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The epidemiology of giant cell arteritis in Otago, New Zealand: a 9-year analysis

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Abstract

Aims To study the epidemiology of biopsy proven giant cell arteritis (GCA) in patients in the Otago region, New Zealand.

Materials and Methods Records of 363 consecutive patients who underwent temporal artery biopsy at Dunedin Hospital between 1996-2005 were reviewed. Annual incidence of biopsy-proven GCA was estimated, epidemiologic characteristics of the biopsy-positive group was compared with the biopsy-negative group.

Results Among the 363 patients who underwent temporal artery biopsy there were 105 (29%) males and 258 (71%) females; biopsy-proven GCA was diagnosed in 70 (19%) patients. The mean age of biopsy-positive group was 72.8 years (range 57-91 years, SD 8.2), which was comparable to the biopsy-negative group 73.4 years (range 50-97 years, SD 9.5), $p<0.2$. The mean annual incidence of GCA in Otago was 12.73/100,000 CI (11.7-14.3, $p<0.5$) for patients ≥50 years over the 9 years of observation

Conclusions The first large study of GCA from Australasia demonstrated that a variation in the annual incidence rate for giant cell arteritis in Otago, New Zealand showed a cyclic pattern. The overall incidence seems to reflect the ethnic origins of the majority of the population from Britain.

The epidemiologic characteristics of giant cell arteritis (GCA) have been studied in large populations from the United States,[1–4], United Kingdom,[5, 6] Sweden,[7–9] Denmark,[10] Norway,[11] Israel[12,13] and Saudi Arabia[14] where incidences have been estimated mainly through retrospective study designs. These studies have demonstrated that GCA occurs more commonly in populations of Nordic descent,[15] fluctuations in incidence with distinctive peaks were reported by some investigators;[2,5,13,16–18] however this was not observed by others.[16,19]

The epidemiologic characteristics of this disease have never been studied in Australasia. We present the first epidemiological study evaluating clinical and laboratory characteristics of biopsy proven GCA in the population of Otago, New Zealand; comparing the characteristics of the biopsy positive with the biopsy negative group and the annual incidence rate in Otago, New Zealand to internationally reported figures.

Materials and Methods

A retrospective analysis of the clinical presentation, management and postoperative course of 363 consecutive patients obtained from the surgical database at Dunedin Hospital, Otago, New Zealand between 1996–2005 was undertaken. All cases of suspected giant cell arteritis in the Otago region are referred to this centre for a temporal artery biopsy. Patients who underwent or were evaluated for a

temporal artery biopsy (TAB) were included in the study, cases lacking sufficient medical information were excluded.

Of a total of 369 cases, 6 cases had insufficient medical information and were excluded from this study. A total of 363 cases fulfilled the inclusion criteria. Variables documented from the medical records included demographic factors such as age and gender, and major symptoms and signs. An ophthalmic trainee or ophthalmologist evaluated all cases within 1 week of starting systemic steroids and temporal artery biopsies were conducted within 2 weeks of starting steroids.

Inflammatory markers were requested for all cases at the time of referral. The decision to undertake a TAB was based on clinical criteria and raised inflammatory markers and / or platelet count. Trainees performed biopsies within 1 week of clinical assessment.

A repeat biopsy was performed within 1 week in histologically negative cases if clinical features were judged to be sufficiently suggestive, inflammatory markers were raised inexplicably or the initial biopsy was shorter than 2 mm.

An experienced pathologist examined all histological specimens. Temporal artery biopsies were processed and cut at 4–5 μm thickness from at least 3 levels. Hematoxylin and eosin stains slides were produced from each paraffin block. Cases with visual involvement were admitted to hospital and biopsy was performed within 24 hours and methylprednisone 1mg/kg/day was administered intravenously for 3 days. Follow-up within 1 week was arranged after the biopsy to ensure the improvement of symptoms, compliance with treatment and wound assessment.

