

Case Report

Acute Rheumatic Fever with Cardiac Tamponade: A Case Report

Kardiyak Tamponad ile Akut Romatizmal Ateş: Olgu Sunumu

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ABSTRACT

Acute rheumatic fever (ARF) is a common condition in children between the five and 15 age. It is of considerable importance to individual and public health in developing countries as being a preventable sequela to untreated or inadequately treated streptococcal tonsillopharyngitis. ARF has various manifestations which can seldom be life-threatening. Here, we present the case of a 12-year-old acute rheumatic fever patient with cardiac tamponade.

Keywords: Rheumatic fever, cardiac tamponade, *Streptococcus pyogenes*

ÖZET

Akut romatizmal ateş (ARF), 5-15 yaş arası çocuklarda sık görülen bir durumdur. Tedavi edilmeyen veya yetersiz tedavi edilen streptokok tonsillofarenjitinin önlenilebilir bir sekeli olarak gelişmekte olan ülkelerde birey ve toplum sağlığı için büyük önem taşımaktadır. ARF'nin nadiren yaşamı tehdit edebilen çeşitli belirtileri vardır. Burada 12 yaşında akut roma-tizmal ateşli bir hastada kardiyak tamponadı sunuyoruz.

Keywords: Romatizmal ateş, kalp tamponadı, *Streptococcus pyogenes*

INTRODUCTION

Acute rheumatic fever (ARF) is a non-suppurative sequela to un- or inadequately treated streptococcal (group A; streptococcus pyogenes) tonsillopharyngitis that often develops three weeks after the primary infection (1). ARF commonly yields extensive systemic involvement, including heart, joints, and brain, and is not only but essentially associated with collagen-reactive autoantibodies (2,3). Although the incidence of ARF has begun to decrease with the discovery of antibiotics (4), it still creates a public health burden in developing co-

untries and socioeconomically disadvantaged societies in developed countries (5,6). Carditis in ARF can be presented with chest pain, tachypnea, palpitations, dyspnea, muffled heart sounds, murmurs, and bibasilar crackles. Although pericardial effusion related to ARF pericarditis is relatively frequent, cardiac tamponade is an extremely rare complication. Here, we present a 12-year-old male patient diagnosed with cardiac tamponade developed due to ARF.

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CASE REPORT

A 12-year-old male patient was admitted with chest pain for two days. Tachypnea (35/min), tachycardia (124/min) and fever (38.5°C) were noted in the physical examination. There were muffled heart sounds accompanied by a 3/6 pansystolic murmur extending from the left apical region to the entire precordium and a mid-diastolic murmur in the right 2nd intercostal space. Corrigan pulse was felt on palpation. In initial echocardiography (echo), grade 2-3 aortic valve insufficiency and grade 2-3 mitral valve insufficiency were detected (Figure 1) alongside a pericardial effusion (23 mm) without cardiac tamponade. Electrocardiography (ECG) showed widespread concave ST elevation and PR depression in the limb leads and precordial leads (V2-6) with sinus tachycardia. Anti-streptolysin O (ASO) and erythrocyte sedimentation rate (ESR) were measured at 501 IU/mL and 66 mm/h, respectively. Additional laboratory findings are presented in Table 1. Due to carditis with fever and supporting laboratory findings, the patient was diagnosed with ARF with one major and two minor criteria according to revised Jones criteria (7) for moderate/high-risk populations (≥ 2 cases per 100000 school-aged children). Primary penicillin treatment (penicillin G; 1,200,000 IU, single dose) and prednisolone (0.5 mg/kg, every 6 hours) were started while Brucella coombs gel test and troponin I measurement were requested for differential diagnosis. Empiric ceftriaxone (50 mg/kg, every 12 hours) plus vancomycin (10 mg/kg, every 8 hours) was administered against purulent pericarditis. Other etiologies for carditis, such as hepatitis, malignancies and HIV infection, were ruled out by appropriate tests. Captopril (0.5 mg/kg, every 12 hours) was also added to the treatment for congestive heart failure. On the 2nd day, hypotension (80/55 mmHg), accelerated tachycardia (147/min) and jugular venous distention were noticed. The patient was immediately referred to an echocardiographic examination that revealed right atrial collapse and enlarged pericardial effusion (25 mm) (Figure 2) compatible with pericardial tamponade in conjunction with hemodynamic findings. The effusion was drained by percutaneous pericardiocentesis and furosemide (1 mg/kg, twice a week) was added to the treatment. The drainage material was requested to be gram-stained, cultured for aerobic bacteria and examined for viral genomes. Meanwhile, ongoing antibiotherapy was maintained. Following the result that no pathogen had been identified in the pericardial fluid, antibiotherapy was stopped on the 4th

