Epidemiology of Giant Cell Arteritis in Waikato, Aotearoa New Zealand

Introduction:

Giant cell arteritis (GCA) is the most common primary vasculitis in adults over 50 years of age. It is a granulomatous vasculitis and classically involves the aorta and its branches, the distribution of which helps to classify as either cranial or extra-cranial [1]. Symptoms include temporal headaches, visual loss, scalp sensitivity, jaw claudication and limb claudication. A feared complication of untreated disease is permanent visual loss.

Ethnicity may be a risk factor for GCA with Scandinavian countries reporting the highest incidence rates [2]. It appears much less common in Asian and African American patients [3-7]. Age also appears to be a risk factor with peaking incidence in the 70-79 years age group with a female predominance[7].

Investigation of GCA has historically been with temporal artery biopsy (TAB) which has been the gold standard. Colour doppler ultrasound of the temporal and axillary arteries (CDUS) is being used with increasing frequency over the last decade depending on the availability and expertise in local centres. Ultrasound features of GCA include the halo sign (a hypoechoic ring around the lumen of the temporal or axillary artery) or vessel wall thickening that can lead to stenosis or occlusion of a blood vessel [8, 9]. Computed tomography (CT), magnetic resonance imaging (MRI) and F-fluorodeoxyglucose positron emission tomography with computed tomography (FDG-PET) supplement these investigations, particularly in extra-cranial large vessel vasculitis.

The American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) have released new classification criteria in 2022 [10]. Patients must be 50 years or older with additional clinical criteria of polymyalgic symptoms, visual loss, jaw or tongue claudication, scalp tenderness and abnormal examination of the temporal artery. Investigations with inflammatory markers, TAB, CDUS and FDG-PET are used in the criteria. Patients with 6 or more points reach a classification for GCA.

The foundation of treatment for GCA is with corticosteroids, and whilst untreated disease is associated with significant morbidity, its treatment is also associated with morbidity for patients [11]. Newer steroid-sparing treatments include tocilizumab, an interleukin-6 inhibitor, or janus kinase (JAK) inhibitors. In resource-limited countries like Aotearoa New Zealand (AoNZ), appreciation of the epidemiology of a condition is crucial when advocating for government support for these evolving therapies, which are currently not routinely available for our patients.

AoNZ is a country of 4.6 million people with 16 regions. As of the 2018 Census, there are 70.2% Europeans (made up mostly of New Zealand Europeans (NZE) with a smaller group of Other Europeans), 16.5% Māori, 15.1% Asian, 8.1% Pacific Islander along with smaller proportions of other nationalities. Waikato is the fourth largest region in AoNZ making up 9.5% of the population [12]. Our primary objective was to examine the incidence of GCA in Waikato, which, given its large population, is likely to be reflective of the epidemiology of GCA nationally.

Methods:

Beginning in 2013, a new process was developed for handling suspected cases of GCA in the Waikato region. General practitioners, physicians and those likely to encounter suspected cases were asked to discuss cases with the on-call rheumatologist. Cases were therefore identified prospectively. In addition, a search of ultrasonography lists in the radiology department and a histology search of all temporal artery biopsies performed from January 2014 to December 2022 were screened for patients suspected of having GCA.

The electronic records were reviewed including clinic letters, primary care referrals, ultrasound request forms, laboratory results, discharge summaries and electronic prescriptions. Data was collected regarding patient demographics, clinical and laboratory features of their GCA, investigations, treatment and mortality. Ethnicity was reported in conjunction with New Zealand Statistics method of reporting and if patients identified as two ethnicities, then both were noted.

Cases of GCA were defined as the final clinical diagnosis recorded by the Rheumatologist or Ophthalmologist. Where a patient had been evaluated more than once over the time period, only the original evaluation was retained for analysis.

CDUS involved ultrasound of both temporal and axillary arteries. Positive CDUS was classified as the halo sign and indeterminate if only vessel wall thickening was noted. TAB was classified as positive if there was evidence of active inflammation and the pathology report supported a diagnosis of GCA.