Major features assessed to determine the presence of GCA were giant cells in the intima and the media, lymphocytes and histiocytes in the media, reduplication/fragmentation of the internal elastic lamina and intimal thickening. Healed GCA was diagnosed in the presence of intimal fibrosis, media scarring with eccentric and segmented disruption of the internal elastic lamina or chronic media inflammation with neovascularisation. However, if one or more of these features were absent, healed arteritis or atherosclerosis was considered [20]. Biopsy positive cases were placed on oral prednisone 1mg/kg/day immediately upon receiving the biopsy results.

Statistical analysis was performed on the Statistical Package for the Social Sciences (SSPS) v16.0. The Exact method was used in calculating the confidence interval for the incidence of GCA. An independent-sample t-test was used to compare mean differences in clinical characteristics and laboratory findings between the GCA positive and negative groups.

Annual age and gender specific incidence rates were calculated / 100,000 population aged ≥50 years using the number of incident cases as the numerator and population estimates based on census counts as the denominator. Linear interpolation was used to estimate population size for intercensal years [21]. Ninety five percent confidence intervals (95% CI) were computed for incidence rates.

Results

Patient demographics—The population in Otago consists ethnically of Europeans (85.9%), Māori (7.1%), Pacific Islanders (1.8%), Asians (4.5%), Middle Eastern and African (0.7%). Over the 9 years of observation there was an estimated increase in total population numbers by 6265 (3.3%), an increase in the population age group ≥50 years by 9484 (16.1%) and a decrease in the population <50 years by 3219 (2.4%).

Temporal artery biopsies were performed on a total of 363 patients, of which 105 (29%) were males and 258 (71%) were females. The mean age of the population was 73.2 years (range 50–97 years, SD 9.3). The average follow up was 51.9 months (range 1–120, SD 24.9).

A total of 70 (19%) patients with biopsy proven GCA (GCA+ve) were identified. The mean age at diagnosis was 72.8 years (range 57–91 years, SD 8.2). There were 52 (74%) females and 18 (26%) males giving a ratio of 2.9:1.

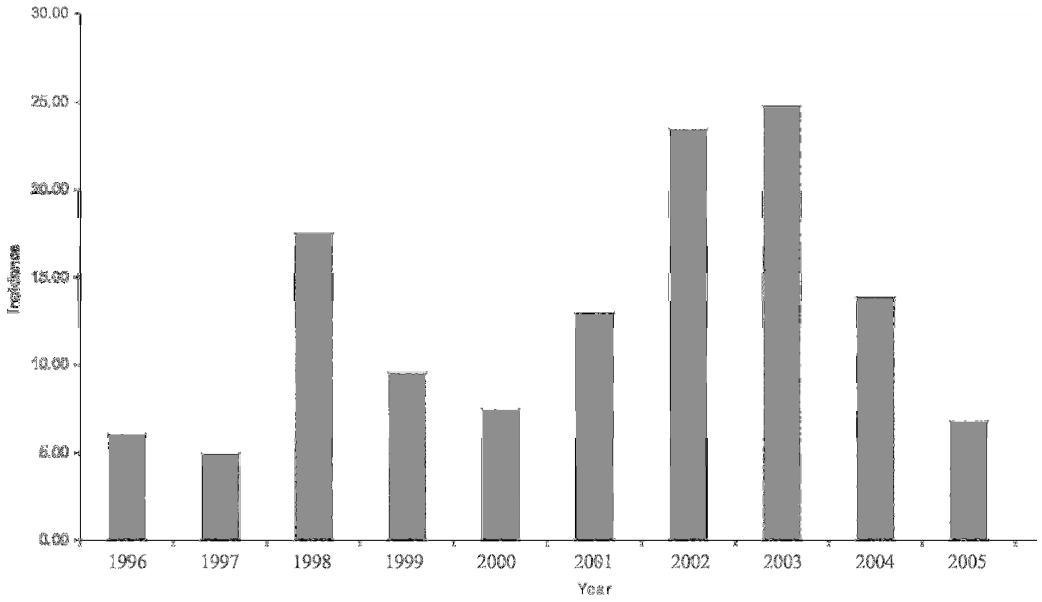
The annual incidence of GCA/100,000 population aged ≥50 years adjusted for gender and age is shown in Table 1, the mean annual incidence in the population ≥50 years was 12.7/100,000 over the 9 years of observation.

