

# Giant Cell Arteritis Presenting as Ischemic Stroke: A Case Report

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**Abstract:** Giant cell arteritis (GCA) is the most common vasculitis over 50 years of age. GCA is an inflammatory condition affecting medium to large sized arteries. GCA is classified into a cranial form (c-GCA) and an extracranial/large vessel form (LV-GCA). Temporal artery doppler ultrasound (TADUS) has become the imaging modality of choice for GCA for rapid diagnosis. Glucocorticoids (GC) remain the primary treatment for GCA. We report the case of a 79-year woman with temporal headache, right hemiparesis and hypoesthesia. She had normocytic normochromic anemia, elevated c reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Brain resonance (BR) revealed bihemispheric infarction. She was diagnosed with left sensorimotor lacunar cerebrovascular accident. TADUS revealed hypoechoic halo in the temporal arteries. c-GCA was newly diagnosed as the aetiology of stroke and treated with GC with resolution of symptoms. Ischemic stroke is a rare complication in GCA and is a life-threatening condition. This case stands out for the importance of GC as an effective therapy in GCA with symptomatic intracranial involvement, improving their prognosis. In daily practice the early diagnosis of GCA can be challenging.

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## 1. Introduction

The most widely used criteria for Giant cell arteritis (GCA) were formulated by the 2022 American College Rheumatology (ACR)/European League Against Rheumatism (EULAR) and includes clinical, laboratory, imaging or biopsy criteria. The diagnosis of GCA should be considered in a patient over the age of 50 years, temporal headache, visual loss, jaw or tongue claudication, morning stiffness in shoulders/neck, scalp tenderness, temporal artery abnormalities (tenderness to palpation, decreased pulse amplitude), elevated ESR  $\geq 50$  mm/hour and/or CRP  $\geq 10$  mg/L, positive temporal artery biopsy or halo sign on Temporal Artery Duplex Ultrasonography (TADUS). Other findings are fever, fatigue, weight loss, limb claudication, asymmetric blood pressures, vascular bruits. A diagnosis of GCA should be considered in all patients with Polymyalgia rheumatica (PMR) [1-9].

Females are affected more frequently than males with a peak incidence between the seventh and eighth decades of life. GCA is commonly found in individuals of Scandinavian descent or in Scandinavian countries and is less common in Latin American, Asian, Arabic and African American populations. GCA is commonly presented with non-specific constitutional symptoms: fever, fatigue, weight loss, anorexia, myalgias, headache. Less often, GCA is a cause for stroke. About half of patients diagnosed with GCA have PMR. The most common ocular manifestation of GCA is visual loss most often secondary to



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arteritic anterior ischemic optic neuropathy. The most feared complication of GCA remains that of permanent loss of vision which commonly is painless and sudden and is rarely reversible [1-9].

We present the case of a 79-year woman with atrial fibrillation (AF) medicated with apixaban, with temporal headache, right hemiparesis and hypoesthesia, normocytic normochromic anemia, elevated CRP/ESR. BR revealed bihemispheric infarction. She was diagnosed with left sensorimotor lacunar cerebrovascular accident. TADUS revealed hypoechoic halo in the temporal arteries and GCA was diagnosed. This case highlights the critical intersection of vasculitis and cerebrovascular disease. This case emphasizes that we should try to improve case finding to reduce delay diagnosis, thereby highlighting the need for increased knowledge and awareness that GCA is a medical emergency.

## 2. Case Report

We present the case of a 79-year-old caucasian woman patient with a history of dyslipidemia, AF, heart failure. She had bilateral temporal pulsatile headache with relief after paracetamol for 5 months before admission. She performed routine analysis that revealed normocytic normochromic anemia. She refused to do any more tests and did not seek more medical help. She was medicated with apixaban 5 mg 12/12h, atorvastatin 40 mg, furosemide 80 mg, spironolactone 25 mg, empagliflozin 10 mg, digoxin 0,25 mg, carvedilol 6,25 mg 12/12 h. The patient was admitted to the emergency department with headache, right hemiparesis and hypoesthesia. These symptoms started four hours before admission. There was no history of trauma. She lived in an urban area and no travel history was reported. She also denied animal exposure and drug or toxin history. On physical examination, the patient had a Glasgow Coma Scale score of 15, right hemiparesis and hypoesthesia, National Institutes of Health Stroke Scale (NIHSS) [4].

