The Impact of Temporal Artery Biopsy at a UK Tertiary Plastic Surgery Unit

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Background: Guidelines recommend temporal artery biopsy (TAB) for patients suspected of having giant cell arteritis (GCA). We evaluated the impact of TAB on the diagnosis and management of patients with suspected GCA at a tertiary plastic surgery unit.

Methods: A retrospective review of all TAB procedures performed at our centre over 7 years was performed. One hundred and one patients were included in the study. Patients were classified into 3 diagnostic groups: confirmed (positive TAB), presumed (negative TAB with high clinical suspicion) and unlikely (negative TAB with low clinical suspicion). The clinical presentation and management for each group were compared.

Results: The average American College of Rheumatology (ACR) score was 3.07. The number of patients with an ACR score of ≥3 before TAB was 72 (71.3%) and remained the same after TAB. The number of patients who remained on steroid therapy was lower in the group with an unlikely diagnosis of GCA compared to the group with a confirmed diagnosis (p<0.05). Conversely, there was no significant difference in steroid therapy between those with a presumed and confirmed diagnosis (p>0.05).

Conclusions: This study found a significant difference in steroid treatment between those with confirmed GCA and those where the diagnosis was unlikely showing that TAB may support decisions regarding steroid therapy. However, TAB was inappropriately requested for patients whose pre-TAB ACR score was ≥3 as this score is sufficient for the diagnosis of GCA. Therefore, the use of TAB should be limited to cases of diagnostic uncertainty. (Plast Reconstr Surg Glob Open 2019;7:e2541; doi: 10.1097/GOX.0000000000002541; Published online 28 November 2019.)

INTRODUCTION

Giant cell arteritis (GCA) is a medium- to large-sized vasculitis commonly affecting patients over 50 years old. The arteries typically affected in this disease are the carotid arteries and its branches such as the superficial temporal artery and central retinal artery.1-3 Inflammation of these blood vessels leads to the arterial stenosis and subsequently reduction in blood supply. Involvement of the central retinal artery is particularly dangerous as it can result in permanent vision loss and consequently, poor patient outcomes.1 Therefore, it is crucial that GCA is diagnosed early and treatment with corticosteroids started.

Histologically, GCA presents with transmural inflammation with mononuclear infiltrates and formation of giant cells within the affected vessel wall.1 These features can be identified using temporal artery biopsy (TAB) which is currently the standard for the diagnosis of GCA.5

According to the guidelines from American College of Rheumatology (ACR), British Society of Rheumatology (BSR), and British Health Professionals in Rheumatology (BHPR), high-dose steroids should be started and referral for TAB considered in all patients with a clinical suspicion of GCA.5-6 However, the biopsy should not delay the immediate initiation of steroid treatment where clinically indicated. Although it has previously been thought that steroid treatment before TAB can mask histological evidence of GCA,4 a large cohort study of 535 patients showed that these histological features of GCA were detectable despite more than 14 days of steroid treatment.5 BSR and BHPR guidelines also recommend continuing steroid treatment despite a negative TAB as long as typical clinical features are present and the response to steroids is positive.5 However, after the TAB procedure, if the diagnosis...
of GCA is ruled out, steroids should be tapered to stop within 2 weeks. ACR guidelines also suggest that GCA can be diagnosed if 3 out of 5 of the following criteria are met: (1) age of more than 50 years, (2) new-onset headache, (3) temporal artery abnormality on clinical examination, (4) erythrocyte sedimentation rate of 50 mm/h or higher, and (5) abnormal TAB. Therefore, if any 3 of these criteria are met before TAB, the patient should be treated with GCA. The sensitivity of TAB has a wide range of about 39%-90%, which suggests that a negative biopsy does not completely rule out a diagnosis of GCA. These indicate that the results from TAB are supportive but not necessary to the diagnosis of GCA and steroid treatment can be initiated irrespective of them.

TAB is also an invasive procedure with complications such as facial nerve injury, hematoma formation, and wound infection. Furthermore, the procedure is resource intensive as it requires referral to a surgical unit experienced in performing regular TABs such as our tertiary plastic surgery unit. There is also increasing evidence on the use of less invasive and cost-effective diagnostic tools for GCA such as duplex ultrasound and Magnetic Resonance Imaging (MRI) and Position Emission Tomography-Computed Tomography (PET-CT) scan. Hence, this study aims to assess the diagnostic significance of TAB and its influence on the management of patients with suspected GCA.

