Review Article

Temporal artery biopsy for suspected giant cell arteritis: A mini review

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Giant cell arteritis (GCA) is a granulomatous inflammation involving medium and large vessels that can lead to serious clinical manifestations associated with tissue ischemia. Temporal artery biopsy (TAB) is currently the gold standard method for the diagnosis of GCA, with a specificity of 100% and a sensitivity of 77%. However, the false-negative rate for TAB ranges from 9% to 61%. False negatives may be related to the timing of biopsy, the length of specimen, and the existence of "skip lesions." We reviewed the relevant evidence for methods to improve the sensitivity and reduce the false-negative rate for TAB. To reduce the false-negative rate for TAB, it is recommended to perform TAB within 1 week of starting corticosteroid therapy. Although there is currently no consensus, we suggest that the temporal artery is cut to a length of 20–30 mm and to prepare serial pathological sections. It is necessary to attach great importance to patients suspected of having GCA, and complete TAB should be performed as soon as possible while starting corticosteroid therapy promptly. We also discuss the clinical value of non-invasive vascular imaging technologies, such as DUS, CTA, MRA, and 18F-FDG-PET/CT, as auxiliary methods for GCA diagnosis that could partially replace TAB.

Key words: Diagnostic methods, giant cell arteritis, temporal artery biopsy, vasculitis

Giant cell arteritis (GCA) is a type of autoimmune inflammation involving the medium and large vessels that leads to proliferation of the vascular intima, thickening of the vascular wall, narrowing and blocking of the lumen, and exacerbation of tissue ischemia. GCA is common in elderly people in Western countries. It is usually accompanied by headache, scalp tenderness, mastication discomfort, fatigue, anorexia, fever, weight loss, and other systemic symptoms.[1] GCA is considered to be a clinical emergency in ophthalmology and neurology. In prior studies, 14%–70% of GCA patients had eye involvement, which can lead to acute and rapidly progressive vision loss, and 20% of patients had partial or total permanent vision loss in one eye or both eyes.[2] Arteritic anterior ischemic optic neuropathy (A-AION) is the most common cause of visual dysfunction caused by GCA, accounting for 81.2% of cases of permanent vision loss due to GCA.[3,4] Beyond serious ocular manifestations, GCA-related stroke was reported in 2.7%–7.4% of cases, and it may be fatal in severe cases.[5,6]

In 1990, the American College of Rheumatology (ACR) published diagnostic criteria for GCA comprising the following five factors[7]: I, age at onset ≥50 years; II, onset of a new headache; III, temporal artery tenderness or reduced pulsation; IV, elevated erythrocyte sedimentation rate (ESR) (≥ 50 mm/h using the Westergren method); and V, histological evidence of necrotizing arteritis in the temporal artery with predominantly mononuclear cell infiltration or granulomatous inflammation usually with multinucleate giant cells. The diagnosis of GCA is made if patients satisfy at least three of these five criteria. In 2016, the ACR published updated diagnostic criteria for GCA and quantified the relative contribution of each criterion to the diagnosis.[8] Notably, the importance of histological evidence was not weakened.

It is reported that the clinical diagnostic sensitivity of the 1990 GCA classification diagnostic criteria is 93.5% and the specificity is 91.2%.[9] Some studies have shown that the diagnostic accuracy rate for GCA patients based on these criteria is just 51.4% without temporal artery biopsy (TAB), but the accuracy rate increased to 73% when combined with pathological diagnosis,[10] suggesting that TAB plays an important role in the diagnosis of GCA. The diagnosis of GCA using TAB is highly specific. The site of the temporal artery showing tenderness or containing nodules should be selected for biopsy. Positive pathological results can confirm the presence of GCA, but negative results cannot exclude its diagnosis.[11] In a recent meta-analysis, the sensitivity of TAB for the diagnosis of GCA was 77%.[12] In prior studies, the false-negative rate ranged from 9% to 61%.[13-15] False negatives may be due to the timing of biopsy, the length of the specimen, or the presence of so-called "skip lesions."[1]

In this review, we discuss the relevant evidence for methods to improve the sensitivity and reduce the false-negative rate, and we describe the clinical value of noninvasive vascular imaging technology as an auxiliary method for the diagnosis of GCA that can partially replace TAB.

