Giant Cell Arteritis as an Uncommon Cause of Chronic Cough: A Case Report

Moiz Salahuddin, Bruce F. Sabath

Patient: Female, 27-year-old
Final Diagnosis: Giant cell arteritis
Symptoms: Cough
Medication: —
Clinical Procedure: —
Specialty: Pulmonology

Objective: Unknown etiology
Background: Chronic cough is a common medical concern. Giant cell arteritis (GCA) is an uncommon cause of chronic cough and is not usually suspected since symptoms can be non-specific. We present a case of chronic cough due to GCA in which symptoms were subtle but imaging was remarkable and clearly disclosed the diagnosis.

Case Report: A 71-year-old woman presented to the pulmonary clinic with a concern of worsening cough for 4 months. She had been treated with proton pump inhibitor, intranasal steroids, and antibiotics, without improvement. Other symptoms were an occasional headache for the prior 5 months, but this had resolved. She had a history of early-stage breast and thyroid cancers, both of which were treated surgically several years earlier and were in remission. Results of a physical examination including flexible video laryngoscopy of the upper airway were completely normal. Laboratory investigations showed normal blood chemistries and blood cell counts. Her C-reactive protein level was 1 mg/L (upper limit of normal <10) but her erythrocyte sedimentation level was 121 mm/hr (upper limit of normal <30). A positron emission tomography (PET) scan was performed as surveillance for her prior cancers. This showed diffuse tracer uptake in the aorta as well as bilateral common carotid, subclavian, and common iliac arteries, revealing GCA as the underlying diagnosis.

Conclusions: Giant cell arteritis is a rare cause of chronic cough. Other symptoms can be subtle or non-specific as in our case, and a high index of suspicion is needed to obtain a temporal artery biopsy. In these cases, imaging adjuncts can provide a non-invasive diagnosis.

Keywords: Cough • Giant Cell Arteritis • Headache Disorders, Primary

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Background

Chronic cough is a common concern encountered in various disciplines. The differential diagnosis is broad and the process of uncovering the etiology can be challenging and prolonged. Giant cell arteritis is a rare cause of chronic cough and, as such, is not usually suspected unless relevant signs or symptoms of vasculitis are readily apparent [1]. In cases where giant cell arteritis is not suspected as the cause of chronic cough, imaging studies can nonetheless reveal the diagnosis. Here, we present a case of chronic cough due to giant cell arteritis in which symptoms were subtle and non-specific but imaging was remarkable and clearly disclosed the diagnosis.

Case Report

A 71-year-old woman presented to the pulmonary clinic with a chief concern of dry cough for the previous 4 months. The severity of the cough had been slowly progressive during that period of time. She had been treated with empiric proton pump inhibitor, intranasal steroids, and oral antibiotics, but did not have any significant improvement. She previously had an occasional frontal headache, but this had resolved; there were no other ongoing symptoms. She had a history of early-stage breast and thyroid cancers, both of which were treated surgically several years earlier, and she did not have any evidence of disease recurrence in follow-up. She was a never-smoker.

On examination, she was afebrile, with a heart rate of 80 beats/min, blood pressure of 130/70 mmHg, respiratory rate of 16 breaths/min, and oxygen saturation of 99% on room air. The physical examination showed clear bilateral air entry into the lungs, with no adventitious breath sounds or increased work of breathing. The oropharynx and nasal turbinates were normal, without evidence of inflammation. Flexible video laryngoscopy of the upper airway was normal. The remainder of the physical examination was normal.

Blood testing showed a white blood cell count 9.2 (73% neutrophils, 17% lymphocytes), mild anemia with hemoglobin 9.0 gm/dL, high platelet count (564×10^3/μL), and creatinine 0.7 mg/dL. C-reactive protein level was 1 mg/L (upper limit of normal <10) but the erythrocyte sedimentation rate (ESR) was elevated at 121 mm/hr (upper limit of normal <30). She had a computed tomography (CT) of the chest, which showed normal lung fields without any mediastinal or hilar adenopathy. Pulmonary function testing was normal with no evidence of obstruction, restriction, or diffusion defect. A nasal swab for SARS-CoV-2 was negative. A fluorodeoxyglucose (FDG) positron emission tomography-CT (PET-CT) was performed to evaluate for occult recurrence of prior malignancy. This showed diffuse increased radiotracer activity throughout the thoracic and abdominal aorta and their branches.

