Case Report / *Olgu Sunumu*

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A Rare Case of Pericarditis Complicated by Pleuro-Pericardial Effusion due to Rhinovirus/Enterovirus Infection in a 16-Year-Old Male

16 Yaşında Bir Erkekte Rinovirüs/Enterovirüs Enfeksiyonuna Bağlı Plevro-Perikardiyal Efüzyonla Komplike Olan Nadir Bir Perikardit Olgusu

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Abstract

A previously healthy 16-year-old male presented with chest pain, dyspnea, and fever. Clinical, radiological, and laboratory assessments confirmed the diagnosis of pericarditis accompanied by pleuropericardial effusion. Reverse transcriptase polymerase chain reaction analysis of a nasopharyngeal swab identified rhinovirus/enterovirus as the causative agent. Extensive investigations ruled out other viral etiologies, malignancies, and collagen vascular diseases. This case highlights a rare presentation of pericarditis with associated pleuropericardial effusion secondary to rhinovirus/enterovirus infection.

Keywords: Rhinovirus, enterovirus, pericarditis, pleural effusion, pericardial effusion

Öz

Daha önce sağlıklı olan 16 yaşındaki erkek hasta göğüs ağrısı, nefes darlığı ve ateş şikayetiyle kliniğimize başvurdu. Klinik, radyolojik ve laboratuvar değerlendirmeler, plevroperkardiyal efüzyonun eşlik ettiği perikardit tanısını doğruladı. Nazofaringeal sürüntüden yapılan ters transkriptaz polimeraz zincir reaksiyonu analizi, etken maddenin rinovirüs/enterovirüs olduğunu ortaya çıkardı. Kapsamlı araştırmalar, diğer viral etiyolojileri, maligniteleri ve kolajen vasküler hastalıkları ekarte etti. Bu olgu, rinovirüs/enterovirüs enfeksiyonuna bağlı plevroperikardiyal efüzyonun eşlik ettiği nadir bir perikardit olgusunu sunmaktadır.

Anahtar Kelimeler: Rinovirüs, enterovirüs, perikardit, plevral efüzyon, perikardiyal efüzyon

Introduction

Viral infections are among the most common causes of pericarditis in children. While enteroviruses are the leading pathogens in such cases, other viruses—including Epstein-Barr virus (EBV), cytomegalovirus (CMV), human immunodeficiency virus (HIV), hepatitis B virus (HBV), and herpes simplex virus—may also contribute to its development (1). Human rhinoviruses, which are closely related to human enteroviruses, are responsible for the majority of acute respiratory tract infections observed throughout the year (2). Although there is currently no specific antiviral therapy or available vaccine, these infections

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are typically self-limiting and managed with supportive care (3). However, recent studies and case reports have highlighted that these viruses may occasionally result in life-threatening complications (4). In this report, it was aimed to present a rare case of pericarditis with pleuropericardial effusion associated with rhinovirus/enterovirus infection.

Case Report

A 16-year-old previously healthy male was admitted to Ondokuz Mayıs University Faculty of Medicine Hospital with a 10-day history of chest pain accompanied by dyspnea and radiation to the left shoulder and neck. His history revealed an upper respiratory tract infection approximately one week prior to symptom onset. There were no signs of acute gastroenteritis. He was a boarding school student, and his family lived in a rural area engaged in animal husbandry. He reported close contact with a friend who had a cough.

On physical examination, his body temperature was= 37.8 °C, heart rate= 104 beats/min, blood pressure= 122/56 mmHg, and respiratory rate= 24/min. Auscultation revealed decreased breath sounds at the bilateral lung bases and distant heart sounds. Other systemic examinations were unremarkable.

Laboratory investigations showed neutrophilic leukocytosis and a marked acute phase response: White blood count= 12.66×10^3/μL, neutrophils= 67.5%, lymphocytes= 7.6%, monocytes= 20.6%, hemoglobin= 12.5 g/dL, hematocrit= 37.6%, red blood cells= 4.58×10^6/μL, platelets= 134×10^3/μL. C-reactive protein was= 104 mg/L (reference <5 mg/L) and erythrocyte sedimentation rate= 42 mm/h (reference <15 mm/h). Procalcitonin was= 0.06 ng/mL at admission and increased to 0.33 ng/mL on day 2. Troponin I was elevated at 140.86 ng/L (reference <47.34 ng/L). Serum electrolytes, renal and liver function tests, and thyroid function were within normal limits.

