Rare Ischemic Complications of Giant Cell Arteritis: Case Series and Literature Review

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Financial support: None declared

Conflict of interest: None declared

Case series
Patients: Female, 75-year-old • Female, 76-year-old • Female, 72-year-old
Final Diagnosis: Giant cell arteritis
Symptoms: Ischemic complications of giant cell arteritis
Medication: —
Clinical Procedure: —
Specialty: General and Internal Medicine • Rheumatology

Objective: Challenging differential diagnosis
Background: Some ischemic complications due to giant cell arteritis (GCA) are rare and underdiagnosed. We describe the clinical features and outcomes of patients with GCA presenting with rare ischemic complications.

Case Reports: Our single-center retrospective database of patients with GCA was reviewed from 1994 to 2020. We describe 3 cases of rare ischemic complications secondary to GCA. We review the literature regarding ischemic complications due to GCA and their outcomes.

All 3 cases met the American College of Rheumatology criteria for GCA. All patients experienced rare ischemic complications due to GCA. In case 1, the patient presented with a sixth cranial nerve palsy. In case 2, the patient presented with tongue and scalp necrosis, and with permanent visual loss due to anterior ischemic optic neuropathy. In case 3, the patient presented with scalp necrosis. In all 3 cases, the patients received glucocorticoids either intravenously and/or orally, which led to improvement. They all improved within the course of their followup visits. A literature review was performed to identify similar cases and outcomes.

Conclusions: Ischemic complications due to GCA can be part of the initial presentation of the vasculitis, making confirmation of the diagnosis more difficult. Physicians should be aware of these rare complications since rapid diagnosis and initiation of glucocorticoids may alter the course of the disease.

Keywords: Giant Cell Arteritis • Ischemic Stroke • Optic Neuropathy, Ischemic • Skin Diseases, Vascular

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/937565

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Background

Giant cell arteritis (GCA) is a systemic vasculitis affecting mostly patients older than 50 years of age. It involves large and medium-sized vessels, predominantly cranial branches of the carotid arteries [1]. Common manifestations include headaches (65-85%), jaw claudication (11-40%), polymyalgia rheumatica (48-53%), and constitutional symptoms (14%) [2]. Inflammation of the affected vessel walls may lead to stenosis, potentially causing ischemic complications such as ophthalmic manifestations (amaurosis fugax, diplopia, blurry vision, permanent visual loss), ischemic stroke, lingual and scalp necrosis, and limb claudication. Rapid diagnosis and treatment with glucocorticoids are important to avoid irreversible ischemic damage. Some of the ischemic features of GCA are less frequently seen; therefore, late recognition of the disease and delay in therapy may occur [2,3].

We describe 3 cases of rare ischemic complications due to GCA and review the literature to further understand the presentation, diagnosis, and outcomes of such complications. Our objective is to raise clinician awareness of these infrequent GCA manifestations to prevent delays in disease recognition and treatment.

Case Reports

Out of the 100 patients with GCA in our retrospective database, we identified 3 patients with rare ischemic complications. Our database of patients with GCA included patients from January 1994 to December 2020. We performed a paper and electronic search and identified patients with the following inclusion criteria: all cases met the 1990 American College of Rheumatology classification criteria for GCA and presented with rare ischemic complications attributed to GCA as their initial presentation. These manifestations included cranial nerve palsy, scalp or tongue necrosis, ischemic strokes, and Charles Bonnet syndrome. Cases with other more commonly described ischemic complications were excluded, such as jaw claudication, amaurosis fugax, vision loss, and transient diplopia with normal neuroophthalmological examination. Data regarding age, sex, medical history, GCA signs and symptoms, timing of ischemic complications, laboratory tests, imaging studies, treatments, and outcomes were collected from medical records. Research ethics board approval was obtained (REB 2019-1754) for this retrospective project.