Statistical analysis:

Descriptive data are presented as frequencies for categorical variables. Continuous variables are presented as mean with standard deviation (SD). Where symptoms were not reported, this was reported as missing data, thus percentage calculations are of valid percent rather than total percent. T-tests were used to compare mean age at diagnosis. Chi-square test was used to compare deaths between ethnicities. Incidence was calculated by examining the number of new instances of GCA in proportion to the population of the Waikato region for

each year over the duration of the study. Population counts for the Waikato region were provided by age groups from Statistics New Zealand, a centralized government agency which collects and manages official statistical data for AoNZ. Yearly population projections for the Waikato region are mostly based around 2018 census data (13). After monitoring incoming patients for GCA over a span of 9 years and meticulous tracking occurrences of deaths, prevalence was calculated by determining the total number of individuals with GCA at the end of the observation period and expressing this count as a proportion of the total population under surveillance (i.e., Waikato region and people over 50 years old). The mean annual mortality rate was calculated from the yearly death count among the changing total population of total known cases of GCA in the Waikato region. Moreover, a standardized mortality ratio (SMR) was derived comparing the observed deaths among cases of GCA versus what would be expected using the age specific rates of the surrounding population. Analyses were conducted in SPSS and R software. All significance tests were two-tailed and values of $p<0.05$ were considered significant.

Results:

Cases of GCA and demographics

From January 2014 to December 2022, a total of 761 evaluations took place for patients with suspected GCA and were referred for either CDUS, TAB or both. Of these, 214 individuals had a final diagnosis of GCA. The majority of the patients with GCA were managed by Rheumatology (65.9%), followed by Ophthalmology 17.8%, a combination of both specialities in 8.5% and other specialities (usually General Medicine or Neurology) in 7.9%. Most patients were female (68.2%) with a mean age of 74.0 (SD 8.3) years.

Patients with GCA were most commonly of European background in 93.9% (201/214), Māori 7.9% (17/214) and Asian 0.5% (1/214). The remaining patients (2.8%) included other minorities or patients who did not state an ethnicity. There were no cases of GCA in patients of Pacific Islander descent. Seventeen patients identified as having both European and Māori ethnicity, thus the total percentage is greater than 100%.

The mean age of European patients with GCA (74.2 (SD 8.3) years) was significantly older than those identifying as Māori (70.1 (SD 8.9) years) (p=0.047).

Table 1. identifies the baseline clinical characteristics of patients with GCA. There was a mean duration of symptoms of 26.4 (SD 29.5) days at the time of referral or initial review.

Analysis of moving averages for annual numbers of cases of GCA showed a statistically significant peak in 2019 and a smaller one in 2021.

Table 1. Clinical characteristics of patients with GCA.

Clinical Symptoms	No. (valid %)
Headache	183/205 (89.3)
Unilateral headache	86/205 (42.0)
Scalp sensitivity	103/155 (66.5)
Jaw claudication	92/181 (50.8)
Visual symptoms (any)	93/183 (50.8)
Typical symptoms (i.e., AION, CRAO)	24 / 214 (11.2) *
Diplopia	14/ 214 (6.5) *
PMR	73/142 (51.4)
Temporal artery tenderness	82/142 (57.7)
ESR mean (SD) mm/hour	46.2 (29.6)
CRP (mean (SD) mg/L	68.7 (70.1)
Platelets above normal range	69/208 (33.2)
Haemoglobin below normal range	66/208 (31.7)
ACR/EULAR 2022 classification criteria ≧ 6	179/214 (83.6%)

* Reported using total population.

AION = anterior ischaemic optic neuropathy; CRAO = central retinal artery occlusion; PMR = polymyalgia rheumatica; ESR = erythrocyte sedimentation rate; CRP = c-reactive protein; Platelet ULN > 400 x10^9/L; Haemoglobin LLN < 115 g/L (women) and < 130 g/L (men). ACR = American College of Rheumatology; EULAR = European League Against Rheumatism;

Diagnostic Tests

In patients diagnosed with GCA, CDUS was performed in 167/214 (78.0%). Of these ultrasounds performed , 17 (10.2%) were positive with a halo sign, 88 (52.7%) were negative and 62 (37.1%) were indeterminate with vessel wall thickening noted.