Table 1. Incidence of giant cell arteritis/100,000 population in the Otago region for the population age ≥50 years. CI, confidence interval

Variables	Incidence /100,000	95% CI (P<0.5)
All patients	12.7	11.7–14.3
Females	21.3	19.8–24.2
Males	8.8	8.3–10.2
Age 50–64	21.0	20.9–21.5
Age 65–74	144.3	143.2–146.4
Age ≥75	342.7	340.2–347.3

The variation in annual incidence is shown in Figure 1. We observed a cyclic annual incidence with 2 peaks 5 years apart.

Figure 1. Annual incidence of giant cell arteritis/100,000 population in the Otago region 1996–2005 for the population age ≥50 years. The distribution is cyclic with a peak in 1998 and 2003



The incidence rates increased with age and there was a female: male ratio of 2.8:1. Seasonal variations were observed with more cases of GCA diagnosed in the spring 23 (32.8%) than the summer 17 (24.3%), autumn 16 (22.9%), and winter 14 (20%), however the difference between the seasons was not statistically significant (P<0.9).

Among the biopsy positive cases 5 (1.4%) showed lesions consistent with healed giant cell arteritis. Unilateral biopsies were performed on 335 patients. These were positive in 67 cases (18.5%). In 28 cases bilateral sequential biopsies were performed of which 2/28 (7.2%) demonstrated active arteritic lesions and the other case was a lesion consistent with GCA.

There were no reported postoperative complications from the procedure.

A total of 293 (81%) patients were biopsy negative (GCA-ve). The mean age in this group was 73.4 years (range 50–97 years, SD 9.5). There were 88 (30%) males and 205 (70%) females. Discharge diagnoses included autoimmune conditions polymyalgia rheumatica, rheumatoid arthritis, systemic lupus erythematosus and other vasculitides in 54 (18.4%), optic neuropathies in 6 (2%), malignancies in 5 (1.7%), non-vasculitic cerebrovascular accident in 4 (1.4%), septicaemia in 3 (1.0%) and other diagnoses 15 (5.1%). No definite diagnosis was recorded in the medical notes of 206 (70%) cases.

Clinical and laboratory differences between the biopsy positive and negative groups—There was no difference in mean age between the GCA +ve and –ve groups ($p<0.2$) or gender ($p<0.6$). The major presenting symptoms and signs are summarised in Tables 2 and 3 respectively.

Headache was the commonest symptom occurring in 198 (54.5%) patients. Fever was the commonest sign occurring in 38 (10.5%) of patients. In analysing the clinical difference in the GCA +ve and –ve groups, jaw claudication, anorexia and scalp tenderness were the most significant discriminating symptoms (Table 2).

Table 2. Major symptoms in the study population

Symptom	GCA +ve cases	GCA -ve cases	Total cases (%)	P value
Headache	47 (67.1%)	156 (53.2%)	198 (54.5%)	0.1
PMR symptoms	22 (31.4%)	69 (23.5%)	91 (25%)	0.1
Jaw claudication	17 (24.3%)	17 (5.8%)	34 (9.4%)	0.0001
Scalp tenderness	16 (22.9%)	39 (13.3%)	55 (15.2%)	0.003
Malaise	12 (17.1)	39 (13.3%)	51 (14.1%)	0.4
Anorexia	10 (14.3%)	22 (7.5%)	32 (8.8%)	0.005
Weight loss	9 (12.9%)	30 (10.2%)	39 (10.7%)	0.6
Permanent visual loss	10 (14.3%)	31 (10.6%)	41 (11.3%)	0.3
Transient visual loss	7 (10%)	24 (8.2%)	31 (8.5%)	0.7

PMR=Polymyalgia rheumatica, GCA+ve= patients with giant cell arteritis, GCA-ve=patients without giant cell arteritis. The P value indicates the statistical significance of the difference in the prevalence of the symptom in the biopsy positive versus the biopsy negative population.

