Clinical Course and Management of a Consecutive Series of Patients with "Healed Temporal Arteritis"

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ABSTRACT. ***Objective.*** To describe the clinical course and management of patients with a pathologic diagnosis of "healed" giant cell arteritis (GCA), and to determine whether previously published histological descriptions of healed arteritis can identify patients with a greater likelihood of clinically significant arteritis.

Methods. All temporal artery biopsy reports between 1994 and 2003 were examined for a diagnosis of "healed arteritis." Two rheumatologists abstracted the medical record for presenting features, physical findings, comorbid conditions, and data on treatment and outcomes. One pathologist, blinded to the clinical data, reviewed all specimens and reinterpreted the biopsies according to published histological descriptions of healed arteritis.

Results. Forty-seven patients with an initial pathologic diagnosis of healed arteritis were identified. In 54% of these patients, corticosteroid therapy did not change after the diagnosis of healed arteritis was documented in the pathology report. Seventy percent were ultimately treated with no corticosteroids or low-moderate corticosteroid regimens. Only 32% of the initial cases were confirmed upon review of the biopsies using standardized histological criteria. Patients with confirmed healed arteritis were more likely to have a documented history of polymyalgia rheumatica/GCA and a longer duration of corticosteroid treatment before biopsy. These patients were not more likely to have adverse outcomes.

Conclusion. In this case series, the diagnosis of healed arteritis had little effect on treatment decisions. In most cases, the initial pathologic diagnosis of healed arteritis was not confirmed when biopsies were reviewed by a single pathologist using uniform histological criteria. (First Release Dec 1 2011; J Rheumatol 2012;39:295–302; doi:10.3899/jrheum.110317)

Key Indexing Terms:
GIANT CELL ARTERITIS TEMPORAL ARTERITIS BIOPSY

Giant cell arteritis (GCA) is the most common systemic vasculitis, with an incidence of 18.5/100,000 among whites over 50 years old[1]. Left untreated, GCA can cause significant morbidity, such as blindness, aortic dissection, and even death[2,3].

Temporal artery biopsy is the "gold standard" for the diagnosis of GCA. Histological interpretations include active arteritis, healed arteritis, arteriosclerosis/atherosclerosis, and normal. Although the histological characteristics of active arteritis are well defined and universally acknowledged[4,5,6], no widely accepted, standardized definitions for healed arteritis exist. Changes seen in healed arteritis, such as irregular intimal thickening, intimal and medial fibrosis, focal areas of persistent chronic inflammation[7], confluent loss of the elastic lamella, and medial neovascularization[8], may also be observed in patients with arteriosclerosis/atherosclerosis or with normal aging in the absence of a history of GCA[9,10,11]. As a result, patients currently labeled with a diagnosis of healed arteritis may be a heterogeneous group, encompassing a wide range of entities, including actual healed arteritis, arteriosclerosis/atherosclerosis, active GCA, and non-GCA diagnoses. It is not clear whether a diagnosis of healed arteritis on a pathology report has useful implications for treating physicians and their patients.

We studied the implications of a pathology report containing a diagnosis of healed arteritis. We described the clinical course and outcomes of patients with a histological diagnosis of healed arteritis at 1 academic center. All biopsies were also reexamined by a single pathologist using uniform criteria based on previously published histological descriptions of healed arteritis. Subgroup analyses were performed to determine whether these histological descriptions could identify a subgroup of patients that differed clinically from patients who did not conform to these descriptions.

MATERIALS AND METHODS

We reviewed the pathology reports of 415 temporal artery specimens from consecutive biopsies performed between 1994 and 2003 at 1 academic center (Figure 1). For the 15 patients who had bilateral temporal artery biopsies during the study period, only the first biopsy was used. All patients with a histo-

