THE AMERICAN COLLEGE OF RHEUMATOLOGY 1990 CRITERIA FOR THE CLASSIFICATION OF GIANT CELL ARTERITIS

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Criteria for the classification of giant cell (temporal) arteritis were developed by comparing 214 patients who had this disease with 593 patients with other forms of vasculitis. For the *traditional format classification*, 5 criteria were selected: age ≥50 years at disease onset, new onset of localized headache, temporal artery tenderness or decreased temporal artery pulse, elevated erythrocyte sedimentation rate (Westergren) ≥50 mm/hour, and biopsy sample including an artery, showing necrotizing arteritis, characterized by a predominance of mononuclear cell infiltrates or a granulomatous process with multinucleated giant cells. The presence of 3 or more of these 5 criteria was associated with a sensitivity of 93.5% and a specificity of 91.2%. A *classification tree* was also constructed using 6 criteria. These criteria were the same as for the traditional format, except that elevated erythrocyte sedimentation rate was excluded, and 2 other variables were included: scalp tenderness and claudication of the jaw or tongue or on deglutition. The classification tree was associated with a sensitivity of 95.3% and specificity of 90.7%.

Giant cell (temporal) arteritis (GCA) was described by Hutchison in 1890 (1) and by Horton et al in 1932 (2). In the years following Horton's description, giant cell arteritis was considered to be an unusual condition, but today, it is recognized more frequently, especially in some geographic areas. Epidemiologic studies have shown an incidence rate as high as 15–30 cases per year per 100,000 persons over the age of 50 in some populations (3,4).

The etiology of GCA is unknown. Reports of its presence in first-degree relatives of GCA patients, a predilection for the disease to occur in Caucasians, and an association with HLA-DR4 suggest a genetic predisposition. Variations of incidence rates in different geographic areas may imply some influence of climate or other geographic factors. The increasing incidence after age 50 and predominance in women suggest a relationship to aging and, perhaps, hormonal changes (5).

Giant cell arteritis produces a broad range of symptoms, but most patients have clinical findings relatable to involved arteries at some time during the illness (4,6). Frequent manifestations include fatigue, fever, headaches, jaw claudication, loss of vision, scalp tenderness, polymyalgia rheumatica, and aortic arch syndrome. GCA differs from other forms of vasculitis in that the skin, kidneys, and lungs are rarely

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involved. The disease may begin abruptly or it may begin gradually, over a number of months, before becoming clinically recognizable.

Blood tests reflect the underlying inflammatory processes. The erythrocyte sedimentation rate is usually highly elevated, but may be normal or only slightly increased in 1% or 2% of patients with active disease (7). Other acute-phase reactant serum proteins are similarly elevated (8). Additional laboratory alterations include a moderate normochromic normocytic anemia, increased platelet count, decreased serum albumin levels, increased factor VIII antigen, and elevated levels of hepatic enzymes (4,6,9).

The medium-sized extracranial arteries are most frequently affected clinically, but in some cases, the aorta and its primary branches to the upper extremities and neck or elsewhere are involved (10). The inflammatory lesions are usually scattered irregularly along the courses of involved vessels, but longer segments may be affected in a continuous manner. Histologically, a granulomatous inflammatory process is seen that is usually focused along the internal elastic lamina. At times, this leads to occlusion of the blood vessel or weakening of the vessel wall and subsequent rupture. The pathologic changes seen in this condition are described by Lie et al elsewhere in this issue of *Arthritis and Rheumatism* (11).

Corticosteroids are highly effective in the treatment of GCA. Reversible symptoms and findings usually become normal within 1 month of treatment with 40–60 mg of prednisone per day or an equivalent dose of a similar corticosteroid (12). Giant cell arteritis tends to run a self-limited course, lasting several months to several years (13). Exacerbations or recurrences are seen in some patients.

For a description of the patient selection and evaluation methods, see the article by Bloch et al (14), which appears elsewhere in this issue of *Arthritis and Rheumatism*.