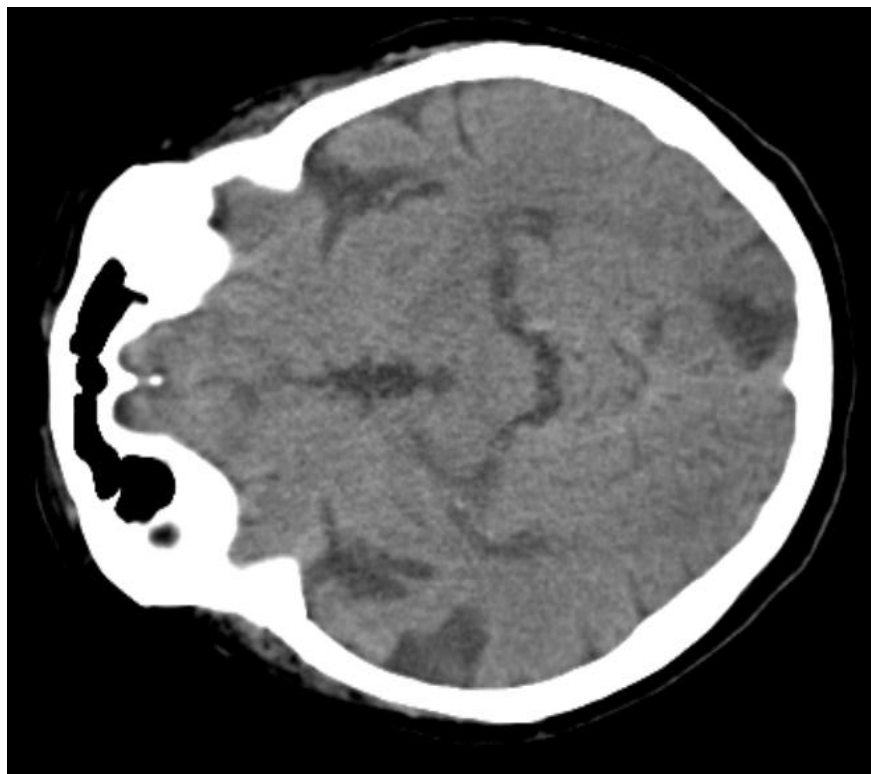
The patient was normotensive (125/65 mmHg), had no asymmetrical blood pressure in arms, pulses were arrhythmic and symmetrical (FC 70 bpm). Oxygen saturation 99% while breathing ambient air. The patient appeared pale. His heart sounds and lungs were normal to auscultation. She had no palpable liver, lymph nodes, abdominal pain or skin rash on examination. Blood tests showed normocytic normochromic anemia (hemoglobin 9,8 g/dl), increased CRP (9,8 mg/dl), increased ESR (> 89 mm/hour), therapeutic levels apixaban. A chest radiograph was normal. An electrocardiogram showed AF. Urinalysis was unremarkable. Brain computed tomography (CT) showed no acute ischemic or hemorrhagic vascular injury, angiographic study of the supra-aortic and intracranial trunks showed no arterial stenosis in the vertebral or common carotid arteries but revealed calcified plaques in the carotid bifurcations, reducing the caliber of the internal carotid arteries by 30% (Figure 1).

She was diagnosed with left sensorimotor lacunar cerebrovascular accident. Not eligible for thrombolysis due to therapeutic levels apixaban. Not eligible for endovascular treatment due to risk greater than benefit (age, comorbidities, location). The patient was admitted for diagnostic investigation and clinical monitoring. There was no iron deficiency, vitamin B12 or folate deficiency. Thyroid function was normal. Serologies for HIV, Hepatitis B and C and Syphilis were negative, as were the blood cultures. Serum protein electrophoresis was normal. Total cholesterol 122 mg/dl, high-density lipoprotein 35 mg/dl, low-density lipoprotein 73 mg/dl, triglycerides 70 mg/dl. She performed carotid and vertebral artery doppler ultrasound, there was no evidence of atheromatous plaque. CT-scan excluded infection or neoplasia. Autoimmune screening was negative (rheumatoid factor, antineutrophil-cytoplasmatic, antinuclear, anti-double stranded DNA and extractable nuclear antigen antibodies). BR showed multiple bihemispheric ischaemic lesions: location cortical, subcortical left temporo-occipital, left frontal parietal, right frontal (Figure 2 e 3).

