# Visual Manifestations of Giant Cell Arteritis

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## Abstract

### Background

The frequency of visual loss in GCA has been variably reported in literature between 12% to 70%. We present the largest study of the frequency and nature of visual complications in a cohort of 350 consecutively diagnosed people with GCA.

### Methods

All individuals suspected to have GCA are referred to our service. They are assessed using structured forms and diagnosed using ~~either~~ ultrasonography, biopsy or PET scan. ~~Data from~~ Our ~~service~~ database was analysed to study frequency and cause of visual loss. Clinical data were~~as~~ analysed for predicting visual loss in a binary logistic regression model.

### Results

Visual symptoms ~~including diplopia and blurring or loss of vision~~ occurred in 101 (29%). Visual loss in one or both eyes occurred in 48 (14%). 4 had binocular visual loss. Anterior ischaemic optic neuropathy (N=31), Retinal artery obstruction (N=8), Occipital stroke (N=2) were the main causes of visual loss. Of the 47 individuals who had repeat visual acuity testing at 7 days, 3 individuals had improvement to 6/9 or better. After the introduction of the fast-track pathway, the frequency of visual loss fell from 19% to 12%. Age at diagnosis (OR 1.12) and Headache (OR 0.22) were significant determinant of visual loss in a multivariate model. Jaw claudication trended to significance (OR 1.96, p=0.054)

### Conclusions

We report the frequency of visual loss in the largest cohort of GCA from a single centre to be 14%. Improvement in vision is rare. A dedicated fast-track pathway reduces visual loss. Age is a poor prognostic marker~~. and having a H~~headache may result in earlier diagnosis and protect against visual loss.

## Introduction

Giant cell arteritis (GCA) is large vessel vasculitis that preferentially affects people of Northern European ancestry, peaking in the 8th decade of life. It has a predilection for branches of the external carotid and/or subclavian artery. However, one of its most feared complications is visual loss which is presumed to be due to direct involvement of the

ophthalmic artery which is usually a branch of the internal carotid artery. The vascular supply of the eye is mainly from two sources, the posterior ciliary arteries, and the central retinal artery. The posterior ciliary arteries supply the optic nerve head, the choroid up to the equator, the retinal pigmented epithelium, and the outer 130 μm of the retina. Their involvement can cause anterior ischaemic optic neuropathy (AION). The central retinal artery runs inside the optic nerve and its blockage presents as central retinal artery obstruction (CRAO). In GCA, these two ocular syndromes can cause permanent visual loss. Posterior circulation strokes because of involvement of the vertebral artery, a branch of the subclavian artery can result in hemifield loss due to involvement of the occipital cortex. Double vision has also been reported commonly in GCA, but it is not clear whether this is because of involvement of the vasculature of the extraocular muscles or the cranial nerves.

Visual loss in GCA has been reported to be between 12% to 70% (1-9). It can be postulated that this wide variation in the data could be in part due to the specialty reporting this data - 12%-16% from internal medicine (4, 7), 14%-26% from rheumatology (5, 9), 20% from neurology (1), 48-70% from ophthalmology (2, 3, 6, 8).

At our centre we run an interdisciplinary fast track service for the diagnosis and management of individuals with suspected GCA. The details of this service are published elsewhere (10). Briefly, referrals are triaged from primary care or internal medicine, and managed jointly by ophthalmology and rheumatology. The patient pathway takes every individual with suspected GCA through a validated ultrasonography service (11). A second test is arranged in those individuals where the pre-test probability remains high after a negative or equivocal result as per international recommendations (12). Our data is likely to be more representative of the entire spectrum of presentations of GCA.

## Methods

### Setting

A tertiary interdisciplinary vasculitis service in a large predominantly rural county.

### Participants

Consecutively diagnosed individuals with GCA diagnosed by ultrasonography, temporal artery biopsy or positron emission tomography. The presence of inflammatory infiltrate in at least one layer of the temporal artery biopsy is accepted as a positive biopsy in the correct clinical context. The presence of a non-compressible halo sign in at least two arteries is accepted as a positive ultrasonography result in the appropriate clinical context. The halo sign is defined as per internationally accepted consensus (13). The PET scan is accepted to be positive when there appears to be greater 18-fluorodeoxyglucose uptake in an artery than in the liver.