RESULTS

Patient population. Two hundred fourteen patients were entered into this study with the diagnosis of giant cell (temporal) arteritis. Table 1 shows the age at onset of symptoms and the sex distribution of the patients. Seventy-five percent were women, and the mean age at disease onset was 69 years. As in previous reports of GCA, approximately 90% of the patients were over the age of 60 when the disease began. The classification criteria that were selected were based on the analysis of findings in these patients compared with those in the 593 control patients with other forms of vasculitis.

Table 1. Sex distribution and age at disease onset in 214 patients with giant cell (temporal) arteritis

Age at disease onset	Men (n = 54)	Women (n = 160)	Total
45–59	7	12	19
60–69	19	63	82
70–79	23	72	95
80+	5	12	17
Undefined	0	1	1

Table 2 lists 33 variables which were chosen initially as potentially important discriminators against other forms of vasculitis. The individual items were selected from inspection of the results of univariate analysis of all items on the data collection form, and the combined items were derived by selecting the best combinations of individual variables. The number of cases and controls (all subjects in whom the particular variable was determined), the sensitivity (the percentage of cases with a defined value in whom that variable was positive or abnormal), and the specificity (the proportion of controls in whom the variable was negative or normal) are also shown in Table 2.

Two single items and 8 combined items were selected from among those in Table 2 as a "short list" of criteria that would have the greatest potential to separate cases of GCA from the control cases.

Traditional format classification. Approximately 30 combinations of the 10 variables in the "short list," varying from sets of 3 criteria to a set using all 10, were tested before a final set was chosen to classify giant cell arteritis in the traditional format. Table 3 lists the final set of criteria with their definitions. A patient shall be classified as having GCA if at least 3 of these 5 criteria are met. The presence of any 3 or more of the 5 criteria is associated with a sensitivity of 93.5% and a specificity of 91.2%. Some other criteria lists (not shown) also had sensitivity and specificity values that approached those of the criteria set chosen. For example, when all 10 criteria in the short list in Table 2 are used, the presence of 4 or more yields a sensitivity of 90.3% and a specificity of 89.8%.

In this traditional classification format, 14 (6.5%) of the subjects entered as a diagnosis of GCA failed to meet the criteria; that is, they were not classified as having GCA. These 14 patients met a total of 26 criteria from Table 3, an average of less than 2

Table 2. Comparison of the sensitivity and specificity of potential criteria variables for giant cell (temporal) arteritis*

Criterion	No. of patients (n = 214)	No. of controls (n = 593)	Sensitivity (%)	Specificity (%)
History				
1. Age at disease onset ≥50 years†‡§	213	588	98.6	63.8
2. Headache, new, localized†‡§	214	590	64.5	81.9
3. Jaw claudication	213	579	38.5	97.9
4. Tongue claudication	213	578	2.8	99.8
5. Claudication on deglutition	212	577	4.2	99.0
6. Claudication, variables 3–5†§	212	576	40.6	97.6
7. AM stiffness neck/torso	211	586	50.2	86.5
8. AM stiffness shoulders/arms	210	586	52.9	77.5
9. AM stiffness hips/thighs	211	586	46.9	79.7
10. Polymyalgia rheumatica, variables 7–9 (2 out of 3)†	210	585	52.9	79.3
11. Diplopia	213	587	11.3	93.9
Physical				
12. Ischemic optic neuritis	212	580	7.5	98.4
13. Amaurosis fugax	214	588	11.2	95.7
14. Partial unilateral loss of vision	213	587	4.2	99.0
15. Complete unilateral loss of vision	212	585	3.3	99.7
16. Partial bilateral loss of vision	214	584	2.3	99.1
17. Optic atrophy	212	586	4.7	99.0
18. Visual abnormality, variables 11–17†	210	573	27.6	88.8
19. Right TA tenderness	212	495	23.1	99.6
20. Decreased right TA pulse	210	477	35.2	97.9
21. Left TA tenderness	211	494	21.3	99.2
22. Decreased left TA pulse	209	476	28.7	97.9
23. TA abnormality, variables 19–22†‡§	211	473	57.3	96.8
24. Scalp tenderness	212	584	40.6	97.9
25. Scalp nodules	212	585	13.7	99.5
26. Scalp tenderness or nodules†§	212	581	43.9	97.4
Laboratory				
27. ESR (Westergren) ≥50 mm/hour†‡	207	514	86.5	47.7
28. Serum alkaline phosphatase >1.5 times normal†	197	449	11.2	81.1
29. Serum aspartate aminotransferase >1.5 times normal	203	461	8.4	80.7
30. Serum alkaline phosphatase or aspartate aminotransferase >1.5 times normal	195	439	17.4	69.7
Artery biopsy				
31. Predominantly mononuclear cell infiltration	211	320	90.0	80.6
32. Presence of granulomatous inflammation with giant cells	210	322	84.3	88.2
33. Abnormal biopsy, variables 31 or 32†‡§	211	320	92.9	73.1

