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Giant Cell Arteritis Presenting with Unilateral Ptosis in a Patient with Schistosomal Cirrhosis: Diagnostic Value of PET/CT in an Atypical Case

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Abstract: Giant cell arteritis (GCA) is a medical emergency in adults older than 50 years because treatment delay may lead to irreversible visual loss or other ischemic injury. We report a 71-year-old man with schistosomal cirrhosis who presented with fever, persistent headache, scalp tenderness, and subsequent right-sided ptosis. The diagnostic process was difficult because his early manifestations were nonspecific, inflammatory abnormalities could initially be interpreted in the context of chronic liver disease, and temporal artery ultrasound was unrevealing. PET/CT subsequently demonstrated diffuse fluorodeoxyglucose uptake involving the bilateral subclavian, carotid, superficial temporal, and peripheral arteries, supporting a diagnosis of large-vessel GCA. After high-dose methylprednisolone was started, the patient experienced rapid symptomatic relief together with marked improvement in inflammatory markers; methotrexate was later added as a glucocorticoid-sparing agent. Management required additional caution because of cirrhosis, abnormal liver biochemistry, and severe hypertension. This case emphasizes three practical points: unilateral ptosis may be an early neuro-ophthalmic signal of GCA even in the absence of overt visual loss, a negative temporal artery ultrasound does not rule out disease when extracranial involvement predominates, and PET/CT can provide decisive evidence when initial cranial imaging fails to explain a highly suspicious presentation.

Keywords: giant cell arteritis; ptosis; schistosomal cirrhosis; PET/CT; large-vessel vasculitis; case report

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1. Introduction

Giant cell arteritis is a granulomatous vasculitis that primarily involves medium-sized and large arteries in adults older than 50 years, with particular predilection for the extracranial branches of the carotid artery and the aorta[1-3]. Although the disorder is classically introduced through the triad of new headache, scalp tenderness, and jaw claudication, the real clinical spectrum is considerably broader. Many patients first come to medical attention with constitutional symptoms, cranial pain without typical ischemic complaints, or laboratory evidence of systemic inflammation that lacks an immediately obvious cause. This variability matters because diagnostic delay can result in permanent visual loss, cerebrovascular events, or later large-vessel complications[1-6]. For that reason, GCA should be considered a time-sensitive inflammatory vascular disease that crosses the usual boundaries between rheumatology, neurology, and ophthalmology.

The ophthalmic manifestations of GCA are best known for arteritic anterior ischemic optic neuropathy, yet the

neuro-ophthalmic spectrum extends beyond abrupt visual decline. Diplopia, ocular motor nerve dysfunction, and ptosis have all been reported, but these findings are substantially less common than optic nerve ischemia and may therefore be underrecognized when they occur in isolation or near the beginning of the illness[8-10]. At the same time, the diagnostic pathway for suspected GCA has changed in recent years. Ultrasound is now recommended as the preferred first-line imaging test for cranial disease, especially when temporal artery involvement is suspected, because it is noninvasive and can be performed rapidly[2,3]. Nevertheless, a normal ultrasound result does not exclude GCA, particularly when inflammation is concentrated in extracranial vessels rather than the superficial temporal arteries. In this context, PET/CT may uncover metabolically active large-vessel vasculitis and substantially increase diagnostic confidence [2,13].

The present case is clinically meaningful because several features simultaneously obscured the diagnosis. The patient initially presented with fever and headache rather than a fully developed cranial vasculitic syndrome, later developed unilateral ptosis without clear visual loss, and had schistosomal cirrhosis with splenomegaly, a chronic condition that complicated interpretation of inflammatory and hepatic laboratory abnormalities. We report his clinical course, diagnostic reasoning, treatment strategy, and short-term outcome, with special emphasis on how PET/CT clarified the diagnosis after a negative temporal artery ultrasound and helped redirect management toward large-vessel GCA.

2. Case Presentation

2.1. Patient information and baseline history

A 71-year-old retired man was admitted on December 31, 2025, because of persistent headache accompanied by fever for more than 2 weeks and right upper eyelid drooping for 5 days. His past medical history included grade 3 hypertension with very high cardiovascular risk, schistosomal cirrhosis with splenomegaly, a right renal simple cyst, previous left eye trauma, and prior left inguinal surgery. He also described a 5-year history of tinnitus associated with mildly decreased hearing in the left ear. There was no known family history of autoimmune disease, vasculitis, or other relevant hereditary disorders.