TAB was performed in 168 (78.5%) of the patients with GCA and 67 (39.9%) had positive histology reported. The ACR/EULAR 2022 classification criteria for GCA were positive in 83.6% of patients.

The sensitivity and specificity of CDUS compared to clinical diagnosis was 10.2% (95% CI: 6.0 – 15.8%) and 99.8% (95% CI: 98.9-99.9%) respectively. The sensitivity and specificity of a non-negative CDUS (i.e., either a positive or indeterminate result) was 47.3% (95% CI: 39.5 – 55.2%) and 91.2% (95% CI: 88.3 – 93.5%) respectively.

For TAB, the sensitivity and specificity compared to clinical diagnosis was 39.9% (95% CI: 32.4-47.7%) and 100.0% (95% CI: 97.5-100.0%) respectively. For the ACR/EULAR 2022 classification criteria, the sensitivity was 83.6% (95% CI: 78.0 – 88.3) and specificity was 53.1% (95% CI: 48.7 – 57.5%).

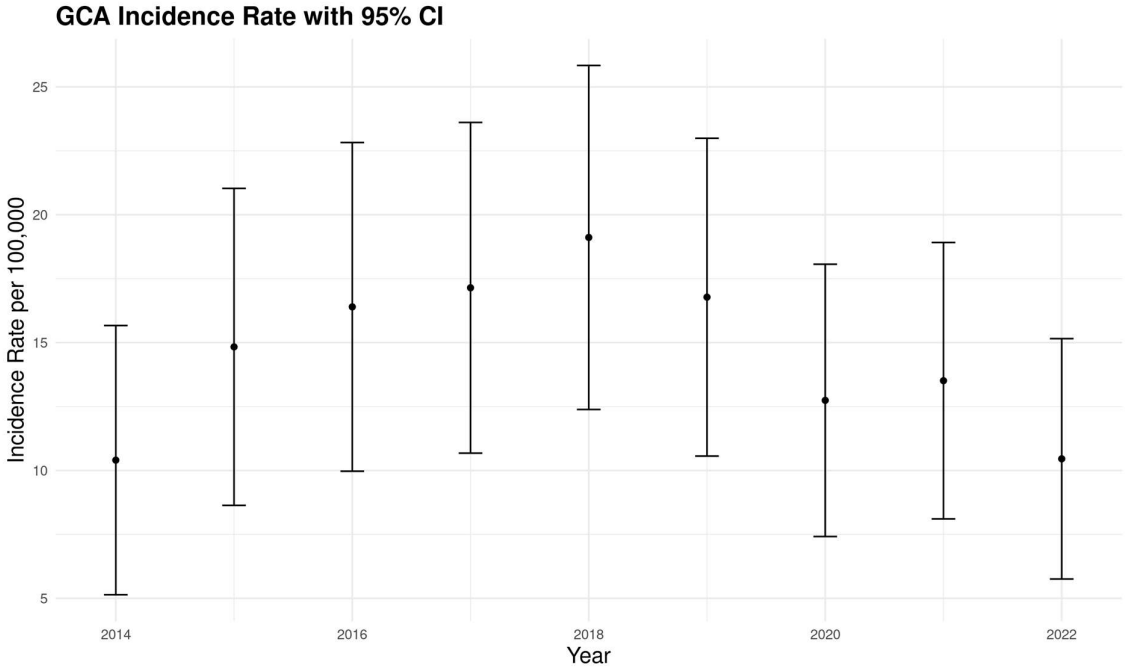
Eleven patients with GCA had a FDG-PET scan performed looking for large vessel vasculitis (LVV). There was evidence of LVV in 5 patients (45.5%) with none (0/5) of these patients having a positive CDUS and 1 (1/3) having a positive TAB.

Two patients with GCA had neither a CDUS nor a TAB. Their data had been collected prospectively as part of the CDUS referral process. Both had significant referral delays where the treating Rheumatologist decided against further tests but that the case was clinically consistent with GCA.