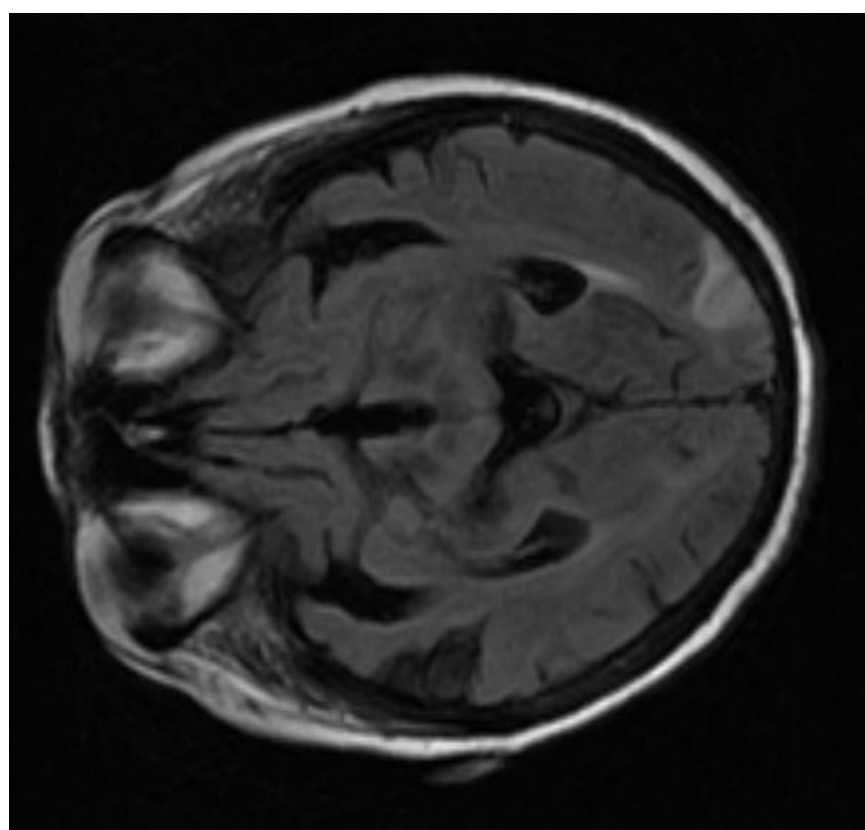
TADUS revealed parietal thickening, lack of compressibility and persisting visibility of the halo during compression of the vessel lumen by the ultrasound probe in the temporal arteries, compatible with a positive hypoechoic halo and compression sign. GCA

was diagnosed using ultrasound. She was evaluated by Ophthalmology who excluded ocular vasculitis.

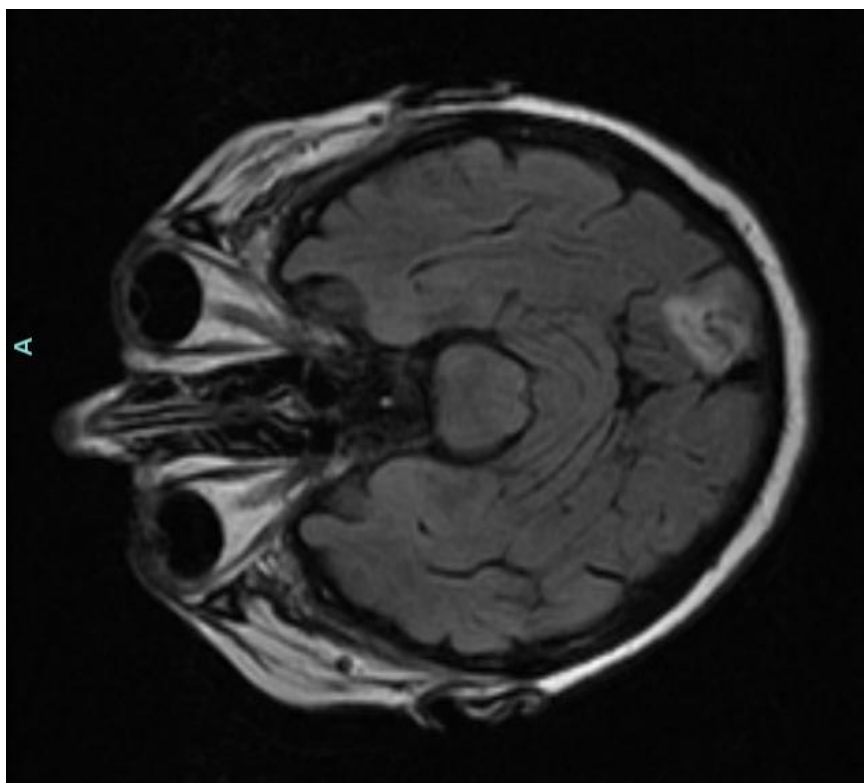
**Figure 1.** Brain CTS shows no acute ischemic or hemorrhagic vascular injury.