Case 1. GCA with Sixth Cranial Nerve Palsy

A 75-year-old woman with known hypothyroidism, osteoporosis, and non-metastatic melanoma presented to the emergency department for new-onset bitemporal headache with horizontal diplopia which started 3 weeks prior to her visit. Her headache was bilateral (both temples), occurred mostly at night with a duration of 3 minutes. Her headache was accompanied by photophobia and phonophobia. There were no other neurologic deficits or systemic symptoms. The headache improved with acetaminophen but was recurrent at night. There was no history of trauma or newly initiated medication. Physical exam showed a deficit of the lateral movement (abduction) of the right eye. The patient also reported mild worsening of chronic shoulder pain. A head computed tomography (CT) scan showed a small left temporal hypodensity. However, a magnetic resonance imaging (MRI) of the brain and a cerebral CT angiography were unremarkable. A right ischemic sixth cranial nerve palsy was confirmed by a neuro-ophthalmologist. The patient did not have jaw claudication, scalp tenderness, or visual symptoms, and both temporal arteries were pulsatile and painless. She did not present any constitutional symptoms (fever or weight loss). Laboratory findings showed a mild chronic anemia (113 g/L, normal: 120-160 g/L), thrombocytosis (633×10^9/L, normal: 150-400×10^9/L), and a C-reactive protein (CRP) level of 5.3 mg/L (normal: <10 mg/L). Prednisone was not started initially as clinical suspicion of GCA was low. The initial diagnosis was idiopathic paresis of the sixth cranial nerve and headache secondary to cervicalgia.

In the following 2 months, she developed jaw and tongue claudication, fatigue, and diaphoresis. Her complete blood count (CBC) was unchanged, but her CRP was slightly increased to 12 mg/L and erythrocyte sedimentation rate (ESR) was at 47 mm/h (normal: <20 mm/h). Daily oral prednisone was started at 50 mg with daily oral aspirin 80 mg. A temporal artery biopsy was performed within 2 weeks of initiation of glucocorticoids, and this biopsy confirmed the diagnosis of GCA.

After 3 months of therapy, the patient showed complete recovery of her sixth cranial nerve palsy. Her CBC and inflammatory markers normalized. Over the next 2 years, the patient was asymptomatic, and no disease flare occurred upon prednisone taper.

Case 2. GCA with Tongue and Scalp Necrosis, and Permanent Visual Loss Due to Anterior Ischemic Optic Neuropathy

A 76-year-old woman with a previous medical history of dyslipidemia, hypertension, chronic obstructive pulmonary disease, osteoporosis, and active smoking consulted an ear, nose, and throat specialist for a painful tongue ulcer measuring 2 cm (Figure 1), which was suspicious for malignancy. She concurrently suffered from new-onset bilateral headache and constitutional symptoms (fatigue and a 10-pound weight loss over the last month). Two weeks later, she presented in the emergency department after developing scalp tenderness, jaw claudication, and a sudden loss of vision in the right eye. On examination, temporal artery pulses were absent, and a diffuse abdominal aorta bruit was present. Small necrotic lesions

![Figure 1](image-url)
on her scalp were found and were painful to touch (Figure 2). Routine blood work revealed inflammatory anemia (112 g/L), thrombocytosis (523×10^9/L), leukocytosis (16.3×10^9/L, normal: 4.5-11×10^9/L), and a CRP of 159 mg/L. The cerebral CT scan was unremarkable. CT angiography of large vessels (from the carotids to iliac arteries) did not show any signs of large or medium-sized vessel vasculitis but showed significant atherosclerosis with stenosis of multiple arteries (renal artery, origin of the celiac trunk, origin of the superior mesenteric artery) and chronic bilateral iliac artery thrombosis. An ophthalmologic exam confirmed the presence of anterior ischemic optic neuropathy (AION) of the right eye. Diagnosis of GCA was confirmed with a positive temporal artery duplex ultrasonography. A positive halo and compression sign was seen on the right superficial (common) and parietal temporal artery. Facial arteries were also affected bilaterally. No temporal artery biopsy was performed. A tongue biopsy was performed and showed ulceration with granulation tissue. There were no signs of malignancy and culture was negative for mycosis and herpes.