METHODS

We retrospectively reviewed all TAB procedures over 7 years from March 2012 to February 2019 that were performed at a UK tertiary plastic surgery unit. The inclusion criteria were all patients who underwent TAB regardless of their demographics, and there were no exclusion criteria. In total, 141 patients were identified as having a TAB procedure at our center. Forty patients had insufficient data, and thus only 101 patients were included in the data collection and analysis.

Data were collected regarding the patient’s age, sex, ACR criteria score, site of TAB, length of TAB specimen, histopathology results of the TAB specimen, duration of steroid treatment before and after TAB procedure, the influence of TAB result on diagnosis, and status of steroid management at follow-up. The diagnosis of GCA was categorized into confirmed, presumed, and unlikely based on the rheumatologist’s decision which was influenced by clinical manifestations and TAB result. Typically, patients will be classified as confirmed if TAB is positive, presumed if clinical suspicion for GCA is high (based on ACR criteria) but TAB was negative, and unlikely if clinical suspicion is low and TAB result was negative for GCA. The duration of each procedure, type of anesthesia used, and any complications of the surgery were also looked at. Kolmogorov–Smirnov test was used to determine the distribution of each data, and the appropriate parametric and nonparametric statistical tests were used with a P value of <0.05 to be considered significant.

RESULTS

The mean follow-up period was 5.4 weeks (SD 3.0). Of the 101 patients, the mean age was 68.3 years (SD 9.4). Seventy-nine (78.2%) patients were women, and 22 (21.8%) patients were men. The mean pre-TAB ACR criteria score was 2.87 (SD 0.74) out of 4, and the mean post-TAB ACR score was 3.07 (SD 0.96) out of 5. The number of patients with an ACR score of ≥3 was the same before and after TAB for 72 patients (71.3%). Of the 72 patients with a pre-TAB ACR score of ≥3, 20 (27.8%) went on to have a positive TAB result and 52 (72.2%) had a negative TAB. The summary of the ACR criteria scores is listed in Table 1.

The median number of days from referral to TAB procedure is 5 days (range: 1–22 days). Twenty patients (19.8%) were found to be positive for GCA on TAB, 78 (77.2%) were negative, and 3 (3.0%) were inconclusive due to a vein being sampled (n = 1) and inadequate samples (n = 2). Regarding the laterality of TAB, 44 (43.6%) patients had right-sided TAB and 57 (56.4%) had left-sided TAB. The mean TAB length was 11.0 mm (SD 5.6). All of the TAB procedures were performed under local anesthesia with a mean procedure duration of 31.8 minutes (SD 13.1). There was only 1 surgical complication (0.99%) which was the unintended biopsy of a vein.

Ninety-five (94.1%) patients were started on prednisolone before the TAB procedure whereas 6 patients (5.9%) were not due to low clinical suspicion. Of these 95 patients, 74 had ongoing prednisolone therapy at follow-up and 21 patients were tapered and stopped on prednisolone due to negative TAB result. Three of the 6 patients who were not initially started on prednisolone were then started on prednisolone after TAB due to a change in diagnosis to systemic vasculitis after exclusion with TAB (n = 1), a presumed diagnosis of GCA (n = 1), and a positive TAB result (n = 1). In summary, at follow-up, 77 patients (76.2%) had ongoing prednisolone therapy, 21 (23.8%) had their steroid treatment stopped, and 3 patients (3.0%) did not start steroids at all. At follow-up, 19 patients (18.8%) developed a total of 24 side effects from steroid therapy which is listed in Table 2.