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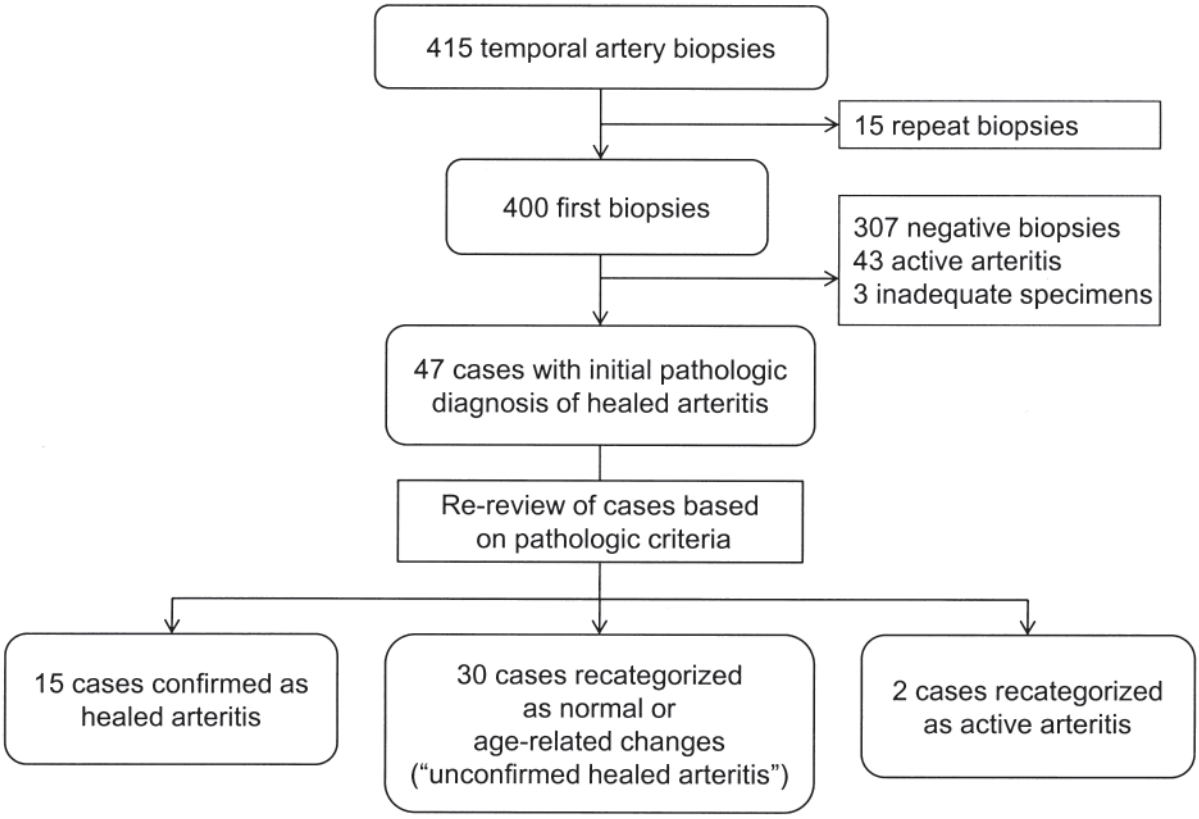

Figure 1. Over 10 years, 415 temporal artery biopsies were performed at our institution.

logical diagnosis of "healed arteritis" were identified. Two board-certified rheumatologists reviewed the available inpatient and outpatient medical records to abstract clinical data, including demographics, signs and symptoms preceding temporal artery biopsy [elevated erythrocyte sedimentation rate (ESR), anemia, headache, visual changes, jaw claudication, fever of unknown origin, proximal arthralgias, and myalgias], corticosteroid dosing at the time of biopsy, corticosteroid dosing postbiopsy, and longterm outcomes (permanent visual loss, aortic aneurysm, large vessel disease causing claudication).

Corticosteroid dosing at the time of biopsy was categorized into 3 groups: (1) no corticosteroids; (2) low/moderate-dose corticosteroids (prednisone < 40 mg daily); and (3) high-dose corticosteroids (prednisone ≥ 40 mg daily). Corticosteroid dosing postbiopsy was categorized as (1) no corticosteroids; (2) low/moderate-dose corticosteroids (prednisone < 40 mg daily); and (3) high-dose corticosteroids (prednisone ≥ 40 mg daily for at least 1 month).

Blinded to the clinical data, a cardiovascular pathologist reviewed the biopsy material (3 H&E-stained slides with 2 intervening elastic Van Gieson-stained slides for each case) of the subjects with an initial diagnosis of healed arteritis. No widely accepted, standardized definitions for healed arteritis exist, so previously published histological descriptions of healed arteritis were used to recategorize biopsies as "confirmed" or "unconfirmed" healed arteritis[7,10,12,13]. Biopsies that showed an absence of ongoing medial chronic inflammation but demonstrated fibrosis, attenuation and/or neovascularization of the media, irregular intimal proliferation, multifocal to complete loss of the internal elastic lamina, and adventitial fibrosis and chronic inflammation were categorized as "confirmed healed arteritis" (Table 1, Figure 2). Biopsies that showed no significant medial pathology but showed regular intimal proliferation with elastosis, focal loss of the internal elastic lamina, or calcification of this structure (Monkeberg's calcification) were reclassified as "atherosclerotic or age-related changes," and biopsies with no significant pathology were classified as normal. Both these histological types were included in the category of unconfirmed healed arteritis. Biopsies that showed ongoing inflammation within the media were separately classified as active arteritis.

