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Femoral-popliteal bypass with femoral artery biopsy concluding giant cell arteritis

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Abstract
Giant cell arteritis is a systemic, inflammatory vasculitis that affects small to medium-sized arteries.[1] A 60-year-old Hispanic female presented to a vascular surgeon with a referral from her rheumatologist. The patient had a femoral biopsy from her recent femoral-popliteal bypass requested by her rheumatologist. The patient had constant claudication from her upper and lower extremities, which resulted from the femoral-popliteal bypass. This case demonstrates a nonclassical presentation of giant cell arteritis. The continuous claudications in both her upper and lower extremities presented in a clinical setting were related to peripheral artery disease that would have been secondary to smoking. If left undiagnosed and untreated, the patient could have ended eye blindness from compression to the temporal artery. Continuous follow-up care with her rheumatologist can lead to better long-term outcomes for her giant cell arteritis. This case study presents the pathogenesis, diagnosis, and treatment the patient received for her giant cell arteritis.

Keywords: Claudication, femoral artery, giant cell arteritis, bypasses

Introduction
A 60-year-old Hispanic female presented to a vascular surgeon with a referral from her rheumatologist for a femoral biopsy due to constant claudication from her upper and lower extremities. Her rheumatologist suggested the biopsy from her recent femoral-popliteal bypass. She had a bypass from continuous claudication in her lower extremities. The patient had continuous claudication in both the upper and lower extremities. The patient had ongoing conservative management but continued claudication, leading to several bypasses. The patient reported weakness in her lower extremities, some numbness, and tingling. The patient took aspirin, clopidogrel, and gabapentin without any symptomatic relief. The patient's past medical history included anxiety, arm and back pain, bilateral knee pain, claudication of the upper extremity, former smoker, GERD, insomnia, and subclavian arterial stenosis. The patient was a smoker for over 40 years. She quit smoking a year before her extensive bypass surgeries; she did not consume alcohol and denied any history of illicit drug use. The patient is divorced and unemployed, with two adult children. Her family was unable to obtain her during her consultation. Before her femoral biopsy, the patient had undergone several surgeries from left common carotid to brachial artery bypass, carotid, brachial bypass, and segmental excision of the right superficial femoral artery with an arterial repair. The patient denied fever, chills, cough, nausea, vomiting, diarrhea, abnormal or dark stool, decreased or painful urination, hematuria, unusual bleeding, or bruising.

Physical Exam
The patient's blood pressure in the outpatient office was unable to be obtained in both the right and left arms due to absent brachial pulses. Heart rate was also unable to be brought in the upper extremities due to absent radial pulses. Tympanic temperature was 97.7 degrees F. The patient was a well-developed female in no acute distress. Her cardiac rhythm and rate were regular, with normal S1 and S2. The bilateral lung fields were clear to auscultation. Her lower extremities were warm with excellent capillary refill and palpable 2+ dorsalis pedis and pedal pulses.

Diagnostic Testing
The patient underwent a series of two bilateral lower extremities ultrasound exams. After she consulted the vascular surgeon, she was referred to an outpatient imaging center. Bilateral ultrasound findings included concentric arterial wall thickening and noncompressible and

hypoechoic signs of vessel walls. Before the surgery, the patient had cardiac clearance and a complete blood count.

Management

After the procedural biopsy, the patient was sent home the same day. She was scheduled for an outpatient follow-up with the vascular surgeon. The patient was seen for a two-week follow-up at his outpatient clinic after her femoral biopsy to conclude the results with her. The biopsy results concluded with Giant Cell arteritis. The patient had several claudications in both the upper and lower extremities and finally understood the main reason. Before her follow-up appointment, the patient had seen her rheumatologist. Her rheumatologist had prescribed her glucocorticoids the following day after her biopsy. At the time of the encounter, the patient was two weeks into taking glucocorticoids. She started to feel better after starting glucocorticoids. She no longer complained of paraesthesias and coolness in her extremities.

Discussion

This case report's goal is not only to signify the risk related to giant cell arteritis but also to educate providers on the risk factors, clinical presentation, and disease screening techniques. Early intervention is essential in patients with giant cell arteritis due to the disease's dermtinatal effects on many organs. For instance, due to its immune-mediated inflammatory changes in the vessel wall, it could primarily lead to sudden vision loss. The immune pathology is still unclear, though; dendrocytes found within the adventitial layer of an artery activate T cells. Thus, causing an immune-mediated response producing Th1 to gamma interferon and Th17, activating Interleukin 17. Intresentling, with the first-course treatment of steroids, gamma interform persists where Th17 and IL17 decrease in the inflammatory response. The most common workup for GCA includes but is not limited to ESR and CRP lab results that are usually classified as elevated with ESR above 50. A typical gold standard for diagnosis is a temporal artery biopsy. However, it can be included in 30% of patients, such a biopsy must be done at least two weeks before steroid treatment. Pathological findings of GCA include a giant cell granuloma formation near a disrupted internal elastic lamina, though arteritis must involve the adventitia and media of the arterial wall. Imagining studies such as an MRI angiogram or CT scan have helped identify edema of the vessel wall with broad vascular areas, though many contraindications. Risk and assessment of GCA would be the main factor in treatment to prevent further damage. A simple tapered 40–60 mg methotrexate dose pack and evaluation. Others include TNF blockers, abatacept, and rituximab, though studies show significant improvement with methotrexate over others. If early prevention is not performed, further progression of occlusion can lead to significant damage. Patients in this situation must be cleared from cardiology before any surgical intervention.

Conclusion

This case demonstrates a nonclassical presentation of giant cell arthritis. After extensive claudications and bypasses, the patient got proper treatment and continued her everyday life with better reassurance. The patient was able to resume her daily activities without any complications. The patient stated she had felt more energized after starting glucocorticoids. The patient's continuous follow-up care with her rheumatologist can lead to better long-term outcomes for her.

Conflict of Interest

Not available

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