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Histopathology of TAB

Although TAB is currently the gold standard method for the diagnosis of GCA,[11] there are no standards for the histopathology of GCA. The main diagnostic histopathological feature is angiogranulomatous inflammation comprising epithelioid cells, T lymphocytes, macrophages, and giant cells. Plasma cells, eosinophils, and fibroblasts can also be found. The presence of giant cells is not essential and, in fact, these cells are absent in 18%–50% of specimens.[16] Other features include narrowing and occlusion of the arterial lumen, intimal proliferation, rupture of the internal elastic layer, and lumen thrombosis [Fig. 1]. These pathological manifestations can be either continuous or segmental,[17] and segmental "skipping lesions" was observed in 12%–28% of cases.[16,17]

Timing of TAB

Early recognition and timely intervention of GCA are very important for the prevention of vision loss. For patients with suspected GCA, TAB should be performed as soon as possible to obtain biopsy samples.[18] Because A-AION usually leads to irreversible visual impairment, timely steroid treatment is valuable for preventing further visual deterioration, reducing disease recurrence, and reducing the incidence of GCA in the fellow eye. Biousse *et al*.[19] reported that ≥50% of untreated patients had visual impairment in the fellow eye shortly after the onset of visual impairments in the first eye. Beri *et al*.[20] reported that 19 of the 20 patients with bilateral A-AION showed involvement of both eyes at the first visit. In the other patient, the fellow eye developed A-AION symptoms within 2 days after starting systemic corticosteroid treatment. In addition, GCA patients without ocular involvement at the initial visit were followed up for many years, but no signs of A-AION were observed after starting corticosteroid therapy. These findings indicate that systemic corticosteroid therapy is effective for preventing A-AION. However, it takes some time for corticosteroids to block the development of arteritis in the

posterior ciliary artery. Therefore, some patients may develop A-AION a few days after starting corticosteroid therapy.

Chaudhuri reported that TAB remained positive for 32 months after starting corticosteroid therapy.[21] However, Allison reported that the positive rate using TAB decreased from 82% before corticosteroid therapy to 60% at 1 week after corticosteroid intervention; therefore, it was recommended to perform TAB within 1 week after starting corticosteroid therapy.[22] Narvaez reported that the positive rate using TAB was 78% after corticosteroid therapy for 2 weeks, 65% after corticosteroid therapy for 2–4 weeks, and only 40% after corticosteroid therapy for ≥4 weeks.[23] For patients with suspected GCA or A-AION, TAB should be performed as soon as possible in order to confirm the diagnosis and avoid delaying treatment, and corticosteroid treatment should not be delayed.[24]

