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Biopsy Findings in Giant Cell Arteritis - Relation to Baseline Factors and Corticosteroid Treatment

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Letter

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The diagnosis of giant cell arteritis (GCA) is based on clinical findings as well as histopathologic evidence of vasculitis. In patients with a clinical picture suggestive of GCA, a temporal artery biopsy (TAB) is often used to confirm the diagnosis. A negative biopsy may be the basis for rejecting the diagnosis in cases with atypical presentation and disease course. There are conflicting data on the impact of corticosteroid treatment on TAB findings in GCA (1-4).

We investigated histopathologic features in TAB from a well defined sample of patients with a clinical diagnosis of GCA, and assessed how these features relate to treatment with corticosteroids and baseline inflammatory markers.

Individuals who developed GCA after inclusion in two population based health surveys [the Malmö Diet and Cancer Study (MDCS; N=30447; performed 1991-1996) and the Malmö Preventive Medicine Project (MPMP; N=33346; performed 1974-1992)] performed in the same catchment area were identified by linking the health survey databases to the local patient administrative register and the national hospital discharge register.

In a structured review of medical records, cases were classified according to the American College of Rheumatology criteria for GCA (5), and clinical data regarding GCA, laboratory parameters and results of temporal artery biopsy were extracted.

Cases with a confirmed clinical diagnosis of GCA and details available at the time of the start of corticosteroid therapy and at the time of the TAB were included in this study. Of 83 incident cases in the MPMP and MDCS, 62 could be included in the present investigation. In a structured review of TAB pathology reports, the presence or absence of the following histopathologic features was recorded: giant cells and/or granuloma, fragmented internal elastic lamina, inflammatory infiltrates

and fibrosis.

Sixty-two cases with confirmed GCA diagnosis were included (Table 1). Patients with a positive biopsy had significantly higher CRP ($p=0.002$) and erythrocyte sedimentation rate (ESR) ($p=0.009$) at diagnosis (Table 1). Biopsy positive cases were slightly younger (Table 1). Patients who started treatment with corticosteroids after the biopsy or on the same day were more likely to have inflammatory infiltrates described in the pathology report than those biopsied at a later time point ($p=0.02$), but there were no significant differences in the proportions with a fragmented internal elastic lamina, giant cells and/or granuloma present, or overall positive biopsy report (Figure 1).

The observation that patients with a positive biopsy had significantly higher baseline CRP and ESR levels is in agreement with previous studies (6-7). We did not find the association between higher age and biopsy positivity reported by others (4, 8), but this may be due to the fact that the number of patients aged over 80 years was limited because of the age distribution of the source population.

Our results are compatible with those of Font and Prabhakaran (9), who observed that patients treated for more than 14 days had inflammation limited to the media-adventitia junction whereas the others were more likely to have diffuse infiltrates in the arterial wall. In contrast to their study, we did not see giant cells in fewer patients after treatment. This could be explained by the fact that none of our patients were treated for more than 13 days prior to biopsy.

In a recent study, Deng and co-workers demonstrated that interleukin (IL)-17 producing T-helper (Th)17 cells were sensitive to steroid treatment and interferon (IFN)- γ -producing Th1 cells persisted in patients who had remaining histologic signs of inflammation despite long term treatment (10). This is in agreement with our findings that steroid treatment affects some features of the inflammation seen in TAB.

Limitations of this study include the small sample size, and the relatively short time from

start of corticosteroid treatment to biopsy in all patients. Strengths of the study include the community based approach, reflecting that the patients are representative for GCA cases managed in the area, and the availability of complete medical information and histopathology specimens.

In conclusion, we have demonstrated that inflammatory infiltrates in GCA may be reduced within a few days after starting corticosteroid treatment, but other features of GCA persist for a longer time despite treatment, leading to a similar diagnostic yield in those treated for a brief period. Patients with a positive biopsy had higher baseline CRP and ESR levels.

Table 1.
Characteristics of included patients with GCA and baseline parameters in giant cell arteritis patients with a positive vs. negative temporal artery biopsy

	All cases (n=62)	Biopsy positive (n=43)	Biopsy negative (n=19)	p ¹
Age at GCA diagnosis (years)	70.7 (SD 6.1; range 56-82)	70.0 (SD 6.3; range 58-82)	72.4 (SD 5.4; range 56-79)	0.16
Female sex	45 (73 %)	32 (74 %)	13 (68 %)	0.54
ESR mm/h; mean (SD)	77.9 (27.7)	84.2 (22.7)	64.0 (33.2)	0.009
CRP mg/l; median (IQR)	97 (58-144)	101 (82-173)	54 (25-92)	0.002
Time (in days) from corticosteroid treatment to TAB (median; IQR)	2 (1-4)	2 (1-4)	3 (1-4)	0.92
Initial steroid dose (mg) (median; IQR)	40 (40-60)	40 (40-60)	40 (30-60)	0.79