2.2. History of present illness

The illness began on December 15, 2025, when he awoke with diffuse headache and fever after emotional distress on the preceding day. The highest documented temperature was 37.9°C. He denied vertigo, focal weakness, numbness, abdominal pain, diarrhea, cough, sputum production, and other localizing infectious symptoms. Rest did not meaningfully relieve the discomfort. During the following days he sought medical attention at outside institutions. Cervical magnetic resonance imaging demonstrated multilevel degenerative changes, but these findings did not adequately explain the severity or persistence of his symptoms. Empirical treatment with cephalosporin and penicillin was administered, yet the fever and headache continued without clear benefit.

As the illness progressed, he developed sharp scalp pain with tenderness, nausea, fatigue, and poor appetite. Laboratory testing at our hospital revealed marked systemic inflammation, including a C-reactive protein (CRP) concentration of 93.2 mg/L. Chest and abdominal computed tomography showed a small pericardial effusion, cirrhotic liver morphology, and splenomegaly. Symptomatic treatment remained ineffective. On December 25, 2025, he was transferred to another hospital, where the erythrocyte sedimentation rate (ESR) was 83 mm/h and gamma-glutamyl transferase (GGT) was 233 U/L. Brain magnetic resonance imaging disclosed only chronic ischemic changes and no acute explanatory lesion. Temporal artery ultrasound was reported as negative, which further complicated the evaluation because the inflammatory syndrome remained striking while first-line vascular imaging failed to confirm cranial arteritis.

On December 26, 2025, he developed right upper eyelid ptosis without obvious diurnal fluctuation. He did not describe definite visual deterioration, and the left eye was unaffected. Because the combination of fever, persistent headache, scalp tenderness, markedly elevated inflammatory markers, and newly developed ptosis raised suspicion for occult vasculitis, PET/CT was arranged. The scan demonstrated diffusely increased fluorodeoxyglucose uptake in the bilateral subclavian, carotid, superficial temporal, and peripheral arteries, particularly in the lower extremities, a pattern strongly suggestive of large-vessel GCA. Intravenous methylprednisolone 40 mg was initiated on December 30, 2025,

and he was subsequently transferred to the Department of Rheumatology, Renji Hospital, Shanghai Jiao Tong University School of Medicine, for continued evaluation and treatment.

2.3. Clinical assessment

At the time of transfer, the fever had resolved and the headache had partially improved after glucocorticoid exposure, but right-sided ptosis and bilateral scalp tenderness were still present. On physical examination he was alert, cooperative, and hemodynamically stable. The right upper eyelid remained visibly drooped, whereas the remainder of the neurologic examination was largely nonfocal. Pupils were equal and reactive to light, limb strength was preserved, and no pathologic reflexes were elicited. Cardiopulmonary findings were unremarkable, and the abdomen was soft without focal tenderness.

Laboratory testing continued to support an active inflammatory process, with CRP 52.8 mg/L and ESR 83 mm/h. Liver biochemistry was abnormal, including alanine aminotransferase (ALT) 48 U/L, aspartate aminotransferase 17 U/L, GGT 207 U/L, and hypoalbuminemia at 31.1 g/L. The white blood cell count was not markedly elevated, although neutrophil predominance was noted. An extensive infectious workup, including assays for HIV, hepatitis B and C, syphilis, and tuberculosis together with blood cultures, was negative. Autoimmune serologies, including antinuclear antibodies, antineutrophil cytoplasmic antibodies, and a myositis panel, were also negative. Cerebrospinal fluid examination was essentially normal, making central nervous system infection, leptomeningeal malignancy, and inflammatory meningoencephalitis less likely and narrowing the differential diagnosis.

2.4. Diagnostic reasoning

The differential diagnosis was initially broad and evolved over time. Infection was strongly considered at first because the presentation was dominated by fever and a marked inflammatory response. Cervicogenic headache and nonspecific chronic ischemic brain disease were also entertained after outside imaging studies. Once ptosis appeared, additional alternatives included ocular myasthenia gravis, isolated oculomotor nerve palsy, microvascular ischemic neuropathy related to hypertension, and intracranial structural disease. However, there was no diurnal fluctuation, cerebrospinal fluid findings were unremarkable, and brain imaging did not reveal a lesion that could explain the ocular symptom. Meanwhile, the persistence of scalp tenderness and the continuing inflammatory pattern kept systemic vasculitis high on the list of possibilities.

