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Letter to the Editor: New Observation

Giant Cell Arteritis Presenting With Myocardial Infarction

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We describe an older woman with extensive cardiovascular history who developed chest pain and was diagnosed with new myocardial infarction (MI). During hospital admission, she lost vision in one eye and was found to have pallid optic disk edema with very elevated inflammatory markers. Temporal artery biopsy (TAB) confirmed the diagnosis of giant cell arteritis (GCA). While uncommon, GCA can cause vasculitis of coronary arteries; thus, all patients with recent diagnosis of GCA and new onset of chest pain should be screened for MI as the etiology of both can be vasculitis.

A 65-year-old woman with past medical history of diabetes mellitus type 2, hypertension, end-stage renal disease, pulmonary embolism, and peripheral arterial disease presented to emergency room with left-sided chest pain. Coronary angiography performed 4 months ago demonstrated nonobstructive coronary artery disease (CAD). She had no dyspnea, orthopnea, or diaphoresis. Her troponin was elevated (492ng/ml), but electrocardiogram showed only equivocal evidence of infarction. She was admitted for observation.

The next morning, she noticed sudden painless loss of vision in the left eye (LE). On bedside, examination vision was 20/20 in the right eye (RE) and counting fingers in the LE with left relative afferent pupillary defect. Anterior segment examination was unremarkable. Posterior segment examination of RE revealed severe nonproliferative diabetic retinopathy (NPDR). In the LE, the optic disk was pale and swollen with peripapillary cotton wool spots, and there was no evidence of inner retinal edema. There was moderate NPDR. On further questioning, she described symptoms consistent with jaw claudication for the past month but had no other symptoms of GCA.

Given her clinical findings, diagnosis of GCA was suspected. Erythrocyte sedimentation rate (ESR) was 116 mm/h, and C-reactive protein (CRP) was 95mg/L. Treatment with intravenous methylprednisolone 1gx3 days commenced followed by oral prednisone 60 mg daily. TAB demonstrated transmural inflammation with giant cells and elastic lamina disruption, confirming diagnosis of GCA. Coronary angiography demonstrated severe multivessel disease, with 70%–99% obstruction of all arteries, and coronary artery bypass grafting was planned. However, 1 week later the patient became hypotensive, developed lactic acidosis, and was transferred to the intensive care unit. Empiric broad-spectrum antibiotics were started. The next day, she developed melena and evidence of multi-organ failure. The patient declined further interventions and expired 2 days later.

While GCA is well known to be a systemic vasculitis predominantly involving large- to medium-sized cranial arteries such as the external carotid branches, central retinal artery, short posterior ciliary arteries, vertebral arteries as well as aorta, involvement of coronary arteries remain underrecognized even though it has been well established that GCA can affect coronary arteries.[1]

MI in GCA can occur through several mechanisms which make a diagnosis of vasculitis of the coronary arteries challenging. First, direct inflammation of the vessels producing vasculitis can occur. Second, accelerated formation of atherosclerotic plaque due to chronic vessel wall inflammation with resultant endothelial changes is an alternative possibility. Lastly, indirect adverse effects from the disease itself such as exposure to long-term treatment with glucocorticoids can negatively affect patient's cardiovascular condition and patients with GCA display cardiovascular risk factors more frequently than the age-matched general population.[2] Thus, when coronary angiography reveals occluded coronary arteries in patients with GCA, it could be the result of either ruptured atherosclerotic plaque or vasculitis, with the autopsy being the only way to definitely establish underlying pathophysiology.[1,2] While it is possible that in our patient occurrence of MI and GCA were unrelated, angiography performed only 4 months prior demonstrated nonobstructive disease with less than 50% stenosis in the worst occluded vessels; thus, we postulate that vasculitis caused by GCA has caused further decrease in the size of vascular lumen producing critical myocardial ischemia.

Overall, GCA-related vasculitis of the coronary artery is rare even among patients with presumed vasculitis-related MI. One retrospective study showed that only 6 out of 251 patients (2.4%) with biopsy-proven GCA had MI. Most of those were due to a mismatch of oxygen supply and myocardial oxygen demand (type 2 MI), and only one showed occlusion of coronary arteries (type 1 MI).[3] Notably, patients with GCA-unrelated MI who suffered from an MI years after their diagnosis of GCA had a higher cumulative dose of glucocorticoids and presented more frequently with type 1

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MI, supporting the fact that prolonged glucocorticoid treatment promotes development of atherosclerosis.

A case series of nine patients with GCA-related deaths reported two cases where coronary arteries were involved by vasculitis with both cases demonstrating a thrombosed coronary artery with ruptured cardiac wall, and in both cases the diagnosis and treatment were delayed.[1,4] There is also a case report describing a 51-year-old patient with initial diagnosis of dilated cardiopathy-opathy who eventually required a heart transplant in whom GCA was diagnosed on histological analysis of the native heart which demonstrated findings of granulomatous vasculitis with muti-nucleated giant cells in the septal branch of the coronary artery without evidence of significant atherosclerosis.[5]

Recent guidelines recommend general screening for aortic involvement in all patients diagnosed with GCA as it may affect long-term prognosis. Our case, however, emphasizes the importance of considering involvement of coronaries arteries as well as it can be a rare manifestation of GCA requiring aggressive immunosuppressive treatment with the potential of immediate lethal outcome. Considering GCA as the cause of adverse cardiac events in all patients with the recent diagnosis of GCA is pivotal and might be life-saving as the incidence of MI and cerebral ischemia in treated GCA patients is low, confirming the role of treatment in lowering heart- and brain-related ischemic events due to vasculitis.[4]

In summary, our case reminds neurologists that in rare cases GCA can be associated with MI. While there are multiple pathophysiological mechanisms that can cause GCA-related MI and disease can affect either multiple coronary arteries or a single branch, early diagnosis and aggressive immunosuppressive treatment is crucial to achieve best possible outcomes. It also emphasizes the importance judicial use of glucocorticoids in patients with GCA, and considering rapid steroid taper as prolonged use of steroids in patients with underlying coronary artery disease can contribute to unfavorable long-term cardiovascular outcomes.

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