

Giant Cell Arteritis as a Cause of Myocarditis and Atrial Fibrillation

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Giant cell arteritis (temporal arteritis; GCA) is a systemic vasculitis of medium and large arteries, affecting predominantly the aortic branches to the head and neck. Temporal arteritis was first described by Horton et al¹ in 1932 and classically presents with a combination of polymyalgia rheumatica, headache, and manifestations of systemic illness (fever, anemia, anorexia, malaise, and weight loss). GCA usually occurs in the elderly with a mean age at diagnosis of 72 years. Patients with GCA seem to be at increased risk for cardiovascular events, with heightened rate of acute myocardial infarction, cerebral vascular attack, and peripheral vascular disease. Myocarditis and myopericarditis are not commonly documented in patients with GCA.

In this report, we present a biopsy-proven GCA presenting with diplopia and new diagnosis of atrial fibrillation (AF) and myocarditis with left ventricular systolic dysfunction. Patient promptly responded to corticosteroid therapy further confirming the diagnosis of GCA.

Case Report

A 57-year-old man with no significant past medical history had a tick bite, and he was treated empirically with a 1-week course of doxycycline for nonspecific symptoms of dizziness and malaise. Three weeks afterward, he developed severe left temporal lancinating headaches refractory to aggressive migraine therapy that ultimately required hospitalization. Extensive workup including lumbar puncture, brain magnetic resonance imaging, and testing for babesia, lyme, and ehrlichia were negative. He was empirically treated with ceftriaxone and discharged home once the pain subsided. A few days later, he developed dyspnea and substernal chest pain. Emergency department evaluation revealed a weakly positive troponin I, and he was hospitalized for acute coronary syndrome. Exercise myocardial perfusion imaging demonstrated normal ejection fraction and a reversible inferior wall defect that was attributed to attenuation artifact. Follow-up coronary angiogram showed no evidence of obstructive coronary artery disease, and no aneurysms were detected. Computed tomographic angiogram of the chest was negative for pulmonary embolism but revealed mediastinal and hilar adenopathy and a 3 mm right-upper lobe nodule. Before discharge, he developed AF with a rapid ventricular rate and worsening chest pain and dyspnea. Transthoracic echo

revealed new reduced ejection fraction of 30% to 35%. He was transferred to our institution for further evaluation.

On admission, he was noted to be in paroxysmal AF with no evidence of overt volume overload. He was started on a low-dose basic heart failure regimen, which included metoprolol, lisinopril, furosemide, and spironolactone. A rhythm control strategy with amiodarone 400 mg PO daily was initiated after it was noted that his symptoms partially improved when he was in sinus rhythm. Beside from dyspnea and fatigue, he complained of vertical diplopia that was only present binocularly and when he was in AF. Laboratory testing was notable for an erythrocyte sedimentation rate of 90 mm/h and C-reactive protein 166 mg/L. Cardiac magnetic resonance imaging revealed an ejection fraction of 33% without late gadolinium enhancement to suggest myocarditis. Cardiac positron emission tomography after a 24-hour carbohydrate-free diet revealed normal resting [¹³N]-ammonia perfusion, diffuse left ventricular [¹⁸F]-fluorodeoxyglucose (FDG) uptake consistent with myocarditis, and right atrial FDG uptake consistent with right atrial inflammation (Figure 1). There was no significant FDG uptake in the aorta. Transbronchial biopsy of mediastinal lymph nodes revealed incidental benign bronchial cells and serum protein electrophoresis, antineutrophil cytoplasmic antibody, and antinuclear antibody testing were unremarkable. Extensive infectious workup was negative. Ophthalmologic examination revealed left optic nerve swelling/edema/pallor with an inferior visual field defect. Left temporal artery biopsy revealed a portion of artery with granulomatous inflammation of the medial layer and mild lymphocytic inflammation; consistent with giant cell/temporal arteritis (Figure 2). Before the final pathology report was available, the patient developed AF with a rapid ventricular rate and hypotension, and he was admitted to the cardiac intensive care unit. He received a bolus of amiodarone 150 mg IV and empirical solumedrol 500 mg IV. Within a few hours, his symptoms completely resolved. Amiodarone was discontinued, and he was discharged home symptom free 3 days later. C-reactive protein levels 1 day after starting solumedrol had dropped to 36.3 mg/L, and 2 weeks later were 0.4 mg/L.

Discussion

To date, there have been 6 reports of GCA causing myocarditis²⁻⁵ and only 1 which included cardiac positron emission

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tomography and magnetic resonance imaging data.⁴ However, that report did not include coronary angiography to rule out acute coronary syndrome. In our patient, acute coronary syndrome was ruled out with coronary angiography and the presence of normal ammonia perfusion on positron emission tomography imaging suggests that microvascular disease was not responsible for causing AF or reduced ejection fraction in our patient. The coincidence of paroxysmal AF and diplopia possibly suggests that flares of systemic inflammation precipitated both symptoms. This is supported by a report that FDG uptake is increased in the atria of patients with AF. Furthermore, the AF in our patient most likely responded to solumedrol and not to amiodarone because arrhythmia activity correlated with C-reactive protein levels. Many inflammatory conditions have been associated with myocarditis including necrotizing vasculitis, scleroderma, systemic lupus erythematosus, and rheumatoid arthritis. This report highlights the contribution of systemic inflammation to myocardial pump dysfunction and atrial arrhythmias and highlights the need for further investigation

for identifying patients with similar disease processes who may benefit from anti-inflammatory pharmacological intervention.

Disclosures

None.