### Data collection

The National Health Service in England has commissioned select centres in England for delivery of specialised care including that of GCA (14). As part of this programme, our centre is required to maintain a secure database to allow returns to the health service of nationally benchmarked metrics. Data entry is done prospectively at time of diagnosis

and every follow-up. For patients with GCA, the recorded dataset was revised after the publication of EULAR recommendations for a minimum core dataset (15).

### Outcomes of interest

The primary outcome of interest was the frequency of permanent visual loss in GCA. Other outcomes of interest were frequency of visual symptoms, the ocular syndrome leading to permanent visual loss, the visual acuities at baseline and 7 days, the effect of a fast-track pathway on incidence of visual loss, and the value of the recorded symptoms, clinical signs, and laboratory markers to predict permanent visual loss.

### Statistics

All statistics were done using SPSS 28 (IBM, Armonk, New York, USA). Continuous variables were tested for normality using the Shapiro-Wilke test. The normally distributed variables were compared across the two groups (with and without visual loss) using the independent samples t test. The non-parametric variables were compared using the Mann-Whitney U test. The effect of the categorical variables in visual loss was calculated using the Chi-squared test or the Fisher's exact test as appropriate. All the variables that were found to have a P value of >0.2 were included in a binary logistic regression using Block Entry method. The categorical variables were denoted using simple contrast with the absence of the variable being the reference.

## Results

350 patients were diagnosed with GCA between 2012 and 2021. The mean (standard deviation) age was 74.6 (7.8). 236 (67.4%) were female. The mean Haemoglobin was 121.1 (14.4) g/dL. The median (IQR) Erythrocyte sedimentation was 63 (44) mm. The median (IQR) C-reactive protein was 65 (77) mg/dL.

#### Visual symptoms and signs

101 (28.9%) had visual symptoms. 27 had diplopia, 80 had blurring or loss of vision. Visual acuity had been recorded for 89 of the individuals with visual symptoms. The visual acuities at baseline are as in Table 1. Of the 80 who complained of blurred vision, 48 (13.7% of cohort) individuals had objective permanent visual loss. The causes of visual loss are as in Table 2.

**Table 1 Visual acuity in 89 of 101 individuals with visual symptoms**

| | Right eye (N) | Left eye (N) |
|---|---|---|
| Better than 6/6 | 6 | 3 |
| 6/6 to 6/7.5 | 16 | 21 |
| 6/7.5 to 6/9 | 34 | 21 |
| 6/9 to 6/12 | 7 | 14 |
| 6/12 to 6/15 | 0 | 0 |

| | | |
|---|---|---|
| 6/15 to 6/18 | 4 | 4 |
| 6/18 to 6/24 | 3 | 2 |
| 6/24 to 6/36 | 2 | 3 |
| 6/48 to 6/60 | 2 | 2 |
| Worse than 6/60 | 15 | 19 |

**Table 2 Aetiology of visual loss in 48 individuals**

| Ocular syndrome | N = 48 |
|---|---|
| Anterior Ischaemic optic neuropathy | 31 (3 of which were binocular) |
| Retinal artery obstruction | 8 (1 of which was binocular) |
| Posterior circulation stroke | 2 (homonymous field loss) |
| Undetermined cause | 7 (all monoocular) |

### Comparing visual acuity at Day 0 and Day 7

Repeat visual acuities were performed at Day 7 in 47 of the 48 individuals who had visual loss. A comparison of the repeat examination is presented in Figure 1 and Figure 2. 21/47 individuals had no difference in the visual acuity at day 0 and 7. 10/47 had worsening of one eye with no change in the other. 2/47 had worsening in both eyes. 9/47 had improvement in 1 eye without any change in the other. 1/47 had improvement in one eye and worsening in the other. 4 had improved visual acuity in both eyes.