Normally distributed continuous data were expressed as means and SD. Continuous data with non-normal distributions were expressed as medians and interquartile ranges. Categorical data were reported as numbers and percentages. Clinical characteristics of participants with confirmed healed arteritis were compared with clinical characteristics of participants with unconfirmed healed arteritis using Fisher's exact test for dichotomous variables and t tests and Wilcoxon signed-rank tests for continuous variables. All analyses were performed using the SAS 9.2 software package (SAS Institute, Cary, NC, USA).

Our study was approved by the Partners Institutional Review Board.

RESULTS

Over 10 years, 415 temporal artery biopsies were performed at our institution (Figure 1). Four hundred were initial biopsies, and 15 were repeat biopsies. Forty-seven pathologists interpreted the specimens from these biopsies. Of the 400 initial biopsies, 47 (11.8%) were identified as healed arteritis. Three hundred seven specimens were negative for GCA. Forty-three were identified as active arteritis, and 3 were inadequate specimens (no artery present).

Healed arteritis on original pathology report. Among the 47 patients with a diagnosis of healed arteritis on biopsy report, 39 (83.0%) were women (Table 2). Mean age was 71.2 years and mean ESR was 78.6 mm/h. Information regarding corticosteroid dose at biopsy was available for 41 out of 47 patients. At biopsy, 19 (46.3%) were not receiving cortico-

Table 1. Definitions of terms used for classification.

Classification	Description
Healed arteritis	Fibrosis, attenuation and/or neovascularization of the media, irregular intimal proliferation, multifocal to complete loss of internal elastic lamina, adventitial fibrosis. Absence of ongoing chronic medial inflammation
Atherosclerotic or age-related changes	Regular intimal proliferation with elastosis, focal loss of the internal elastic lamina, Monkeberg's calcification. No significant medial pathology
Active arteritis	Ongoing inflammation of the media
Normal	No significant pathology

steroids, 6 (14.6%) were receiving a low to moderate dose of corticosteroids (median dose 10 mg daily), and 16 (39.0%) were treated with high-dose corticosteroids (median dose 60 mg daily). Median prednisone dose was 6 mg daily. Median treatment duration before biopsy was 1 day.

Data on corticosteroid dosing before and after biopsy were available for 37 patients. The majority (n = 20, 54.1%), experienced no changes in corticosteroid regimen after pathology reports were provided to clinicians. Eight (21.6%) had no corticosteroid treatment before or after biopsy, and 12 (32.4%) had corticosteroid therapy maintained at the same level before and after biopsy (Figure 3). Nine (24.3%) experienced an increase in corticosteroid dose, 4 (10.8%) experienced a decrease in corticosteroid dose, and 4 (10.8%) stopped corticosteroids completely. Ultimately, 12 (32.4%) received no additional corticosteroids, 14 (37.8%) were treated with a low to moderate-dose regimen, and 11 (29.7%) received high-dose corticosteroids for ≥ 1 month.

Followup information was available for 44 patients (93.6%), ranging from 24 days to 11.7 years. The median duration of followup was 4.4 years. During the study period, 3 patients developed sequelae that may have been consistent with GCA, but alternative explanations were also noted. One patient was a 66-year-old woman with a history of arthritis and pleurisy who had sudden-onset bilateral visual loss. She was treated with prednisone 60 mg daily with unilateral recovery of vision. She was subsequently diagnosed with lupus optic neuropathy, given positive tests for antinuclear and dsDNA antibodies. The second patient was an 85-year-old white woman who developed permanent, unilateral visual loss, associated with an ESR of 55 mm/h. A fluorescein angiogram was suggestive of retinal/ophthalmic artery embolism. She was not treated with corticosteroids. A third patient, a 76-year-old white man, presented with malaise, weight loss, shoulder discomfort, and an ESR of 99 mm/h. He was treated with high-dose corticosteroids but 1 year later was diagnosed with a distal thoracic aortic aneurysm. The pathology report described a lymphoplasmacytic infiltrate throughout the vessel wall and adipose tissue, suggestive of possible IgG4-related aortitis[14]. No giant cells or granulomatous inflammation were noted.

