Pericarditis as an initial symptom in Takayasu arteritis

Takayasu Arteriti’nde başlangıç semptomu olarak perikardit

Takayasu arteritis (TA) is a chronic inflammatory vasculitis with unknown etiology, affecting the large and medium sized arteries with a striking predilection for aorta and its major branches. Because of the non specific initial clinical presentation, the disease remains undiagnosed for a long period of time. Cardiac manifestations in TA are rarely reported in the literature. They determine the disease prognosis. Pericardial effusion was rarely reported in TA, and is exceptionally the first manifestation. We report a case, of TA presented initially with acute pericardial effusion.

A 18- year- old female was admitted to the hospital with chest pain and dyspnea. The 2-dimensional echocardiography showed moderate pericardial effusion measuring 1.1 cm anteriorly and 1.2 cm posteriorly. Laboratory finding showed elevated erythrocyte sedimentation rate (ESR; 120 mm/hr) and C-reactive protein (CRP; 6 mg/dl), hemoglobin 11.2 g/dl and platelet count 413.000/ml. She received non steroidal anti-inflammatory drugs (NSAID) for 2 months under the impression of acute viral pericarditis. Her symptoms did not improve and the echocardiography revealed the persistence of pericardial effusion.

The nature of pericardial fluid was an exudate. Protein counts were 5600 mg/dl. Lactic dehydrogenase was 538 UI/L and WBC counts was 204/μl, with 15% lymphocytes, 10% neutrophil and 75% others. Gram and AFB stains of pericardial fluid showed no organisms and the cytology was negative for malignancy. Pericardial biopsy revealed neither evidence of tuberculosis nor signs of malignancy. Viral and bacterial serology was negatives (B and C hepatitis, Epstein Barr Virus, Herpes Simplex Virus, tuberculosis).

On physical examination, there were pulse differences and carotid tenderness, with asymmetric blood pressure (110/60 mm Hg in the left arm and 135/80 mm Hg in the right arm) and bilateral systolic subclavian and carotid murmurs. The electrocardiogram showed low voltage in the precordial leads.

Table 1. Cases of pericardial effusion in Takayasu disease reported in the literature

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Sex/age, years</th>
<th>Clinical features</th>
<th>2-D echocardiography</th>
<th>MR imaging</th>
<th>Biological abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duclos (1991) (1)</td>
<td>F/21</td>
<td>Miscarriage 5th month, fever</td>
<td>Left ventricular dilatation and hypokinesia, lesions of the aortic and mitral valves</td>
<td>large coronary aneurysms</td>
<td>Elevated CRP and ESR</td>
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<tr>
<td>Lee (1998) (2)</td>
<td>F/ 25</td>
<td>Fever, chill, chest discomfort, dyspnea II, acute pericardial, no pulse difference, no bruit, no carotid tenderness</td>
<td>Posterior pericardial effusion; no intracardiac abnormalities</td>
<td>Segment of luminal irregularity and dilatation in descending thoracic and abdominal aorta</td>
<td>CRP: 5.7 mg/dl; ESR 144 mm/hr</td>
</tr>
<tr>
<td>Narita (1999) (3)</td>
<td>F/ 40</td>
<td>Fever, fatigue, malaise and severe chest pain.</td>
<td>Massive pericardial effusion and bilateral pleural effusion, cardiomegaly</td>
<td>-</td>
<td>CRP: 22 mg/dl, ESR 88 mm/hr</td>
</tr>
<tr>
<td>Moghadam (2009) (4)</td>
<td>F/ 32</td>
<td>Back pain, fatigue, malaise, fever and adynamia</td>
<td>Cardiomegaly, pericardial effusion, a mitral valve prolapsed with mild regurgitation and a supravalvular pulmonary artery stenosis</td>
<td>Concentric thickening of the common carotid arteries with discrete luminal narrowing of the right common carotid artery and thickening of the right subclavian artery, homogeneous concentric thickening of the main pulmonary artery and of the entire ascending and descending aortic wall</td>
<td>CRP: 14.2 mg/dl; ESR: 125 mm/hr</td>
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CRP - C-reactive protein, ESR - erythrocyte sedimentation rate, MR - magnetic resonance
Ultrasound Color Doppler of the extra cranial arteries showed an intimal thickening interesting essentially the right subclavian artery.

A computed tomography angiography showed a circumferential intimal thickening interesting the right, subclavian artery, vertebral artery and the primitive carotid artery reducing their diameter (Fig. 1).

The clinical manifestations of our patient including high ESR and the vascular lesions fulfilled the diagnostic criteria of TA. She received oral prednisolone 60 mg per day for 4 weeks. Clinical improvement was noted. Her ESR and CRP became rapidly normalized within 2 weeks following oral prednisolone therapy.

To the best of our knowledge only four cases of TA with pericardial effusion have been reported so far (1-5); they are summarized in the Table 1. This pericardial effusion was the first manifestation in only 2 cases, as reported in our case (2-4).

In our case, the acute pericardial effusion developed in the absence of other intracardiac abnormalities as an initial manifestation of TA. Other cardiac abnormalities were found in the others cases (1-3-4) including myocarditis, left ventricular dilation, aortic and mitral valve lesions and endocardial invagination.

TA should be considered as one of causes of acute febrile pericardial effusion in especially young women with enhanced CRP and elevated ESR.

Amira Hamzaoui, Randa Salem, Rim Klii, Olfa Harzallah, Olfa Berriche, Mondher Golli, Silvia Mahjoub
From Departments of Internal Medicine and Radiology, Fattouma Bourguiba Hospital, Monastir-Tunisia

References


Address for Correspondence/Yazışma Adresi: Dr. Amira Hamzaoui
Department of Internal Medicine, Fattouma Bourguiba Hospital, Monastir-Tunisia
Phone: +90 216 986 16 195 Fax: +90 216 734 60 678
E-mail: hamzaoui.amira@yahoo.fr

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