Case Report

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Post-Cardiac Injury Syndrome (Dressler Syndrome) Following Atrial Septal Defect (ASD) Repair: A Case Report

Ali Mirabi[1], Reihane Tabaraii[2], Morteza Sedaqat[3], Saba Gaeini[3], Maryam Masoumi[2]*

1. Resident of Cardiology, Clinical Research and Development center, Shahid Beheshti Hospital, Qom University of Medical Sciences, Qom, Iran.
2. Clinical Research and Development Center, Shahid Beheshti Hospital, Qom University of Medical Sciences, Qom, Iran.
3. Medical Student, Student Research Committee, Qom University of Medical Sciences, Qom, Iran.

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ABSTRACT

Dressler syndrome is a type of secondary pericarditis that can be accompanied by pleural effusion or pericardial effusion resulting from injury of the pericardium or heart tissue.

A 33-year-old male was admitted to the emergency department with pleuritic chest pain radiating to both shoulders and fever. Two months before the admission, the patient underwent traditional open-heart surgery with median sternotomy and pericardiectomy for atrial septal defect (ASD) closure. Blood tests showed elevated acute phase reactants (leukocytosis, high erythrocyte sedimentation rate, and C-reactive protein). Left-sided pleural effusion was revealed on chest CT scan. Diagnosis of Dressler syndrome was established. The patient's pleuritic chest pain and shoulder pain improved clinically with a short course of colchicine.

Although Dressler syndrome is rarely seen, it should be considered in the differential diagnosis of pleuritic chest pain. Special attention should be paid to Dressler's syndrome because pericarditis can develop following cardiac surgery.

Introduction

Post-cardiac injury syndrome also known as Dressler syndrome was first described by William Dressler in 1956 (1). Dressler syndrome is a type of secondary pericarditis that can be accompanied by pleural effusion or pericardial effusion resulting from injury of the pericardium or heart tissue. It is an immune-mediated disease that appeared to be initiated with damage to mesothelial pericardial cells which release cardiac antigens and stimulate immune response and inflammatory cascades (2).

*** Corresponding Author:**
Maryam Masoumi, MD.
Address: *Clinical Research and Development Center, Qom University of Medical Sciences, Qom, Iran.*
E-mail: *m.masoumiy@gmail.com*

Case Presentation

A 33-year-old male was admitted to the emergency department with pleuritic chest pain and fever for 5 days duration. Two months before admission, the patient underwent general anesthesia for median sternotomy and pericardiectomy for atrial septal defect (ASD) closure (with 4x4 centimeter ASD patch). He had a retrosternal left hemithorax and pleuritic chest pain radiating to both shoulders that worsened by deep breathing. He did not have cough, dyspnea, hemoptysis, pedal edema, palpitation, nausea or vomiting.

On admission, physical examination revealed a blood pressure of 110/65 mm Hg, pulse rate of 105 beats/min, respiratory rate of 19 breaths/min, temperature of 38.1°C and pulse oximetry measured 97% blood oxygen saturation in room air. No jugular venous distention was observed. A normal S1 and S2 sound and soft systolic murmur was audible in cardiac examination. There were diminished breath sounds and vocal resonance on the left lower thorax, dullness to percussion and decreased tactile fremitus.

The electrocardiogram (EKG) showed normal sinus rhythm, normal axis, right bundle branch block (RBBB), ST-segment depression in leads I, aVL, v1-v6 and T-inversion in leads II, III, aVF, v1-v6 with No ST-elevation. Chest X-ray showed mild left pleural effusion and chest spiral computerized tomography (CT) scan revealed small left sided pleural effusion. There was no sign of mediastinitis or osteomyelitis in chest CT scan (Figure 1). Ultrasound-guided thoracentesis was performed and only 5 cc of yellow fluid was aspirated which was not adequate for biochemistry analysis. Transthoracic echocardiogram

revealed normal LV size (LVED= 56mm, LVESD= 38mm, IVsd= 9mm, PWD= 8mm), no LVH, mildly reduced systolic function with LEVF=45-50%, normal LA dimension (LA size= 39mm), mild RA enlargement (RA area= 20cm2), mild RV enlargement, moderate RV dysfunction, mild doming of anterior mitral valve leaflet (AMVL), mild to moderate MR, trivial TR, PAP=43 mmHg, no residual ASD by CFD study and no pericardial effusion.

Laboratory tests revealed mild leukocytosis (WBC=14.400 /µL) and elevated acute phase reactants (CRP=55.4 mg/L and ESR=37 mm/hr). Other laboratory test results were as follows:

Hb=13.2 mg/dl, Platelet= 366.000, urea= 20 mg/dl, creatinine= 1.1 mg/dl, Na=141 mmol/L and K=4.2 mmol/L.