The comparison of demographic data, ACR score, time taken to TAB, TAB length, and prednisolone therapy between those with positive and negative TAB is summarized in Table 3. Of note, we found that the mean ACR score before TAB was higher in the positive TAB group compared to those with a negative TAB (3.4 versus 2.8, P < 0.001). There was no difference between the mean TAB length of

| TABLE 1. Summary of Number of Patients with Each ACR Criteria Score Including Total Number of Patients with a Score of ≥3 before and after TAB Procedure |
|--------------------------|--------------------------|
| ACR Score | No. Patients |
| ≥3 | 72 (71.3%) |
| ≥2 | 26 (25.7%) |
| ≥1 | 3 (3.0%) |
| 4 | 25 (24.8%) |
| 3 | 41 (40.6%) |
| 2 | 26 (25.7%) |
| 1 | 6 (5.9%) |

| TABLE 2. Summary of Side Effects Due to Prednisolone Therapy |
|--------------------------|--------------------------|
| Side Effect | No. Patients |
| Osteoporosis | 3 (3.0%) |
| Gastrointestinal | 11 (10.9%) |
| Depression | 8 (7.9%) |
| Hypertension | 11 (10.9%) |
| Insomnia | 4 (4.0%) |
| Glaucoma | 2 (2.0%) |

| TABLE 3. Comparison of Demographic Data, ACR Score, Time Taken to TAB, TAB Length, and Prednisolone Therapy |
|--------------------------|--------------------------|
| ACR Score | Time to TAB (days) | TAB Length (mm) | Prednisolone Therapy |
| ≥3 | 4.7 (SD 4.1) | 11.0 (SD 5.6) | Ongoing (77.2%), Stopped (21.8%), Not Started (1.0%) |
| ≥2 | 5.2 (SD 4.0) | 10.5 (SD 5.4) | Ongoing (76.2%), Stopped (23.8%), Not Started (0.9%) |
both groups ($P = 0.854$). Furthermore, we found no difference between the mean dose of steroid therapy before TAB between the 2 groups ($P = 0.084$). On the other hand, after TAB, the mean dose of steroid therapy was higher in the group that had a positive TAB compared to those who had a negative TAB (40.8 versus 17.0 mg, $P < 0.001$).

The diagnosis of GCA was confirmed in 20 patients (19.8%), all of whom had positive TAB. Of those who had negative TAB, GCA was presumed in 26 patients (25.7%) and was unlikely in 55 patients (54.5%). Table 4 summarizes the comparison of ACR scores and change in prednisolone therapy of these 3 different diagnosis groups to show how TAB influences diagnosis and management. It was found that there was no difference in the percentage of patients who had a pre-TAB ACR score of ≥3 between the confirmed diagnosis group and presumed diagnosis group ($P = 0.021$). However, a higher percentage of patients had a pre-TAB ACR score of ≥3 in the group with a confirmed diagnosis compared to those with an unlikely diagnosis ($P = 0.001$). Regarding steroid therapy, there was no significant difference between steroid therapy ($P = 0.373$). In comparison, it was found that a higher percentage of patients remained on ongoing steroid therapy in the confirmed group compared to the unlikely group (100% versus 58.2%, $P = 0.001$).

**DISCUSSION**

The number of patients with positive TAB biopsy in our cohort is low (19.8%) but consistent with other similar studies which also reported a relatively low pickup rate with TAB. Various reasons for the low positive rates have been hypothesized including treatment with corticosteroids before TAB procedure, the length of biopsy specimen, and skip lesions which are a feature of GCA.

The wide range of clinical manifestations of GCA, as well as the high index of suspicion of clinicians, may result in an overreferral for TAB. Recognizing the variety in clinical presentations, previous authors have attempted to identify clinical features and laboratory results which have the strongest correlation with a positive TAB to guide decision-making regarding biopsy and diagnosis. A prospective cohort study of 251 patients studied over 3 years showed that the clinical features most predictive of a positive TAB result were diplopia, jaw claudication, and abnormal temporal artery pulse. Interestingly, the same symptoms of jaw claudication, neck stiffness, temporal cutaneous hyperalgesia, and new-onset headache were present more frequently among patients with positive temporal biopsy compared to those with a negative result.

The mean ACR score in this study is 3.07 which meets the ACR guidelines for GCA diagnosis which is a score of 3 or more. In fact, 72 (71.3%) patients had a total ACR score of 3 or more before TAB and therefore, by ACR guidelines, would have been sufficient for a diagnosis of GCA and did not need a referral for TAB to warrant corticosteroid treatment. A previous study evaluating TAB showed similar percentage of cases with an ACR score of ≥3 before undergoing biopsy. Interestingly, the number of patients in our study with an ACR score of ≥3 after TAB procedure did not change as the 20 patients who had a positive TAB result already had a score of ≥3.