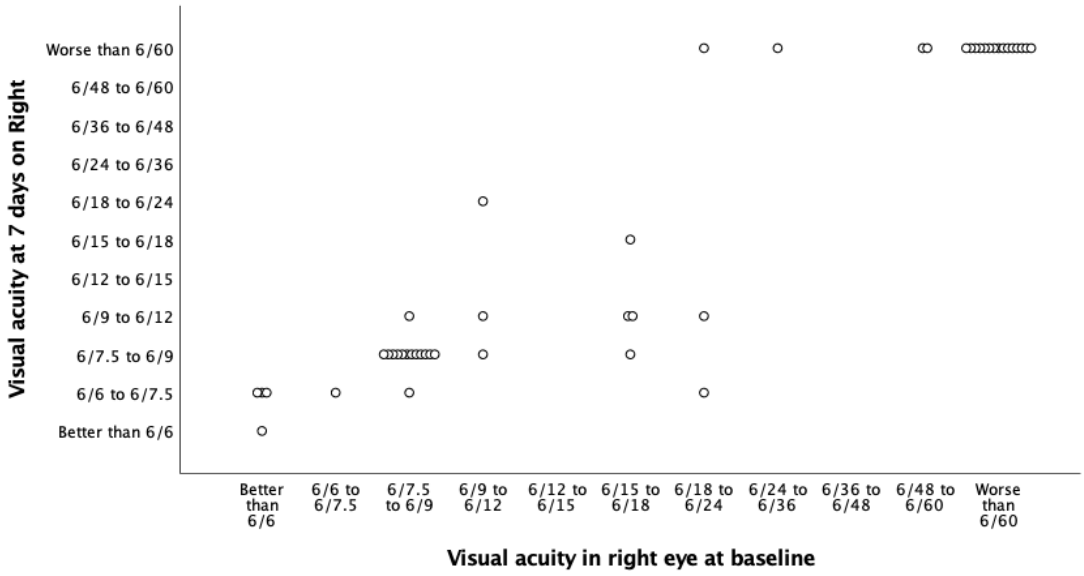


**Figure 1 Visual acuity at Day 0 and 7 in the right eye in 47 individuals with permanent visual loss**

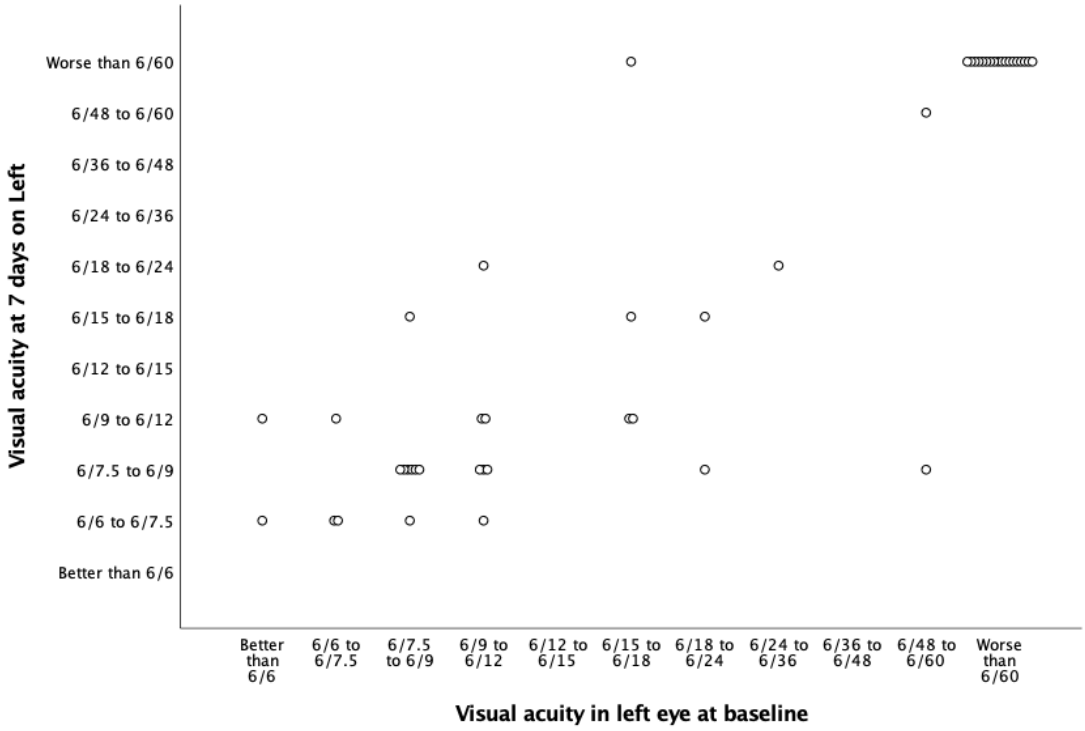


**Figure 2 Visual acuity at Day 0 and 7 in the left eye in 47 individuals with permanent visual loss**

## Effect of fast-track clinic on incidence of visual loss

Between 2012-2016, 107 individuals were diagnosed with GCA. 20 (18.7%) had visual loss at diagnosis. From 2017 onwards, the fast-track pathway picked up 243 new diagnoses, of which 28 (11.5%) had visual loss at diagnosis.

### Identifying predictors of visual loss

Table 3 shows all the factors that were tested separately for their status in predicting visual loss in our cohort. The 48 individuals with visual loss were statistically significantly older by 6.4 years. Jaw claudication appeared to be positively related to visual loss and headache appeared to be negatively related. 6 individuals with visual loss did not have any systemic symptoms.

**Table 3 Univariate analysis testing significance of variables to predict visual loss (Unless stated the numbers are for 48 individuals with visual loss and 302 individuals without visual loss)**

| Variable | With visual loss | Without visual loss | Test used | P value |
|---|---|---|---|---|
| Age (Mean ± SD) | 80.1 ± 6.3 | 73.7 ± 7.6 | t-test | <0.001 |
