Morphological features of temporal arteritis

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Although it varies from center to center, the frequency of temporal artery biopsy in patients suspected of having temporal arteritis (TA) is relatively small. Most commonly, patients suspected of having TA are placed on prednisone for varying periods of time, and if symptoms disappear or lessen the diagnosis is made. During a recent 13-year period at Baylor University Medical Center at Dallas, 15 patients with TA had the diagnosis of TA confirmed by histological examination of a biopsy of one temporal artery. The length of the biopsied artery varied from 0.7 to 5.5 cm (mean 2.7). The 15 patients ranged in age from 68 to 94 years (mean 82, median 85), and 11 (73%) were women. In 13 of the 15 patients (87%), the lumen of the temporal artery was narrowed >95% in cross-sectional area by the panarteritis, and the temporal artery was associated with giant cells in 11 patients (73%). Large collections of erythrocytes were present in the inflamed arterial walls in 5 patients (33%). All 15 patients were treated with varying doses of prednisone with favorable response in each. Eight patients (53%) died from 1 to 105 months (mean 52, median 57) after biopsy of the temporal artery. We have neither positive nor negative evidence that the TA played a role in the patients' death. Despite the present study and numerous others in the last 70 years, the cause of TA remains a mystery.

In 1932 and in 1934, Bayard T. Horton, a vascular specialist at the Mayo Clinic, and others (1, 2) reported two patients with headache, scalp tenderness, weight loss, fever, and night sweats, and histologic examination of one biopsied temporal artery disclosed granulomatous panarteritis. Thereafter, the condition was called *temporal arteritis* (TA) by some and *Horton's disease* by others. In 1937, Horton and Magath (3) described visual loss, jaw claudication, and elevated erythrocyte sedimentation rates in several additional patients with the disease. According to Boes (4), Horton in 1942 was the first to give a patient with TA Kendall's adrenocorticoid extract (nonpure), but apparently it had no effect on the patient's disease. Shick and colleagues (5), in 1950, also at the Mayo Clinic, reported clinical improvement in two patients with TA using a pure form of cortisone. The present study summarizes findings in 15 patients with TA seen at Baylor University Medical Center at Dallas (BUMC) in the last 13 years and describes in detail the various histological features in the temporal artery in these patients.

METHODS

Cases coded as TA by the surgical pathology division of the Department of Pathology of BUMC from 1997 through 2012 were retrieved. Fifteen such cases having biopsy of one temporal artery were found. The paraffin blocks of the temporal artery in each patient were retrieved and recut. The resulting 6-micron-thick sections were stained by both the hematoxylin-eosin method and by the Movat method, and the sections were examined. The clinical records in each patient were retrieved and examined in the BUMC record room, and pertinent findings were tabulated in each patient. Finally, the Social Security Death Index was searched to determine how many of the 15 patients had died.

RESULTS

Pertinent findings in the 15 patients are summarized in *Tables 1 and 2*. The 15 patients ranged in age from 68 to 94 years (mean 82) at the time of the temporal artery biopsy; 11 were women and 4 were men. The age at biopsy in all 15 patients corresponded to the age at which symptoms and/or signs of TA appeared. The symptoms at the time of temporal artery biopsy are displayed for each patient in Table 1: headache in 12, visual disturbance in 10, mastication pain in 7, and temporal artery tenderness in 6. At the time of biopsy, the indirect systemic arterial pressure was ≥ 140 mm Hg systolic and/or ≥ 90 mm Hg diastolic in 11 patients (73%). The body mass index was >25 kg/m² in 8 of the 15 patients, but in none was it ≥ 30 kg/m². Anemia (hematocrit <35%) was present in 8 (57%) of the 14 patients in which the result of this test was available. The platelet count was >250 mm³ in 9 of the 11 patients in whom it was performed. The erythrocyte sedimentation rates were elevated (>20 mm/hour) in all 10 patients where the results were available. The serum C-reactive protein was elevated in all 5 patients in which it was done. One patient (#2) had an aortic aneurysm, and one patient (#8) had had a stroke a few months before

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Table 1. Clinical and laboratory findings in the 15 patients with temporal arteritis confirmed by biopsy and treated with prednisone