Our median of 5 days from the time of referral to TAB is less than 6 weeks which is how long a TAB can remain positive for according to BSR and BHPR guidelines. The guidelines also recommend that biopsy samples should be at least 10 mm which the mean in our cohort has met (11.0 mm). Specimens as short as 3 mm were found in our cohort and may explain the low incidence of positive TABs. However, in our comparison between those with positive and negative TABs, no significant difference in the mean length of TABs was found ($P = 0.854$).

Ideally, TAB should be performed before any steroid therapy is initiated. But due to the delays that can happen in arranging the surgery, it is recommended that high-dose steroid therapy should be promptly initiated where there is clinical suspicion for GCA as it is unlikely to affect the biopsy result. If the TAB result is negative, steroid management can be stopped, ideally within 2 weeks. In our cohort, only 21 patients out of 78 (26.9%) who had negative TAB were stopped on steroid therapy at follow-up. Bowling et al also reported a very low percentage (7.8%) of their TAB-negative patients who were stopped on steroids at 6-week follow-up. However, the limitation of this study is that the TAB results were not studied in context with the clinical presentation as we have done by classifying patients into the different diagnostic groups. In fact, BSR and BHPR guidelines recommend that patients with a high clinical suspicion of GCA but negative TAB should be treated as biopsy-positive GCA. Therefore, it is prudent to consider both the clinical manifestations and TAB result in making a diagnosis of GCA. Our study is the first to look at different groups of diagnosis given to patients at follow-up and subsequently compare steroid management between these groups. We found no significant difference between patients with a presumed diagnosis and those with a confirmed diagnosis ($P = 0.573$). This is consistent with the guidelines that suggests that these 2 groups should be treated the same. Conversely, a significantly higher percentage of patients in the unlikely diagnosis group had stopped steroid therapy compared to those who had a confirmed diagnosis ($P = 0.002$). Nonetheless, more than half (58.2%) of patients in this group were still on steroid therapy at follow-up. This is in

**TABLE 2. List of All Steroid Side Effects Developed by 19 Patients at Follow-up**

| Side Effects from Steroid Therapy | No. Patients |
|----------------------------------|-------------|
| Diabetes worsened                | 5 (5.0%)    |
| Sleep disturbance                | 4 (4.0%)    |
| Edema                            | 4 (4.0%)    |
| Cushingoid features              | 3 (3.0%)    |
| Dizziness                        | 3 (2.0%)    |
| Mood disorder                    | 2 (2.0%)    |
| Myalgia                          | 1 (1.0%)    |
| Restless legs                    | 1 (1.0%)    |
| Paresthesia                      | 1 (1.0%)    |
| Bruising                         | 1 (1.0%)    |
| **Total**                        | **24 (23.8%)** |
TABLE 3. Comparison of Demographic Data, ACR Criteria Score before TAB, Median Time to TAB, Mean TAB Length, and Prednisolone Therapy between Patients with Positive and Negative TAB

|                          | Positive (n = 20) | Negative (n = 78) | P     |
|--------------------------|------------------|-------------------|-------|
| Mean age                 | 73.5 ± 7.2       | 67.2 ± 9.4        | 0.007 |
| Sex ratio                | 3.1              | 3.1               | 0.665 |
| Mean ACR (pre-TAB) score | 3.4 ± 0.5        | 2.8 ± 0.7         | <0.001|
| Median time to TAB       | 3.5              | 5                 | 0.269 |
| Mean TAB length          | 10.8 ± 4.3       | 11.2 ± 5.9        | 0.854 |
| Median pre-TAB duration of prednisolone therapy | 7.0 | 6.5 | 0.913 |
| Mean pre-TAB dose of prednisolone (mg) | 48.4 ± 15.7 | 41.0 ± 16.7 | 0.084 |
| Mean post-TAB dose of prednisolone (mg) | 40.8 ± 12.2 | 17.0 ± 17.2 | <0.001|