Intravenous methylprednisolone (1000 mg daily for 3 days) was administered, followed by oral prednisone with a standard tapering schedule.

At 1-month followup, her headache, scalp tenderness and necrosis, jaw claudication, and tongue ulcer completely resolved. Her CBC and CRP were also normal. Unfortunately, her right eye vision loss did not recuperate over the next 2 years of followup.

**Case 3. GCA with Scalp Necrosis**

A 72-year-old woman who was an active smoker and was known for diverticulosis, thyroid cyst, and a resected basal cell carcinoma, initially consulted a dermatologist for left and right frontal painful cutaneous lesions. She also complained of non-pulsatile occipital and temporal headache which were more severe on her left side. She received a diagnosis of necrobiosis associated with a *Staphylococcus epidermidis* infection and was treated with cephalaxin, which did not improve her lesions. After a month, her headache worsened, and she developed jaw claudication and tongue paresthesia. The patient did not have visual symptoms, but noted a 12-pound weight loss, nocturnal diaphoresis, and major fatigue. Physical examination showed erythematous violet serpiginous lesions on her scalp, scalp tenderness, and arterial bruits on auscultation of the carotids and subclavian arteries. Her temporal arteries were tender and pulseless. Routine blood work showed thrombocytosis (773×10^9/L), an ESR of 32 mm/h, and a CRP of 18 mg/L. A cutaneous biopsy was done and showed epidermal necrosis with superficial perivascular dermatitis. Temporal artery biopsy was positive for the diagnosis of GCA. Oral prednisone was initiated at 80 mg daily (1 mg/kg).

At 1-month followup, the patient had drastically improved and had no more headache or jaw claudication. The epidermal necrosis had disappeared with prednisone therapy and no relapse was seen in the following 2 years.

**Discussion**

We describe 3 patients with rare ischemic complications of GCA. All 3 cases met the 1990 ACR classification criteria for GCA. Temporal artery biopsy was performed in 2 of the cases (cases 1 and 3 in the current report). In case 2, the diagnosis was confirmed by temporal artery duplex ultrasonography. All patients were treated with glucocorticoids, which improved the ischemic symptoms in the following months.

A high index of suspicion with availability of rapid investigation modalities is required to diagnose GCA when patients initially present with atypical, rare, or nonspecific symptoms. Existing diagnostic modalities include temporal artery biopsy,
color doppler ultrasound of cranial arteries, high-resolution MRI of cranial arteries, high-resolution PET with CT of cranial arteries, and extracranial large vessel imaging. Accessibility and availability of the required expertise often dictates which modality will be used. For example, the European League of Associations for Rheumatology recommends temporal artery duplex ultrasound as a first approach for establishing a diagnosis in patients with suspected GCA, but the American College of Rheumatology does not recommend temporal artery duplex ultrasound until a physician with specific expertise is available to oversee the procedure [4,5]. Our academic center has externally validated color doppler ultrasound in GCA and we have been using it as the first method of diagnosis of GCA since 2018 [6]. The patient in case 1 was seen in 2011 and the patient in case 3 was seen in 1995. At that time, temporal artery biopsy was performed as the first method of diagnosis.

Visual Complications

Visual manifestations related to GCA, including permanent visual loss (PVL), are usually due to the vasculitis affecting the ophthalmic branches of the internal carotid artery [7]. Ophthalmic manifestations including diplopia, blurry vision, transient visual loss (amaurosis fugax), and PVL have been described with a frequency that ranges between 10-30%. Some studies report visual manifestations in up to 70% of patients [8-10]. Transient visual symptoms can precede PVL in up to 50-65% of patients [11]. Diplopia, which results from extra-ocular muscle ischemia or cranial nerve ischemia, is reported in about 10-15% of patients with GCA and is usually transient [12,13].

In a retrospective study of 100 patients with GCA, 53% of patients had visual manifestations at presentation which included blurry vision (30%), diplopia (16%), amaurosis fugax (14%), and blindness (19%) [13].