It should be noted that the patient had a recent hospitalization four weeks before this hospitalization due to fever, chills and sweating. He had no chest pain or dyspnea. His evaluations were described in Table 1. The patient was discharged with personal consent despite being advised to continue treatment in his previous hospitalization.

According to the patient's clinical features, laboratory tests and imaging, other clinical conditions such as pulmonary embolism, blunt chest trauma, pneumothorax and infectious or noninfectious causes such as pneumonia were unlikely to have occurred and pointed to post-cardiac injury syndrome. After diagnosis of Dressler's syndrome, the patient was treated with colchicine in this center and was discharged with a prescription of 0.5 mg of colchicine every 12 hours for 1 month. During hospitalization and after receiving colchicine, the patient's clinical symptoms improved; the patient's shoulder pain subsided, there was no fever, pleuretic chest pain diminished, and inflammatory factors such as ESR, CRP and WBC were reduced. After 3 months of follow-up, the patient became asymptomatic.

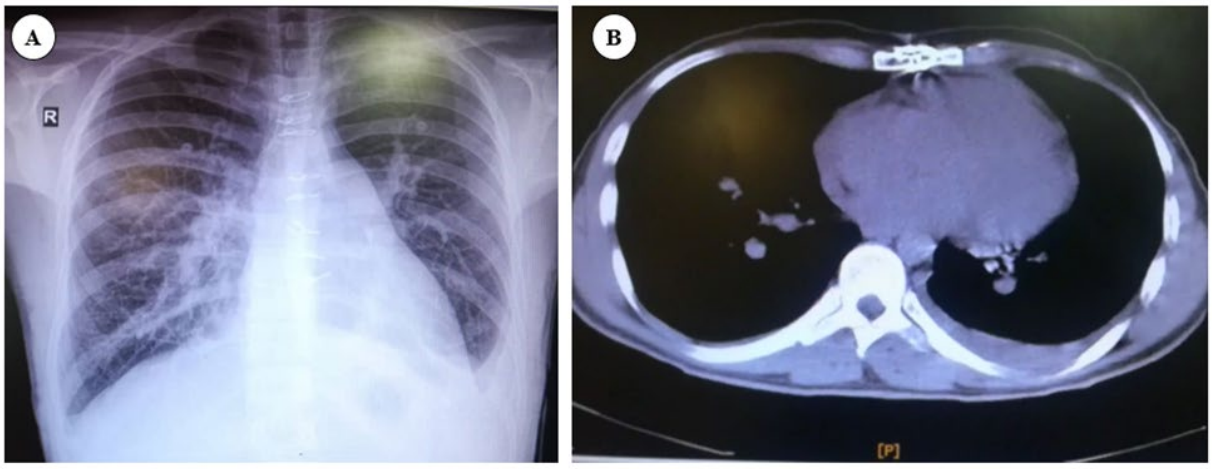

Figure 1. A (Chest radiography and B) Spiral chest CT scan revealing small left sided pleural effusion on the admission

Table 1. Patient's characteristics in this and previous hospitalization

Variables		Previous hospitalization	This hospitalization
Symptoms		Fever, chills, diaphoresis	Pleuritic chest pain, fever, cough, headache,
Vital signs	BP (mmHg)	115/70	110/65
	PR (beats/min)	102	105
	RR (breaths/min)	17	19
	T (°C)	37.8	38.1
	SPO2 (%)	98	97
Laboratory tests	WBC (/µL)	11.800	14.400
	ESR (mm/hr)	40	37
	CRP (mg/L)	56	55.4
Troponin (ng/ml)		Positive (15, 82)	Negative (<14)
Electrocardiogram (EKG)		RBBB, right axis deviation, No ST-T change	RBBB, T inversion, ST depression, No ST elevation
Echocardiography		LEVF= 50%, abnormal septal motion, moderate TR, no pericardial effusion	LEVF=45-50%, trivial TR, mild-moderate MR, no pericardial effusion
Chest CT scan		Mild bilateral pleural effusion (exudative with 70% PMN), alveolar opacification in left lower lobe and mild pericardial effusion	Left sided small pleural effusion (not adequate for analysis), No pericardial effusion
Blood culture		Negative	Negative
PCR-covid-19 test		Negative	Negative

Abbreviations: BP, blood pressure; PR, pulse rate, RR; respiratory rate; T, temperature; SPO2, pulse oximetry; WBC, white blood cell; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; LVEF, left ventricle ejection fraction

Discussion

Dressler's syndrome also known as post-cardiac injury syndrome was first described by William Dressler in 1956. Pericarditis with or without a pericardial effusion resulting from injury to the pericardium constitutes the post-cardiac injury syndrome (3). Patients with Dressler syndrome have common symptoms such as pleural chest pain (worse with breathing or lying down), dyspnea, weakness, arthralgia, heart palpitation, and weaknes. Friction rub can be heard in patients with Dressler syndrome and their pulse is mainly tachycardic (4). Our patient had a low-grade fever, radiating chest pain with deep inspiration and tachycardia. Incidence of Dressler syndrome is estimated at 3-4% in patient with myocardial infection. Due to th improvement in patient therapy management, prevalence of this disease is significantly reduced (4).