**Figure 2.** Brain MRI showing multiple bihemispheric ischemic lesions involving cortical and subcortical regions: left temporo-occipital, left frontoparietal, and right frontal areas.



**Figure 3. (Continued).** Brain MRI showing multiple bihemispheric ischemic lesions involving cortical and subcortical regions: left temporo-occipital, left frontoparietal, and right frontal areas.



Transthoracic echocardiogram revealed left ventricular systolic function at the lower limit of normal. The patient started on intravenous (iv) methylprednisolone (MTP) pulses (1 g daily for three days) followed by prednisolone (PDN) 60 mg (1 mg per kilogram) once daily. After one week of GC, the patient was clinically improved (NIHSS 0), inflammatory markers were normalized (CRP 5 mg/dl, ESR 20 mm/h), anemia improved (Hb 11 g/dl). She was discharged on the 7th day of hospitalization under apixaban 5 mg 12/12h, bisoprolol 5 mg, digoxin 0,125 mg, rosuvastatin 20 mg and PDN 60 mg once daily on tapering. Now she is being followed on an outpatient basis, tolerating steroids taper. GC was slowly tapered by 10 mg every two weeks until 20 mg/day is reached, then reduced by 2.5 mg every 2–4 weeks to 10 mg and afterward by 1 mg every 1–2 months according to clinical response. The patient presented sustained clinical improvement, without recurrence of any ischemic events at three months follow-up. The patient continued corticoid tapering allowing a dose reduction to a minimum of PDN 2.5 mg once daily.

### 3. Discussion and Conclusion

This case report describes an important and clinically relevant case of GCA presenting with an ischemic stroke. In this case the onset of symptoms in GCA was subacute and indolent. Although the clinical manifestation of GCA was nonspecific, some characteristic findings strongly suggested the diagnosis: over the age of 50 years, headache, normocytic normochromic anemia, increased ESR/CRP and stroke. Underlying risk factor for cerebrovascular disease, such as dyslipidemia present in this case increased the risk of cranial ischemia in patients with GCA. GCA is an unusual cause of ischemic stroke. Stroke due to GCA has two notable clinical features: vertebrobasilar involvement is frequent and intracranial internal carotid involvement is rare. The most affected vessels in GCA are the superficial temporal artery, ophthalmic artery, posterior ciliary arteries and vertebral arteries. Less commonly, the aorta, coronary arteries, and carotid circulation are involved,

intracranial arteries are usually spared from the vasculitic attack of GCA, presumably because of the paucity of elastic tissue in their walls [1-9]. This case is rare because GCA had intracranial internal carotid involvement. Stroke is a potential complication related to GCA and can serve as an early warning sign of GCA. Raising awareness regarding these aspects empowers patients to participate in their care actively and promotes optimal outcomes. The granulomatous nature of GCA contributes to the loss of vascular smooth muscle cells and elastic fibers, potentially leading to vascular remodeling. Intimal hyperplasia and lumen occlusion contribute to ischemic complications. The diagnosis of GCA is complicated by the fact that there is no single lab value, imaging result or biopsy that will always be positive in all patients. Therefore, no test can be used to rule it in or out. Patients can present with a broad range of symptoms, none of which are pathognomonic for GCA. It is crucial that clinicians maintain a high index of suspicion for GCA in any elderly patient with stroke.

In this case there was a discrepancy between the results of brain CT and BR. Brain CT showed no acute ischemic or hemorrhagic vascular injury, angiographic study of the supra-aortic and intracranial trunks showed no arterial stenosis in the vertebral or common carotid arteries but revealed calcified plaques in the carotid bifurcations, reducing the caliber of the internal carotid arteries by 30%. BR showed multiple bihemispheric ischemic lesions with location cortical, subcortical left temporo-occipital, left frontal parietal, right frontal. BR has been utilized in the diagnosis of GCA. Active vasculitis is shown by blood vessel wall thickening and/or increased mural contrast enhancement (in the same area as the halo-sign on ultrasound). Findings include mural edema in the temporal arteries as well as the ability to visualize other arteries. BR is much less operator dependent compared to TADUS. BR is comparable to biopsy in diagnostic value and may spare biopsy in patients with normal BR findings. Limitations to this method include limited availability and cost as well as the limited detection of mural inflammation once GC has started because of diminishing sensitivity after initiation of GC [1-9].