Length of the specimen

Due to the segmental distribution of lesions in GCA, there is no consensus on the minimum length of the temporal artery that should be biopsied to achieve the best sensitivity.[1] In order to avoid negative results caused by "skip lesions," Murchison suggested that a fixed specimen length of 20 mm should be used as the minimum sample length.[25] Several studies have shown that the mean length of positive specimens was greater than that of negative specimens.[26,27] Oh reviewed 545 TAB specimens and found that, compared with vessel specimens <15 mm long, the positive rate for vessel specimens ≥15 mm long was 2.25 times greater, and the positive rate of TAB increased by 3.4% for each 1 mm increase in specimen length.[28] Breuer reported that the positive rate for TAB was 19% for vessel specimens ≤5 mm long, 71%–79% for lengths of 6–20 mm, and 89% for lengths >20 mm.[29] The British Society of Rheumatology (BSR) guidelines recommend an arterial biopsy length of 10 mm.[30] In the TABUL study, 7.3% of the specimens <10 mm long did not contain any
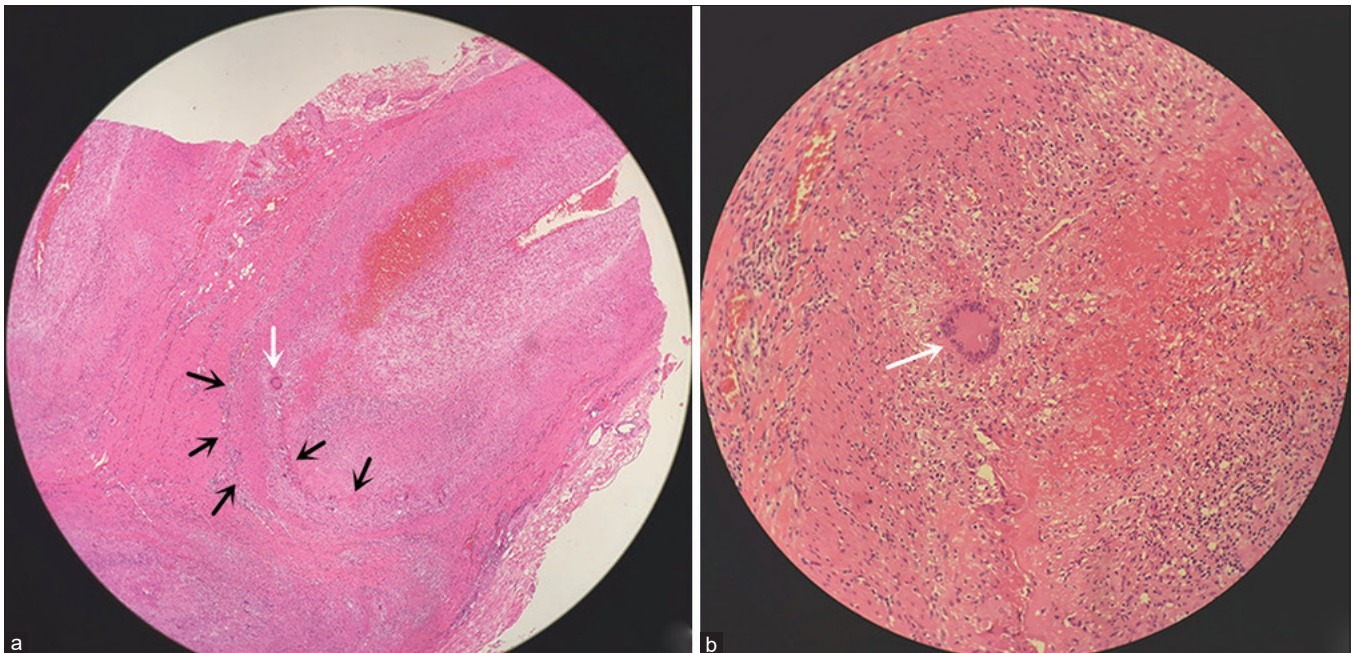
Figure 1: Histopathology of temporal artery showing infiltration of lymphocytes, plasma cells, and histiocytes (black arrow), and multinucleated giant cells (white arrow) (hematoxylin and eosin staining; original magnification, 40× in a and 200× in b)

arterial tissue, suggesting that shorter specimens and greater difficulty in obtaining a specimen increased the false-negative rate for pathological examination.[13] However, Mahr reported that a fixed specimen length of ≥5 mm could be sufficient for diagnosis.[31] Grossman reported that the specimen length was not related to the positive rate for TAB after comparing the lengths of specimens with different pathological results in GCA patients.[32] A study with temporal artery biopsies over an 11-year period from Mayo Clinic also indicated that biopsy length was uniformly noted to have no significant effect on biopsy positivity.[33] The authors explained that the reasons may be due to rich clinical experience and bilateral TAB to increase the positive rate.

Management strategies for TAB-negative patients

For patients with suspected GCA, unilateral TAB is recommended first. Although TAB is positive in most cases of cranial GCA, the negative rate for TAB is about 40% in patients with extracranial/large vessel GCA (LV-GCA).[34,35] Butendieck examines 603 patients with a bilateral biopsy for the diagnosis of GCA in Mayo Clinic and found 7% patients had a negative initial biopsy followed by a positive result on the contralateral side.[33] Mehta reviewed 310 patients who underwent bilateral TAB and observed a 12.1% discordance rate between pathology results.[36] The discordance rates in TAB specimens suggest that performing a bilateral TAB offers additional clinical benefit, such as increasing the positive rate of TAB and reducing the missed diagnosis rate of GCA. If the result of unilateral biopsy is negative, even after corticosteroid therapy, contralateral TAB should be performed to confirm the diagnosis.[18] Furthermore, for patients with a negative result for the initial TAB, no studies have evaluated the effects of steroid or immunosuppressive therapy on the results of subsequent TAB.