Healed arteritis confirmed using published descriptions of healed arteritis. Of the 47 cases with an initial pathologic diagnosis of healed arteritis, only 15 were confirmed as healed arteritis. All the features listed in Table 1 were present to some degree in 13 cases, although the severity or extent of involvement varied. The 2 other confirmed cases of healed arteritis lacked only adventitial fibrosis (Table 3).

Thirty cases were recategorized as normal or atherosclerotic/age-related changes. Among cases of atherosclerotic/age-related changes, focal loss of the internal elastic lamina, regular intimal proliferation, and elastosis were uniformly present, although calcification of the internal elastic lamina was not observed in all cases. Two cases were recategorized as active GCA (Figure 1).

Compared to patients recategorized with normal biopsies or biopsies with age-related changes, patients with confirmed healed arteritis had similar clinical characteristics at the time of biopsy, including similar prevalence rates of headache and visual symptoms (Table 2). In 2 of the confirmed cases, visual symptoms occurred while patients were taking low doses of prednisone (between 0.5 and 15 mg daily) for treatment of mixed connective tissue disease or polymyalgia rheumatica (PMR). In contrast, all visual symptoms among patients with biopsies recategorized as normal/age-related changes occurred prior to corticosteroid treatment. In all but 2 cases, other potential causes of visual problems existed (e.g., diabetic retinopathy, glaucoma, cataracts, macular degeneration, stroke, and lupus optic neuropathy), and in most cases, physicians ascribed the visual loss to other causes or remained uncertain whether the cause was GCA. Little documentation was available for the 2 cases without other explanations for visual symptoms.

Patients with confirmed healed arteritis had a higher prevalence of previous PMR/GCA than patients recategorized as having normal biopsies or biopsies with age-related changes (53.3% vs 13.3%; p = 0.01; Table 2). Patients with confirmed healed arteritis were also more likely to be treated with corticosteroids at the time of biopsy (76.9% vs 44.8%; p = 0.09) and after biopsy (86.7% vs 63.3%; p = 0.16), although these differences were not statistically significant. Among the 13 patients with data on corticosteroid therapy before and after biopsy, the majority (n = 8, 61.5%) had no changes in corticosteroid regimen after pathology reports were provided to clinicians. One (7.7%) had no corticosteroid treatment before or after biopsy, and 7 (53.8%) had corticosteroid therapy maintained at the same level before and after biopsy. For 3 of them (23.1%), corticosteroid dose was increased; for 1