Incidence and Prevalence of GCA in Waikato

The incidence of GCA was calculated for cases identified with TAB and also for cases identified clinically (i.e., on the experience of the rheumatologist with or without supporting imaging or histological data). The mean annual incidence (MAI) of biopsy positive GCA was 4.6 per 100,000 people over 50 years (95% CI: 3.5-5.7). The MAI of clinical GCA was 14.7 per 100,000 people over 50 years (95% CI:12.7-16.6). The annual incidence for clinical GCA is reflected in Figure 1. The confidence intervals are overlapping and thus there does not appear to be any change in the annual incidence rate over the 9-year period. The estimate of the lower bound of prevalence of clinically positive GCA was 94.6 per 100,000 people over 50 years.

Figure 1. GCA Annual Incidence.



GCA = giant cell arteritis; CI = confidence interval.

Covid-19

AoNZ had an initial lockdown period due to the covid-19 pandemic in March-May 2020 and a subsequent lockdown in August-September 2021 when there was community transmission. During these time periods, the service remained in place where patients and physicians had access to CDUS and TAB. Reviews occurred via telephone consultation unless the patient was unwell, in which case a face-to-face review was organized.

Mortality

There were 47 deaths in the cohort over the time period in patients with GCA. The mean age at time of death was 81.1 (SD 7.5) years with a range of 64-95 years of age. Females made up 59.6% of deaths. Mean time to death from referral was 29.1 (SD 23.6) months. The mean annual mortality rate was 38.4 per 1000 cases over 50 years. The standardized mortality ratio (SMR) was 1.18 (95% CI: 0.83–1.52). Most deceased patients were of European descent (42/47, 89.4%) with 4 deaths in Māori patients (8.5%) and 1 not stated. The difference between deaths between Europeans and Māori was not statistically significant (p=0.27).

Infection was the most common cause of death accounting for 16.7% (8/48) deaths, followed by malignancy 12.5% (6/48), cerebrovascular disease 12.5% (6/48) and cardiovascular disease in 10.4% (5/48). Half of these deaths due to infection (4/8) occurred prior to the onset of the Covid-19 pandemic and of the 4 infection-related deaths occurring in 2020 onwards, none of these patients were positive for Covid-19 at the time of their death. In addition to causes of

mortality already described, there was 1 patient who died from an aortic dissection, 1 patient from venous thromboembolism (VTE) and 2 from peptic ulcer disease.

Discussion:

This is the largest epidemiological study on GCA in AoNZ published to date and certainly the first to document the epidemiology of GCA in Waikato.

We report a MAI for clinically diagnosed GCA of 14.7 per 100,000 people over 50 years and a lower incidence of TAB positive GCA of 4.6 per 100,000 people over 50 years. This latter finding is likely due to only 78.5% of GCA patients having a TAB performed, and only 39.9% positive. There are three studies to date looking at the incidence of GCA in AoNZ and cover Otago [14], Canterbury [15] and Counties Manukau [16]. Collectively, these report a MAI of biopsy positive GCA of 10.5-12.73 per 100,000 people over 50 years [14, 15] and a MAI of clinically positive GCA of 11.4-15 per 100,000 people over 50 years old [15, 16].

Data on sensitivity and specificity for TAB and CDUS in AoNZ is only available in one of the three studies (Counties Manukau [16]) with a slightly higher sensitivity for TAB of 57% and CDUS 26%. Specificity is similar with 100% for TAB and 97% for CDUS.

This unity between four epidemiology studies in AoNZ supports the notion that cases of GCA are largely stable over the last decade. Certainly, over our study period in Waikato, cases of GCA did not appear to be rising.

Worldwide, pooled incidences of GCA are 10.00 (95% CI: 9.22 - 10.78) cases per 100,000 over 50 years which is lower than our study but likely reflects the combination of high-risk countries (Scandinavian), moderate-risk (European, American) with low-risk countries (Asian) in different proportions to what we see in AoNZ [2]. It is likely that given AoNZ is made up predominantly of Europeans, that the epidemiology of GCA in our country will reflect other European countries.