| Gender (Females) | 30/48 | 206/302 | Chi-square | 0.43 |
| Haemoglobin in g/L (Mean ± SD) | 119.0 ± 15.3 (for N=46) | 121.4 ± 14.2 | t-test | 0.30 |
| C-reactive protein in mg/L (Median (IQR)) | 67.5 (75) | 65 (78) (for N=289) | Mann Whitney U | 0.89 |
| Erythrocyte Sedimentation Rate in mm (Median (IQR)) | 68 (43) (for N=47) | 62 (42) (for N=281) | Mann Whitney U | 0.60 |
| Smoking status | | | | |
| • Never smoked | 10/16 | 59/134 | Fisher's exact | 0.408 |
| • Ex-smokers | 4/16 | 52/134 | | |
| • Current smoker | 2/16 | 23/134 | | |
| Scalp tenderness | 7/48 | 65/302 | Chi-square | 0.27 |
| Headache | 24/48 | 229/302 | Chi-square | <0.001 |
| Jaw claudication | 25/48 | 105/302 | Chi-square | 0.02 |
| Shoulder girdle pain | 5/48 | 63/302 | Chi-square | 0.09 |
| Temporal artery abnormality | 12/48 | 85/302 | Chi-square | 0.65 |
| Fever >38° C | 1/48 | 12/302 | Fisher's exact | 1.00 |
| Weight loss >2 kg | 11/48 | 73/302 | Chi-square | 0.85 |
| Drenching night sweats | 4/48 | 58/302 | Chi-square | 0.07 |
| Loss of appetite | 9/48 | 58/302 | Chi-square | 0.94 |

## Logistic Regression

A model was created using age, headache, jaw claudication, shoulder girdle pain and drenching night sweats. The Nagelkerke $R^2$ was 0.26. The classification improved from 86.3 to 89.4 with the entry of those variables. The odds ratios are as in Table 4.