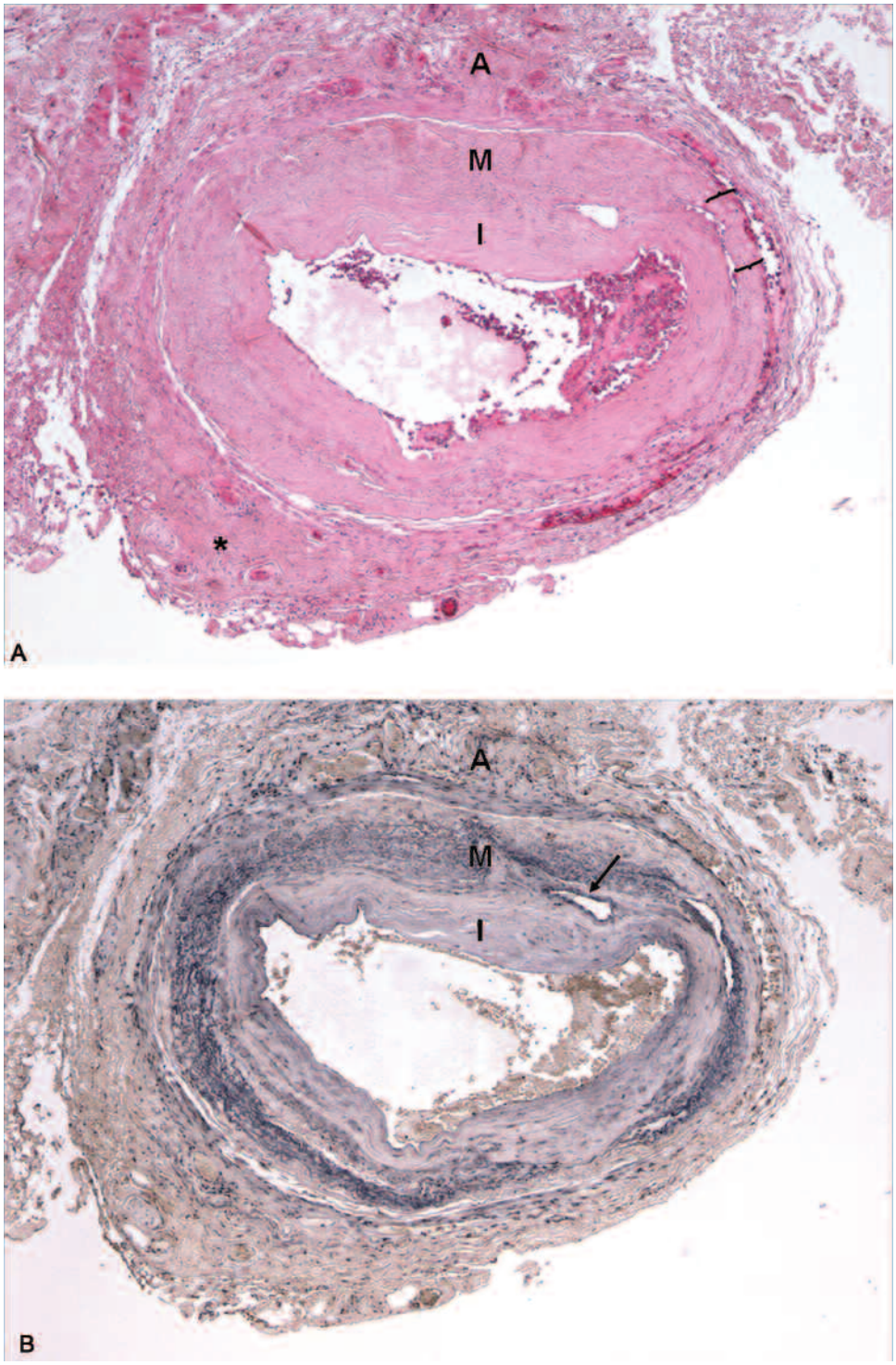

Figure 2. Histopathology of healed arteritis. A. Complete cross-section of a temporal artery biopsy with adventitia (A), media (M), and intima (I). Note the intimal thickening, medial attenuation, and fibrosis (brackets) and adventitial fibrosis (*). H&E stain, 4× objective. B. Serial section of the same biopsy showing a complete, circumferential loss of the internal elastic lamina between the intima and media. Neovascularization at the intimal-medial junction (arrow) is present. Elastic Van Gieson stain, 4× objective.

(7.7%), corticosteroid dose was decreased; and 1 (7.7%) stopped corticosteroids completely. The duration of corticosteroid treatment prior to biopsy was significantly longer among patients with confirmed healed arteritis compared to

Table 2. Clinical characteristics of patients with healed arteritis.

Clinical Characteristics	Total Cohort, n = 47	Confirmed Healed Arteritis, n = 15	Unconfirmed Healed Arteritis, n = 30	p
Mean age, yrs (SD)	71.2 (16.8)	75.1 (9.0)	69.4 (19.8)	0.19
White, n (%)	30 (63.8)	9 (60.0)	21 (70.0)	0.28
Women, n (%)	39 (83.0)	12 (80.0)	25 (83.3)	1.00
Characteristics leading to temporal artery biopsy				
Mean ESR, mm/h (SD)	78.6 (34.3)	79.3 (33.7)	76.0 (34.6)	0.79
Headache, n (%)	29 (61.7)	9 (60.0)	20 (66.7)	0.75
Visual symptoms, n (%)	13 (27.7)	4 (26.7)	8 (26.7)	1.00
Jaw claudication, n (%)	3 (6.4)	0 (0.0)	3 (10.0)	0.54
Fever of unknown origin, n (%)	2 (4.3)	1 (6.7)	1 (3.3)	1.00
Constitutional symptoms, n (%)	16 (34.0)	5 (33.3)	11 (36.7)	1.00
Anemia, n (%)	8 (17.0)	2 (13.3)	6 (20.0)	0.70
History of PMR/GCA, n (%)	12 (25.5)	8 (53.3)	4 (13.3)	0.01
Patients taking corticosteroids at biopsy*, n (%)	24 (55.8)	10 (76.9)	13 (44.8)	0.09
Median dose, mg (IQR)	6.0 (0.0, 60.0)	20.0 (6.0, 60.0)	0.0 (0.0, 50.0)	0.09
Median duration, days (IQR)	1.0 (0.0, 7.0)	10.0 (1.0, 120.0)	0.0 (0.0, 3.0)	0.008
Patients taking corticosteroids after biopsy†, n (%)	33 (71.7)	13 (86.7)	19 (63.3)	0.16