Figure 1. Positron emission tomography-computed tomography. (A) Subclavian (arrows), brachiocephalic (arrowhead), and left common carotid (asterisk) arteries with radiotracer uptake indicating diffuse vessel wall inflammation. (B) Aorta (asterisk) with radiotracer uptake indicating diffuse aortitis.
including bilateral common carotid, subclavian, and common iliac arteries (Figure 1). There was no evidence of cancer recurrence.

Based on these findings, a diagnosis of giant cell arteritis was made without a temporal artery biopsy. She was started on 60 mg of prednisone daily, with rapid improvement in her cough. At 2 weeks of follow-up, her cough had completely resolved. She was referred to Rheumatology, who started her on tocilizumab as a steroid-sparing agent given the suspected need for ongoing therapy for some time. Her symptoms remain well-controlled in follow-up.

**Discussion**

Giant cell arteritis (GCA) is the most common primary systemic vasculitis in the United States, with an incidence of 18 per 100,000 and a prevalence of 228,000 [2]. It is a vasculitis of the large vessels, usually involving the cranial branches that originate from the aortic arch. However, it can involve any large artery in the body, leading to multiple systemic manifestations. GCA is seen in patients above the age of 50 years, with typical symptoms being new headache, acute visual changes, and jaw claudication. In patients with fever of unknown origin and constitutional symptoms, vasculitis such as GCA should be considered in the differential diagnosis [3]. In patients with an ESR >100 mm/h with no acute infectious etiology, the differential should include vasculitides and other connective tissue diseases as well as malignancy. Our patient did not have any obvious systemic signs and symptoms suggestive of vasculitis, but the ESR >100 was a relevant clue.

GCA-related pulmonary involvement can include pulmonary embolism, pulmonary infarction, pulmonary nodules, and pleural effusion [4,5]. In one study, 13% of cases had cough related to GCA and a correlation was found between inflammatory biomarkers and presence of dry cough [1]. Often, patients will have cough related to their pulmonary parenchymal or pleural involvement. However, our case did not have any parenchymal or pleural involvement and presented with persistent coughing. In patients with GCA without parenchymal or pleural involvement, the pathophysiology of cough is likely related to vasculitis involving bronchial arteries that supply cough receptors in the lower airways, or the ascending pharyngeal artery, which supplies the pharynx [6].

GCA is typically diagnosed based on clinical features combined with histopathological evidence from a temporal artery biopsy [7,8]. However, patients with primarily large vessel GCA (e.g., aorta and central vessels) often lack classic symptoms such as headache and jaw pain because the vascular inflammation does not involve the smaller, peripheral vessels that supply these areas. As a result, such patients are more likely to have a negative temporal artery biopsy. In fact, in approximately one-third of patients, the temporal arteries are not involved; therefore, imaging of larger arteries can be helpful in diagnosis [7]. Appropriate imaging modalities include Doppler ultrasonography, CT with angiography (CTA), magnetic resonance imaging with angiography (MRA), and PET scan [7]. Doppler ultrasound of axillary and subclavian arteries typically shows circumferential vessel wall thickening or incompressibility of the arterial lumen [9]. CTA and MRA may demonstrate mural thickening or edema as well as other complications of vasculitis such as vascular stenoses or aneurysms. PET scan shows signs of vessel wall inflammation such as diffusely increased tracer uptake along vascular borders and can also be used in patients with a high pretest probability of GCA. Our case highlights GCA appearing as smooth, linear and contiguous FDG avidity in the thoracic aorta and its main branches.

**Conclusions**

Giant cell arteritis is a rare cause of the common concern of chronic cough. Other symptoms can be subtle or non-specific, as in our case, and a high index of suspicion is needed to obtain a temporal artery biopsy. However, a substantial proportion of cases lack clear signs or symptoms that the temporal artery is involved. In such presentations, imaging adjuncts can provide a diagnosis.

**Declaration of Figures Authenticity**

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