**Table 4 Odds ratios for visual loss of factors in multivariate logistic regression**

| Variable | Adjusted Odds ratio (95% Confidence Interval) for visual loss | P value |
|---|---|---|
| Age at diagnosis (for each year rise in age) | 1.12 (1.06, 1.17) | <0.001 |
| Headache | 0.22 (0.11, 0.46) | <0.001 |
| Jaw claudication | 1.96 (0.99, 3.90) | 0.05 |
| Shoulder girdle pain | 0.38 (0.13, 1.10) | 0.08 |
| Drenching night sweats | 0.47 (0.15, 1.46) | 0.19 |

## Discussion

We present the largest study of the incidence and nature of visual complications in a cohort of 350 consecutive individuals objectively diagnosed with GCA either by imaging or biopsy. Yates et al have published a larger cohort, but these were not consecutively diagnosed, nor were all of them diagnosed based on objective tests (16). Our work has several strengths. It represents all individuals diagnosed with GCA within a 10-year period in our catchment area without any case selection bias. Our hospital serves a population of 900,000 using a common pathway agreed between all internal medicine specialities as well as ophthalmology. We have thus ensured that the full spectrum of GCA is captured. We have structured assessment clerking sheets that are used for the assessment of individuals with suspected GCA, thus having very little missing data. All the ultrasonography examinations are done by one experienced sonographer (CBM) who has participated in local and international validation exercises for this technique (11, 13, 17, 18).

We also recognise that our work has some limitations. The incidence of visual loss because of GCA is dependent on social factors as well as biological factors. Biological factors like signs, symptoms and blood test results are generalisable, but social factors like the distance from a hospital, access to public transport, access to primary care are not. Our data is from a predominantly rural county in the UK without any motorways (controlled-access vehicular highways). This potentially result in reduced and/or delayed access to healthcare resulting in an altered incidence of visual manifestations.

Visual symptoms including diplopia and blurred, or lost vision occur in 29% of our cohort of GCA. The aetiology of diplopia is unknown. The potential anatomical sites for the lesion could be the vasculature of the mid-brain, the vasa nervosa of the cranial nerves or the vasculature of the extraocular muscles. There has been some evidence that it may be the

nerve that is affected (19). But GCA is a disease affecting large vessels and therefore it would be unusual for the micro-circulation of the cranial nerve or its nuclei to be affected. Also, the recovery of diplopia after the commencement of glucocorticoid is usually rapid and unlike the prolonged recovery typical of a nerve injury.

48 (13.7%) people were reported to have suffered permanent visual loss. This is similar to the visual involvement reported by other interdisciplinary services. Gonzalez-Gay et al reported the incidence of visual loss in Lugo, Spain to be 12.5% (20). Similarly, when Font et al studied all the GCA diagnosed in internal medicine, they reported the incidence of visual loss to be 15.8% (21). Pure rheumatology units have reported incidence of visual loss to be as low as 5% (22) and Neuro-ophthalmology units have reported visual loss incidence to be 50% (23). Our inter-disciplinary work has shown that the true incidence of visual loss is likely in the region of the 12-15% mark with a greater confidence by virtue of reporting from a larger cohort. AION and CRAO are the commonest ocular syndromes that cause blindness (23). In our study we have reported that AION was about 3 times as common as CRAO. This is similar to the finding by Baalbaki et al from a cohort of 100 cases from a vasculitis clinic (24). But we believe that we are the first clinicians to report homonymous field loss in 2 cases perhaps as a result of posterior circulation compromise due to vertebro-basilar insufficiency (Table 2). Seven individuals with decreased visual acuity had a normal fundoscopic appearance. Putative causes include posterior ischaemic optic neuropathy, or choroidal infarction due to involvement of the posterior ciliary (25, 26).