day of his hospitalization. Since the tamponade was resolved following the drainage without any detectable wall injury and the hemodynamic status was improved in follow-up examinations for two weeks, the patient was discharged on the 17th day with the prescription of high-dose corticosteroid (prednisolone; 0.5 mg/kg, every 6 hours), angiotensin-converting enzyme inhibitor (captopril; 0.5 mg/kg, every 12 hours) and diuretic (furosemide; 1 mg/kg, twice a week). A relative improvement was observed in the aortic and mitral insufficiencies (both grade 2) in the pre-discharge echocardiography. The outpatient corticosteroids treatment was gradually decreased and stopped at the 4th week of discharge. Secondary penicillin treatment was prescribed to be continued life-long with the recommendation of follow-up visits at six months. The consent of the patient's parents was obtained in this case study.

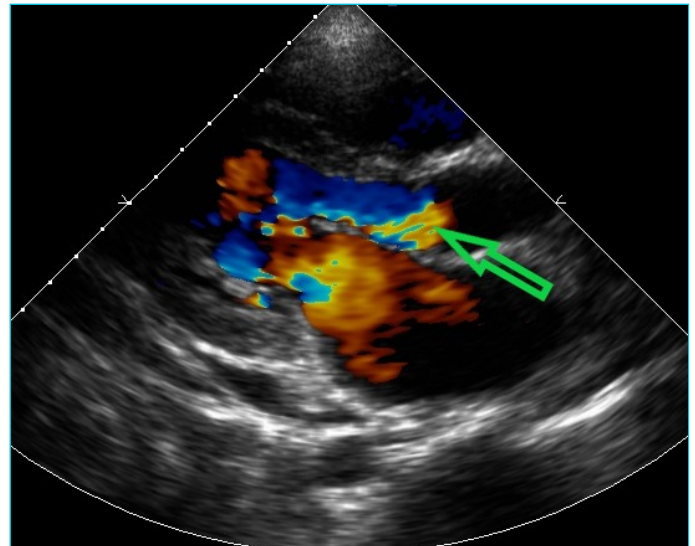


Figure 1. Heart valve dysfunctions in initial echocardiography

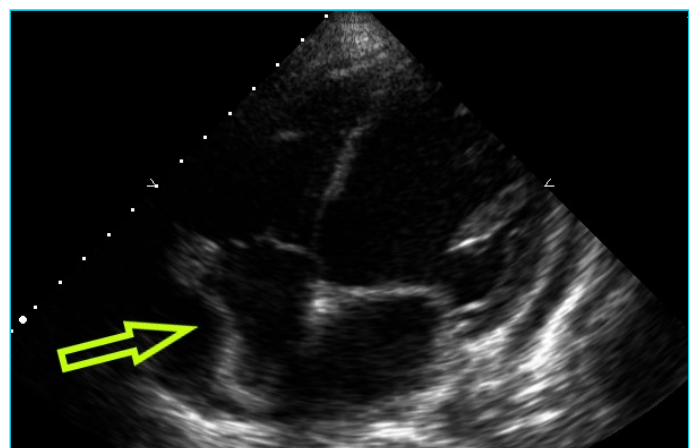


Figure 2. Cardiac tamponade in echocardiography

Table 1. Laboratory values of the patient

Analyte	Value	Reference Range
White blood cell	13.320 cells/ μ L	4.000-11.000 cells/ μ L
Neutrophil	10.000 cells/ μ L	1.500-8.500 cells/ μ L
Lymphocyte	1.840 cells/ μ L	1.500-6.500 cells/ μ L
Hemoglobin	10.8 g/dL	13.5-17.6 g/dL
Platelet	354.000 cells/ μ L	150.000-450.000 cells/ μ L
Monocyte	1.440 cells/ μ L	0-400 cells/ μ L
Eosinophil	40 cells/ μ L	0-200 cells/ μ L
C-reactive protein	1.51 mg/dL	0-0.5 g/dL
Urea	19 mg/dL	10.8-38.4 mg/dL
Creatinine	0.6 mg/dL	0.5-1.2 mg/dL
Albumin	3.09 g/dL	3.5-5.2 g/dL
Corrected calcium	8.9 mg/dL	8.8-10.6 mg/dL
Sodium	136 mEq/L	136-146 mEq/L
Potassium	4.41 mEq/L	3.5-5.1 mEq/L
Chlorine	102 mEq/L	101-109 mEq/L
Magnesium	1.8 mg/dL	1.8-2.6 mg/dL
Phosphorus	4.3 mg/dL	4-7 mg/dL
Uric acid	3.9 mg/dL	3.5-7.2 mg/dL
Alanine transaminase	24 U/L	0-50 U/L
Aspartate transaminase	20 U/L	0-50 U/L