The prevalence of GCA in our study was 94.6 per 100,000 over 50 years. This is also higher than the pooled prevalence of 51.7 per 100,000 people over 50 years from the meta-analysis by Li et al [2] yet very similar to the prevalence of 87.9 per 100,000 people over 50 years reported by a European study [17]. It is likely that given AoNZ is made up predominantly of Europeans that the epidemiology of GCA in our country will reflect other European countries. Whilst the methods of the study were not dedicated to prevalence calculations, we believe that the duration of the study, life expectancy and average age of admittance means it is possible to make an estimate on the lower bound for prevalence.

Our study has similar baseline demographics compared to other New Zealand studies that have noted a female predominance of 65.5-71% [14-16] and mean age of 72.8-74.2 years [14, 15].

Despite Māori and Pacific Islanders making up 23.9% and 4.5% of the Waikato population respectively [12], only 7.0% of our patients with GCA were Māori and none were of Pacific Islander origin. Other AoNZ studies have also demonstrated that GCA is uncommon in Māori and Pacific Islanders [15, 16]. One factor contributing to this may be the lower life expectancy in these groups [12] and the known risk of age and GCA [7]. Asians make up 9.5% of the population of Waikato, yet we noted very few Asians with GCA, a finding consistent with the very low incidence in this group noted by other studies [4].

Our study documented lower sensitivities of TAB and CDUS compared to those in Counties Manukau [16] and other worldwide studies (18-22). TAB is a difficult investigation to acquire in Waikato Hospital due to local referral issues and an under-resourced vascular department, where biopsies commonly occur out of the optimal window. This may account for the lower positive biopsy rates. The lower sensitivity of CDUS was also seen by the Counties Manukau study and the cause remains unclear (16). A separate study analysing the efficacy of a fast-track pathway to investigate GCA in Waikato has noted that early commencement of corticosteroids resulting in less positive CDUS scans, which has been noted in other studies as well (6). However, early corticosteroids are mandatory to prevent complications of disease. We also suspect that ultrasonographers have underreported some positive CDUS scans describing findings as 'suggestive of GCA' rather than noting the presence of a halo sign. This has been noted on a separate retrospective review of several CDUS scans used in the data collection, however, the significance remains unclear as static ultrasound appears inferior to real-time ultrasound though not formally evaluated in GCA (23).

The ACR/EULAR 2022 classification criteria also performed at lower sensitivity and specificity in our study compared to a recent review of their performance in the United States (22) which noted a sensitivity of 92.6% and specificity of 71.8%.

The mortality rate was 38.4 per 1000 people over 50 years old. This compares to a pooled annual mortality rate of 20.4 (95% CI: 17.8, 23.0) cases per 1000 people over 50 years (2). Whilst higher than the pooled mortality rate, it is similar to some of the European studies included in this meta-analysis and thus is probably consistent given our predominantly European population.

Our study noted no increase in the SMR for patients with GCA, which is concordant with the literature. Several meta-analyses looking at all-cause mortality in GCA compared to the general population have found that SMR is not increased with GCA (24, 25). However, on subgroup analysis in these studies, there appears to be an increase in mortality if patients are

recruited through a hospital setting (25). An increase in mortality is also noted during the first 2 years of treatment, which disappears after 10 years (26, 27).

From the literature, the leading cause of death in GCA is cardiovascular disease followed by cerebrovascular disease, infection and malignancy (25). Our study showed causes of death were due to infection, followed by malignancy and cerebrovascular disease, and lastly by cardiovascular disease. This discrepancy may be due to the low numbers of deaths (n=47) and shorter duration of follow up for patients recruited in the latter half of the study period, which may not reflect causes of death for patients who died later than 2023. Despite the study period crossing the Covid-19 pandemic, death due to Covid-19 was uncommon and unlikely to explain the higher number of infection-related deaths. Over half of deceased patients had a time from referral to death of under 2 years (53.2%) consistent with the known increased mortality risk noted in the first 2 years (26, 27).