In our study we report a 12% rise in the odds of developing visual loss with each advancing year (Table 4). Liozon et al found a rise of a similar magnitude of 6% in the odds for visual loss with every advancing year (27). Other workers have also found that the age of those with visual loss in their cohorts was older than those who did not have visual loss (24, 28, 29). We wonder if the cause for this this is not just having biologically older arteries but may also include social factors including the likelihood of greater dependence to access healthcare. Further work is needed to investigate the relationship between socio-economic conditions and visual loss. We have reported that the presence of a headache is associated with a reduction in the odds of developing visual loss. This surprising finding has also been reported by at least 4 other workers (24, 28-30). Salvarani et al have reported adjusted odds ratio (95% CI) of 0.41 (0.07-2.5) which is comparable with our adjusted odds ratio of 0.22 (0.11, 0.46) (30). This is inexplicable anatomically and we wonder whether the absence of headache makes individuals less likely to seek appropriate medical attention. Jaw claudication has been traditionally believed to be a risk factor for visual loss in GCA. We have reported that there appears to be a trend for this, but the confidence interval crosses 1 and p value was 0.054. To date this association remains controversial. Two studies from Canada and Italy have both found that Jaw claudication increased the odds or developing visual loss (27, 30). But like us, three other workers have not found this to be a statistically significant finding (24, 28, 29). Anatomically, it does make sense for jaw claudication to be associated with visual involvement. The maxillary artery is responsible for the vascular nourishment of the muscles of mastication. Its terminal branch is the infraorbital artery which supplies some of the extraocular muscles. But in a small number of individuals, the ophthalmic artery has been known to arise from the middle meningeal artery which itself is a branch of the

maxillary artery (31). Thus, this relationship may rely on anatomical variations leading to a trend towards association rather than unequivocal relationship.

The development of fast-track pathways leading to rapid diagnosis using ultrasonography appear to be cost-effective (10, 32) and lead to reduction of visual loss (29, 32). Diamantopoulos et al report that they reduced the frequency of visual loss from 6/32 in a conventional pathway to 1/43 (32). Patil et al report that the frequency of visual loss dropped from 17/46 to 6/67 (29). In this paper we have reported that the incidence of visual loss dropped from 20/107 to 28/243. Combining the results of the three studies, we have a frequency of visual loss of 43/185 (23%) using conventional pathways and 35/353 (10%) using fast-track pathways. We accept that this is a crude analysis because the three conventional pathways may have been vastly different. But that would also make this figure more representative of the frequency of visual loss if rapid diagnostics are not available. By the same token, fast-track pathways halve the risk of visual loss, and their development and proliferation should be encouraged. The more troublesome problem is that of the 10% where sight loss remains an issue despite rapid diagnostics and probably represents social and primary care factors which need qualitative work to understand this better.

47/48 individuals who had visual loss had repeat visual acuity testing after 7 days of glucocorticoids. 13 individuals had an improvement in the visual acuity. Of those 7/13, the worst eye still had a visual acuity of 6/60 or worse. 3 still had visual acuity of worse than 6/12 in at least one eye which is the legal requirement to be able to drive in the UK. 3 individuals had improvement of vision to 6/9 or better. None had been treated with intravenous methylprednisolone. The role of intravenous methylprednisolone to improve vision is contentious and international recommendations admit to the quality of evidence being low and only recommend that their use be considered (12). In our centre, we use a lean body mass based regimen of oral prednisolone from diagnosis (33).

In conclusion, we present the frequency of visual manifestations of GCA in the largest cohort of objectively diagnosed GCA. 29% of our cohort had visual symptoms, 13% suffered visual loss. The main predictors of visual loss were increasing age and absence of headache. The predictive value of jaw claudication remains equivocal. The frequency of visual loss has been brought down to 11.5% with the introduction of the fast-track pathway but more work needs to be done to understand the causes of the visual loss which may include social factors.

## References


1. Sorensen S, Lorenzen I. Giant-cell arteritis, temporal arteritis and polymyalgia rheumatica. A retrospective study of 63 patients. Acta Med Scand. 1977;201(3):207-13.
2. Jonasson F, Cullen JF, Elton RA. Temporal arteritis. A 14-year epidemiological, clinical and prognostic study. Scott Med J. 1979;24(2):111-7.
3. Graham E, Holland A, Avery A, Russell RW. Prognosis in giant-cell arteritis. Br Med J (Clin Res Ed). 1981;282(6260):269-71.
4. Bengtsson BA, Malmvall BE. The epidemiology of giant cell arteritis including temporal arteritis and polymyalgia rheumatica. Incidences of different clinical presentations and eye complications. Arthritis Rheum. 1981;24(7):899-904.