Elevation of classic inflammatory markers continues to be one of the cornerstones of both, new-onset GCA diagnosis and relapse identification. Although the ESR and/or CRP are almost always high in GCA, they are not specific. Their role is more important to rule out GCA when they are normal rather than to confirm GCA when they are elevated. It is extremely rare that both CRP/ESR are normal in new onset GCA. This finding has a fundamental importance since GCA symptoms (headache) are often not specific and potentially misleading like this case. Normal levels do not definitely rule out GCA but should raise suspicion of alternative diagnoses. Even if the CRP is more sensitive than the ESR, an elevated ESR with a normal CRP can also infrequently be observed. The CRP has certain advantages over the ESR, including a lack of variation with age, sex or hematological factors. CRP proves to be a more sensitive marker for inflammation in GCA, as ESR levels can increase with age and the presence of comorbidities such as anemia. A normal CRP carries a high negative predictive value. The CRP and ESR can be normalized by GC. Other laboratory indices that reflect systemic inflammation include a normochromic normocytic anemia, thrombocytosis, reduced albumin and elevated  $\alpha$ -2 fraction on serum protein electrophoresis and a raised fibrinogen. It is usually the alkaline phosphatase that is often mildly elevated. These alterations revert to normal with GC. Normochromic anemia is often present prior to therapy and improves promptly after the institution of GC. The leukocyte count is usually normal or minimally elevated, even in the setting of widespread systemic inflammation [1-9].

GCA is a clinical diagnosis, but it is usually supported with lab results, imaging and/or pathology. Proceeding with TADUS is appropriate when the clinical suspicion is high. The improvement in the diagnosis of GCA can be attributed to the introduction of highly sensitive diagnostic tools TADUS. TADUS can have higher sensitivity over temporal artery biopsy (TAB). TADUS has several advantages: it is noninvasive, safe, cost, provides simultaneous image acquisition and interpretation, no exposure to radiation, visualization of multiple arteries (facial, occipital, vertebral, carotid, axillary, subclavian

arteries) and has become widely available. Four signs can be found by TADUS in GCA: halo sign, compression sign, stenosis and vessel occlusion. When viewed using TADUS, inflammatory tissue is hypoechoic, allowing a skilled sonographer to detect halo sign (hypoechoic ring of inflamed and edematous vessel walls surrounding the Doppler signal in the lumen of an artery) and compression sign (hypoechoic vessel wall infiltrate in the presence of arterial lumen occlusion). In this case, TADUS was a valuable tool and revealed parietal thickening, lack of compressibility and compression sign (persisting visibility of the halo during compression of the vessel lumen by the ultrasound probe in the temporal arteries), positive hypoechoic halo (mural edema is indicative of active inflammation). These findings suggested vasculitis for the cause of stroke because they have high specificity for the diagnosis. GCA was diagnosed using TADUS. It may be possible for TADUS, if performed by experienced operators, to substitute for TAB as a diagnostic procedure and has been gaining popularity as an alternative. EULAR currently recommend the use of TADUS to confirm the diagnosis of GCA, given the low invasiveness, rapid result availability and comprehensive inflamed vessel visualization.

TADUS has been found to be a cost-effective alternative to TAB in reducing false negatives. Some studies propose TADUS as the initial diagnostic modality, followed by biopsies performed only in negative cases. TAB might be avoided like in this case. Because of the high incidence of false negative with biopsy it has low negative predictive value. In the EULAR recommendations, TADUS should be chosen as the first diagnostic test at disease onset instead of biopsy. A suspected diagnosis of GCA should be confirmed by TADUS but should not delay initiation of glucocorticoids. TADUS should be performed before or as early as possible after initiation of GC because treatment with GC rapidly reduces the sensitivity of TADUS. Temporal arteries can be spared in 40% of patients with GCA, risking misdiagnosis when relied upon in isolation for diagnosis. TADUS has disadvantages, is operator-dependent and lack of experience and standardization of image acquisition can hinder its potential assistance in diagnosis. Adequate and structured training is an important consideration to improve the reliability of TADUS in GCA diagnosis. In addition to its diagnostic role, TADUS may also serve as a prognostic tool and should be the tool of choice for monitoring patients and assessing response to treatment. Halo sign correlates with disease activity markers and GC cumulative dose; it is present in more than 90% with disease relapses. Once treatment is initiated, the halo sign and intima-media thickness are reported to shrink within seven days, eventually disappearing after eight weeks in most patients while recurrence of halo predicts flare. The number of temporal artery segments with halo correlates with ESR/CRP [7-9].

