Giant cell arteritis from a cardiologist's perspective: You put out a fire, but it goes with a wind

Jelena Petrovic[1], Marija Radomirovic[1], Ivana Petrovic Djordjevic[1], Danijela Trifunovic[1, 2], Branislava Ivanovic[1, 2]

[1]Clinic for Cardiology, University Clinical Center of Serbia, Belgrade, Serbia
[2]University of Belgrade, Faculty of Medicine, Belgrade, Serbia

Correspondence to:
Jelena Petrovic, MD,
Clinic for Cardiology,
University Clinical Center
of Serbia,
Koste Todorovica 8,
11000 Belgrade, Serbia,
phone: +38 166 830 28 36,
e-mail:
jelenapetrovic2212@gmail.com

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A 69-year-old female presented with worsening dyspnea and fatigue. She had a history of giant cell arteritis (GCA) diagnosed and treated 16 years before. She also reported well-controlled hypertension lasting for two years prior to admission and no other diseases or cardiovascular risk factors. Physical examination showed neck vein distention, diminished heart sounds and blood pressure of 100/70 mm Hg. Except for sinus tachycardia, electrocardiogram was normal. Chest X-ray revealed cardiomegaly and mediastinal widening with tracheal deviation (Figure 1A). Two-dimensional transthoracic echocardiography (2D-TTE) showed significant pericardial effusion (PE) with early signs of cardiac tamponade together with an ascending aortic aneurysm (AA) and the finding suggestive of hypertrophic obstructive cardiomyopathy (HOCM) with increased pressure gradient at the left ventricular outflow tract (LVOT) (septum 1.7 cm, posterior wall 1.4 cm, PG LVOT 46 mm Hg) and systolic anterior motion of the mitral valve (Figure 1B1–B3). Contrast computed tomography confirmed large PE (23 mm) and discovered unruptured and non-dissecting thoracoabdominal AA (ascending part-61 mm, arch-60 mm, descending thoracic part-74 mm, suprarenal abdominal part-80 mm) with thrombosis (Figure 1C1–C2). Laboratory findings showed systemic inflammation without evidence of infection (C-reactive protein 262 mg/l, erythrocyte sedimentation rate 54 mm/hr, white blood cell count 13.4 × 10^9/l, procalcitonin <0.05 ng/ml, negative blood cultures, urine culture and extensive serologic tests for myopericarditis) or findings suggestive of myocardial necrosis. The Heart Team led by cardiovascular surgeons and interventional cardiologists assessed the patient. Since she was considered ineligible either for pericardiocenthesis or AA repair due to extremely high periprocedural risk, corticosteroid treatment was initiated. Gradually, it led to significant symptomatic improvement. Control 2D-TTE showed complete disappearance of PE and confirmed HOCM with preserved left ventricular systolic function and no segmental wall motion abnormalities. Due to these results and normal levels of myocardial necrosis markers, coronary angiography was not considered. Cardiac magnetic resonance imaging was planned for additional evaluation. Unfortunately, two weeks after the admission, the patient suddenly died. Autopsy was not performed.

GCA is the most common form of vasculitis in people older than 50 years, more frequently affecting women [1]. This type of large-vessel vasculitis increases the risk of death from cardiovascular diseases [1, 2]. AA is a well-known complication, but associated cardiac involvement is rare (<5%) [1, 2]. Pericarditis is atypical manifestation and hemodynamically significant PE is an extremely unusual finding, especially in the late course of the disease [1, 2]. Despite being even more rare in GCA patients, myocarditis should also be kept in mind [1]. Although there have been few reported cases of HOCM in patients with arteritis [3, 4], to the best of our knowledge, this is the first reported case of HOCM in a patient with GCA. Since significant PE may increase LVOT gradient and cause LVOT obstruction with systolic anterior motion [5], repeated echocardiography is necessary after the regression of PE to confirm HOCM in such a patient. Regardless of clinical improvement with corticosteroids in critical GCA patient with inflammatory PE, large AA with thrombosis poses inevitable risk for fatal outcome either due to embolic

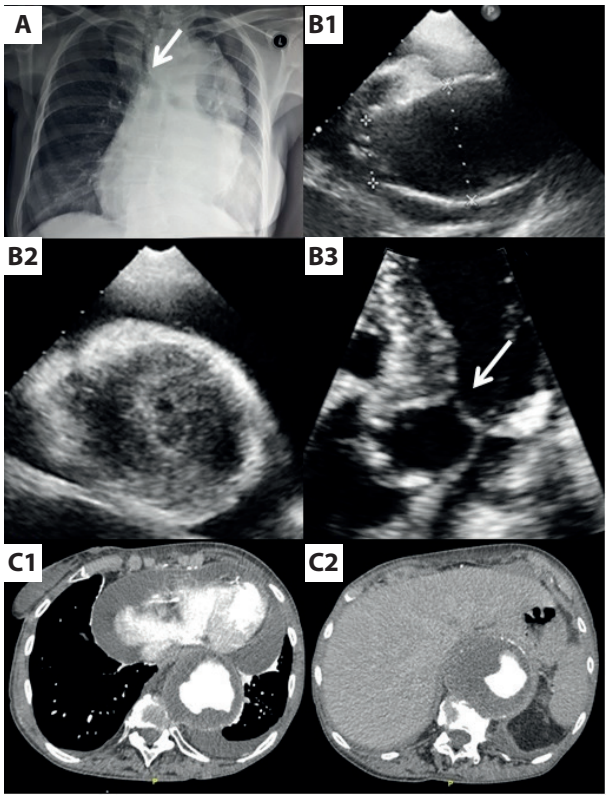

Figure 1. A. Chest X-ray showing cardiomegaly and mediastinal widening with tracheal deviation (arrow). **B1–B3.** 2D-TTE showing ascending AA, parasternal long axis view (**B1**) and HOCM: left ventricle during systole, parasternal short axis view (**B2**) and systolic anterior motion of the mitral valve (arrow), zoomed apical four-chamber view (**B3**). **C1–C2**. Contrast computed tomography scan showing thoracic AA with thrombosis and PE (**C1**) and abdominal AA with thrombosis (**C2**)

Abbreviations: 2D-TTE, two-dimensional transthoracic echocardiography; AA, aortic aneurysm; HOCM, hypertrophic obstructive cardiomyopathy; PE, pericardial effusion

complications or aortic rupture or dissection, with HOCM making the additional risk for sudden cardiac death.

Article information

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