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ORIGINAL ARTICLE

Baseline clinical predictors of an ultimate giant cell arteritis diagnosis in patients referred to temporal artery biopsy

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Abstract The diagnosis of giant cell arteritis (GCA) is based on clinical grounds and confirmed by characteristic histological findings on temporal artery biopsy (TAB). Patients may be diagnosed with GCA based on clinical grounds only, despite negative histological findings. We aimed to investigate which baseline clinical and laboratory features best predict an ultimate diagnosis of giant cell arteritis among patients referred to TAB. We retrospectively analyzed 224 patients who underwent TAB in our hospital between 2000 and 2014. Patients were diagnosed with GCA if TAB was positive for GCA, or by clinical grounds only despite a negative biopsy, provided they fulfilled the American College of Rheumatology 1990 criteria. Baseline clinical and laboratory features were obtained from medical records. Predictors of an ultimate GCA diagnosis were investigated. Overall, 82 patients were diagnosed with GCA—57 had histological evidence of GCA and 25 were diagnosed with GCA despite a negative biopsy. One hundred and forty-two patients were not diagnosed with GCA. Predictors of an eventual diagnosis of GCA in a multivariate logistic regression analysis were headache (OR = 6; $p < 0.001$), jaw claudication (OR 4.5; $p = 0.007$), erythrocyte sedimentation rate (ESR) (OR = 1.5; $p = 0.032$) and platelet count (OR = 1.74; $p = 0.004$). Among patients referred to TAB, headache, jaw claudication, ESR, and thrombocyte levels are predictors for an ultimate diagnosis of GCA. These clinical and laboratory features should be considered when contemplating the diagnosis and treatment of GCA.

Keywords Giant cell arteritis · Temporal artery biopsy · Ultimate diagnosis of giant cell arteritis

Introduction

Giant cell arteritis (GCA) is a vasculitis that involves large and medium-sized vessels, with a predilection for the extra cranial branches of the carotid artery in the elderly [1–3]. The clinical manifestations of GCA are quite varied and can be classified into four subsets: symptoms related to cranial arteritis, extra cranial arteritis, systemic manifestations, and polymyalgia rheumatica (PMR) [4–6]. The diagnosis of GCA is based on clinical grounds. Temporal artery biopsy (TAB) remains the gold standard for the diagnosis of GCA [7], yet it may be normal in up to 20–40 % of the patients [8–13]. In cases of a negative TAB, the American College of Rheumatology (ACR) 1990 criteria may be used to assist in classifying patients as having biopsy-negative GCA [14], although their use as diagnostic criteria in GCA is controversial [15–20]. Previous studies have examined the factors associated with a positive temporal artery biopsy and have found several laboratory and clinical predictors of a positive TAB. Among these are jaw claudication, abnormal temporal artery on physical

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examination, anemia, and thrombocytosis [21–24]. Baseline clinical features of biopsy-proven and biopsy-negative GCA have also previously been examined [12, 25]. Visual complications, abnormal temporal artery on physical examination and jaw claudication were found to be more frequent in the biopsy-proven than in the biopsy-negative group, whereas headaches and PMR were more frequent in the biopsy-negative group. Accordingly, it seems that biopsy-negative GCA constitutes a subset of patients with less severe ischemic complications and distinct features among the wide clinical spectrum of GCA. Therefore, when investigating the features of patients with GCA, we must include the biopsy-negative GCA subgroup. The aim of this study was to investigate the presenting clinical and laboratory predictors of an ultimate diagnosis of GCA, including both subset of patients—biopsy-positive and biopsy-negative.

Materials and methods

We retrospectively reviewed all patients who underwent TAB in the Chaim Sheba medical center between the years 2000 and 2014. Patients' clinical and demographic data were extracted from computerized medical records and manual medical files. We included only cases with complete clinical and laboratory information, including initial clinical presentation, erythrocyte sedimentation rate (ESR), values of complete blood count, and chemistry results, as well as information on whether the diagnosis of GCA was determined and therapy initiated. Post-fixation TAB specimen length was recorded. The research protocol was approved by the local institutional review and complies with the Declaration of Helsinki.