In an autopsy study of GCA patients, it was found that the temporal artery was not always involved and that other arteries may be involved.[37] In patients with negative results of bilateral TAB and there is a strong clinical indication for another biopsy, the temporal artery bifurcation or the posterior temporal branch are suitable sites. Other studies proposed biopsy of other branches of the external carotid artery, such as the occipital artery or maxillary artery, but these sites are more difficult to access and there is a risk of complications, especially nerve injury.[38]

Because of the seriousness of GCA, negative TAB reports should not change the clinical management if GCA is highly suspected based on the patient's clinical manifestations. Sait proposed a simple management plan based on the revised ACR 2016 criteria for GCA: (1) biopsy may not be required if the score does not exceed 2, as these patients are unlikely to have GCA; (2) TAB is not required in patients with a score of ≥5, as they are likely to have GCA and should continue corticosteroid therapy; and (3) biopsy is necessary for cases with a score of 3 and 4, as the results of TAB show the greatest variability in these patients.[39]

Possible alternatives to TAB

TAB is a traumatic procedure with a risk of complications such as parotid gland injury, surgical site hematoma, local infection or sepsis, and overlapping infection.[40] The utility of noninvasive diagnostic methods as alternatives to TAB remains widely debated.[41,42] Patients and many doctors have welcomed novel imaging techniques for GCA in clinical practice that avoid the risks associated with invasive interventions. The European League Against Rheumatism (EULAR) 2018 guidelines recommend that the diagnosis of GCA should be confirmed by imaging or histology.[43] Doppler ultrasound (DUS), computed tomography angiography (CTA), magnetic resonance angiography (MRA), and ^{18}F-deoxyglucose positron emission tomography/computed tomography (18F-FDG-PET/CT) are currently the most commonly used alternative methods.