* Values are the number of cases or controls with the variable described or tested. The sensitivity is the proportion of cases positive for the variable tested or described. The specificity is the proportion of controls negative for the variable tested or described. TA = temporal artery; ESR = erythrocyte sedimentation rate.
† Criterion is one of the final "short list" of variables (n = 10) (see text).
‡ Criterion is used for the traditional format classification.
§ Criterion is used for the tree classification.

per patient. The criteria that were present included headache in 1 patient, biopsy showing arteritis in 8 patients, erythrocyte sedimentation rate >50 mm/hour in 5 patients, and age ≥50 years at disease onset in 12 patients. Other than age ≥50 at disease onset, the patients met a total of 14 criteria, an average of 1 per patient.

Also in the traditional format, 52 (8.8%) of the

Table 3. 1990 criteria for the classification of giant cell (temporal) arteritis (traditional format)*

Criterion	Definition
1. Age at disease onset ≥50 years	Development of symptoms or findings beginning at age 50 or older
2. New headache	New onset of or new type of localized pain in the head
3. Temporal artery abnormality	Temporal artery tenderness to palpation or decreased pulsation, unrelated to arteriosclerosis of cervical arteries
4. Elevated erythrocyte sedimentation rate	Erythrocyte sedimentation rate ≥50 mm/hour by the Westergren method
5. Abnormal artery biopsy	Biopsy specimen with artery showing vasculitis characterized by a predominance of mononuclear cell infiltration or granulomatous inflammation, usually with multinucleated giant cells

* For purposes of classification, a patient shall be said to have giant cell (temporal) arteritis if at least 3 of these 5 criteria are present. The presence of any 3 or more criteria yields a sensitivity of 93.5% and a specificity of 91.2%.

593 controls were misclassified as having GCA because they met 3 or more of the criteria shown in Table 3. Entry diagnoses in these cases included polyarteritis nodosa in 21, Churg-Strauss syndrome in 1, Wegener's granulomatosis in 21, hypersensitivity vasculitis in 4, Henoch-Schönlein purpura in 1, Takayasu arteritis in 1, leukocytoclastic vasculitis in 1, and unspecified vasculitis in 2. The 52 misclassified control patients met a total of 163 criteria from Table 3, slightly more than 3 per patient. Criteria present were headache in 29, positive artery biopsy in 35, elevated erythrocyte sedimentation rate ≥50 mm/hour in 46, age ≥50 years at disease onset in 43, and temporal artery tenderness in 10. Only 3 misclassified controls had undergone a temporal artery biopsy.

Tree classification. Figure 1 shows the best of several tree classifications derived using the computer program CART (15). All 10 variables in the "short list" in Table 2 were included as potential discriminators. Criteria that are used in the tree classification are

