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Giant cell arteritis manifesting as acute sinusitis- A case report

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Abstract
Giant cell arteritis is a common form of vasculitis in the elderly. This disease is characterized by granulomatous inflammation of the arteries, which can lead to serious and fatal conditions, including aortic aneurysm, rupture and dissection, and blindness. Because GCA can be treated with immunosuppressive therapy such as corticosteroids, early diagnosis and prompt treatment can reduce the risk of disability and prevent serious complications. Although tissue biopsy is considered the gold standard for the diagnosis of GCA, it is still risky and unreliable due to the uncertainty of the accuracy of the biopsied tissue sample. Imaging studies, such as computed tomography, which are non-invasive, have been shown to detect vasculitis, including GCA. We present the case of an 85 year old female who had an unusual presentation of giant cell arteritis.

Keywords: Acute sinusitis, temporal headaches, vasculitis, giant cell arteritis

Introduction
Giant cell arteritis (GCA/ Horton disease/ cranial arteritis/ temporal arteritis) is a vasculitis involving the large- and medium-sized vessels. It is considered the most prevalent idiopathic systemic vasculitis in patients older than 50 years. The clinical scenario range is broad, and the signs and symptoms are secondary to ischemia in the territory of the superficial temporal artery (STA) or any of its branches.[1] In the described report, authors present a case of GCA with an unusual clinical manifestation.

Case report
An 85 year-old female presented to an outpatient clinic during winter with a chief complaint of new onset headaches for a week. She described the headaches as frontal and bitemporal in location, but worse on the right side. The pain was described as sharp in character and intermittent in nature. There were no reported aggravating or relieving factors. Headaches were associated with neck stiffness, rhinorrhea and jaw tightness. There was no associated nausea, vomiting, vision changes, fever, chills, cough, arthralgias, myalgias, photophobia or aural symptoms. She denied any recent sick contacts and had no significant travel history. She had a past medical history of hyperlipidemia and was on pravastatin 40mg for the same, hypothyroidism for which was on levothyroxine 75mg, osteoporosis, colonic polyp and CKD Stage 3B. Her vitals on presentation were temperature of 98.1 F, blood pressure of 118/78mmHg, pulse of 81 bpm, SpO2 of 98% on RA and respiratory rate of 12/min. Her physical examination was unremarkable. A COVID test, rapid influenza A & B test were performed and were all negative. She was initially treated with cefdinir 300mg daily for 7 days for a suspected sinus infection. At her follow-up visit in a week, her symptoms had still not improved. Patient reported no change in the severity or pattern of her headaches.Over the counter pain medications did not provide any relief to her. She denied having any other symptoms and had noted an increase in the frequency of her headaches. She was sent for a CT of the sinus without contrast which was normal. Her CBC was unremarkable except for a mildly elevated WBC count of 11,300/µL. Her CMP was indicative of decreased eGFR and elevated Cr consistent with her diagnosis of CKD. Her ESR was remarkably elevated at 92 mm/hr and CRP level at 7.44 mg/dl. Patient denied having any visual changes at this time. She was started on oral prednisone 30mg BID for a suspected diagnosis of giant cell arteritis. Urgent general surgery referral was made for a temporal artery biopsy. Pathology report from her right temporal artery biopsy showed a segment of artery with mild to moderate lymphocytic inflammation involving the intima, media and adventitia with rare possible giant cells consistent with temporal arteritis. Focal atherosclerotic medial calcification was also noted.
Patient was referred to a rheumatologist for further management. She was then started on prednisone 60mg daily and she reported resolution of her symptoms with the medication. Her dose of steroids was gradually tapered down and she was also started on Actemra (Tocilizumab) infusion once every 4 weeks.

Discussion
Giant cell arteritis (GCA) is a granulomatous vasculitis of the aorta and its main branches with tropism for the extracranial tributaries of the carotid arteries. It is the most common primary form of vasculitis in adults. Aging is the commonest risk factor associated with GCA, with the disease almost never occuring before the age of 50. In addition to age, ethnicity has been found to be an important risk factor for GCA with the highest incidence in Scandinavian countries and among Americans of Scandinavian descent. Females are affected more frequently than males. [2]
Clinical features include constitutional symptoms like fever, fatigue, and weight loss. [4] Headache is a prominent feature of GCA. Typically, headaches due to GCA are located over the temples, but they can also be frontal, occipital, unilateral, or generalized. They can progressively worsen, wax and wane, or sometimes even abate before treatment is started. Enlargement, nodular swelling, tenderness, and loss of pulse of the temporal artery, either unilateral or bilateral, are classically attributed to patients with underlying cranial