TABLE 4. Comparison of the Number of Patients with an ACR Score of ≥3 and the Changes in Prednisolone Therapy between Patients Who had a Confirmed Diagnosis of GCA, Those Whose Diagnosis Was Presumed or Unlikely, and Patients Whose Diagnosis Was Excluded Entirely

|                          | Confirmed (n = 20) | Presumed (n = 26) | Unlikely (n = 55) |
|--------------------------|--------------------|-------------------|-------------------|
| ACR score ≥3             | 20 (100%)          | 20 (76.9%)        | 32 (58.2%)        |
| Ongoing                  | 20 (100%)          | 25 (96.2%)        | 32 (58.2%)        |
| Stopped                  | 0 (0%)             | 1 (3.8%)          | 20 (36.4%)        |
| Not started              | 0 (0%)             | 0 (0%)            | 3 (5.5%)          |

*P-value significant to <0.05 using z-score calculator in comparison with “confirmed” group.

contrast to the BSR and BHPR guidelines that recommend stopping steroid therapy within 2 weeks after the diagnosis of GCA is excluded. The high number of patients still on steroid therapy could be explained by a longer steroid weaning regimen or a shorter follow-up period. In fact, the mean dose of steroid treatment was significantly lower in the group with a negative TAB compared to those who had positive biopsy. Being on unnecessary steroid management may have implications in the form of side effects from the steroid therapy such as those identified in 19 patients (18.8%) in this study. However, a limitation to this result is the retrospective nature of the study and the subjectivity of steroid side effects.

Some of the complications of TAB include hematoma, scalp necrosis, wound infection, facial nerve damage, ptosis, and unintended biopsies of veins and nerves.4,23 However, these complications are very rare, and it is a very simple and safe procedure. Only 1 patient out of 101 (0.99%) in our study had a vein biopsied instead of an artery, and therefore, had an inconclusive TAB result. None of the patients suffered any other complications from the procedure. Also, all procedures were performed under local anesthesia which avoids the risks associated with general anesthesia.

Recognizing that TAB is still an invasive procedure, the evidence base for using imaging modalities to diagnose TAB is ever increasing. For example, ultrasound can pick up vessel wall inflammation by detecting the appearance of a “halo sign” caused by hypoechoic region in the area where the intima–media complex is usually found.23,25 Furthermore, color Doppler ultrasound is able to provide more information regarding vessel abnormalities such as stenosis, occlusion, and compressibility of the vessel wall.25,26 It was interesting to note that none of our patients had any imaging modalities before TAB. Previous studies showed a wide range of sensitivities when using Doppler ultrasound to diagnose GCA ranging from 10% to 82% and specificities ranging from 78% to 100%.13,27-20 A prospective multicenter cohort study (TABUL study) involving 381 patients found that, when comparing ultrasound against TAB for the diagnosis of GCA, ultrasound had a higher sensitivity (54% versus 39%) compared to TAB but a lower specificity (81% versus 100%). Furthermore, the study showed that the cost of TAB is almost 9 times the cost of ultrasound (£514 versus £58) and the incremental net benefit for using ultrasound was £485 per patient if clinical judgment strategy was factored in.10 Other diagnostic benefits of using color Doppler ultrasound include having a high resolution and therefore being able to identify vessels as small as 1 mm, and it can be used to image longer segments of arteries as well as other vascular areas. From the patient’s point of view, ultrasound is well tolerated, is able to be performed at the bedside, and is noninvasive with no complications.23 On the other hand, one of the biggest disadvantages of using ultrasound is that it is operator dependent which could explain the wide range of sensitivities among the different studies.25

18F-Fluorodeoxyglucose (18F-FDG) Positron Emission Tomography scan has shown that it can be useful in supporting the diagnosis of GCA, especially in cases with large vessel involvement (large vessel giant cell arteritis, LV-GCA) as it has been shown that the positive rates of TAB are lower in this population.9,11 18F-FDG accumulates in foci of inflammation in arteries which is one of the first pathological processes that occur in large vessel vasculitis and thus allows early diagnosis. However, for this very reason, the diagnostic performance of 18F-FDG-PET declines very quickly after the use of corticosteroids or other immunosuppressive treatment. It is also more expensive and not readily available at all centers.21

Also, shown to be useful in the diagnosis of GCA involving large vessels is CT and MRI angiography. Both these imaging modalities have shown to be useful in identifying aortic involvement in GCA.34,25 This is particularly important as it has been shown that aortitis can lead to severe complications such as aortic aneurysm, rupture,
dissection, and aortic valve incompetence. A population-based study showed that patients with GCA are 17.3 times more likely to develop thoracic aortic aneurysm compared to age-matched controls. The disadvantages of CT angiography are the exposure to radiation and its limited use in patients with poor renal function and an allergy to contrast. Although MRI angiography does not expose patients to radiation, it still requires the use of contrast and is also contraindicated in patients with metal implants.