GCA diagnosis

Temporal artery biopsies were performed under local anesthesia by general or ophthalmic surgeons. All patients underwent unilateral biopsies. Diagnosis of biopsy-proven GCA required the histological findings of interruption of the internal elastic laminate with infiltration of mononuclear cells into the arterial wall [26]. Patients were diagnosed with TAB-negative GCA based on clinical judgment of the treating physician, provided the patient's symptoms and signs improved within 3 days of corticosteroid treatment (40 mg of prednisone or more), no other better alternative diagnosis could be reached after a thorough evaluation and clinical follow-up, and the patients fulfilled the ACR 1990 classification criteria for GCA [14].

Clinical and laboratory data

The clinical information collected included the presence of constitutional symptoms, headache, jaw claudication,

Table 1 Baseline clinical and laboratory findings in 224 patients referred to temporal artery biopsy

Variable	
Males—no. (%)	88 (39)
Age (years) ± SD (range)	72 ± 9.6 (34–91)
Headache—no. (%)	116 (52)
Constitutional syndrome—no. (%)	121 (54)
Abnormal temporal artery—no. (%)	39 (17)
Jaw claudication—no. (%)	25 (11)
Polymyalgia rheumatica—no. (%)	58 (26)
Visual manifestations—no. (%)	51 (23)
Cerebrovascular accidents—no. (%)	14 (6.3)
Elevated liver enzymes no. (%)	43 (19)
ESR (mean ± SD) mm/1st hour (range)	82 ± 28 (5–163)
Hemoglobin (g/Dl) ± SD (range)	11.4 ± 1.7 (4.7–16.2)
Platelet count/mm^3—mean ± SD (range)	348 ± 156 (7–980)
Leukocyte count/mm^3—mean ± SD (range)	10.2 ± 5.6 (3.5–66.7)
Leukocyte count > 11,000 microL—no. (%)	68 (30)
Anemia (hemoglobin < 12 g/dl)—no. (%)	146 (65)
Thrombocytosis (platelets > 450x10^3/μl)—no. (%)	48 (21)
Length of temporal artery specimen—cm ± SD (range)	1.12 ± 0.7 (0.2–7.0)
Temporal artery specimen length < 1—cm (%)	96 (43)

Constitutional syndrome: asthenia, anorexia, and weight loss of at least 4 kg

Abnormal temporal artery: tenderness or decreased pulse of the temporal artery

ESR erythrocyte sedimentation rate

symptoms compatible with PMR, visual manifestations, cerebrovascular manifestations and an abnormal temporal artery on physical examination. The following laboratory data was collected: hemoglobin, leukocytes, and platelets levels, ESR and the presence of elevated liver enzymes.

Statistical analysis

Data were analyzed with Statistical Package for the Social Sciences (IBM SPSS statistics) software version 21.0. Continuous variables were expressed as mean ± standard deviation. Categorical variables were expressed as frequencies (percentage). The clinical characteristics of study subjects were compared with chi-square tests for categorical variables and independent *t* tests for continuous variables between two groups: patients who were diagnosed with GCA and patients eventually not diagnosed with GCA. Patients with biopsy-proven GCA and patients with biopsy-negative GCA were similarly compared. Univariate and multivariate Cox proportional hazards regression modeling were performed to predict eventual diagnosis of GCA. All tests were two-tailed, with p values <0.05 being considered as significant.

Results

During the study period, 224 TAB were performed. The mean age of the patients was 72 (±9.6), and 88 (39 %) were males. The most common presenting symptoms were constitutional syndrome (54 %), headache (52 %), and PMR (26 %) (Table 1). Eighty-two patients (36.6 %) were diagnosed with GCA. Among them, 57 (69.5 %) patients were diagnosed based on a positive TAB, and 25 (30.5 %) patients were diagnosed based on clinical grounds despite a negative TAB. Within the group of patients diagnosed with GCA, headache and PMR were more frequent among patients with biopsy-negative GCA than among patients with biopsy-proven GCA. Length of temporal artery specimen was similar among patients with biopsy-proven and biopsy-negative GCA (Table 2).

Among all patients referred to TAB, patients who were diagnosed with GCA had a higher rate of headaches and jaw claudication than patients who were eventually not diagnosed with GCA. In addition, patients diagnosed with GCA had higher ESR, thrombocyte levels, leukocyte levels, and a higher rate of leukocytosis compared to patients who were not diagnosed with GCA. Length of TAB specimen was not significantly different between the groups (Table 3).