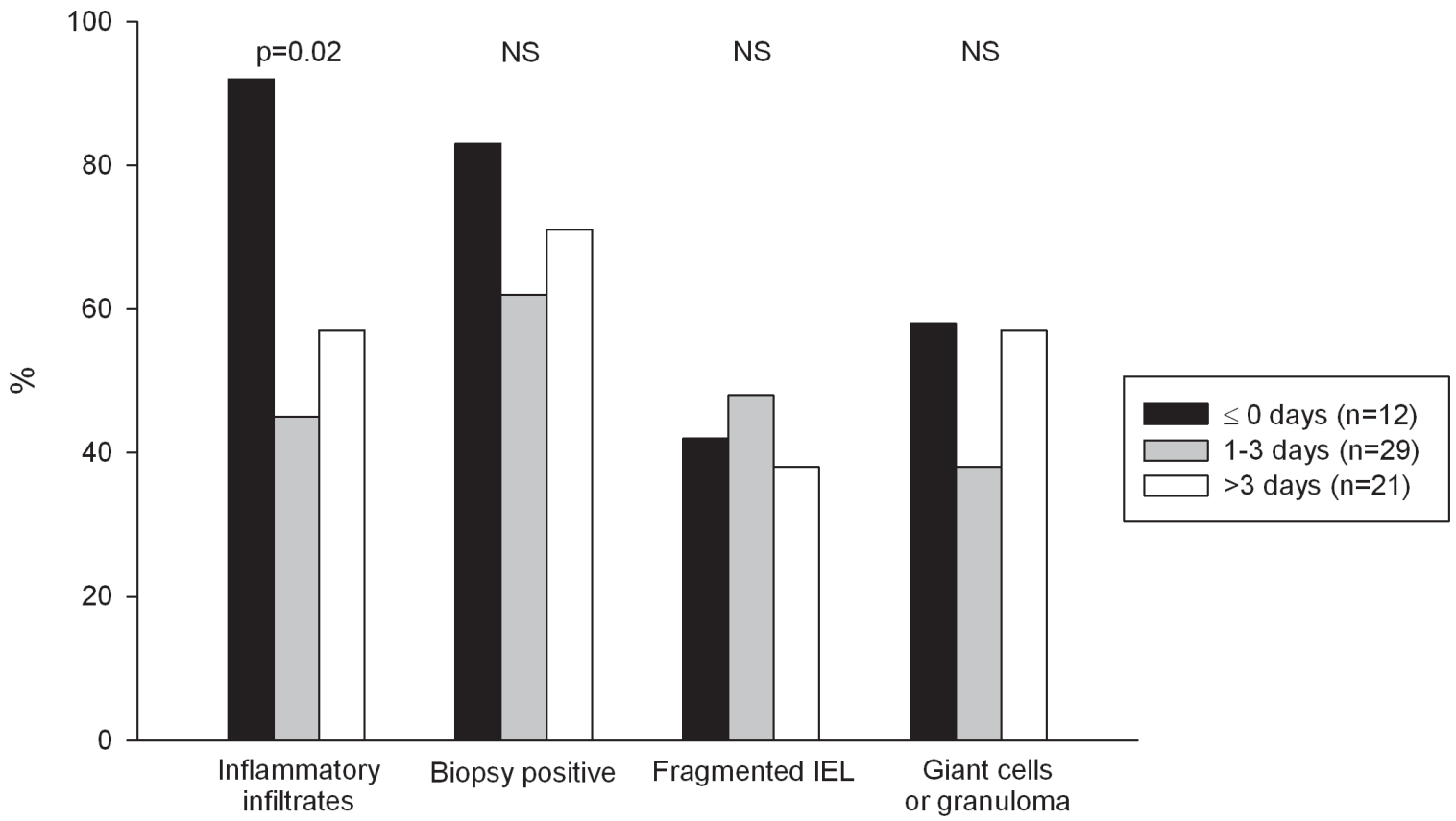
¹ p-value for biopsy positive vs negative

GCA, Giant cell arteritis; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; TAB, temporal artery biopsy; SD, standard deviation; IQR, interquartile range.

Figure legend

Figure 1.

Proportions of patients with features of giant cell arteritis recorded in the pathology reports, stratified by time from initiation of corticosteroid treatment to biopsy. IEL, Internal elastic lamina; NS, not significant.



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