DUS is recommended as a first-line method in the EULAR guidelines.[43] The typical manifestation of GCA observed on DUS is the halo sign, a low echo ring around the arterial lumen, depicting edematous thickening of the arterial wall owing to inflammation.[44] Segmental or diffuse thickening, stenosis, and occlusion of the vascular wall can also be detected by DUS [Fig. 2].[45] In a recent meta-analysis, the sensitivity and specificity of temporal artery DUS were 67% and 95%, respectively, based on the clinical diagnosis.[46] DUS can also be used to guide TAB in terms of determining the path of the temporal artery, the side of the branch that should be collected, and whether the involved side is unilateral or asymmetric. However, the interpretation of DUS is highly dependent on the radiologist's diagnostic skills because they need special training to provide accurate reports.[13] Moreover, the halo signals related to GCA can also be caused by significant atherosclerosis, leading to false-positive results.[47] Furthermore, there are some differences between countries and regions in terms of judging the effectiveness of DUS for the diagnosis of GCA.[36,42,48]
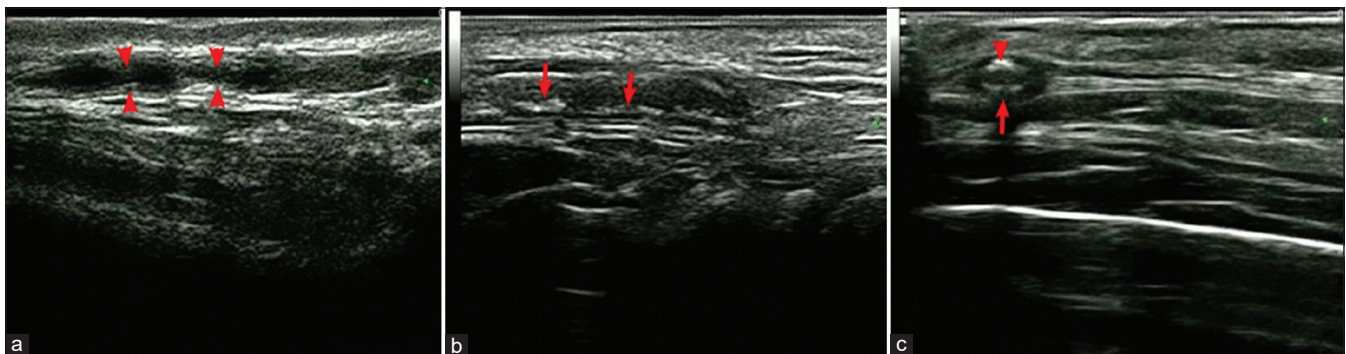
Figure 2: Superficial temporal artery ultrasound. (a) Segmental stenosis (arrows); (b) segmental thickening with calcification (arrows), and (c) hypoechoic halo (large arrow) with calcification (small arrow). (Cited from Chen Q, *et al.* Front Med (Lausanne) 2022.)
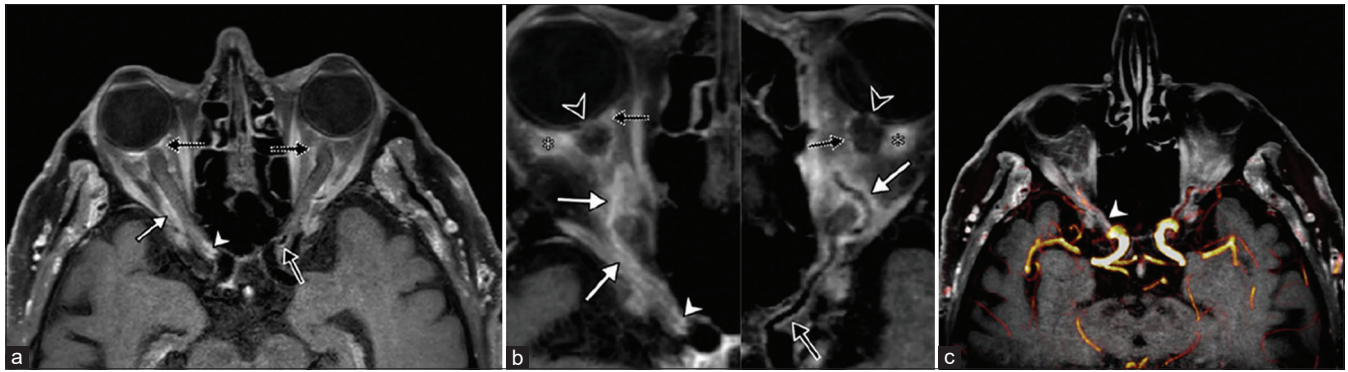
Figure 3: High-resolution vessel wall (HR-VW) imaging in a patient with bilateral A-AION. MRI reformatted in an axial plane (a) and in a curvilinear plane along the axis of each ophthalmic artery (b) showing marked wall thickening and strong mural enhancement with perivascular inflammatory infiltration (white arrow) in the whole course of the right artery, with severe involvement of its proximal part (white arrowhead), and in the left artery wall thickening and substantial mural enhancement (black arrow). MRI also shows bilateral enhancement of the optic disc (black arrowhead) and of the orbital fat (asterisk), as well as inflammatory changes of posterior ciliary arteries (dashed black arrows). Fused magnetic resonance angiography and HR-VW imaging (c) showing stenosis of the proximal part of the ophthalmic artery, due to the severe arteritis-related inflammatory changes (white arrowhead). (Cited from Mohammed-Brahim N, *et al.* Invest Radiol 2019)
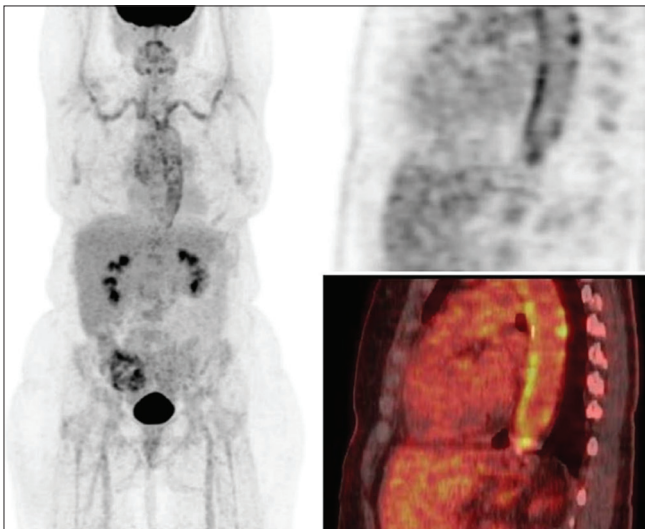
Figure 4: FDG-PET whole-body MIP image (left) and sagittal PET (upper right) and fused PET/CT (lower right) images of the aorta shows increased FDG uptake in the thoracic aorta, subclavian arteries and axillary arteries. FDG uptake is clearly higher than liver uptake and thus categorized as pathologic. Note nodular calcification in the aorta without FDG uptake. This finding together with the diffuse FDG uptake along the vascular wall also points to vasculitis rather than atherosclerosis as the underlying etiology for FDG uptake. (Cited from Emamifar A, *et al.* ACR Open Rheumatol 2020.)