Chest radiography revealed obliteration of the left costophrenic angle. Thoracic computed tomography confirmed pericardial and pleural effusion (Figure 1). Echocardiography showed a high-density pericardial effusion measuring approximately 19-20 mm in thickness anterior to the right ventricle. The thickness of the left ventricular wall and interventricular septum was at the upper limit of normal. Based on these findings, pericardiocentesis was performed for diagnostic and therapeutic purposes.

Analysis of the pericardial fluid revealed a leukocyte count of 3251/μL and an erythrocyte count of 0.014×10^3/μL. Differential cell counts showed 2496×10^3/μL polymorphonuclear and 755×10^3/μL mononuclear cells. Biochemical analysis showed lactate dehydrogenase 504 U/L, albumin 29 g/L, and total protein 48.4 g/L.

Rhinovirus/enterovirus RNA was detected in the nasopharyngeal swab using real-time polymerase chain reaction (RT-PCR), while all other viral pathogens were negative. EBV, CMV, HIV, and HBV were not detected in the virological panel. Interferon-gamma release assay (Quantiferon), acid-fast bacilli (AFB) staining, and PCR for *Mycobacterium tuberculosis* complex were all negative. Brucella agglutination tests (Rose-Bengal and Coombs) were also negative. No growth was observed in blood or pericardial fluid cultures on day one or after one week. Mycobacterial cultures (automated system) were also negative.

All autoimmune serological tests, including anti-dsDNA, antinuclear antibody (ANA), c-ANCA, p-ANCA, and ANA subgroups, were negative. Familial Mediterranean fever (MEFV) gene mutation screening for MEFV yielded no mutations.

Given the presence of extensive pleural effusion, neutrophilic predominance, and systemic inflammation, empirical meropenem and vancomycin were initiated to

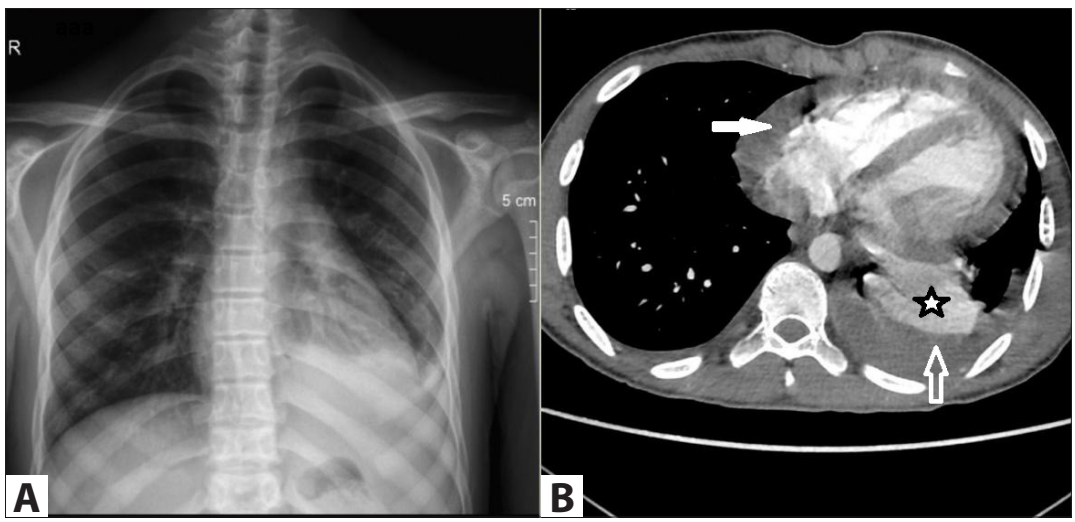

Figure 1. A. Chest radiography shows the appearance of pleural effusion on the left. **B.** Thorax CT scan shows pericardial (white arrow) and left pleural effusion (hollow arrow). There is also compressive atelectasis in the lower lobe of the left lung adjacent to the effusion (asterisk).

cover potential bacterial pathogens. Colchicine was added for its anti-inflammatory effect. The patient was followed up with weekly echocardiography and thoracic ultrasonography.