Patients with visual symptoms may have fewer constitutional symptoms, less concomitant polymyalgia rheumatica, and lower inflammatory markers, but these predictive factors have not been reproduced in all studies [10].

GCA is considered a medical emergency due to its potential risk of PVL, the most feared complication, which is reported in up to 15-30% of GCA patients [9,13-15]. The incidence of PVL has decreased in recent years, suggesting that increased awareness by physicians has led to earlier diagnosis and prompt initiation of glucocorticoids [12].

The most common cause of PVL due to GCA is AION, which is the result of an inflammatory occlusion and an interruption of blood flow in the posterior ciliary arteries to the optic nerve [7,9,16]. AION accounts for 60-90% of visual loss in patients with GCA [9,10,13,14], and is usually described as a sudden painless loss of vision. In its acute phase, the optic disc is pale, and the retina is normal. Edema is usually noted due to ischemia of the optic nerve occurring most frequently near the junction of the optic nerve and the eye. Within 6-8 weeks, optic nerve atrophy develops and is associated with optic disc cupping [7,11,16].

Other causes of vision loss related to GCA include central retinal artery occlusion, posterior ischemic optic neuropathy, and occipital stroke [9,16].

Central retinal artery occlusion is the second leading cause of visual loss in patients with GCA and is reported in 15-20% of GCA patients with PVL [9,10,13,17]. When the vasculitic process involves the central retinal artery, the retina is damaged and appears edematous and greyish. The retinal edema will cause a blunting of choroidal pigmenary detail in the macula which will lead to a contrasting red zone in the macula called the “cherry red spot” [7,16].

If ischemia occurs more distally in the retrobulbar segment, it causes visual loss without an edematous optic nerve. This presentation is referred to as posterior ischemic optic neuropathy (PION) and is unusual in GCA, occurring in less than 3-6% of cases [3,12,18]. Usually, PION presents with sequential vision loss when an arteritic process is involved, compared with monocular vision loss when non-arteritic [18]. The optic disc examination is normal in the acute setting, with the development of optic atrophy around 6 weeks after the event [12,16].

Phosphenes (or photopsia) are perceptions of brief flashes of light that occur independently of the classical stimulation of the retina. They are sometimes reported by patients in the acute phase of retinal or optic nerve ischemia [16]. Patients with PVL may develop chronic visual hallucinations of often well-formed images; this is referred to as Charles Bonnet syndrome. Patients maintain insight that these hallucinations are not real. This phenomenon is thought to result from damage along the visual pathway [16,19,20]. Visual hallucinations are rarely considered in the differential diagnosis of GCA, and older patients who experience them are usually considered as having cognitive impairment or a psychiatric illness [16]. Charles Bonnet syndrome is possibly underdiagnosed; it is reported in 0.4-30% of patients who are visually impaired [20]. One in 5 patients with low vision might experience visual hallucinations [21].

Once it is suspected that visual symptoms are related to GCA, glucocorticoids must be started immediately, to avoid contralateral eye involvement and occurrence of other ischemic events [10]. It is reported that 1-4% of patients will experience a new ischemic event despite initiation of glucocorticoids. Bilateral PVL can occur in 20-62% of patients, either
simultaneously or sequentially [10,16]. In untreated and/or undiagnosed patients, the involvement of the other eye may occur within 1 to 10 days after the initial vision loss [7]. Unfortunately, once PVL occurs, the visual prognosis is poor, with only 15-20% of patients showing visual improvement with treatment [10,13,16].

**Central Neurologic Vascular Complications**

Cerebrovascular accidents (CVA) occur in 2-7% of patients with GCA. Although uncommon, it is the leading cause of mortality in GCA [22-26]. A meta-analysis reported an association between GCA and CVA, with an overall 1.4-fold increased risk compared with patients without GCA [27]. However, another study on 244 patients with GCA compared the incidence of stroke to non-GCA patients and found that the incidence of stroke and transient ischemic attack was similar between these 2 groups [8].