Table 2 Baseline clinical and laboratory findings in 82 patients diagnosed with giant cell arteritis: comparative analysis between patients with positive and negative temporal artery biopsy

Variable	All patients diagnosed with GCA $N=82$	Biopsy-positive GCA $N=57$	Biopsy-negative GCA $N=25$	p value
Males – no. (%)	28 (34)	20 (35)	8 (32)	0.786
Age (years ± SD)	73 ± 8.5	73 ± 8.3	73 ± 9.0	0.683
Headache—no. (%)	62 (76)	38 (67)	24 (96)	0.004
Constitutional syndrome—no. (%)	50 (61)	38 (67)	12 (48)	0.111
Abnormal temporal artery—no. (%)	20 (24)	14 (25)	6 (24)	0.957
Jaw claudication—no. (%)	20 (24)	16 (28)	4 (16)	0.241
Polymyalgia rheumatic—no. (%)	26 (32)	14 (25)	12 (48)	0.036
Visual manifestations—no. (%)	22 (27)	15 (26)	7 (28)	0.874
Cerebrovascular accidents—no. (%)	3 (3.7)	3 (5.3)	0	0.243
Elevated liver enzymes no. (%)	17 (21)	11 (19)	6 (25)	0.565
ESR (mean + SD) mm/1st hour ± SD	89 ± 19.5	88 ± 18	91 ± 22	0.473
Hemoglobin (g/Dl) ± SD	11.4 ± 1.43	11.5 ± 1.50	11.2 ± 1.25	0.457
Platelet count/mm^3—mean ± SD (range)	400 ± 150	420 ± 140	353 ± 162	0.063
Leukocyte count/mm^3 mean ± SD (range)	11.4 ± 7.5	10.8 ± 4.2	12.0 ± 2.4	0.388
Leukocyte count >11,000 microL—no. (%)	29 (35)	19 (33)	10 (40)	0.561
Anemia (hemoglobin < 12 g/dl) - no. (%)	54 (66)	37 (65)	17 (68)	0.786
Thrombocytosis (platelets >450x10^3/μl)—no. (%)	24 (29)	18 (32)	6 (24)	0.487
Length of temporal artery specimen—cm ± SD	1.23 ± 0.92	1.27 ± 1.03	1.15 ± 0.62	0.590
Temporal artery specimen length <1 cm (%)	36 (44)	24 (42)	12 (48)	0.620

Constitutional syndrome: asthenia, anorexia, and weight loss of at least 4 kg

Abnormal temporal artery: tenderness or decreased pulse of the temporal artery

ESR erythrocyte sedimentation rate

Table 3 Baseline clinical and laboratory findings in 224 patients referred for temporal artery biopsy: comparative analysis between patients according to eventual diagnosis of GCA

Variable	Patients diagnosed with GCA	Patients not diagnosed with GCA	*p* value
Number	82	142	
Males—no. (%)	28 (34)	60 (42)	0.231
Age (years ± SD)	73 ± 8.5	72 ± 10.2	0.406
Headache—no. (%)	62 (76)	54 (38)	<0.001
Constitutional syndrome—no. (%)	50 (61)	71 (50)	0.112
Abnormal temporal artery—no. (%)	20 (24)	19 (13)	0.036
Jaw claudication—no. (%)	20 (24)	5 (3.5)	<0.001
Polymyalgia rheumatica—no. (%)	26 (32)	32 (22.5)	0.131
Visual manifestations—no. (%)	22 (27)	29 (20)	0.271
Cerebrovascular accidents—no. (%)	3 (3.7)	11 (7.7)	0.223
Elevated liver enzymes no. (%)	17 (21)	26 (19)	0.680
ESR (mean + SD) mm/1st hour ± SD	89 ± 20	78 ± 31	0.002
Hemoglobin (g/Dl) ± SD	11.4 ± 1.43	11.3 ± 1.82	0.643
Platelet count/mm^3—mean ± SD	400 ± 150	318 ± 152	<0.001
Leukocyte count/mm^3—mean ± SD	11.4 ± 7.5	9.5 ± 4.0	0.014
Leukocyte count >11,000 microL—no. (%)	29 (35)	39 (28)	0.014
Anemia (hemoglobin <12 g/dl)—no. (%)	54 (66)	92 (65)	0.872
Thrombocytosis (platelets >450x10^3/μl)—no. (%)	24 (29)	24 (17)	0.530
Length of temporal artery specimen—cm.. ±SD	1.23 ± 0.92	1.05 ± 0.52	0.094
Temporal artery specimen length <1 cm. (%)	36 (44)	60 (42)	0.810