Patient	Date of biopsy	Age at biopsy (years)	Interval from biopsy to death (mo)	Age at death (yr)	Age at symptom onset (yr)	Symptoms at time of biopsy					Temporal artery tenderness			Platelet count (mm ³)			CRP (mg/dL)	BMI (kg/m ²)	Minimal time on prednisone (mo)	Maximal dose of prednisone (mg)
						H	MP	VD	CS	PR	LC	BP s/d (mm Hg)	Hgb (g/dL)	HCT (%)	ESR (mm/hr)					
1	8/6/1998	75	–	–	75	+	+	+	0	0	+	0	130/80	11.7	35.2	–	–	20.8	73	70
2	8/3/2001	75	50	79	75	+	+	+	+	0	+	+	170/75	8.5	26.9	–	–	20.0	26	60
3	6/25/1997	77	–	–	77	+	0	+	0	0	0	0	140/80	11.5	37.0	76	–	27.1	–	60
4	10/23/2001	77	105	85	77	+	0	0	+	0	0	0	180/90	13.3	40.1	–	–	26.2	17	40
5	11/21/2002	80	65	85	80	+	+	0	+	+	+	+	130/60	10.5	32.0	121	–	24.6	–	60
6	1/29/2002	82	87	90	82	+	0	+	0	0	0	0	130/90	12.4	38.2	–	–	23.0	73	60
7	3/22/2001	85	1	85	85	+	+	+	0	+	+	0	140/80	10.6	32.2	92	26	24.3	0.5	60
8	11/22/2010	86	–	–	86	0	0	+	0	0	0	0	150/55	12.1	35.8	↑	↑	26.0	0.25	60
9	3/26/2002	86	–	–	86	+	0	0	0	0	0	+	140/60	11.2	33.5	↑	–	20.8	11	60
10	9/8/2004	89	74	96	89	+	+	+	0	0	0	+	220/80	9.8	29.9	130	28	29.1	14	60
11	2/10/2000	90	28	92	90	+	0	0	0	0	0	0	175/80	13.6	32.4	↑	–	21.8	24	60
12	9/9/2010	68	3	68	68	0	+	0	0	0	+	0	110/70	11.0	32.0	57	–	25.1	4	60
13	4/25/1998	81	–	–	81	+	0	+	+	0	+	+	155/95	–	–	–	–	29.8	–	60
14	8/22/2008	83	–	–	83	0	0	+	0	0	0	0	115/70	9.9	29.5	103	19	25.4	3	50
15	4/13/2009	94	–	–	94	+	+	+	0	+	+	+	160/90	14.3	40.0	41	6	26.2	19	60

BMI indicates body mass index; BP, blood pressure; CRP, C-reactive protein; CS, cigarette smokers; ESR, erythrocyte sedimentation rate; H, headache; HCT, hematocrit; Hgb, hemoglobin; LC, leg claudication; MP, mastication pain; PR, polymyalgia rheumatica; S/D, peak systole/end diastole; VD, visual disturbance; -, not done, not applicable, or no information.

biopsy. The length of the temporal artery biopsied ranged from 0.7 to 5.5 cm (mean 2.7); sample images and descriptions appear in *Figures 1 to 9*. In 13 of the 15 patients (87%), the lumen of the temporal artery was narrowed >95% in cross-sectional area. The temporal artery was associated with giant cells in 11 patients (73%). All 15 patients received prednisone (maximal dose 40–70 mg) for 0.25 to 73 months (mean 22), and all had symptomatic improvement, including 5 with loss or virtual loss of symptoms.

DISCUSSION

It might seem a bit inappropriate in 2013 to report a series of only 15 patients with biopsy-proven TA when others have reported such large series of patients with biopsy-proven TA (6–35). Gonzalez-Gay and colleagues (13, 19, 20, 25, 26, 28, 31), for example, in 7 articles from 1998 to 2011 described anywhere from 161 to 255 patients with biopsy-proven TA (called “giant cell arteritis” by the authors), but none contained a photomicrograph of a temporal artery. Indeed, of the 30 studies presented in *Table 3* (6–35), only two included a photomicrograph of a temporal artery, and in both only hematoxylin-eosin-stained sections had been used. It is not possible to demonstrate the locations of the panarteritis, i.e., how much of the process involved the intima, media, and adventitia, without an elastic tissue stain that readily identifies the internal and external elastic membranes allowing clear demonstration of media, thus separating it from the intima and adventitia. We employed the Movat stain for this purpose in our study (36).

We prefer the phrase “temporal arteritis” to the phrase “giant cell arteritis” because giant cells are not seen in all TA patients having biopsies of the temporal arteries. Among our 15 patients, we found giant cells in only 11. Mahr and colleagues (37) suggested that finding giant cells in patients with TA is determined in part by the lengths of the temporal arteries examined. These authors examined surgical

Table 2. Morphological findings in the 15 patients with biopsy-proven temporal arteritis

Patient	Length (cm) of excised TA	Number of histological cross-sections	Maximal diameter (mm) of cross-sections	Cross-sectional narrowing	Collection of red blood cells in intima	Lymphocytes	Giant cells
1	4.2	8	3	>95%	0	+++	++
2	4.7	11	2	>95%	+	+++	++
3	2.4	8	2	>95%	0	+++	++
4	2.0	8	2	>95%	+	+++	++
5	2.4	12	2	>95%	+	+++	++
6	0.7	3	2	>95%	0	+	0
7	2.6	8	2	51%–75%	+	+	0
8	2.2	4	2	>95%	0	+++	++
9	5.5	19	6	>95%	0	+++	++
10	3.0	8	4	>95%	0	+++	+
11	0.7	2	2	>95%	0	+	+
12	1.9	4	2	>95%	+	+++	++
13	2.1	6	3	>95%	0	+++	++
14	3.5	7	4	>95%	0	+++	0
15	3.0	7	3	51%–75%	0	+++	0

TA indicates temporal arteritis.