* Data regarding corticosteroid therapy (yes/no) at biopsy were available for 43 participants, but dosing information was available for only 41 participants. Median doses and durations are calculated among all patients in the cohort (not restricted to those on corticosteroid therapy). † Data regarding corticosteroid therapy (yes/no) after biopsy were available for 46 participants. ESR: erythrocyte sedimentation rate; IQR: interquartile range; PMR: polymyalgia rheumatica; GCA: giant cell arteritis.

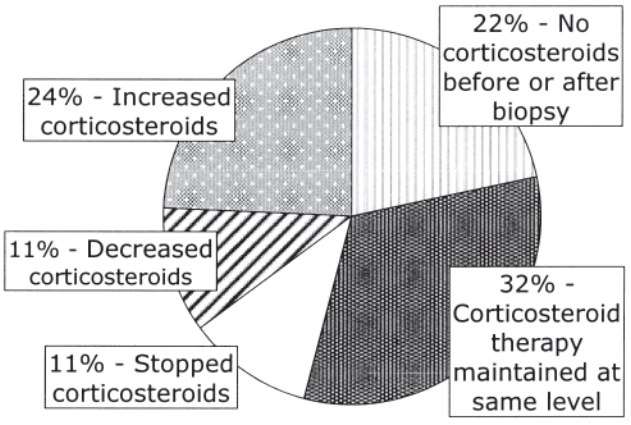

Figure 3. Physician response to pathologic diagnosis of healed arteritis.

those with normal biopsies or biopsies with age-related changes (median 10 days vs 0 days; p = 0.008).

Although there were a few adverse outcomes in the primary cohort of patients initially diagnosed with healed arteritis, no patients with confirmed healed arteritis had a poor outcome associated with GCA (e.g., visual changes consistent with ischemia, large artery disease, or aortic aneurysms; Table 3). Of the 2 patients whose histological diagnoses were changed to active arteritis after review, 1 patient did not have followup information about corticosteroid dosing or adverse outcomes. The other was not treated with corticosteroids, and in the 15 months of available followup, there was no documentation of adverse outcomes associated with active GCA.

DISCUSSION

In this cohort of 400 independent temporal artery biopsies performed as a part of routine clinical care, 11.8% were given an initial diagnosis of healed arteritis. Compared to previously reported cohorts of patients with active GCA[15], classic signs and symptoms of GCA, such as elevated ESR and anemia, were less common in this series. Most physicians did not change treatment decisions based on this histological diagnosis, and irrespective of treatment regimen, adverse outcomes (e.g., visual loss and aortic aneurysms) were less common in this cohort, compared to cohorts of classic, active GCA[16,17,18,19,20].

To our knowledge, the only other study that has reported outcomes of healed arteritis was a series of 44 patients with a clinical diagnosis of GCA[21]. Of the original 44 patients, 7 had healed arteritis characterized by histological findings of focal inflammation without giant cells, 23 had classic GCA characterized by chronic granulomatous inflammation with giant cells, and 14 had atypical arteritis associated with less dense chronic inflammation and occasional giant cells. None of the patients with healed arteritis developed permanent blindness, compared to 5 patients (21.7%) with typical GCA and 1 patient (7.1%) with atypical arteritis. These results are consistent with our findings[21].

Alternative explanations for the lower rates of inflammation and adverse outcomes may be heterogeneity due to diagnostic uncertainty and/or underestimation of adverse events. A previous study showed that trained pathologists disagreed with the consensus diagnosis of healed arteritis in 26.7% to 56.7% of cases[10]. Thus, misclassification of atherosclerosis or arteriosclerosis as healed arteritis may have diluted the fre-

Table 3. Clinical and histological characteristics of the 15 patients with confirmed healed arteritis.