GCA: giant cell arteritis

Constitutional syndrome: asthenia, anorexia, and weight loss of at least 4 kg

Abnormal temporal artery: tenderness or decreased pulse of the temporal artery

* ESR: erythrocyte sedimentation rate

Prevalence of GCA was analyzed according to thrombocyte levels and sex. The study population was divided to tertiles according to their thrombocyte levels (Fig. 1). Prevalence of GCA was significantly higher in both men and women in the higher thrombocyte levels tertiles compared to the lowest tertile ($p < 0.0001$). Using a multivariate logistic regression model, independent predictive variables for eventual diagnosis of GCA among patients referred to TAB were headache (OR = 6, 95 % confidence interval 2.96–12.11), jaw claudication (OR = 4.5, 95 % confidence interval 1.49–13.37), ESR

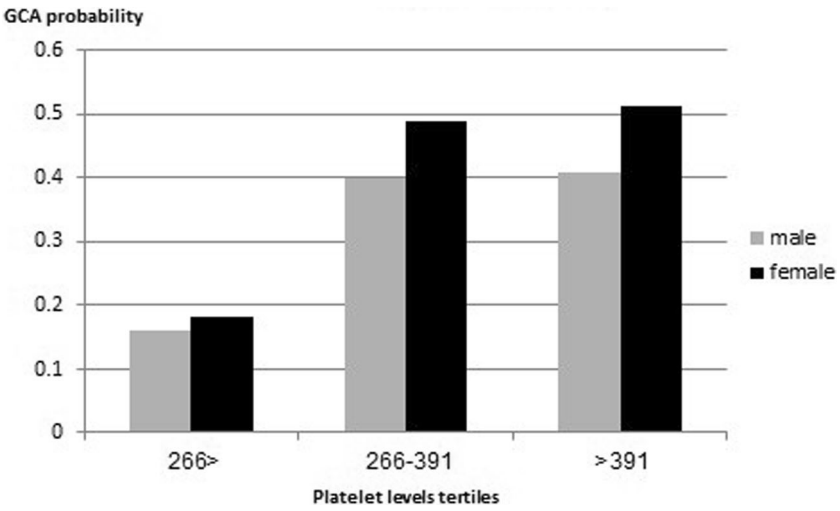

Fig. 1 GCA probability by tertiles of platelet levels and by gender

(OR = 1.5, 95 % confidence interval 1.04–2.17), and platelet levels (OR = 1.74, 95 % confidence interval 1.2–2.52) (Table 4).

