

Cricopharyngeal Muscle Weakness Secondary to Giant Cell Arteritis: A Rare GCA Mimic Presentation Not to Be Missed

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Abstract

INTRODUCTION: Giant Cell Arteritis (GCA) is a systemic granulomatous vasculitis involving medium and large vessels. It has a predilection for older adults aged ≥ 50 years, who usually present with temporal headache, scalp tenderness, jaw claudication, and constitutional symptoms. However, the presented case of GCA had symptoms of dysphagia and dysphonia initially diagnosed as atypical myositis. **PRESENTATION of CASE:** A 77-year-old man who initially presented with dysphagia and dysphonia. Videofluoroscopy showed cricopharyngeal muscle weakness. A right thigh muscle biopsy showed focal endomysial lymphocytic infiltration with myoinvasion reflective of myositis. As a result, he was treated for atypical myositis. He later presented after two years with dysarthria and dysphagia. The initial clinical suspicion was possible cerebral ischemia. MRI/MRA of the brain: suspected cerebral vasculitis. Eventually, a temporal artery biopsy confirmed findings consistent with GCA. **DISCUSSION:** Our patient has presented with uncommon GCA signs of dysphagia and dysphonia, leading to the initial diagnosis of atypical myositis. This indicates the disease's heterogeneous nature and broad clinical spectrum. **CONCLUSION:** GCA presentation is protean, and clinicians must be aware of atypical presentations to ensure timely diagnosis and treatment, preventing severe complications that can lead to irreversible outcomes.

Keywords

Giant Cell Arteritis, Dysphagia, Dysphonia, Dysarthria

1. Introduction

Giant Cell Arteritis (GCA) is a vasculitis disease characterized by granulomatous inflammation affecting large- and medium-sized arteries [1]. GCA leads to vascu-

lar endothelial injury, lymphocyte proliferation, and giant cell formation, resulting in acute and chronic damage. It affects individuals aged ≥ 50 years. Suspected GCA patients usually present with a new onset of temporal headache, scalp tenderness, jaw claudication, and constitutional symptoms such as fever, weight loss, night sweats, polymyalgia symptoms, and/or visual loss [2]. However, vasculitis has been shown to lead to a variety of clinical presentations, such as secondary myopathies [3] [4].

The introduced case is a GCA patient who presented with symptoms of dysphagia and dysphonia, initially diagnosed as atypical myositis.

2. Case Report

A 77-year-old man with a background medical history of Type 2 Diabetes Mellitus, hypertension, and hyperlipidemia who initially presented with dysphagia and dysphonia. Videofluoroscopy at that time revealed weakness of the cricopharyngeal muscle. A right thigh muscle biopsy showed focal endomysial lymphocytic infiltration with myoinvasion, and sparse perivascular lymphocytic inflammation around capillaries/small arteries in the epimysium. Antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, C-reactive protein, complements, immunoglobulins, creatine kinase level, and myositis panel were all normal. The malignancy screen with computed tomography (CT) of the thorax, abdomen, and pelvis, gastroscopy, and tumor markers was normal. Despite that the findings were matching possible myositis, all the serology workup was negative. Thus, the patient was diagnosed with atypical myositis. The patient was pulsed with IV methylprednisolone 500 mg daily for 3 days, IV Immunoglobulins 0.4 g/Kg per day for 5 days, methotrexate 10 mg weekly for 4 weeks, and then 15 mg weekly, and folic acid 5 mg daily. Later, he was started on Rituximab infusion every 6 months. His clinical symptoms improved with this treatment regimen. However, in his latest attendance, he presented as a FAST positive call with general weakness and slurred speech associated with increased episodes of dysarthria and dysphagia. On admission, he had blood tests which showed mildly raised CRP 11 mg/L. He also underwent CT and Magnetic Resonance Imaging/Magnetic Resonance Angiography of the brain, which revealed multifocal supratentorial and infratentorial lesions, predominantly seen at subcortical white matter with areas of juxtacortical involvement at the right temporal lobe and left superior frontal lobe middle gyrus (**Figure 1**). A multidisciplinary team, including neurology and radiology, suspected the images looked more like cerebral vasculitis than stroke. He was pulsed with IV Methylprednisolone followed by oral Prednisolone 60 mg daily. Due to the unusual presentation, the patient underwent a temporal artery biopsy (TAB), which demonstrated focal intimal thickening with mononuclear cell infiltration extending transmurally which raises the possibility of GCA. He was diagnosed with GCA, and his Rituximab treatment was switched to tocilizumab (IL-6 inhibitor) subcut weekly. Dramatic clinical improvement was observed.

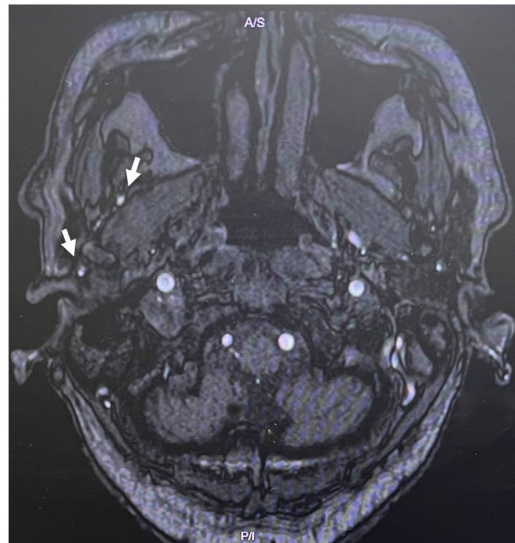


Figure 1. MRI scan showing (arrows) multifocal supratentorial and infratentorial lesions.

3. Discussion

Our patient has been diagnosed with GCA based on the gold standard method of TAB. His temporal artery ultrasound was deferred as he was unwell and COVID-positive in the ward, which prevented him from transferring for the scan. The initial diagnosis of atypical myositis based on a muscle biopsy revealed early stages of vasculitis of the capillaries/small arteries in the epimysium. Anatomically, the blood supply of the cricopharyngeal muscle is through the inferior thyroid artery arising from the subclavian artery, which is most commonly secondarily affected by the cranial GCA [2]. Atypical GCA presentations, as in our case, involving vasculitis of the aortic arch branches supplying the neck and brain have been reported in Australia and Portugal. This case finding correlates with a case report from the Canberra Hospital of a 73-year-old man with odynophagia and a non-healing ulcer over the tip and lateral border of his tongue, reported to be affected by GCA [5]. Another case from the Local Health Unit of São João presented with holocranial headaches and tongue pain with partially detachable whitish plaques, reported to be affected by GCA [6].

4. Conclusion

Clinicians need to reconsider the clinical spectrum of large-vessel vasculitis and the atypical symptoms of GCA to ensure a thorough diagnosis and prompt initiation of treatment. Our patient did not show any “classic” features of GCA such as headache, jaw claudication, or scalp tenderness. The key learning point from introducing this case is the need to have a high clinical suspicion of GCA in the presence of atypical manifestations such as dysphagia and dysphonia.

Informed Consent

The patient has provided informed consent.

Author Contributions

Writing-original draft preparation, Nada Elsheikh; writing-review and editing, Alwin Sebastian. Both authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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