The patient's clinical status improved rapidly. Follow-up echocardiography on hospital day 10 showed complete resolution of the pericardial effusion (Figure 2). Antibiotic therapy was discontinued on day 28, and the patient was discharged after completing one month of supportive treatment.

Discussion

Viral pericarditis is the most common cause of acute pericarditis in the pediatric population (5). Among viral agents, enteroviruses are the most frequently implicated; however, adenovirus, parvovirus B19, HIV, EBV, CMV, other herpesviruses, and coronavirus disease-2019 may also be involved (6). Patients with viral pericarditis typically present with fever and precordial chest pain, and they generally appear less toxic than those with bacterial pericarditis (5). In our case, the patient presented with precordial chest pain, dyspnea, and low-grade fever.

Other important infectious causes of pericardial effusion include bacteria. The most isolated organisms in purulent bacterial pericarditis are *Staphylococcus aureus*, group A *Streptococcus*, *Salmonella* spp., *Pseudomonas aeruginosa*, and other gram-negative bacilli (6). While acute idiopathic or presumed viral pericarditis is the leading cause of pericardial effusion in developed countries, tuberculosis remains a major etiology in developing regions, with rates exceeding 70% in some series (6,7). In our case, tuberculous pericarditis was considered in the differential diagnosis. However, AFB staining, interferon-gam-