GCA. About one-half of patients experience jaw claudication, a symptom that involves mandibular pain or fatigue brought on by mastication and relieved by stopping. About 20% to 30% of the patients experience visual disturbances. GCA-associated visual loss could be transient or permanent. Transient visual changes usually manifest as an abrupt partial field defect or as if a curtain covers the field of vision of an eye. Permanent vision loss, commonly resulting from anterior ischemic optic neuropathy, is painless and sudden. It can be unilateral, bilateral, partial, or total. [3] Current studies estimate that the incidence of permanent vision loss is 8.2% in patients with GCA. Vascular abnormalities may manifest as limb claudication, asymmetric blood pressures, abnormal radial pulses, vascular bruits, and temporal artery abnormalities such as tenderness to palpation, decreased pulse amplitude, and the occurrence of nodules. [4]

Polymyalgia rheumatica (PMR) is characterized by aching and morning stiffness around the shoulders and hip girdles, in the neck, and in the torso. PMR is closely linked to GCA, occurring in approximately 40 to 50 percent of patients with GCA. Conversely, GCA is found in about 10 percent of patients with PMR.

Diagnosis of GCA is done with the help of laboratory and imaging modalities. Initial laboratory testing includes complete blood count, ESR, CRP levels, complete metabolic panel (to include serum creatinine, liver function tests, glucose),urine dipstick analysis, serum protein electrophoresis and a bone profile panel (to include calcium, phosphorous, albumin, total protein, alkaline phosphatase, 25-hydroxyvitamin D).

Gold standard for diagnosing GCA is a temporal artery biopsy. The classic histologic picture of GCA is a transmural inflammatory infiltrate consisting of lymphocytes, macrophages, and, in approximately 75 percent of cases, giant cells. Ideally, the biopsy should be performed prior to starting glucocorticoid therapy. However, given the threat of permanent vision loss, clinicians should begin glucocorticoid therapy as soon as the diagnosis of GCA is suspected. Temporal artery biopsy should then be performed within 2 weeks of starting glucocorticoid therapy.

Colored doppler US of the head, neck, and upper extremities can serve as a diagnostic surrogate for temporal artery biopsy. In GCA, the temporal arteries show a circumferential dark area in the vascular lumen, thought to represent mural edema, dubbed as the "halo sign". The presence of bilateral halo signs of the temporal arteries is specific for GCA. [5]

Initial management for patients without any signs of ischemic organ damage (eg, visual loss) is to initiate glucocorticoid therapy with prednisone 40 to 60 mg/day (or equivalent) with the goal of relieving symptoms and preventing visual loss.

For patients with newly diagnosed GCA who have an increased risk of glucocorticoid-related adverse effects, the addition of tocilizumab to glucocorticoids is suggested. Patients who are at an increased risk of developing glucocorticoid-related side effects or complications include those with osteoporosis, diabetes, hypertension, or glaucoma.

Tocilizumab is not initiated if ANC is <2,000/mm^3, platelets are <100,000/mm^3, or if ALT or AST are >1.5 times ULN. If a patient develops a serious infection, therapy should be interrupted until the infection is controlled. Methotrexate is an alternative for patients who are unable to receive tocilizumab therapy due to the risk of infections, history of gastrointestinal perforations, or cost.

Patients who present initially with visual manifestations (amaurosis fugax or unilateral visual loss) or with cerebrovascular events (eg, stroke or transient ischemic attack) potentially attributed to GCA, require a higher initial dose of glucocorticoids, which should be administered promptly. 500-1000 mg/day of intravenous methylprednisolone daily for three days (followed by 40 to 60 mg/day of oral prednisone) or 1 mg/kg/day of oral prednisone (if intravenous pulse cannot be rapidly initiated). Subsequent management involves a gradual taper of the glucocorticosteroids. The goal is to taper glucocorticoid therapy to zero over a period of 12 to 18 months while maintaining clinical remission and normal inflammatory markers.

Conclusion

Clinicians should look out for GCA in any patient older than 50 who complaints of a new onset headache or change in the features of their preexisting headache. [1] Other symptoms that warrant suspicion in such patients are jaw claudication, unexplained fever, abrupt visual disturbance or loss, and signs of vascular abnormalities.

Any patient with a pre existing diagnosis of polymyalgia rheumatica should undergo evaluation for GCA. At a minimum, they should undergo carotid and subclavian artery auscultation for bruits and bilateral blood pressure measurements.

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