The crucial interpretive step was to integrate the PET/CT pattern with the overall clinical syndrome. Although temporal artery ultrasound was negative, PET/CT demonstrated metabolically active inflammation in multiple large arteries, including the superficial temporal arteries, a distribution highly compatible with GCA rather than isolated infection or another organ-limited disorder [2,3,13]. The diagnosis was further supported by his age, the new-onset headache, scalp tenderness, markedly elevated ESR and CRP, and the subsequent rapid response to glucocorticoid therapy. Schistosomal cirrhosis complicated interpretation because it could plausibly contribute to constitutional complaints and abnormal biochemical indices, but it could not account for the vascular uptake pattern on PET/CT or the neuro-ophthalmic manifestation. Taken together, the evidence favored large-vessel GCA presenting in an atypical manner.

Table 1. Timeline of key diagnostic and therapeutic events

Date	Clinical event	Diagnostic or therapeutic significance
Dec 15, 2025	Onset of headache and low-grade fever	Beginning of systemic inflammatory illness
Dec 19–25, 2025	Empirical anti-infective and symptomatic treatment	Poor response argued against a simple infectious process
Dec 25, 2025	ESR 83 mm/h; temporal artery ultrasound negative	Persistent inflammation despite negative first-line vascular imaging
Dec 26, 2025	Right upper eyelid ptosis developed	Important neuro-ophthalmic clue raising suspicion for vasculitic ischemia

Table 1 (Continued)

Date	Clinical event	Diagnostic or therapeutic significance
Dec 30, 2025	PET/CT showed diffuse arterial FDG uptake; methylprednisolone initiated	PET/CT strongly supported large-vessel GCA and prompted targeted therapy
Dec 31, 2025	Transferred to the Department of Rheumatology at Renji Hospital	Comprehensive reassessment and treatment intensification
Jan 6, 2026	Transient peak in liver enzymes during treatment	Required hepatoprotective therapy and close monitoring
Jan 9–10, 2026	CRP normalized; ESR decreased to 18 mm/h; ptosis improved	Biochemical and clinical response supported diagnostic confidence

2.5. Therapeutic intervention

After transfer, intravenous methylprednisolone 80 mg daily was administered from December 31, 2025, through January 9, 2026, to suppress arterial inflammation rapidly and reduce the risk of ischemic complications. Intravenous immunoglobulin (20 g daily for 3 days) was also given as adjunctive immunomodulatory therapy. Supportive measures included proton-pump inhibition for gastric protection, calcitriol and calcium supplementation for bone protection, and antihypertensive treatment with sacubitril/valsartan. Given the coexistence of severe hypertension and chronic liver disease, treatment intensity and adverse-effect monitoring were weighed carefully throughout hospitalization.

During treatment, liver enzyme levels rose transiently, with ALT peaking at 122 U/L on January 6, 2026. In the setting of underlying cirrhosis, this change required close monitoring and a cautious balance between disease control and hepatic safety. Polyene phosphatidylcholine and reduced glutathione were introduced, after which liver biochemistry gradually improved. Following multidisciplinary discussion, methotrexate 10 mg once weekly was added as a glucocorticoid-sparing agent together with folic acid supplementation, and a plan was made for close outpatient rheumatologic and hepatic follow-up. This step was intended to help maintain remission while limiting cumulative steroid exposure.

2.6. Outcome and follow-up

The clinical response was rapid and clinically meaningful. Fever did not recur, headache diminished substantially, and the right-sided ptosis improved markedly during the admission. Inflammatory markers declined in parallel with symptomatic recovery, reaching CRP 0.8 mg/L and ESR 18 mm/h by January 9, 2026. At discharge on January 10, 2026, his overall condition was good. Eyelid strength had essentially normalized, cardiopulmonary examination remained stable, and no focal neurologic deficit was detected.

He was discharged with a tapering regimen of oral methylprednisolone, weekly methotrexate, folic acid, esomeprazole, calcitriol, nifedipine, hepatoprotective therapy, and ursodeoxycholic acid. Follow-up plans included review in the rheumatology clinic within 2 weeks, serial monitoring of blood pressure, complete blood count, liver and renal function, ESR, and CRP, and continued hepatology surveillance for schistosomal cirrhosis. The short-term outcome was favorable, but the need for ongoing monitoring remained clear because both relapse of vasculitis and medication-related toxicity were plausible concerns in this complex clinical setting.

3. Discussion

This case is instructive because it brings together several features that can independently delay recognition of GCA and, in combination, create substantial diagnostic uncertainty. The early presentation was dominated by constitutional symptoms and persistent headache rather than an immediately recognizable cranial ischemic syndrome. In addition, the patient had a chronic hepatosplenic disorder that could easily distract attention toward infection, metabolic disturbance, or decompensated liver disease. Only later did the pattern become more suggestive of vasculitis, particularly after scalp

tenderness and unilateral ptosis emerged. The clinical lesson is that GCA should remain in the differential diagnosis of older adults with unexplained inflammatory headache syndromes even when the initial presentation appears nonspecific. This pattern is especially important in older patients whose symptoms fail to improve with empiric anti-infective treatment or whose investigations repeatedly reveal inflammation without a convincing infectious or structural explanation.