5. Aiello PD, Trautmann JC, McPhee TJ, Kunselman AR, Hunder GG. Visual prognosis in giant cell arteritis. Ophthalmology. 1993;100(4):550-5.
6. Glutz von Blotzheim S, Borruat FX. Neuro-ophthalmic complications of biopsy-proven giant cell arteritis. Eur J Ophthalmol. 1997;7(4):375-82.
7. Cid MC, Font C, Oristrell J, de la Sierra A, Coll-Vinent B, Lopez-Soto A, et al. Association between strong inflammatory response and low risk of developing visual loss and other cranial ischemic complications in giant cell (temporal) arteritis. Arthritis Rheum. 1998;41(1):26-32.
8. Hayreh SS, Podhajsky PA, Zimmerman B. Ocular manifestations of giant cell arteritis. Am J Ophthalmol. 1998;125(4):509-20.
9. Gonzalez-Gay MA, Garcia-Porrua C, Llorca J, Hajeer AH, Branas F, Dababneh A, et al. Visual manifestations of giant cell arteritis. Trends and clinical spectrum in 161 patients. Medicine (Baltimore). 2000;79(5):283-92.
10. Mukhtyar C, Ducker G, Fordham S, Mansfield-Smith S, Jones C. Improving the quality of care for people with giant cell arteritis. Clinical Medicine. 2021;21(4):e371-e4.
11. Mukhtyar C, Myers H, Scott DGI, Misra A, Jones C. Validating a diagnostic GCA ultrasonography service against temporal artery biopsy and long-term clinical outcomes. Clin Rheumatol. 2020;39(4):1325-9.
12. Hellmich B, Agueda A, Monti S, Buttgereit F, de Boysson H, Brouwer E, et al. 2018 Update of the EULAR recommendations for the management of large vessel vasculitis. Ann Rheum Dis. 2020;79(1):19-30.
13. Chrysidis S, Duftner C, Dejaco C, Schafer VS, Ramiro S, Carrara G, et al. Definitions and reliability assessment of elementary ultrasound lesions in giant cell arteritis: a study from the OMERACT Large Vessel Vasculitis Ultrasound Working Group. RMD Open. 2018;4(1):e000598.
14. Mukhtyar C, Hodgson H. The need to establish standards of care for Giant Cell Arteritis. Rheumatology (Oxford). 2020;59(4):702-4.
15. Ehlers L, Askling J, Bijlsma HW, Cid MC, Cutolo M, Dasgupta B, et al. 2018 EULAR recommendations for a core data set to support observational research and clinical care in giant cell arteritis. Ann Rheum Dis. 2019;78(9):1160-6.
16. Yates M, MacGregor AJ, Robson J, Craven A, Merkel PA, Luqmani RA, et al. The association of vascular risk factors with visual loss in giant cell arteritis. Rheumatology (Oxford). 2017;56(4):524-8.
17. Schafer VS, Chrysidis S, Dejaco C, Duftner C, Iagnocco A, Bruyn GA, et al. Assessing Vasculitis in Giant Cell Arteritis by Ultrasound: Results of OMERACT Patient-based Reliability Exercises. J Rheumatol. 2018;45(9):1289-95.
18. Schafer VS, Chrysidis S, Schmidt WA, Duftner C, Iagnocco A, Bruyn GA, et al. OMERACT definition and reliability assessment of chronic ultrasound lesions of the axillary artery in giant cell arteritis. Semin Arthritis Rheum. 2021.
19. Mournet S, Sene T, Charbonneau F, Poillon G, Vignal C, Clavel G, et al. High-resolution MRI demonstrates signal abnormalities of the 3rd cranial nerve in giant cell arteritis patients with 3rd cranial nerve impairment. Eur Radiol. 2021;31(7):4472-80.
20. Gonzalez-Gay MA, Miranda-Filloy JA, Lopez-Diaz MJ, Perez-Alvarez R, Gonzalez-Juanatey C, Sanchez-Andrade A, et al. Giant cell arteritis in northwestern Spain: a 25-year epidemiologic study. Medicine (Baltimore). 2007;86(2):61-8.
21. Font C, Cid MC, Coll-Vinent B, Lopez-Soto A, Grau JM. Clinical features in patients with permanent visual loss due to biopsy-proven giant cell arteritis. Br J Rheumatol. 1997;36(2):251-4.
22. Hocevar A, Rotar Z, Jese R, Semrl SS, Pizem J, Hawlina M, et al. Do Early Diagnosis and Glucocorticoid Treatment Decrease the Risk of Permanent Visual Loss and Early Relapses in Giant Cell Arteritis: A Prospective Longitudinal Study. Medicine (Baltimore). 2016;95(14):e3210.
23. Hayreh SS, Podhajsky PA, Zimmerman B. Occult giant cell arteritis: ocular manifestations. Am J Ophthalmol. 1998;125(4):521-6.