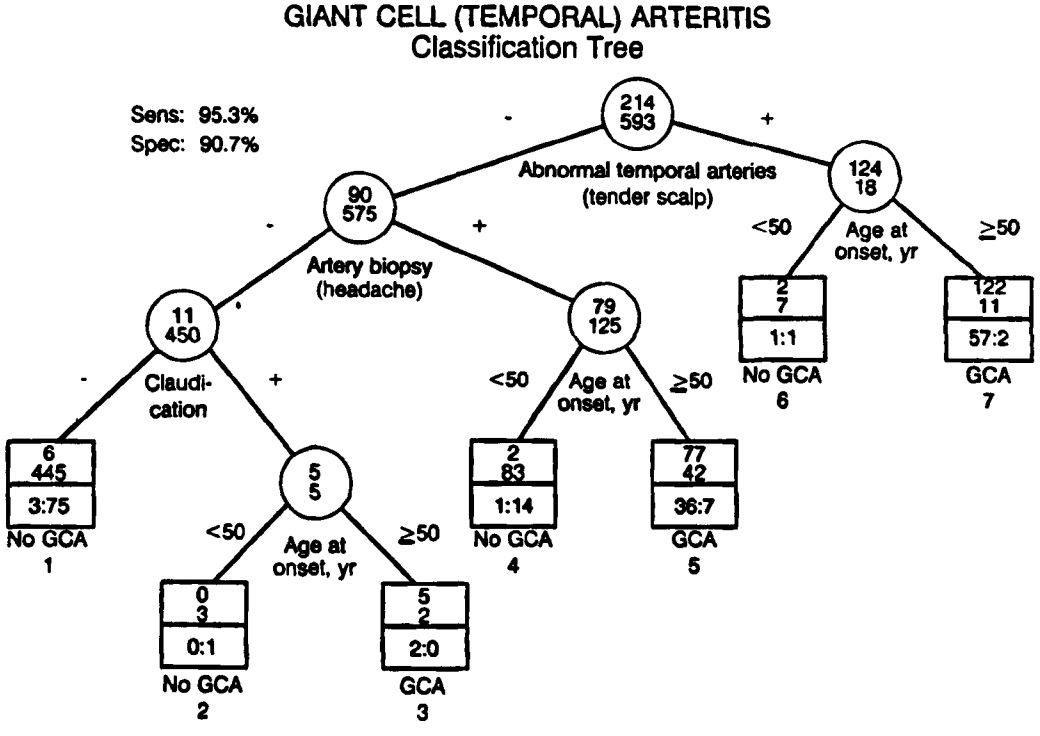

Figure 1. Classification tree for giant cell (temporal) arteritis (GCA). The circles and boxes contain the number of patients with GCA (top number) and the number of control patients with other forms of vasculitis (bottom number). The bottom half of the boxes shows the percentage of patients with GCA (out of all GCA cases) (left number) and the percentage of controls (out of all controls) (right number). Boxes specify whether patients are classified as having GCA or not having GCA (No GCA). The numbers under these specifications are the subset numbers (see Table 4 for definitions of criteria and Table 5 for explanations of subsets). Parentheses indicate the surrogate variable to be used when the first variable is not defined.

Table 4. Criteria and definitions used for the classification of giant cell (temporal) arteritis (tree format)

Criterion	Definition
1. Age at disease onset ≥50 years	Development of symptoms or findings beginning at age 50 or older
2. New headache*	New onset of or new type of localized pain in the head
3. Claudication of jaw, tongue, or on deglutition	Development or worsening of fatigue or discomfort in muscles of mastication, tongue, or swallowing muscles while eating
4. Temporal artery abnormality	Temporal artery tenderness to palpation or decreased pulsation, unrelated to arteriosclerosis of cervical arteries
5. Scalp tenderness or nodules*	Development of tender areas or nodules over the scalp, away from the temporal artery or other cranial arteries
6. Abnormal artery biopsy	Biopsy specimen with artery showing vasculitis characterized by a predominance of mononuclear cell infiltration or granulomatous inflammation, usually with multinucleated giant cells

* Used as a surrogate if artery biopsy is not available (criterion 2) or if temporal artery abnormality is not present (criterion 5).

defined in Table 4. In the classification tree, the presence of temporal artery tenderness or decreased pulsation separated cases from the controls better than any other criterion. When this information was not available, scalp tenderness was used as a surrogate, since it separated the patients in a manner that best approximated the original variable. When biopsy was not done, headache served as a surrogate. The classification tree resulted in an overall sensitivity of 95.3% and specificity of 90.7%.

The classification tree in Figure 1 contains 3 case subsets (numbers 3, 5, and 7) and 4 control subsets (numbers 1, 2, 4, and 6) (see also Table 5). All cases were classified by the presence of 2 criteria. In subset 7, in which 122 cases were classified, criteria were either temporal artery tenderness or decreased pulse (119 cases) or the surrogate of scalp tenderness (3 cases), and age ≥50 years (122 cases). These 122 cases had a total of 789 short-list criteria or an average of 6.5 criteria per patient. In case subset 5, all 77 cases had a positive artery biopsy; these 77 met an average of 4.8 criteria. The 5 cases correctly classified in subset 3 had a total of 28 criteria, or 5.6 criteria per case. Thus, although classification of cases as GCA was based on the presence of only 2 criteria, several other additional criteria were also present in most. Cases in which a positive biopsy was used for classification had fewer total criteria.