The ptosis deserves special emphasis. Although diplopia and ptosis have been described in GCA, they are uncommon in comparison with arteritic anterior ischemic optic neuropathy and therefore may not immediately trigger suspicion for vasculitic disease [8-10]. In this patient, the most plausible mechanism is ischemia affecting the oculomotor nerve or its vasa nervorum rather than a primary neuromuscular junction disorder[8,9]. That interpretation is supported by the absence of diurnal fluctuation, the lack of other convincing features of myasthenia gravis, the concomitant inflammatory syndrome, and the rapid improvement after glucocorticoid therapy. From a practical standpoint, isolated or predominant ptosis in an older patient with new headache and elevated inflammatory markers should prompt careful reconsideration of GCA, even if visual acuity is initially preserved.

A second important lesson concerns imaging strategy. Current EULAR-based practice places ultrasound at the center of the initial diagnostic evaluation for suspected cranial GCA, but the choice of imaging should always be informed by the likely vascular territory [2]. A negative temporal artery ultrasound does not close the diagnostic pathway, particularly when symptoms are systemic, the presentation is atypical, or extracranial large-vessel involvement is suspected. In this case, PET/CT proved decisive because it demonstrated widespread arterial inflammation in a pattern characteristic of large-vessel GCA[2,13]. This finding did more than confirm a tentative suspicion; it shifted the diagnostic momentum away from alternative explanations and supported immediate, targeted immunosuppressive therapy. In diagnostically ambiguous cases, PET/CT may therefore serve as a problem-solving modality rather than a merely confirmatory test. It is particularly valuable when biopsy is unavailable, delayed, or potentially less informative because the disease burden lies predominantly outside the cranial branches typically assessed by ultrasound.

The background of schistosomal cirrhosis added another layer of complexity. Chronic hepatosplenic schistosomiasis may be associated with persistent hepatic dysfunction, portal hypertensive changes, and abnormal laboratory parameters that complicate interpretation when a patient becomes acutely ill[7]. Moreover, hepatic abnormalities have occasionally been described in GCA itself, which can further blur the boundary between primary liver disease and systemic vasculitis[11]. In this patient, elevated inflammatory markers and deranged liver biochemistry could initially be attributed to infection, chronic liver disease, or both. However, cirrhosis could not explain the characteristic vascular FDG uptake pattern, the scalp tenderness, or the steroid-responsive improvement in ptosis. The case therefore illustrates the danger of premature diagnostic closure when a longstanding comorbidity seems to offer an easier explanation for a new clinical syndrome.

Treatment also required individualized judgment. High-dose glucocorticoids remain the cornerstone of remission induction in GCA and should be initiated promptly when ocular or other ischemic complications are a concern [1,2]. Even so, this case shows that successful treatment involves more than simply following a standard steroid protocol. The coexistence of cirrhosis and severe hypertension narrowed the therapeutic margin and increased the importance of close biochemical and clinical surveillance. The transient rise in liver enzymes after treatment initiation reinforced the need for multidisciplinary management. The later introduction of low-dose methotrexate as a glucocorticoid-sparing agent reflected an attempt to reduce relapse risk and cumulative steroid toxicity while recognizing that hepatotoxicity remained a relevant concern in a patient with pre-existing liver disease.

Finally, the early treatment response strengthened diagnostic confidence. Clinical and biochemical improvement after glucocorticoid therapy is not specific and cannot replace imaging or histologic evidence. Nonetheless, when a compatible symptom complex, characteristic PET/CT findings, and prompt steroid responsiveness occur together, the overall case for GCA becomes highly persuasive. A limitation of this report is the absence of histologic confirmation by temporal artery biopsy. However, contemporary practice increasingly accepts an imaging-supported diagnosis when biopsy is impractical or when the totality of evidence is compelling [2,3,13].

4. Conclusion

Giant cell arteritis can present with unilateral ptosis rather than overt visual loss, and diagnosis may be particularly challenging when chronic liver disease obscures the inflammatory background. This case demonstrates that a negative temporal artery ultrasound should not be used to exclude GCA when clinical suspicion remains high. In complex presentations, PET/CT can reveal large-vessel inflammation and provide the missing evidence needed to justify timely treatment. Early recognition, persistence in diagnostic reasoning, and individualized immunosuppressive management were central to the favorable short-term outcome in this patient.

Disclosure statement

The author declares no conflict of interest.

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