Patient	Age, yrs	Sex	Race/ Ethnicity	Histology Findings: Medial Attenuation and/or Fibrosis	Intimal Proliferation	Loss of Internal Elastic Lamina	Adventitial Inflammation and Scarring	Medial Inflammation	Duration of Pre-biopsy Cortico-Steroid Therapy, days	Prior Diagnosis of GCA or PMR	Duration Between Diagnosis and Biopsy	Post-biopsy Therapy	Outcome
1	58.5	F	Unknown	Focal	Mild	Multifocal	Mild	None	122	PMR: proximal myalgias; ESR 60	6 yrs	Slowly tapered corticosteroids but was later on and off cortico-steroids multiple times for PMR	Multiple flares of PMR
2	91.5	F	White	Multifocal	Mild	Diffuse	Mild	None	0	No	NA	Started prednisone 15 mg daily, tapered to 10 mg daily after 1 wk	No known adverse outcomes but minimal followup
3	76.0	F	Hispanic	Multifocal	Mild	Multifocal	Mild-moderate	None	793	PMR: proximal myalgias; ESR 57	1 yr	No change in prednisone dose; continued prednisone 20 mg daily	No known adverse outcomes
4	88.3	M	White	Multifocal	Mild	Diffuse	Mild	None	10 (but was also treated with chronic corticosteroids for 2-5 yrs 10 yrs ago)	GCA: no symptom data	~15 yrs	No change in prednisone dose; continued prednisone 40 mg daily and tapered to 10 mg daily after 2 mo	No known adverse outcomes
5	84.6	F	White	Multifocal	Moderate	Diffuse	Mild	None	1	PMR: no symptom data	7 yrs	Tapered to prednisone 20 mg daily over 1 wk; then slowly tapered over next 3 mo	No known adverse outcomes
6	75.6	F	White	Multifocal	Mild	Multifocal	Mild	None	Unknown	PMR: no symptom data	Unknown	Treated with low-moderate dose prednisone and tapered over yrs	No known adverse outcomes but minimal followup
7	68.2	M	Hispanic	Focal	Mild	Focal	Mild	None	0	No	Unknown	No cortico-steroids	No known adverse outcomes
8	82.2	F	Hispanic	Focal	Mild	Multifocal	Mild-moderate	None	3	No	Unknown	Continued prednisone 60 mg daily; tapered over yrs	No known adverse outcomes
9	64.8	F	White	Focal	Mild	Focal	None	None	14	PMR: proximal myalgias; no ESR recorded	3 mo	Continued prednisone 60 mg daily; tapered over yrs	No known adverse outcomes
10	74.7	F	White	Focal	Moderate	Multifocal	Mild	None	7	No	55	Increased to methylprednisolone 48 mg daily; tapered over yrs	No known adverse outcomes
11	72.7	F	African American	Multifocal	Moderate	Diffuse	Mild	None	Unknown	GCA: headache, visual blurring, ESR 50	5 yrs	Unknown	Flare of PMR

Table 3. Continued.

Patient	Age, yrs	Sex	Race/ Ethnicity	Histology Findings: Medial Attenuation and/or Fibrosis	Intimal Proliferation	Loss of Internal Elastic Lamina	Adventitial Inflammation and Scarring	Medial Inflammation	Duration of Pre-biopsy Cortico-Steroid Therapy, days	Prior Diagnosis of GCA or PMR	Duration Between Diagnosis and Biopsy	Post-biopsy Therapy	Outcome
12	77.0	F	White	Multifocal	Mild	Multifocal	None	None	120	PMR: proximal myalgias; ESR 109	5 mo	Increased to prednisone 20 mg bid but decreased to 20 mg daily after 1 wk	No known adverse outcomes
13	76.2	M	Asian	Multifocal	Moderate	Complete	Mild	None	0	No	NA	Decreased to prednisone 40 mg daily and tapered over 1 mo	No known adverse outcomes
14	67.0	F	White	Focal	Mild	Diffuse	Mild	None	548	No	NA	Decreased to prednisone 60 mg daily and tapered over yrs	No known adverse outcomes
15	69.5	F	White	Focal	Mild	Focal	Mild	None	13	No	3 mo	Continued prednisone 40 mg daily; tapered over yrs	No known adverse outcomes

GCA: giant cell arteritis; PMR: polymyalgia rheumatica; ESR: erythrocyte sedimentation rate; NA: not available.

quency of inflammatory characteristics and adverse outcomes associated with GCA in our study. In addition, the calculation of adverse event rates may have been limited by the use of medical record review to identify these events. Although medical record review likely identified major outcomes managed at this academic institution, this strategy may have missed episodes that were treated at other sites. In addition, some outcomes may not have occurred for several years after the initial diagnosis, and others, such as aortic aneurysms, may have been present but clinically silent[22].