In this case, TAB was not performed because was unavailable in our hospital. TAB has some limitations. Several factors can affect the diagnostic sensitivity of TAB with false negative results: biopsies are sampled from spared segments of arteries due to skip lesions and the segmental nature of arteritis (discontinuous character of the histopathologic changes), lack of temporal artery involvement (disease phenotype not associated with cranial arteritis), inadequate sample length, incorrect tissue sampled and the initiation of steroids prior to biopsy can lead to resolution of the vascular inflammatory infiltrate. The TAB should be performed before GC or within 2 weeks of initiating GC. False-negative results occur in up to 44% of patients with an established diagnosis of GCA. The temporal arteries may be spared by the inflammatory process, especially in patients with large vessel involvement, in whom temporal artery biopsy findings are negative in a substantial number of cases [9].

In this case, an older female (79-year) had dyslipidemia and AF hypocoagulated with therapeutic levels apixaban. She presented cranial symptoms (headache, right hemiparesis and hypoesthesia), normocytic normochromic anemia, increased CRP and ESR suggestive of GCA. BR revealed bihemispheric infarction. Transthoracic echocardiograms excluded endocarditis and intraventricular thrombus formation because there is evidence in literature that non-infective endocarditis may be a complication of GCA. She had classic

risk factors for ischemic stroke like dyslipidemia and AF but other causes must be investigated like GCA. TADUS showed hypoechoic halo and compression sign highly specific for GCA. In this case, stroke had multiple causes: advanced age, atherosclerosis, AF and GCA. GCA was a co-incidental finding in a patient who suffered a cardioembolic event despite she had AF hypocoagulated with therapeutic levels apixaban. Despite a history of dyslipidemia, carotid and vertebral artery doppler ultrasound had the disadvantage operator-dependent and didn't revealed atheromatous plaque but angiographic study of the supra-aortic and intracranial trunks revealed calcified plaques in the carotid bifurcations reducing the caliber of the internal carotid arteries by 30%. Atherosclerosis has overlapping pathophysiology with GCA as cytolytic, proteolytic and reactive oxygen species are deposited in arterial adventitia, causing chronic low-grade inflammation, angiogenesis and fibrosis, subsequently leading to arterial remodeling. Vascular remodeling may occur early in atherosclerotic disease. Due to the shared pathological processes, one might predict that the presence of GCA or atherosclerosis could precipitate or accelerate the development of the other. Vascular ageing may play a central role in the initial immune activation in GCA. Ageing has been known to make blood vessels vulnerable to inflammation and atherosclerosis [1-6].

The patient was evaluated for malignancy because there is evidence that GCA can be a paraneoplastic phenomenon and the first manifestation of malignancy, particularly lung carcinoma, carcinoid tumor and breast carcinoma [1-6]. Given the evidence of disease progression with new vascular events, treatment with iv MTP (1 g/day for 3 days) followed by PDN (60 mg/day in a tapering schedule) was started resulting in anemia and neurologic improvement and normalization of ESR/CRP. This clinical case stands out for the importance of GC as therapy in GCA with symptomatic intracranial involvement, improving their prognosis. For three months follow-up consultations, the patient continued to show good neurologic progress. Follow-up TADUS performed at 3 months found complete resolution of temporal artery halo signs.

Once diagnosed, GCA treatment is guided by disease stratification. Stratifying patients is based on clinical, laboratory and TADUS and is needed to reduce relapses and prevent long-term vascular damage. Stratification of patients into clinical-serological phenotypes and their associated outcomes (risks of ischemic complications, relapse and treatment adverse events), facilitating selection of the minimal, individualized effective glucocorticoid dose for each patient. GC are still the mainstay of treatment of GCA with three main objectives: dampening the deranged inflammatory process to avoid acute ischemic complications, preventing disease relapses using the lowest effective dose of GC, preventing long-term vascular damage (aneurysm and stenosis). All treatment recommendations are well considered by EULAR and ACR guidelines. GC relieves symptoms (headache), as well as lower or even normalize the acute phase reactants and might prevent recurrence of stroke. Even with treatment, relapses are common. The ACR recommends a comprehensive approach, considering individual patient characteristics and tailoring treatment strategies leading to improved patient outcomes and enhanced team performance in the management of GCA. The prognosis is poor for untreated individuals. Major mortality stems from stroke. The contribution of this case is the significant diagnostic delay despite preceding symptoms headache and anemia five months before admission because initial misinterpretation of her symptoms. The activity of GCA was indolent. Stroke, a severe complication due to delayed diagnosis, underscores the importance of recognizing GCA. GCA was associated with delayed diagnosis and worse clinical outcome with many requiring a higher cumulative GC dose and are at higher risk of relapse. Vascular complications of GCA can be abrupt and irreversible like cerebrovascular accident but these are not inevitable if GCA is diagnosed early and treated appropriately. High dose GC must be started once GCA is suspected to prevent ischemic complications. GC have been the cornerstone of GCA treatment. Early GC treatment is associated with improved stroke outcomes [1-15].