In the tree classification, 10 cases (4.7%) with an entry diagnosis of GCA failed to fulfill criteria requirements and were not classified as having giant

Table 5. 1990 classification tree for giant cell (temporal) arteritis (GCA)*

GCA subsets	No. of patients GCA/non-GCA	% correctly classified	% GCA patients in subset	Non-GCA subsets	No. of patients GCA/non-GCA	% correctly classified	% non-GCA patients in subset
7. Age ≥50 at disease onset and tenderness or decreased temporal artery pulsation	122/11	92	57	1. Vasculitis, with normal temporal arteries, no claudication, and negative biopsy	6/445	99	75
5. Age ≥50 at disease onset and artery biopsy showing mononuclear cell or granulomatous inflammation, with clinically normal temporal arteries	77/42	65	36	2. Vasculitis, age <50 at disease onset, claudication, normal temporal arteries, and biopsy without specified findings	0/3	100	<1
3. Age ≥50 at disease onset, claudication, normal temporal arteries and negative biopsy	5/2	71	2	4. Vasculitis, age <50 at disease onset, clinically normal temporal arteries, and biopsy with specified findings	2/83	98	14
				6. Vasculitis, with abnormal temporal arteries, age <50 at disease onset	2/7	78	1

* The subset numbers also appear below the subset boxes in Figure 1. Missing data rules: If temporal artery findings are not available, substitute scalp tenderness; if biopsy findings are not available, substitute headache; if other findings are not available, the vasculitis cannot be classified. The classification tree yields a sensitivity of 95.3% and a specificity of 90.7%. See Table 4 for definitions of criteria.

cell arteritis. Manifestations of these 10 patients included headache in 4, temporal artery tenderness or decreased pulse in 2, scalp tenderness in 3, polymyalgia rheumatica in 5, elevated erythrocyte sedimentation rate ≥50 mm/hour in 7, elevated serum alkaline phosphatase or aspartate aminotransferase levels in 1, abnormality of vision in 2, positive artery biopsy in 4, and age ≥50 years at disease onset in 6. Thus, although these patients had an average of slightly over 3 criteria per patient, individually, none of them fulfilled classification requirements.

Fifty-five control patients (9.3%) were misclassified as having GCA by the tree method. Entry diagnoses in these included polyarteritis nodosa in 18, Churg-Strauss syndrome in 4, Wegener's granulomatosis in 18, hypersensitivity vasculitis in 6, Henoch-Schönlein purpura in 2, Takayasu arteritis in 1, unspecified arteritis in 4, arteritis associated with a myeloproliferative disease in 1, and lymphomatoid granulomatosis in 1. Criteria manifested by these patients included headache in 18, temporal artery tenderness or decreased pulse in 10, scalp tenderness in 9, polymyalgia rheumatica in 15, elevated erythrocyte sedimentation rate ≥50 mm/hour in 32, elevated alkaline phosphatase or aspartate aminotransferase levels in 18, jaw, tongue, or deglutition claudication in 6, abnormalities of vision in 11, age ≥50 years in 53, and positive artery biopsy in 40. Only 2 misclassified controls had undergone temporal artery biopsy.

DISCUSSION

Although giant cell arteritis may present in a wide variety of ways, the majority of cases form a definable clinical process. This is reflected by the identification of 2 classification criteria rules with sensitivity and specificity values higher than 90% that require only 5 items in the traditional format and 6 in the tree format. The criteria were derived by a careful study of information on the data collection forms obtained from a large number of patients (n = 1,000). After formulating a list of important clinical findings as potential criteria (Table 2), a "short list" of 10 criteria was selected and analyzed more extensively. Some criteria, although characteristic manifestations of GCA, were not useful in separating this disease from other forms of vasculitis because of low sensitivity or specificity. These criteria were not included in the final criteria sets. Thus, typical symptoms, such as abnormal vision and polymyalgia rheumatica, were not used. Although the erythrocyte sedimentation rate was included as a criterion in the traditional format, its low specificity (48%) excluded it as a criterion in the tree format.