Among the signs abnormalities of the superficial temporal arteries, a clinical diagnosis of arteritic anterior ischemic optic neuropathy (AAION) and raised inflammatory markers without suggestive clinical symptoms and signs were significant discriminators (Table 3).

Table 3. Major signs in the study population

Signs	GCA +ve cases	GCA -ve cases	Total cases (%)	P value
Fever	9 (12.9%)	29 (9.9%)	38 (10.5%)	0.4
STA tenderness	9 (12.9%)	18 (6.1%)	27 (7.4%)	0.002
STA reduced pulse	6 (8.6%)	1 (0.3%)	7 (1.9%)	0.001
CVA	4 (5.7%)	11 (3.8%)	15 (4.1%)	0.3
Anemia	2 (2.9%)	4 (1.4%)	6 (1.7%)	0.2
Raised inflammatory markers without suggestive clinical symptoms and signs	1 (1.4%)	14 (4.8%)	15 (4.1%)	0.004

STA=superficial temporal artery, AAION=arteritic anterior ischemic optic neuropathy, CVA=cerebrovascular accident, GCA+ve= patients with giant cell arteritis, GCA-ve= patients without giant cell arteritis. The P value indicates the statistical significance of the difference in the prevalence of the sign in the biopsy positive versus the biopsy negative population.

Table 4. The distribution of inflammatory markers in the study population

ESR mm/hr	GCA +ve cases	GCA -ve cases
≤20	3 (4.3%)	34 (11.6%)
21–70	31 (44.3%)	160 (54.6%)
71–120	30 (42.9%)	84 (28.7%)
121–170	6 (8.6%)	14 (4.8%)
≥171	3 (4.3%)	1 (0.3%)
Total	**70**	**293**
CRP mg/dl		
≤5	3 (4.3%)	43 (14.7%)
6–50	18 (25.7%)	64 (21.8%)
51–95	31 (44.3%)	150 (51.2%)
96–140	10 (14.3%)	17 (5.8%)
≥141	8 (11.4%)	19 (6.5%)
Total	**70**	**293**

ESR=erythrocyte sedimentation rate, CRP=C-reactive protein, GCA+ve=patients with giant cell arteritis, GCA-ve=patients without giant cell arteritis.

The mean erythrocyte sedimentation rate (ESR) was 80.4mm/hr (range 14–137, SD 30.1) in the biopsy positive group compared to 66.2 mm/hr (range 1–217, SD 37.9) in the biopsy negative group (P<0.01). The mean C-reactive protein (CRP) was 86.5mg/dl (range 1–441, SD 75.1) in the biopsy positive group compared to 54.2 mg/dl (range 1–328, SD 66.3) in the biopsy negative group (P<0.98).

Table 5 shows the distribution of selected systemic diseases in this series; only polymyalgia rheumatica (PMR) reached statistical significance.

Table 5. Past history of systemic diseases in the study population

Systemic diseases	GCA +ve cases	GCA -ve cases	Total cases (%)	P value
CVA	8 (11.4%)	28 (9.6%)	36 (7.7%)	0.5
Neoplasia	6 (8.6%)	31 (10.6%)	37 (8.5%)	0.2
Diabetes	11 (15.7%)	37 (12.6%)	48 (10.1%)	0.2
IHD	16 (22.8%)	71 (24.2%)	87 (19.6%)	0.6
Hypertension	25 (35.7%)	94 (32.1%)	119 (32.8%)	0.4
PMR	10 (14.3%)	20 (6.8%)	30 (8.3%)	0.0001

CVA=cerebrovascular accident, IHD=ischemic heart disease, PMR=polymyalgia rheumatica, GCA+ve= patients with giant cell arteritis, GCA-ve= patients without giant cell arteritis.. The P value indicates the statistical significance of the difference in the prevalence of the disease in the biopsy positive versus the biopsy negative population.

Visual loss—The cause of permanent visual loss in the GCA +ve population was mainly due to AAION in 9 eyes (8 unilateral cases and 1 bilateral case). The presenting and final visual acuities of these cases ranged between hand movements to no light perception. The optic disc showed a chalky white swollen optic disc in all cases. Central retinal artery and branch retinal artery occlusion were diagnosed in 1 case each. There were no reported cases of improvement in vision in those that presented with AAION.