The presence of a strong inflammatory response at the time of presentation is a risk factor for a protracted treatment course and a high relapse rate. In this case at admission ESR 89 mm/h and hemoglobin 9,8 g/dl indicated a strong inflammatory response. The patient was started on high-dose iv GC with excellent clinical and biochemical response. The ACR recommends initiating treatment with high-dose oral GC in patients with GCA and stroke. GC are still essential in both the induction and maintenance phase according to current clinical practice and international recommendations. Due to the crucial importance of preventing complications of stroke, the ACR recommends administering MTP 1000 mg iv for 3 days followed by 1–2 mg/kg/day of oral PDN. If MTP is unavailable, 1 mg/kg/day of oral PDN should be given to patients with GCA and evidence of cranial ischemia. Older individuals are at high risk of serious adverse reactions to high-dose iv GC pulses, however, iv MTP has been shown to demonstrate higher serum concentrations more rapidly than oral dosing and thus may be useful in strokes. The role of iv GC pulse has been associated with successful and rapid GC tapers. GCA is a systemic disease of variable duration. Most patients taper and discontinue GC after a few years of treatment. Some may require long-term use of low-dose GC. Lifestyle recommendations should be provided to all patients to prevent metabolic complications and reduce cardiovascular risk. GC are currently regarded as crucial for maintenance therapy. Slower tapering of GC dose seems to prevent relapses [1-15].

The induction dose of oral GC must be maintained ideally for two to four weeks and in any case at least until remission is established. After that, tapering should be started. Upon clinical remission, clinicians decrease the GC dose by 10 mg every 2 weeks to reach 20 mg/d of PDN after the patient has been in remission for 4 to 8 weeks. At this point, the daily PDN dose decreases by 2.5 mg every 2 to 4 weeks until the patient achieves 10 mg/d. Clinicians can continue to taper the PDN 1 mg every 1 to 2 months if clinical remission continues. The goal is to taper GC to zero over a period of 12 to 18 months while maintaining clinical remission and normal inflammatory markers. Patients at risk of GC toxicity may warrant a more rapid GC taper. Slow tapering of GC is necessary to mitigate the risk of adrenal insufficiency. While reducing the dose of GC, clinical monitoring and serial measurement of acute phase reactants are paramount to identify disease relapses. If a flare is diagnosed, GC should be increased to the last effective dose or, in case of ischemic manifestations, to the induction dose. The risks associated with the prolonged use of GC at moderate-high doses are particularly accentuated in patients with GCA due to the old age of this population [1-15].

Current EULAR recommendations suggest tapering the GC to a target dose  $\leq 5$  mg prednisone equivalent daily after 12 months of treatment but admit that this goal is hard to reach and most patients require longer treatment. Disease stratification will probably be necessary to identify patients with a higher probability of requiring a standard 1–2 years course and those needing slower dose tapering. Future approaches must also be addressed if a strategy of trying to stop GC at all costs, with the risk of disease flare and subsequent need for increased dosing is a better strategy than maintaining a low dose (PDN equivalent  $\leq 5$  mg daily) indefinitely. Only a small fraction of patients with GCA achieve definitive, long-lasting ( $\geq 3$  years) treatment-free disease remission [1-15].

Once GC treatment is initiated, the patient must be monitored closely to determine the response to therapy. Some have recommended following the ESR and CRP every 3–7 days, depending on the severity and route of administration of the GC. Although symptomatic improvement may be reached within days of therapy initiation, the ESR can take several weeks to decrease. Once an effective dose of GC is reached (based upon improvement in symptoms), the treatment at this dose should be continued for a minimum of 4–6 weeks. However, treatment courses typically last for 1–2 years, given the importance of a very slow steroid taper to avoid a relapse of the GCA. The patient should be monitored closely for any relapse of symptoms or increase in ESR or CRP. The effectiveness of GC is often monitored by measuring ESR and CRP over time, but these inflammatory markers may not consistently reflect disease activity. Patients usually require at least 2 years of GC

and most are able to taper off by 5 years after diagnosis. Some patients suffer relapses of GCA either during or after initial treatment. The reason for this variability is multifactorial. Signs and symptoms of relapse can vary from patient to patient and inflammatory markers may be normal during the flare. Physicians should ask patients about new-onset ischemic complications, symptoms like original presentation or classic manifestations of GCA. EULAR recommendations suggest treating major relapses defined by clinical symptoms with high dose GC of 40-60 mg/day.