We acknowledge that patients being investigated in the private healthcare system were not captured in this study and that the private health community in NZ does account for a significant portion of the Rheumatology workforce (28). The access to CDUS in the Waikato private sector is limited and unlikely to occur at short notice except via the Waikato fast-track pathway which was set up in 2013 to enable rapid access (i.e., same day or next working day) to CDUS for patients. Thus, most patients in the private sector are referred to the public system and would be captured in this dataset. It is unlikely that patients living out of Waikato would be investigated by the Waikato Hospital because access to healthcare was usually strictly limited to District Health Board zones. This may not be the cases for private cases of GCA which may have crossed boundaries. Overall, it is likely that we have recorded most cases of GCA over a 9-year period. Other minor limitations of this study include its retrospective nature along with some missing data due to inadequate documentation of clinical symptoms.

Conclusion

The epidemiology of giant cell arteritis in Waikato is comparable to other epidemiological studies in AoNZ, reflecting a stability in the incidence of GCA nationally over the past decade. Giant cell arteritis appears to be less common in Māori, Pacific Islander and Asian patient groups and occurs at a younger age in Māori patients compared to European. We noted no increase in mortality with common causes of mortality being infection followed by malignancy and cerebrovascular disease.

Funding

The first author has been employed part-time by the Waikato Hospital, Te Whatu Ora, for 12 months in a research position to carry out this research project amongst others as well as perform a clinical-role. There is no other specific funding towards the project.

Conflicts of Interest

None.

Ethics Statement

National ethics approval was granted by HDEC (Reference: 2023 EXP 15448) and there was a local assessment through the Waikato Hospital who also approved the project (RD023025) which included review by Te Puna Oranga Māori Consultation Research Review Committee.

References

1. Simon S, Ninan J, Hissaria P. Diagnosis and management of giant cell arteritis: Major review. Clin Exp Ophthalmol. 2021;49(2):169-85.
2. Li KJ, Semenov D, Turk M, Pope J. A meta-analysis of the epidemiology of giant cell arteritis across time and space. Arthritis Res Ther. 2021;23(1):82.
3. Smith CA, Fidler WJ, Pinals RS. The epidemiology of giant cell arteritis. Report of a ten-year study in Shelby County, Tennessee. Arthritis Rheum. 1983;26(10):1214-9.
4. Kobayashi S, Yano T, Matsumoto Y, et al. Clinical and epidemiologic analysis of giant cell (temporal) arteritis from a nationwide survey in 1998 in Japan: the first government-supported nationwide survey. Arthritis Rheum. 2003;49(4):594-8.
5. Yu E, Chang J. Giant Cell Arteritis: Updates and Controversies. Frontiers in Ophthalmology2022.
6. Pinnell J, Tiivas C, Mehta P, Dubey S. Corticosteroids reduce vascular ultrasound sensitivity in fast- track pathways (FTP): results from Coventry Multi-Disciplinary FTP for cranial Giant Cell Arteritis. Scand J Rheumatol. 2023;52(3):283-92.
7. Gonzalez-Gay MA, Vazquez-Rodriguez TR, Lopez-Diaz MJ et al. Epidemiology of giant cell arteritis and polymyalgia rheumatica. Arthritis Rheum. 2009;61(10):1454-61.
8. Rinagel 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(1):56-61.
9. Jianu DC, Jianu SN, Dan TF, et al. Ultrasound Technologies and the Diagnosis of Giant Cell Arteritis. Biomedicines. 2021;9(12).
10. Ponte C, Grayson PC, Robson JC, et al. 2022 American College of Rheumatology/EULAR classification criteria for giant cell arteritis. Ann Rheum Dis. 2022;81(12):1647-53.
11. Curtis JR, Westfall AO, Allison J, et al. Population-based assessment of adverse events associated with long-term glucocorticoid use. Arthritis Rheum. 2006;55(3):420-6.
12. New Zealand Census 2018 [internet] New Zealand. Stats NZ Tatauranga Aotearoa. Cited 2023, 31st July. Available from: https://www.stats.govt.nz/tools/2018-census-place-summaries/new-zealand