Although there is a growing evidence base for new imaging modalities, it is still not routinely used at our center. Therefore, as a retrospective study, we are still unable to compare these imaging modalities with the current standard that is TAB. Future studies such as a randomized controlled trial will be able to provide more information on which diagnostic tool is more effective.

CONCLUSIONS

This study shows that TAB can support the diagnosis and management of GCA when combined with clinical assessment. However, a large percentage of patients were inappropriately subjected to TAB as they had an initial ACR score of ≥3 before TAB. Despite being a simple procedure, TAB can be resource intensive for the plastic surgery department. Therefore, we believe that although TAB still has an important role to support clinicians in the diagnosis and management of GCA, its use should be limited to patients with diagnostic uncertainty rather than requested for every patient with a suspicion of GCA.

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REFERENCES

1. Patil P, Karia N, Jain S, et al. Giant cell arteritis: a review. Eye. 2015;3:25–33.
2. Weyand CM, Goronzy JJ. Giant-cell arteritis and polymyalgia rheumatica. Ann Intern Med. 2003;139:505–515.
3. Maleszewski JJ, Younge BR, Fritzlen JT, et al. Clinical and pathological evolution of giant cell arteritis: a prospective study of follow-up temporal artery biopsies in 40 treated patients. Mod Pathol. 2017;30:788–796.
4. Winkler A, Wudel J. Temporal artery biopsy. Medscape Web site. https://emedicine.medscape.com/article/1520091-overview. Updated 2018. Accessed March 19, 2019.
5. Hunder GG, Bloch DA, Michel BA, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum. 1990;33:1122–1128.
6. on behalf of the BSR and BHPR Standards, Guidelines and Audit Working Group, on behalf of the BSR and BHPR Standards, Guidelines and Audit Working Group, on behalf of the BSR and BHPR Standards, on behalf of the BSR and BHPR Standards.
7. Allison MC, Gallagher PJ. Temporal artery biopsy and corticosteroid treatment. Ann Rheum Dis. 1984;43:416–417.
8. Achkar AA, Lie JT, Hunder GG, et al. How does previous corticosteroid treatment affect the biopsy findings in giant cell (temporal) arteritis? Ann Intern Med. 1994;120:987–992.
9. Borchers AT, Gershwin ME. Giant cell arteritis: a review of classification, pathophysiology, geoepidemiology and treatment. Autoimmun Rev. 2012;11:A54–A554. Available at: http://www.sciencedirect.com/science/article/pii/S1568997212000880. Accessed March 19, 2019.
10. Luqmari R, Lee E, Singh S, et al. The role of ultrasound compared to biopsy of temporal arteries in the diagnosis and treatment of giant cell arteritis (TABUL): a diagnostic accuracy and cost-effectiveness study. Health Technol Assess. 2016;20:1–238.
11. Ringel M, Chatelus E, Jousse-Joulin S, et al. Diagnostic performance of temporal artery ultrasound for the diagnosis of giant cell arteritis: a systematic review and meta-analysis of the literature. Autoimmun Rev. 2019;18:56–61. Available at: http://www.sciencedirect.com/science/article/pii/S1568997218302611. Accessed March 19, 2019.
12. Schmidt WA. Ultrasound in the diagnosis and management of giant cell arteritis. Rheumatology (Oxford). 2018;57:ii2–ii31. Available at: https://academic.oup.com/rheumatology/article/57/suppl_2/ii2/4898141. Accessed March 19, 2019.
13. Arida A, Kyprianou M, Kanakis M, et al. The diagnostic value of ultrasonography-derived edema of the temporal artery wall in giant cell arteritis: a second meta-analysis. BMC Musculoskelet Disord. 2010;11:44.
14. Chong EW, Robertson AJ. Is temporal artery biopsy a worthwhile procedure? ANZ J Surg. 2005;75:388–391.
15. Davies C, Frost B, Eshan O, et al. Temporal artery biopsy who needs one? Postgrad Med J. 2006;82:476–478.
16. Kaltsoonoudis E, Pelechas E, Papoudou-Bai A, et al. The impact of temporal artery biopsy for the diagnosis of giant cell arteritis in clinical practice in a tertiary university hospital. PloS One. 2019;14:e0210845.
17. Leeborn J, Donnelly R, Nash JR. Does temporal artery biopsy influence the management of temporal arteritis? QJM. 2006;99:33–36.
18. Younge BR, Cook BE Jr, Bartley GB, et al. Initiation of glucocorticoid therapy before or after temporal artery biopsy? Mayo Clin Proc. 2004;79:483–491.
19. Rieck KL, Kermani TA, Thomsen KM, et al. Evaluation for clinical predictors of positive temporal artery biopsy in giant cell arteritis. J Oral Maxillofac Surg. 2011;69:36–40. Available at: http://www.sciencedirect.com/science/article/pii/S0278239110002570. Accessed March 19, 2019.
20. Toren A, Weis E, Patel V, et al. Clinical predictors of positive temporal artery biopsy. Can J Ophthalmol. 2016;51:476–481. Available at: https://www.sciencedirect.com/science/article/pii/S1568997217302318. Accessed March 19, 2019.
21. González-López JJ, González-Moraleja J, Burdasal-Moratilla A, et al. Factors associated to temporal artery biopsy result in suspects of giant cell arteritis: a retrospective, multicenter, case-control study. Acta Ophthalmol. 2013;91:763–768.
22. Bowling K, Rait J, Atkinson J, et al. Temporal artery biopsy in the diagnosis of giant cell arteritis: does the end justify the means? Ann Med Surg (Lond). 2017;20:1–5.
23. Davies CG, May DJ. The role of temporal artery biopsies in giant cell arteritis. Ann R Coll Surg Engl. 2011;93:4–5.
24. Muratore F, Pipitone N, Salvarani C, et al. Imaging of vasculitis: state of the art. *Best Pract Res Clin Rheumatol*. 2016;30:688–706.