GCA is a chronic disease in which relapses are common and long-term monitoring is required. Relapses are usually treated by escalating GC doses 10–15 mg above the previous effective dose, thus increasing the cumulative GC dose and the likelihood of GC long-term side effects. Minor relapses defined by recurrence of active disease without fulfilling the criteria for a major relapse are recommended to be treated 5-15 mg above the last effective dose of GC. Most cases of GCA relapse have been shown to occur at PDN doses below 20 mg/day, especially during the first year of treatment. Patients with GCA can rarely develop sequelae of large vessel vasculitis after GC treatment has been tapered. Some have recommended follow-ups at weeks 0, 1, 3, and 6, and then monthly thereafter for the first year. Physicians should individualize management and follow-up schedules, focusing on patient disease progression, symptoms and complications. Each visit should screen for progressing GCA, including cranial symptoms and ischemic symptoms. Adverse effects to GC should also be monitored closely. As GCA is a chronic condition, proper patient education and access to support should not be overlooked. Patient education is crucial in the comprehensive treatment approach for individuals with GCA. Focusing on strategies that help mitigate complications associated with glucocorticoid therapy is imperative [1-15].

Healthcare professionals must recognize the potential adverse effects and complications related to the long-term use of GC: cataract, glaucoma, fragility fractures, osteoporosis, osteoporosis, hypertension, adrenal infection, hypertension, immunosuppression, weight gain. Clinicians must evaluate the risk of these complications and implement measures to reduce their occurrence. Clinicians should universally advise lifestyle interventions, including diet, regular exercise, weight loss, strength training, smoking cessation. Calcium and vitamin D supplementation and H2 antagonist or proton pump inhibitor should be provided when appropriate with regular monitoring of bone mineral density and initiation of bisphosphonate therapy when indicated. Monitoring glucose intolerance through blood glucose checks before initiating therapy and every 3 months is crucial [1-15].

In GCA the frequency of vascular events (stroke, coronary and peripheral arterial disease, venous thromboembolism) is higher. While current therapies for GCA can control disease symptoms and reduce the occurrence of severe complications, they are not curative of the disease. Even in patients who clinically appear to be in longer term, treatment-free remission, the reappearance of disease after sometimes quite prolonged periods and the occurrence of long-term disease-related complications is evidence of a more chronic disease course over many years [1-15]. All patients with GCA should be vaccinated against *Streptococcus pneumoniae*, influenza-virus and SARS-CoV-2.

We present a case of GCA which affected cranial arteries and illustrate pitfalls in the diagnosis of this polymorph condition. This case illustrates the difficulty of differentiating the different vasculitis due to overlapping symptoms. This case supports the hypothesis that this type of vasculitis could be related to the occurrence of ischemic stroke. Review of her charts revealed a substantial delay from the onset of symptoms headache and anemia to diagnosis. This case demonstrates the need for additional efforts to reduce delay in referring patients with GCA to prevent serious complications. GCA is a medical emergency as it can cause severe complications such as stroke if not treated in a timely manner. These complications can be prevented by prompt GC treatment. Therefore, swift referral, diagnosis and treatment are essential. This case involved a long diagnostic delay in a patient with GCA leading to ischemic cerebral vascular accident, highlighting the need for

and the difficulty of early diagnosis. The delay in diagnosis in our case can be attributed to different factors. First, delay can occur because the patient does not immediately seek medical attention. Based on the information available, our patient was not keen on visiting a general practitioner with general complaints and only attended after the onset of cranial complaints. Second, delays can occur during the referral of a patient when physicians do not consider GCA. This is often due to the generic nature of symptoms. In this case, there was an approximately 5-month delay from first consultation to diagnoses. A swift diagnosis was made using TADUS, avoiding further diagnostic delay. Delays can occur because of unique or atypical manifestations, making recognition and diagnosis of GCA difficult. Delay was substantially lengthened for our patients. The elevated suspicion of CGA contributes to a decrease in diagnostic delay and significantly reduces neurologic symptoms.

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**Research Ethics Committee Approval:** We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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