Differential diagnoses of Dressler syndrome include acute coronary syndrome, pulmonary embolism, pneumonia, heart failure, pleuritic, pneumothorax, and malignancy which depend on the patient's clinical symptoms. In patients with chest pain, especially pleural chest pain, special attention should be paid to Dressler's syndrome in which patients also developed pericarditis following cardiac surgery. Our case suggests post-cardiac injury syndrome that occurs following cardiac operation. Persons more at risk are those who have had a myocardial infection, heart surgery, or trauma to the chest following an accident or any other injury. For this reason, these people should be given special attention, especially if they have the clinical symptoms mentioned earlier.

The main pathogenic mechanisms of Dessler syndrome are diverse and include autoimmune reactions, viral infections and local inflammation. Etiology of this disease is still indeterminate, but in most studies, the emphasis is on reactions related to the immune system (5). It is thought that cardiac antigens are released following myocardial surgery, which results in production of anti-heart antibodies (6). Also, manipulation during pericardiectomy can play a role in development of post-cardiac injury syndrome.

Cardiac tamponade is the most important complication of Dressler syndrome but rare. If Dressler syndrome is diagnosed in time, cardiovascular collapse can be prevented.Other complications include pleural effusion and constrictive pericarditis (4, 7). A case was presented by Dylan et al. in 2006: he was 82 years old with history of hypertension, dyslipidemia, glaucoma, and cigarette smoking who complained of persistent pleural effusion at which time parapneumonic pleural effusion was diagnosed. He underwent therapeutic thoracocentesis and received antibiotics. At the next visit to the hospital, he had fever, weakness, pleuritic chest pain, an elevated ESR, pericardial effusion, and exudative pleural effusion and at this time, postcardiac injury syndrome was diagnosed (8). Our case also had pleural effusion in both hospitalizations. Therefore, pleural effusion is a very important and common finding in patients with Dressler syndrome deserving of special attention and patients should be evaluated in this regard.

In most of the various studies conducted on Dressler syndrome, it has been shown that ESR, CRP increased and also leukocytosis was documented as our case illustrated such findings. Also, in patients who participated in these studies, in their electrocardiograph, ST elevation was observed. In contrast, no ST elevation was seen on our patient's ECG and ST depression with T inversion was seen.

To date, no case has been reported with ASD in the diagnosis of Dressler's syndrome. Patients with ASD disease are usually asymptomatic in the first and second decades of life. In the third decade, a large number of patients develop shortness of breath during exercise, palpitation due to atrial arrhythmias, and enlarged heart on CXR. With these explanations, fever and pleuritic chest pain in Dressler syndrome do not correspond to the clinical manifestations of ASD. This is likely to occur following surgical manipulation of the infection that follows from stimulation of the immune system. Excessive increases in immune response can also trigger a reaction against heart antigens, resulting in production of antibodies.

In a study conducted by Nomoto et al. in treatment of Dressler syndrome, it was found that colchicine and acetaminophen together can be effective in remission and recurrence of Dressler syndrome. More precisely, colchicine, because of its role in inhibiting cytokines such as interleukin-1 beta and interleukin 18, can reduce inflammation such as pericarditis and even its recurrence (9). Glucocorticoids do not play a significant

role in prevention or treatment of Dressler's syndrome. In the clinical trial performed by Bunge JJ et al. on 822 patients who underwent valvular surgery (half of the patients were treated with dexamethasone and the other half with placebo), it was shown that this class of drugs cannot be used as prophylaxis or treatment of post-pericardiotomy (10).

NSAIDs can be used in Dressler's syndrome because they are anti-inflammatory, but they have more side effects than

colchicine. For this reason, if a drug from the NSAID family is used, it should have the least side effects. In various studies, it has been recommended to use NSAIDs such as ibuprofen, aspirin, and indomethacin in Dressler's syndrome, but because of the side effects ese, colchicine and

acetaminophen can be used as first line drugs. We only used colchicine without steroids in treatment of our patient, which resulted in relative complete recovery. Although Dressler syndrome is rarely seen, it should be considered in the differential diagnosis of pleuritic chest pain. Special attention should be paid to Dressler's syndrome patients who also develop pericarditis following cardiac surgery.

Ethical Considerations

Compliance with ethical guidelines

The ethics committee of Qom University of Medical Sciences approved this study with a pproval id IR.MUQ.REC.1402.051 and it fulfilled the guidelines of the World Medical Association Declaration of Helsinki, 2013 edition. The participant completed and signed a written informed consent form, and all personal information was kept private.

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Conflict of interest

The authors declared no conflict of interest.

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