13. Abdul-Rahman AM, Molteno AC, Bevin TH. The epidemiology of giant cell arteritis in Otago, New Zealand: a 9-year analysis. N Z Med J. 2011;124(1329):44-52.
14. Lyne SA, Ruediger C, Lester S, et al. Giant cell arteritis: A population-based retrospective cohort study exploring incidence and clinical presentation in Canterbury, Aotearoa New Zealand. Front Med (Lausanne). 2022;9:1057917.
15. Nagarajah R, Gupta R, Kumar S. Diagnostic use of ultrasound in giant cell arteritis in Counties Manukau District Health Board, New Zealand. Rheumatol Adv Pract. 2022;6(2):rkac040.
16. Catanoso M, Macchioni P, Boiardi L, et al. Incidence, Prevalence, and Survival of Biopsy-Proven Giant Cell Arteritis in Northern Italy During a 26-Year Period. Arthritis Care Res (Hoboken). 2017;69(3):430-8.
17. Lee YH, Song GG. Overall and cause-specific mortality in giant cell arteritis : A meta-analysis. Z Rheumatol. 2018;77(10):946-51.
18. Hill CL, Black RJ, Nossent JC, et al. Risk of mortality in patients with giant cell arteritis: A systematic review and meta-analysis. Semin Arthritis Rheum. 2017;46(4):513-9.
19. Baslund B, Helleberg M, Faurschou M, Obel N. Mortality in patients with giant cell arteritis. Rheumatology (Oxford). 2015;54(1):139-43.
20. Mohammad AJ, Nilsson J, Jacobsson LT, et al. Incidence and mortality rates of biopsy-proven giant cell arteritis in southern Sweden. Ann Rheum Dis. 2015;74(6):993-7.

1. Simon S, Ninan J, Hissaria P. Diagnosis and management of giant cell arteritis: Major review. Clin Exp Ophthalmol. 2021;49(2):169-85.
2. Li KJ, Semenov D, Turk M, Pope J. A meta-analysis of the epidemiology of giant cell arteritis across time and space. Arthritis Res Ther. 2021;23(1):82.
3. Smith CA, Fidler WJ, Pinals RS. The epidemiology of giant cell arteritis. Report of a ten-year study in Shelby County, Tennessee. Arthritis Rheum. 1983;26(10):1214-9.
4. Kobayashi S, Yano T, Matsumoto Y, Numano F, Nakajima N, Yasuda K, et al. Clinical and epidemiologic analysis of giant cell (temporal) arteritis from a nationwide survey in 1998 in Japan: the first government-supported nationwide survey. Arthritis Rheum. 2003;49(4):594-8.
5. Yu E, Chang J. Giant Cell Arteritis: Updates and Controversies. Frontiers in Ophthalmology2022.
6. Pinnell J, Tiivas C, Mehta P, Dubey S. Corticosteroids reduce vascular ultrasound sensitivity in fast- track pathways (FTP): results from Coventry Multi-Disciplinary FTP for cranial Giant Cell Arteritis. Scand J Rheumatol. 2023;52(3):283-92.
7. Gonzalez-Gay MA, Vazquez-Rodriguez TR, Lopez-Diaz MJ, Miranda-Filloy JA, Gonzalez-Juanatey C, Martin J, et al. Epidemiology of giant cell arteritis and polymyalgia rheumatica. Arthritis Rheum. 2009;61(10):1454-61.
8. Rinagel M, Chatelus E, Jousse-Joulin S, Sibilia J, Gottenberg JE, Chasset F, 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(1):56-61.
9. Jianu DC, Jianu SN, Dan TF, Munteanu G, Bîrdac CD, Motoc AGM, et al. Ultrasound Technologies and the Diagnosis of Giant Cell Arteritis. Biomedicines. 2021;9(12).