25. Berger CT, Sommer G, Aschwanden M, et al. The clinical benefit of imaging in the diagnosis and treatment of giant cell arteritis. *Swiss Med Wkly*. 2018;148:w14661.

26. Aschwanden M, Daikeler T, Kesten F, et al. Temporal artery compression sign—a novel ultrasound finding for the diagnosis of giant cell arteritis. *Ultraschall Med*. 2013;34:47–50.

27. Ball EL, Walsh SR, Tang TY, et al. Role of ultrasonography in the diagnosis of temporal arteritis. *Br J Surg*. 2010;97:1765–1771.

28. Maldini C, Dépinay-Dhellemmes C, Tra TT, et al. Limited value of temporal artery ultrasonography examinations for diagnosis of giant cell arteritis: analysis of 77 subjects. *J Rheumatol*. 2010;37:2326–2330.

29. Monti S, Floris A, Ponte CB, et al. The proposed role of ultrasound in the management of giant cell arteritis in routine clinical practice. *Rheumatology (Oxford)*. 2018;57:112–119.

30. Puppo C, Massolo M, Paparo F, et al. Giant cell arteritis: a systematic review of the qualitative and semiquantitative methods to assess vasculitis with 18F-fluorodeoxyglucose positron emission tomography. *Biomed Res Int*. 2014:2014:574248.

31. Salvarani C, Soriano A, Muratore F, et al. Is PET/CT essential in the diagnosis and follow-up of temporal arteritis? *Autoimmun Rev*. 2017;16:1125–1130. Available at: http://www.sciencedirect.com/science/article/pii/S1568997217302318. Accessed March 19, 2019.

32. Evans JM, O’Fallon WM, Hunder GG. Increased incidence of aortic aneurysm and dissection in giant cell (temporal) arteritis. A population-based study. *Ann Intern Med*. 1995;122:502–507.