis.



**Figure 2.** Computed tomography pulmonary angiography scan (A) axial, (B, D) coronal and (C) sagittal views. Large pleural effusion is seen (right lung greater than left) in addition to cardiac tamponade with large pericardial effusion with no enlargement of the heart. Large mildly complex pericardial effusion with subdiaphragmatic reflux of contrast into the intrahepatic IVC and hepatic veins are clearly illustrated in (D). There were no filling defects to indicate pulmonary embolism.