Although magnetic resonance imaging (MRI) is mainly used to exclude differential diagnoses, such as compressive optic neuropathy, it has recently been used to identify the positive signs of GCA-related A-AION, for which typical features include circular vessel wall thickening and strong contrast enhancement of the vessel wall [Fig. 3].[49-51] Previous studies have determined the sensitivity (68%–89%) and specificity (73%–97%) of high-resolution MRI for the evaluation of cranial GCA.[52-55] Siemonsen used 3T MRI to study intracranial vessels and reported a medium sensitivity (50%) but high specificity (100%) for the diagnosis of GCA.[56] Mohammed-Brahim used 3T

high-resolution vascular wall MRI to accurately determine the inflammatory changes in the ophthalmic artery, and the sensitivity, specificity, positive predictive value, and negative predictive value were all 100%. That method was also more likely to detect posterior ciliary arteritis in patients with A-AOIN.[57] Sommer used high-resolution black blood MRI to evaluate posterior ciliary arteritis in GCA patients and reported sensitivity and specificity of 92.9% and 92.3%, respectively.[51] Other studies have reported that high-resolution MRA of the temporal artery is a reliable method for the diagnosis of cranial GCA, with a sensitivity of 88.7% and a specificity of 75.0%.[54,58] High-resolution vascular wall (HR-VW) MRI and extracranial artery MRI can also be used for the accurate diagnosis of GCA showing typical inflammatory changes.[54,59,60]

18F-FDG-PET/CT has been proposed to assist in the diagnosis of GCA. The high metabolic activity of inflammatory cells, such as macrophages and lymphocytes, causes high uptake of FDG at arterial inflammatory sites [Fig. 4].[61,62] Compared with TAB, the coincidence rate of 18F-FDG-PET/CT in GCA was 69.2%.[61] Extracranial vasculitis, such as arteritis, can also be evaluated by 18F-FDG-PET/CT, which is helpful to clarify the diagnosis of vasculitis.[63,64] However, it is often difficult to perform MRA or 18F-FDG-PET/CT in a timely manner in some hospitals, and the specificities of these imaging modalities are lower than that of TAB. In addition, the sensitivity rapidly decreases after only a few days of high dose steroid exposure.[65]

Dejaco expressed support for the recently published EULAR proposal for large vessel vasculitis imaging in clinical practice.[64] The authors suggested that GCA could be diagnosed without biopsy or further imaging in patients with high clinical suspicion of GCA and positive DUS results. PET and/or MRI were more valuable in patients with cranial GCA without head symptoms or signs. It is worth noting that EULAR emphasized the need for TAB if the clinical, laboratory, and imaging results could not confirm or exclude the presence of GCA. In addition, TAB remains the preferred diagnostic method if vascular imaging expertise is in doubt.[64]

Conclusion

In conclusion, TAB remains an important tool for the diagnosis of GCA. It is necessary to attach great importance to patients suspected of having GCA, and complete TAB should be performed as soon as possible while starting corticosteroid therapy promptly. To reduce the false-negative rate for TAB, it is recommended to perform TAB within 1 week of starting corticosteroid therapy. Although there is currently no consensus, when we consider that tissues contract during fixation, we suggest that the temporal artery is cut to a length of 20–30 mm and to prepare serial pathological sections. New imaging methods, particularly DUS, CTA, MRA, and 18F-FDG-PET/CT, can partially replace TAB to assist in the diagnosis of GCA.

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Conflicts of interest

There are no conflicts of interest.

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