Discussion

We present the first large epidemiological study in Australasia of patients referred for temporal artery biopsy. The mean annual incidence rate in this study is intermediate between studies reported in populations of European origin[22,23] and those rates reported in studies from Mediterranean countries.[18,24,25] Although the predominant ethnic origin of the population in the Otago province is from the United Kingdom, incidence rates were nearly half of those reported in a population in the United Kingdom by Smeeth et al.[5]

Factors influencing the incidence rates such as the high proportion of ethnic Europeans in Otago, the increasing population age group ≥50 years and increase in awareness of this disease among referring practitioners are likely to be relevant factors.

A cyclic nature of annual incidence was noted in our series; Salvarani et al noted over an observation period of 50 years on the population of Minnesota five peaks in the incidence rate, each of which lasted about 3 years with peaks occurring approximately every 7 years.[2] A close concurrence between the observed incidence peaks of PMR/GCA and epidemics of *Mycoplasma pneumoniae*, parvovirus B19, and Chlamydia pneumoniae has been found in different areas of Denmark[23] A possible link to parvovirus B19 was found in the Olmsted population.[2] We did not investigate this possibility in our study.

The seasonal variation is also suggestive of an environmental factor, this has been reported by other investigators,[5,16] Smeeth et al reported higher rates of both GCA and PMR in during late spring and early summer.[5] Other studies, as ours, did not find any seasonal effect or this effect did not achieve statistical significance.[18,19,26]

Headache was the most common presenting manifestation of GCA as it is in other major epidemiological studies. Jaw claudication was the symptom most characteristic of the GCA+ve versus –ve group. This is in keeping with the traditional clinical teaching that jaw claudication, although somewhat insensitive, is a relatively specific feature for GCA.[27]

Gonzalez-Gay et al in a series of 240 patients reported differences in isolated PMR and PMR associated with GCA. Patients with isolated PMR were significantly younger than those with PMR associated with biopsy-proven GCA and had a lower frequency of anorexia, malaise and weight loss.[28] In our series the age difference was not statistically significant. This may reflect the limited numbers of patients with these symptoms or the limitation of a retrospective study design in which information regarding clinical variables was neither always available nor complete. This could also account for the large proportion of biopsy negative patients without a recorded discharge diagnosis.

ESR and CRP were highly correlated in our study ($P<0.0001$) but only ESR was significantly different in comparing GCA +ve and –ve groups. Both ESR and CRP are sensitive tests for the diagnosis of GCA and their combination increases the sensitivity further [29]. Costello et al in a retrospective analysis of 121 GCA+ve patients compared the sensitivity and specificity of tests used in the diagnosis of GCA and found ESR achieved a sensitivity of 94.2% and a specificity of 80.5%. For CRP these values were 98.6% and 75.7% for the sensitivity and specificity respectively. The failure of CRP to achieve statistical significance in the GCA +ve group may be attributed to the lower specificity of the latter test.[30]

Table 3 demonstrates a statistically significant difference in the prevalence of a positive TAB in the subgroup of 15 cases presenting with a raised inflammatory markers in the absence of suggestive clinical symptoms and signs of GCA; this indicates the low possibility of a positive biopsy in this clinical setting; although high inflammatory markers are a hallmark of GCA, are a nonspecific indicator of inflammation.[28]

The results of the study should be interpreted with caution, as potential sources of bias are detection and collection bias, increasing awareness of GCA among clinicians could increase referral and biopsy rates. In addition the retrospective design of this study limits its yield; however the long period of observation, inclusion only of biopsy proven cases in the GCA +ve group, clinical and laboratory comparisons drawn from the GCA-ve group and the first large epidemiologic study on this disease from a population in Australasia generates interest in its findings.

Conclusion

The first large study of GCA from Australasia demonstrated that a variation in the annual incidence rate for giant cell arteritis in Otago, New Zealand showed a cyclic pattern. The overall incidence seems to reflect the ethnic origins of the majority of the population from Britain.

Competing interests: None.

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