24. Baalbaki H, Jalaledin D, Lachance C, Febrer G, Rheaume M, Makhzoum JP. Characterization of visual manifestations and identification of risk factors for permanent vision loss in patients with giant cell arteritis. Clin Rheumatol. 2021;40(8):3207-17.
25. Ghanchi FD, Williamson TH, Lim CS, Butt Z, Baxter GM, McKillop G, et al. Colour Doppler imaging in giant cell (temporal) arteritis: serial examination and comparison with non-arteritic anterior ischaemic optic neuropathy. Eye (Lond). 1996;10 ( Pt 4):459-64.
26. Hayreh SS, Zimmerman B, Kardon RH. Visual improvement with corticosteroid therapy in giant cell arteritis. Report of a large study and review of literature. Acta Ophthalmol Scand. 2002;80(4):355-67.
27. Liozon E, Dalmay F, Lalloue F, Gondran G, Bezanahary H, Fauchais AL, et al. Risk Factors for Permanent Visual Loss in Biopsy-proven Giant Cell Arteritis: A Study of 339 Patients. J Rheumatol. 2016;43(7):1393-9.
28. Chen JJ, Leavitt JA, Fang C, Crowson CS, Matteson EL, Warrington KJ. Evaluating the Incidence of Arteritic Ischemic Optic Neuropathy and Other Causes of Vision Loss from Giant Cell Arteritis. Ophthalmology. 2016;123(9):1999-2003.
29. Patil P, Williams M, Maw WW, Achilleos K, Elsideeg S, Dejaco C, et al. Fast track pathway reduces sight loss in giant cell arteritis: results of a longitudinal observational cohort study. Clin Exp Rheumatol. 2015;33(2 Suppl 89):S-103-6.
30. Salvarani C, Cimino L, Macchioni P, Consonni D, Cantini F, Bajocchi G, et al. Risk factors for visual loss in an Italian population-based cohort of patients with giant cell arteritis. Arthritis Rheum. 2005;53(2):293-7.
31. Liu Q, Rhoton AL, Jr. Middle meningeal origin of the ophthalmic artery. Neurosurgery. 2001;49(2):401-6; discussion 6-7.
32. Diamantopoulos AP, Haugeberg G, Lindland A, Myklebust G. The fast-track ultrasound clinic for early diagnosis of giant cell arteritis significantly reduces permanent visual impairment: towards a more effective strategy to improve clinical outcome in giant cell arteritis? Rheumatology (Oxford). 2016;55(1):66-70.
33. Mukhtyar C, Cate H, Graham C, Merry P, Mills K, Misra A, et al. Development of an evidence-based regimen of prednisolone to treat giant cell arteritis - the Norwich regimen. Rheumatol Adv Pract. 2019;3(1):rkz001.