DISCUSSION

Acute rheumatic fever (ARF), a delayed multisystemic inflammatory response to streptococcal tonsillopharyngitis in which diagnosis and treatment are crucial, is an important cause of morbidity and mortality if left untreated. ARF may be presented with intricate manifestations. For example, cardiac conduction system involvement may result in any type of atrioventricular blocks (8) and mesenteric inflammation may bring about abdominal pain, which is frequently mild and self-limiting but occasionally become severe enough to cause acute abdomen (9). Recent revisions in the diagnos-

tic criteria for ARF, including the consideration of low- and moderate/high-risk populations, provided physicians with finer means to distinguish this potentially catastrophic condition (10). According to the revised Jones criteria (7), alongside the evidence of preceding group A streptococcal infection (*Streptococcus pyogenes*), the major risk factors are clinical and/or subclinical carditis, mono-/polyarthritits or polyarthralgia, Sydenham chorea, subcutaneous nodules, and erythema marginatum, whereas prolonged rate-adjusted PR interval without carditis, monoarthralgia, fever ($\geq 38^{\circ}\text{C}$), ESR ≥ 30 mm/h and/or CRP ≥ 3 mg/dl (must be greater than the upper limit of the laboratory reference) are the minor risk factors in moderate/ high-risk populations as in the present case report. ARF can involve all main layers of the heart and lead to pancarditis. Endocarditis in ARF can result in clinically inapparent heart valve dysfunctions, termed subclinical (silent) carditis. The patient presented in this report had pansystolic and mid-diastolic murmurs pointing out clinical (evident) carditis and was fulfilling a major (clinical carditis) and two minor criteria (fever and prolonged ESR) with validated past streptococcal infection (elevated ASO).

In pediatric practice, collagen vascular diseases, bacterial and viral infections and metabolic disorders are the most common non-idiopathic causes of pericarditis. The definitive diagnosis is established by echocardiography, although pericardial rub and electrocardiographic changes support the diagnosis. Pericarditis occurs in up to one-tenth of patients with ARF and is oft related to pancarditis (11,12). Acute pericarditis manifested with chest pain, fever, tachypnea and tachycardia in our patient.

The most interesting part of the present case was ARF-related cardiac tamponade which is extremely rare in the medical literature. We are aware of only five previous cases with tamponade (see Table 2) (13-17). All five cases have been subjected to pericardiocentesis, and no pathogen has been identified in drainage fluid in any case. Similarly, neither bacterial nor viral infection was detected in our case. We observed a pericardial effusion with 23 mm of separation in initial echocardiography; however, the successive echocardiography performed due to clinical deterioration along with jugular distention demonstrated a slight increase in effusion (25 mm) compressing the right atrium that suggests the development of cardiac tamponade. Regarding medical imaging, 2 mm of enlargement in the effusion might be considered insignificant. However, the case we presented here emphasizes even such a minute increase in the

effusion volume can engender a risk for cardiac tamponade when the pericardial reserve is exceeded.

Conclusively, the present case underlines that ARF can involve all layers of the heart, including the pericardium, and ARF-related pericarditis can provoke cardiac tamponade, which is seldom but unignorable as being a life-threatening complication.

Table 2. Previous case reports of rheumatic fever with cardiac tamponade

Case	Age	Sex	Major Findings	Minor Findings	Origin	Ref.
1	11	Female	Clinic carditis	Elevated ESR & CRP, fever	Brazil	13
2	12	Female	Clinic carditis	Elevated ESR & CRP, fever	Turkey	14
3	7	Female	Clinic carditis	Elevated ESR & CRP, fever	Turkey	15
4	10	Male	Clinic carditis	Elevated CRP, fever	USA	16
5	12	Female	Clinic carditis, a antis	Elevated ESR, fever	Singapore	17

Patient Consent Form / Hasta Onam Formu

The parents’ of this patient consent was obtained for this study.

Conflict of Interest / Çıkar Çatışması

The authors declared no conflicts of interest with respect to authorship and/or publication of the article.

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