GCA has a predilection for vertebrobasilar arteries; the prevalence of stroke involving the vertebrobasilar territory is estimated at 40-75% of GCA-related strokes, vs 15-20% of atherosclerosis-related strokes [3,17,22-24,26]. Vertebrobasilar strokes tend to occur at diagnosis or within the first month after initiation of treatment with glucocorticoids [25,27]. In contrast, anterior circulation strokes tend to occur before the onset of other GCA symptoms or after the first month of diagnosis [3,27]. The median time between GCA diagnosis and stroke was reported to be 1.6 months in a French retrospective study [24].

Patients with GCA and stroke are mostly older men, smokers, and people with an increased prevalence of diabetes, hypertension, and history of ischemic heart disease. It was also reported that patients with severe ischemic events, such as CVA, had an increased incidence of ophthalmic ischemic symptoms and presented more often with AION [17,22-24,28]. Other predictive factors identified for the occurrence of stroke in GCA patients were the presence of visual symptoms at diagnosis, low inflammatory markers, and the absence of anemia [22].

The presence of systemic symptoms, especially fever, has been reported to be protective in the development of GCA-related CVA. The use of antiplatelet therapy in GCA patients is debated, but low-dose aspirin was identified as a protective factor in a few studies [17,29], while others have not found a beneficial effect [25,30,31].

The most common neurological manifestations in GCA-related stroke are focal motor or sensory deficit (41%), followed by cerebellar syndrome (34%), and facial palsy (32%) [22]. At the time of diagnosis, about 77% of patients with CVA have other GCA-related symptoms. Amaurosis fugax and transient ischemic attacks were frequently reported [17]. Patients with PVL or jaw claudication are more likely to suffer from an ischemic stroke in the future, affecting the carotid or vertebrobasilar territory [2]. Ischemic stroke of the occipital lobe occurs in up to 7% of patients, which may cause homonymous hemianopsia [16].

Ischemic cerebrovascular events are mainly due to vasculitis (occlusion or stenosis) of extradural blood vessels rather than intradural arteries. Intracranial involvement is rare in GCA since intracranial arteries contain little or no internal elastic lamina [22,23]. The exact incidence of intracranial involvement is unknown, but studies have observed that these patients usually present with multiterritory CVA [23]. In a review describing 47 patients with intracranial GCA, the overall mortality was high: it was reported in 53% of patients, and only 22% of patients had complete neurologic recovery. These strokes occurred, at a median time of 60 days after the diagnosis of GCA, in 64% of patients. Mortality was found to be lower in patients who were started on immunosuppressives in combination with glucocorticoids than glucocorticoids alone [23]. One hypothesis to explain these findings is that prednisone and methylprednisolone do not effectively cross the blood-brain barrier, which raises the question of whether dexamethasone should be used instead in these patients [23].

Physicians should be aware that CVA are among the initial manifestations of GCA, but may occur at a later stage, despite glucocorticoid treatment.

**Tongue and Scalp Necrosis**

Scalp and tongue necrosis are uncommon ischemic complications of GCA and are associated with an increased mortality rate [2,32,33]. They may represent the initial manifestations of the disease, which usually leads to a diagnosis delay [32]. Tongue and scalp necrosis tend to occur more frequently in older patients [32,33].

Scalp necrosis has been associated with a higher incidence of visual complications, including PVL, and is usually a sign of active GCA involving multiple blood vessels [32]. Scalp necrosis has also been associated with a higher incidence of tongue necrosis, and both manifestations can occur simultaneously. Simultaneous tongue and scalp necrosis was previously reported in only 2 cases in the literature [32,34].