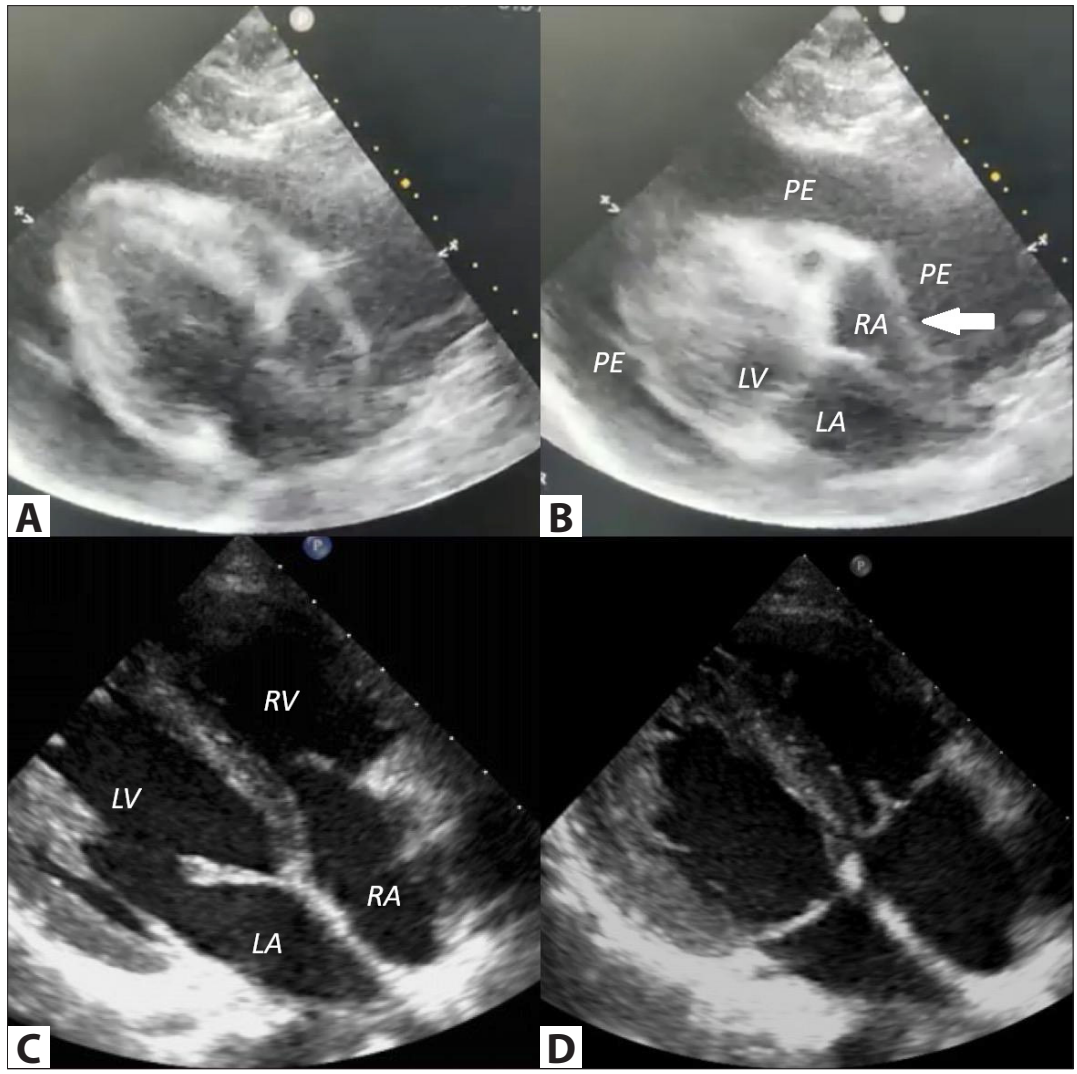

Figure 2. The patient's parasternal four-chamber views: **(A,C).** During ventricular diastole **(B,D).** During ventricular systole. The right atrial wall is markedly compressed (**B** – white arrow) and excessive pericardial effusion is observed with the free movement of the heart **(A,B).** After the treatment is completed and the patient's pericardial effusion has resolved **(C,D)**, the atria and pericardium appear normal **(D)**.
PE: Pericardial effusion, RA: Right atrium, RV: Right ventricle, LA: Left atrium, LV: Left ventricle.

ma release assay (Quantiferon), mycobacterial cultures, and PCR for *M. tuberculosis* complex were all negative. In addition, brucellosis was excluded based on negative Rose-Bengal and Coombs tests in a patient with known zoonotic exposure.

Non-infectious etiologies such as autoimmune diseases, malignancies, metabolic disorders, rheumatologic conditions, and trauma were also considered and systematically excluded.

Enteroviruses can cause a wide spectrum of clinical presentations in children, ranging from non-specific febrile illness, upper respiratory tract infections, and hand-foot-mouth disease to severe manifestations such as encephalitis, hepatitis, myocarditis, and pericarditis (8). Rhinovirus, which belongs to the same family (Picornaviridae), is generally associated with mild upper respiratory symptoms, including rhinorrhea, nasal congestion, cough, sneezing, sore throat, and headache. However, recent evidence suggests that rhinovirus is not limited to the upper respiratory tract and can occasionally lead to severe complications such as lower respiratory tract infections, acute respiratory distress syndrome, and myocarditis (9).

Moreover, it has been reported that while enterovirus RNA typically becomes undetectable in the nasopharyngeal mucosa within 2-3 weeks (approximately 14-21 days), rhinovirus RNA may persist for up to 5-6 weeks (approximately 35-42 days) (10). In the present case, there was a history of upper respiratory tract infection approximately 7-10 days prior to presentation. Rhinovirus/enterovirus RNA was detected in a nasopharyngeal swab sample via RT-PCR. However, due to the inability to perform viral typing and quantitative PCR in our center, this represents a main limitation of this report.

Cardiac involvement due to rhinovirus is extremely rare. Thanh et al. have reported a case of non-fulminant myocarditis secondary to rhinovirus infection in a 13-year-old girl who fully recovered with medical treatment and did not require extracorporeal membrane oxygenation (11). Kanık-Yüksek et al. from Türkiye have described a case of spontaneous pericardial effusion in a four-month-old infant with bronchiolitis due to rhinovirus (12). Similarly, Tapparel et al. have reported rhinovirus-associated pneumonia and pericarditis in a 14-month-old boy (13).

In this context, the present case adds to the existing limited literature by documenting a rare instance of pericarditis complicated by pleuropericardial effusion associated with rhinovirus/enterovirus infection.

Informed Consent: Patient consent was obtained.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - EK; Design - EHE; Supervision - EHE; Resource - EHE, İOŞ; Data Collection and/or processing - EK; Analysis and/or interpretation - EHE, İOŞ; Literature search - EK; Writing - EK; Critical review - EHE, İOŞ, DB, FRK.

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