Discussion

According to previous studies, yield of TAB for the diagnosis of GCA is 60–80 %. Namely, up to 40 % patients may be diagnosed with GCA based solely on clinical grounds, despite a negative TAB [8–13]. In our study, 30.5 % of the patients diagnosed with GCA had a negative TAB, indicating a relatively low yield of TAB. This may be attributed to the relatively short length of temporal artery biopsies in our cohort (Table 1), as only 55.6 % of the patients who underwent TAB had a temporal artery length ≥1 cm, which has previously been described to be associated with increased diagnostic yield of GCA [27]. Clinical spectrum of biopsy-negative GCA, as well as distinction between biopsy-proven and biopsy-negative GCA, has previously been described [12, 25]. These studies have demonstrated that several clinical differences exist between patients with biopsy-proven GCA and biopsy-negative GCA. Predictors for biopsy-proven GCA were abnormal temporal artery on physical examination, a history of constitutional syndrome, and visual complications. On the other hand, PMR and headaches were more frequent among patients with biopsy-negative GCA. According to these studies, it seems that among patients with GCA, those with biopsy-negative GCA constitute a subset with a less severe disease at the time of diagnosis and during follow-up. These findings are partially compatible with our findings. In our study, PMR and headaches were indeed more frequent in the biopsy-negative GCA group, but no clinical or laboratory variables were found to be more frequent in the biopsy-proven group. This may be attributed to the large study populations in previous studies as compared to our study population. Several studies have examined the factors associated with a positive temporal artery biopsy, and have found some laboratory and clinical predictors of a positive TAB [21–24]. Gonzalez-Lopez et al. found the following clinical and laboratory factors independently associated with a positive TAB: temporal cutaneous hyperalgesia, decreased temporal artery pulse, jaw claudication, recent-onset headache, PMR, weight loss, age, length of surgical specimen, and ESR [21]. Rieck's group found that among patients suspected of GCA, only weight loss and jaw claudication were predictive of a positive TAB. They found no laboratory findings predictive of a positive TAB [22]. Chmelewski et al. compared biopsy-positive and biopsy-negative patients and found an increased incidence of headache, jaw claudication, and prior PMR in biopsy-positive patients, but the sensitivity and specificity of these indicators were relatively low. Other clinical and laboratory parameters were similar between the two groups. Based on their findings, they concluded that presenting features are seldom helpful in predicting biopsy results [23]. Matthew's groups retrospectively analyzed a very large population study of patients who had a TAB performed, in order to identify laboratory predictors of a positive TAB. They found that the odds of a positive biopsy were 1.5 greater with an ESR of 47 to 107 mm/h., 5.3 times greater with a C-reactive protein level >2.45 mg/dl, and 4.2 times greater with platelets $>400{,}000 \times 10^3/\mu l$ [24]. The finding that ESR and thrombocyte levels are predictors of a positive TAB is similar to our findings. We found that ESR and thrombocyte levels are independent predictors for eventual diagnosis of GCA. Moreover, we found that the prevalence of GCA was significantly higher in both men and women in the higher thrombocyte levels tertiles compared to the lowest tertile (Fig. 1). The fact that these laboratory findings were not found to predict a positive TAB in some of the previous studies may be related to the relatively small number of patients with a positive TAB in these studies, which precluded reaching a significant difference in laboratory parameters among these patients [22, 23]. Regarding clinical features-jaw claudication was consistently found to predict a positive TAB. Other clinical features were not consistent between different studies [21–23]. As mentioned above, Gonzalez-Lopez's group found several clinical features predictive of a positive TAB. In our study, we found only two clinical features predictive for a diagnosis of GCA: jaw claudication and headache. Our study differs from previous studies mentioned above, as we did not focus only on patients with a positive TAB only. As previously mentioned, up to 40 % of the patients may be diagnosed with GCA based solely on clinical grounds, despite a negative TAB [8–13]. This subset of biopsy-negative GCA patients are probably somewhat different from the biopsy-positive GCA patients [12, 25]. Therefore, when analyzing patients with GCA, we must include this relatively large subgroup of patients with a negative biopsy which are diagnosed solely on clinical grounds. In our study, we included all patients eventually diagnosed with GCA. Accordingly, we analyzed the predictors of eventual diagnosis of GCA among all patients referred to TAB. In daily practice many patients are diagnosed with GCA

Table 4 Predictive variables for eventual diagnosis of GCA among patients referred to temporal artery biopsy

Variable	OR (95 % CI)	P - value
Headache	6.0 (2.96-12.11)	<0.001
Jaw claudication	4.5 (1.49-13.37)	0.007
ESR (SD=)	1.5 (1.04-2.17)	0.032
Platelet (SD=)	1.74 (1.20-2.52)	0.004

* GCA: Giant cell arteritis

** All univariate significant variables, including age and gender, were entered into the model using forward likelihood method

despite a negative biopsy. Therefore, when contemplating the diagnosis of GCA, the clinical and laboratory predictors for eventual diagnosis of GCA are of great significance. To the best of our knowledge, this is the first study that analyzed the predictors of an eventual diagnosis of GCA, including biopsy-negative GCA, among patients referred to TAB. We believe the results of our study are more relevant to daily clinical practice, as they reflect the whole spectrum of GCA patients. In conclusion, we found that among patients referred to TAB, those with headache, jaw claudication, elevated ESR and thrombocyte levels are more likely to be eventually diagnosed with GCA. Thus, when contemplating the diagnosis and treatment of GCA, these clinical and laboratory features should be considered.

Compliance with ethical standards

Disclosures None.

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