Tongue necrosis is rare since the tongue has a rich vascular supply from the lingual artery, the tonsillar branch of the facial artery, and the ascending pharyngeal artery (which all have the external carotid artery as their common origin). This makes bilateral lingual necrosis even more uncommon [33,35]. Lingual manifestations (edema, pallor, pain, claudication) have been reported in up to 25% of patients with GCA and are associated with an increased risk of other ischemic complications [33,35].
It is suggested that patients (>50 years of age) with tongue necrosis should undergo an expedited workup for GCA in the absence of an alternative cause [34]. Physicians should be aware of this rare complication since diagnosis and early glucocorticoid treatment may alter the course of the disease and reduce its morbidity [33].

**Peripheral Arterial Complications**

GCA can involve extracranial large vessels, including arteries of the lower and/or upper extremities, and can cause clinical manifestations such as lower and upper limb claudication [3]. Involvement of these blood vessels can lead to severe complications including acute limb ischemia and amputation. Patients with extracranial large vessel involvement usually have fewer cranial manifestations of the disease, such as headache, jaw claudication, and scalp tenderness [36].

Several studies have shown that patients with GCA have an increased risk of peripheral artery disease (PAD). This is likely due to accelerated atherosclerosis related to chronic inflammation and/or secondary to vasculitis of the lower extremities. A meta-analysis including 9789 patients showed that patients with GCA had an increased risk of PAD, with an 88% excess risk. In this meta-analysis, statistical heterogeneity was high, and the results were likely affected by detection and publication bias; thus, the precise causal association between PAD and GCA is still unclear [37].

In GCA patients with large vessel involvement, upper extremity vasculitis seems to affect 60% of patients, while lower extremity vasculitis affects 20-30% of patients [36]. Subclavian and axillary arteries are the most frequently involved blood vessels in upper extremity vasculitis. Arterial involvement in lower extremity vasculitis mostly affects superficial femoral arteries followed by deep femoral arteries, posterior tibial arteries, and popliteal arteries [36,38].

Among patients with GCA, vasculitic involvement of lower extremity arteries is rare, but small studies using ultrasonography have shown that the prevalence is probably underestimated, as patients with GCA do not usually undergo systemic vascular imaging [39,40]. A study using positron emission tomography (PET) scan to assess vascular uptake in patients with GCA showed that 37% of patients had vascular uptake in the femoral arteries [41,42]. However, it was recently shown in the same longitudinal cohort that there was no difference in arterial uptake in iliac and femoral arteries between patients with large vessel vasculitis and the comparator group consisting of healthy patients or patients with dyslipidemia [41,42].

To our knowledge, specific predictive factors have not yet been recognized to identify GCA patients at risk of symptomatic PAD. Some studies have shown that upper or lower extremity vasculitis occurs more often in women [36,38]. Another study showed that hypertension, dyslipidemia, and smoking increase the risk of developing symptomatic lower limb vasculitis in the setting of GCA [38].

Clinical manifestations of peripheral artery involvement in patients with GCA have been described in 3-16% of patients, including intermittent claudication (87%), Raynaud’s phenomenon (19%), limb ischemia (18%), and gangrene (6%). In a previously published study, 10.25% of the 351 patients had symptomatic upper and/or lower extremity vasculitis [36]. Of these patients, 20% were symptomatic before GCA diagnosis, 36% had new-onset limb claudication at the time of GCA diagnosis, and 44.4% became symptomatic during followup, mainly during steroid taper or discontinuation. Severe ischemic complications of the extremities occurred in 28% of affected patients and all required surgery [36].

In GCA patients with upper/lower extremity vasculitis, the aorta may also be involved (70% of patients) and should be systematically investigated to detect complications, such as ectasia or aneurysm [36].

These findings underscore the importance of a focused history and thorough physical exam to detect and screen for PAD in patients with GCA.

**Conclusions**

Uncommon ischemic manifestations related to GCA may occur before the classic disease presentation and therefore cause a delay in diagnosis. As most patients with these rare presentations are more prone to other ischemic complications such as vision loss, early recognition and rapid glucocorticoid initiation is important to avoid irreversible damage.

**Acknowledgements**

We would like to thank our research coordinator, Mrs. Guylaine Marcotte, for her administrative support.

**Declaration of Figures’ Authenticity**

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.
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