The classification tree analysis divided the entire population of cases and controls into 7 subsets. Three of these represent cases of GCA, and 4 represent vasculitis control subsets (Figure 1 and Table 5). More than half of the cases were classified correctly and with high accuracy by the presence of age ≥50 years at disease onset and tenderness or decreased pulsation of a temporal artery. This basic combination of findings is uncommon in other forms of vasculitis. An additional 36% who did not have normal temporal artery findings were classified by the presence of age ≥50 years at disease onset and an artery biopsy showing vasculitis with a predominance of mononuclear cells or granulomatous inflammation. A small number of other cases were also classified with age ≥50 years at disease onset and the presence of claudication of the jaw or tongue or on deglutition; they had normal temporal arteries and negative biopsy. Thus, the tree classification defines giant cell (temporal) arteritis in relatively simple terms, as a vasculitis beginning at age 50 or older, with abnormal temporal arteries on physical examination or claudication of the jaw or tongue or on deglutition, and biopsy of an artery showing vasculitis with a predominance of mononuclear cells or granulomatous inflammation. Conversely, vasculitis without an abnormality of a temporal artery, claudication of the jaw or tongue or on deglutition, and an absence of predominantly mononuclear cells or granulomatous inflammation on an artery biopsy excludes most giant cell (temporal) arteritis patients with a high degree of accuracy (Table 5).

In this series, 196 of the 214 GCA patients had positive biopsies (showed arteritis), and 18 lacked biopsy proof. Fifteen of these 18 had biopsies performed, but there was no arteritis, and in 3 patients, a biopsy was not performed. Twelve of the 18 patients without biopsy proof were classified as having GCA by the traditional format rule. In 11 of these 12, the biopsy was negative, and in 1, a biopsy was not done. These 12 correctly classified patients had an average number of 5.4 short-list criteria, as follows: headache in 11, scalp tenderness in 2, temporal artery tenderness in 6, polymyalgia rheumatica in 10, elevated erythrocyte sedimentation rate in 11, elevated aspartate aminotransferase or alkaline phosphatase levels in 4, claudication in 6, abnormal vision in 3, and age ≥50 at disease onset in all 12.

With the tree format, 12 of the 18 patients who lacked biopsy proof of GCA were also classified as

having giant cell arteritis. In 10 of them, the biopsy was negative for GCA, and in 2, a biopsy was not done. These 12 patients met a total of 106 short-list criteria, a high average of 6.2 criteria per patient. As noted earlier, in the tree format, patients classified as having GCA without using biopsy findings as a criterion tended to have met more criteria than those in whom a biopsy was used for classification. Thus, patients without biopsy proof of GCA who were classified as having GCA with both the traditional and tree formats appeared to be similar in other respects to those who had a positive biopsy. Moreover, proportionately fewer cases were classified as GCA by the traditional rule (12 of 200, or 6%) and the tree format (12 of 204, or 5.9%) when biopsy proof was lacking than in the overall group of 214 GCA patients (18 of 214, or 8.4%). If temporal artery biopsy had been specified, fewer control patients would have been misclassified. A number of patients with Wegener's granulomatosis, polyarteritis nodosa, and other forms of vasculitis had biopsies of lung or other tissues that showed chronic inflammatory granulomatous arteritis, along with enough additional criteria to misclassify them as having GCA. However, biopsy of a temporal artery was not specified as a criterion because few controls had undergone this procedure and comparative analysis would not have been possible.

The use of either criteria set is relatively straightforward and easy to apply. Biopsy is the only invasive test; this procedure can be done under local anesthesia, and there is a relatively low morbidity rate associated with it. Routine histologic staining is adequate for determining the presence of necrotizing arteritis with mononuclear cell infiltration or granulomatous arteritis. There are no exclusions other than the presence of a connective tissue disease, which helps to simplify the use of the criteria. In the tree classification, the surrogates of scalp tenderness and headache provide greater flexibility for the use of these criteria when some clinical data are not available.

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