References

- Horton BT, Magath TB, Brown GE. An undescribed form of arteritis of the temporal vessels. *May Clin Proc.* 1932;7:700–701.
- Pugnet G, Pathak A, Dumontel N, Sailler L. Giant cell arteritis as a cause of acute myocarditis in the elderly. *J Rheumatol.* 2011;38:2497. doi: 10.3899/jrheum.110609.
- Teixeira A, Capitaine E, Congy F, Herson S, Cherin P. Myopericarditis during Horton disease. *Rev Med Interne.* 2003;24:189–194.
- Kennedy LJ Jr, Mitchinson MJ. Giant cell arteritis with myositis and myocarditis. *Calif Med.* 1971;115:84–87.
- Daumas A, Rossi P, Jacquier A, Granel B. Myopericarditis revealing giant cell arteritis in the elderly. *J Rheumatol.* 2012;39:665–666. doi: 10.3899/jrheum.110934.

KEY WORDS: atrial fibrillation ■ giant cell arteritis ■ myocarditis

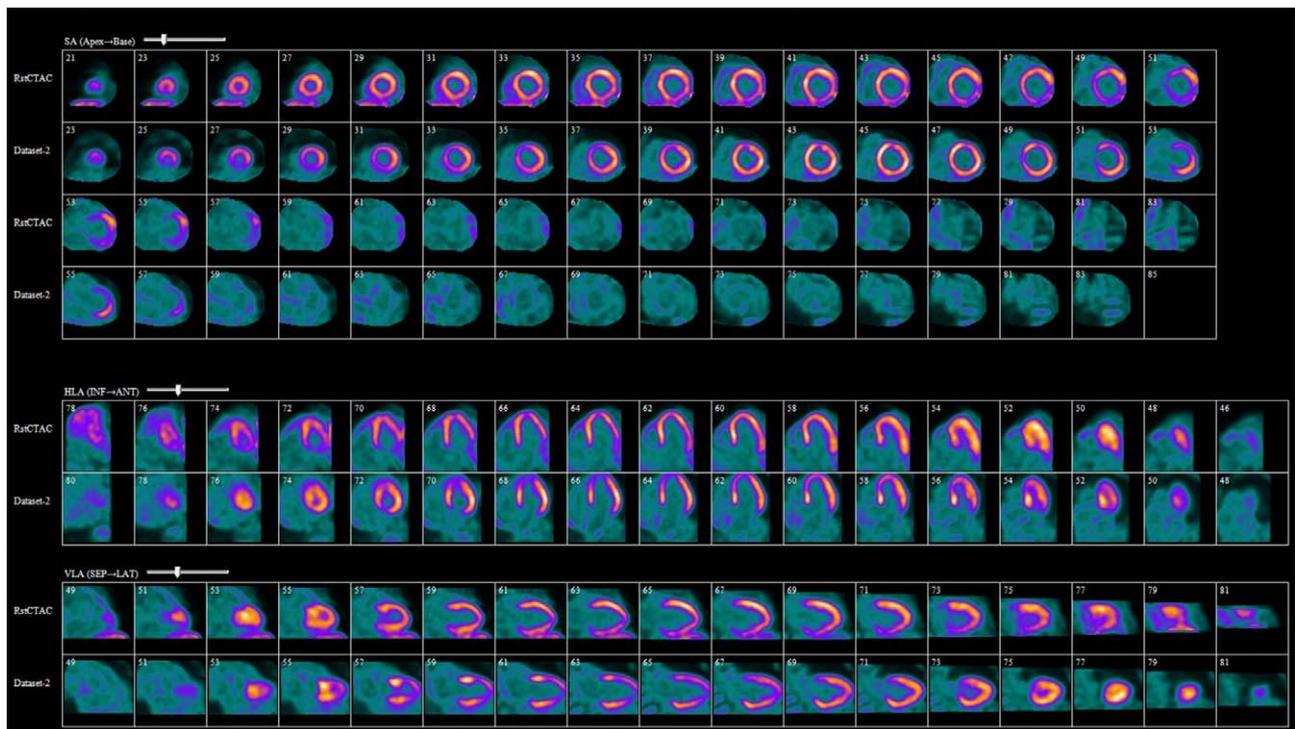


Figure 1. Positron emission tomography imaging with and without CT attenuation correction demonstrating increased left ventricular and right atrial uptake of [¹⁸F]-fluorodeoxyglucose.

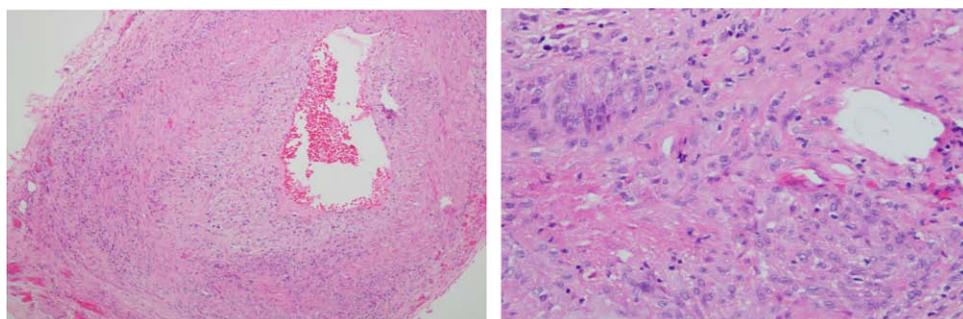


Figure 2. Temporal artery, left, biopsy, and H and E staining: portion of artery with granulomatous inflammation of medial layer and mild lymphocytic inflammation; consistent with giant cell/temporal arteritis. Image on right at ×400 magnification.

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