In the second portion of our study, we reviewed all biopsies to determine whether the original diagnoses were consistent with previously published descriptions of histological features characteristic of healed arteritis. Similar to a retrospective study in which 2 out of 10 cases were confirmed as healed arteritis after evaluation according to study-derived criteria[7], we were able to confirm only 31.9% of cases initially categorized as healed arteritis, highlighting the heterogeneity of the interpretation of the histology.

Compared to patients with unconfirmed healed arteritis, patients with confirmed healed arteritis had a higher prevalence of previous PMR/GCA and longer duration of previous corticosteroid treatment. These data could imply that the histological findings of healed arteritis are a consequence of prior episodes of active GCA that resolved with corticosteroid treatment. However, of the 13 patients with data regarding corticosteroid dosing prior to biopsy, 3 had no history of corticosteroid use, and 2 had < 1 week of corticosteroid treatment. These findings suggest that active GCA either resolved on its own, with minimal corticosteroid therapy, or that these histological findings represent a distinct phenotype of GCA — one that is associated with lower levels of inflammation and with a better prognosis than classic active GCA. Our study, however, was unable to distinguish between these possibilities, and ambiguity remains whether "healed arteritis" is an apposite designation of these histological changes.

Previous studies regarding the effect of corticosteroids on histological findings have yielded differing results. In a study of 132 patients with clinical symptoms of GCA, corticosteroid treatment for > 1 week was associated with lower positive biopsy rates compared to no treatment[23]. In contrast, a study of 523 patients revealed no difference in positive biopsy rates among patients treated with corticosteroids compared to those who were untreated[4]. Similarly, subgroup analyses revealed no difference in biopsy positivity rates among those treated with ≥ 14 days of corticosteroids compared to those who were untreated; however, biopsies of patients treated with longer durations of corticosteroids were more frequently, although not significantly, associated with atypical features (e.g., lack of giant cells and adventitial inflammation).

In our study, the comparisons between confirmed cases of healed arteritis and unconfirmed cases of healed arteritis were limited by the retrospective study design, small sample size, and possible misclassification of patients with unconfirmed

healed arteritis. The designation of confirmed versus unconfirmed healed arteritis was made by a single study pathologist who reviewed the specimens based on published histological descriptions of healed arteritis and active arteritis. If specimens did not conform to these descriptions, the biopsy was given a diagnosis of unconfirmed healed arteritis. However, other pathologists may interpret these findings differently, classifying them as actual healed arteritis or mild, active arteritis. Although bias from misclassification should influence results toward the null, these data should be considered hypothesis-generating, rather than conclusive evidence of an association between histological changes of healed arteritis and previous inflammatory disease.

In addition to these factors, confounding by indication may also have exaggerated the differences between the 2 groups. In our retrospective study, patients were not randomized to corticosteroids versus placebo; rather, their treatment was based on clinical signs and symptoms. Because of their history of PMR/GCA, patients may have been treated with corticosteroids for longer periods due to these preexisting conditions. In addition, given the patients' medical histories, physicians may have been more likely to treat them with corticosteroids than to watch and wait, a strategy appropriate to patients with no history of inflammatory disease.

Interestingly, none of the adverse outcomes occurred among patients with a confirmed histological diagnosis of healed arteritis. It is not clear whether the benign outcomes are due to more aggressive steroid treatment among patients with confirmed healed arteritis or whether this histological diagnosis does not have clinically significant prognostic value.

Our study shows that the diagnosis of healed arteritis in usual clinical practice was unreliable. Compared to patients in studies of active GCA, patients with healed arteritis tended to have lower ESR and lower rates of anemia. Presented with a diagnosis of healed arteritis on a pathology report, most physicians did not change therapy. There was no consistent treatment regimen for healed arteritis, with about one-third of patients not receiving any corticosteroids, one-third receiving a low to moderate dose of corticosteroids, and one-third receiving a high-dose corticosteroid regimen. Irrespective of the treatment, adverse outcomes were rare. With the use of uniform histological characteristics to define healed arteritis, we identified a subgroup of patients who had a higher prevalence of previously documented PMR/GCA and a longer duration of corticosteroid use. Whether these histological characteristics are clinically useful in distinguishing patients with a higher risk for poor outcomes is unclear.

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