10. Ponte C, Grayson PC, Robson JC, Suppiah R, Gribbons KB, Judge A, et al. 2022 American College of Rheumatology/EULAR classification criteria for giant cell arteritis. Ann Rheum Dis. 2022;81(12):1647-53.
11. Curtis JR, Westfall AO, Allison J, Bijlsma JW, Freeman A, George V, et al. Population-based assessment of adverse events associated with long-term glucocorticoid use. Arthritis Rheum. 2006;55(3):420-6.
12. New Zealand Census 2018. https://www.stats.govt.nz/tools/2018-census-place-summaries/new-zealand: Stats NZ; 2018.
13. Aotearoa NST. Subnational population component changes and median age (RC, TA), at 30 June 2018-2023 2023 [Available from: https://nzdotstat.stats.govt.nz/wbos/Index.aspx?DataSetCode=TABLECODE7979#.
14. Abdul-Rahman AM, Molteno AC, Bevin TH. The epidemiology of giant cell arteritis in Otago, New Zealand: a 9-year analysis. N Z Med J. 2011;124(1329):44-52.
15. Lyne SA, Ruediger C, Lester S, Chapman PT, Shanahan EM, Hill CL, et al. Giant cell arteritis: A population-based retrospective cohort study exploring incidence and clinical presentation in Canterbury, Aotearoa New Zealand. Front Med (Lausanne). 2022;9:1057917.
16. Nagarajah R, Gupta R, Kumar S. Diagnostic use of ultrasound in giant cell arteritis in Counties Manukau District Health Board, New Zealand. Rheumatol Adv Pract. 2022;6(2):rkac040.
17. Catanoso M, Macchioni P, Boiardi L, Muratore F, Restuccia G, Cavazza A, et al. Incidence, Prevalence, and Survival of Biopsy-Proven Giant Cell Arteritis in Northern Italy During a 26-Year Period. Arthritis Care Res (Hoboken). 2017;69(3):430-8.
18. Sebastian A, Tomelleri A, Kayani A, Prieto-Pena D, Ranasinghe C, Dasgupta B. Probability-based algorithm using ultrasound and additional tests for suspected GCA in a fast-track clinic. RMD Open. 2020;6(3).
19. Moreel L, Betrains A, Doumen M, Molenberghs G, Vanderschueren S, Blockmans D. Diagnostic yield of combined cranial and large vessel PET/CT, ultrasound and MRI in giant cell arteritis: A systematic review and meta-analysis. Autoimmun Rev. 2023;22(7):103355.
20. Nakajima E, Moon FH, Junior NC, Macedo CR, de Souza AWS, Iared W. Accuracy of Doppler ultrasound in the diagnosis of giant cell arteritis: a systematic review and meta-analysis. Adv Rheumatol. 2023;63(1):5.
21. Rubenstein E, Maldini C, Gonzalez-Chiappe S, Chevret S, Mahr A. Sensitivity of temporal artery biopsy in the diagnosis of giant cell arteritis: a systematic literature review and meta-analysis. Rheumatology (Oxford). 2020;59(5):1011-20.
22. Molina-Collada J, Castrejón I, Monjo I, Fernández-Fernández E, Torres Ortiz G, Álvaro-Gracia JM, et al. Performance of the 2022 ACR/EULAR giant cell arteritis classification criteria for diagnosis in patients with suspected giant cell arteritis in routine clinical care. RMD Open. 2023;9(2).
23. Van Holsbeke C, Yazbek J, Holland TK, Daemen A, De Moor B, Testa AC, et al. Real-time ultrasound vs. evaluation of static images in the preoperative assessment of adnexal masses. Ultrasound Obstet Gynecol. 2008;32(6):828-31.
24. Lee YH, Song GG. Overall and cause-specific mortality in giant cell arteritis : A meta-analysis. Z Rheumatol. 2018;77(10):946-51.
25. Hill CL, Black RJ, Nossent JC, Ruediger C, Nguyen L, Ninan JV, et al. Risk of mortality in patients with giant cell arteritis: A systematic review and meta-analysis. Semin Arthritis Rheum. 2017;46(4):513-9.

26. Baslund B, Helleberg M, Faurschou M, Obel N. Mortality in patients with giant cell arteritis. Rheumatology (Oxford). 2015;54(1):139-43.
27. Mohammad AJ, Nilsson J, Jacobsson LT, Merkel PA, Turesson C. Incidence and mortality rates of biopsy-proven giant cell arteritis in southern Sweden. Ann Rheum Dis. 2015;74(6):993-7.
28. Harrison AA, Tugnet N, Taylor WJ. A survey of the New Zealand rheumatology workforce. N Z Med J. 2019;132(1507):70-6.