Figure 1. Patient 1. Various views of the temporal artery. (a) Movat-stained section ($\times 40$) showing relatively intact media (pink), very thickened intima (green) with severe luminal narrowing, and severely thickened fibrous tissue of the adventitia (tan). (b) Hematoxylin-eosin (H&E)-stained section showing numerous inflammatory cells involving the outer intima, media, and inner adventitia ($\times 100$). (c) A close-up showing intimal granulomatous-type cells adjacent to the media with penetration of the media (H&E, $\times 400$). (d) Another view showing an inflamed nodule in brackets in the adventitia (H&E, $\times 40$). (e) A close-up of that nodule (H&E, $\times 400$).

reports of temporal artery biopsies in 223 patients with TA and found that 164 (74%) of the reports mentioned the presence of giant cells. These authors also mentioned that a temporal artery length of at least 0.5 cm was sufficient for diagnosis of TA. Our smallest length among the patients was 0.7 cm. Among the four patients in whom we did not see giant cells, the lengths of the temporal artery biopsied were 0.7, 2.6, 3.0, and 3.5 cm; the latter three lengths were among the longest in the patients we studied.

Although the present study focuses on the histologic features of TA, one might reasonably ask if biopsy of this artery is a useful

routine endeavor. A near universal observation in TA is a rapid, sometimes dramatic, diminution or loss of symptoms after corticosteroid therapy has been initiated. If there is not a quick symptomatic response, biopsy can then be performed. There appears to be little change in the histologic features of the TA before corticosteroid therapy and up to about 6 weeks after initiation of therapy (10). A report by Guevara et al described a positive biopsy after 6 months of prednisone treatment (38). If a patient with suspected TA has a negative biopsy of the temporal artery, is it useful then to biopsy the contralateral temporal artery? According to a study by Boyev and colleagues (18), biopsy of one temporal artery in a patient

with TA provides a 97% chance that the same findings would be present in the contralateral temporal artery, so that the additional biopsy would rarely be useful diagnostically.

On occasion, TA resolves without corticosteroid therapy. Horton et al, in their original two patients, described temporary remissions with relapses (1), and they later described seven additional patients in whom remission occurred without drug therapy months after diagnosis (3). Patients have been described where headaches and local symptoms have disappeared simply by removal of a portion of the temporal artery for diagnostic purposes (39, 40).

Figure 2. *Patient 2.* (a) A Movat-stained section ($\times 100$) of temporal artery with severely narrowed lumen (*within the green portion*) of the intima with blood (*red*) within the intimal plaque and marked disruption of the internal elastic membrane (*black*). The adventitia is thickened by dense fibrous tissue (*tan*). (b) The same section stained by hematoxylin-eosin ($\times 100$). (c) A close up of a portion of the media showing numerous mononuclear cells ($\times 400$).

Figure 3. *Patient 4.* Movat-stained section ($\times 40$) of the temporal artery showing near-total occlusion of the lumen, blood (*red*) within the intimal plaque, disruption of the internal elastic membrane (*black*), and severely thickened adventitia by dense fibrous tissue (*tan*). The absence of much lumen and the marked thickening of the adventitia by dense fibrous tissue makes these arteries, by external palpation, quite firm and nodular.

Figure 4. *Patient 6.* Two views of Movat-stained sections of the temporal artery showing (a) virtual occlusion ($\times 100$) and (b) severe narrowing ($\times 100$). The internal elastic membrane (*black*) is interrupted and the quantity of fibrous tissue in the adventitia is considerably less than in previously illustrated cases.

Figure 5. *Patient 8.* (a) A Movat-stained section of the temporal artery showing near occlusion of the lumen by fibrous tissue and mucopolysaccharide material (*green*) and dense fibrous tissue causing considerable thickening of the adventitia. (b) Hematoxylin-eosin stain ($\times 40$) of another section of the same artery showing numerous inflammatory cells between the 8:00 and 11:00 positions. (c) Close-up ($\times 400$) of a portion of the inflammatory cells.

Figure 6. Patient 9. (a) View of a hematoxylin-eosin-stained section ($\times 20$) of the temporal artery with virtual total occlusion of its lumen. The darkened area represents collections of inflammatory cells. The adventitia is thickened by fibrous tissue. (b) A close-up ($\times 400$) of a minute portion of the inflammatory infiltrates. Granulomatous-type cells and a giant cell are visible.

Figure 7. Patient 9. (a) A Movat-stained section ($\times 40$) of three branches of a temporal artery with narrowing of each branch, marked disruption of the media in two of the branches, and dense fibrous tissue in the adventitia. (b) A close-up of a hematoxylin-eosin-stained section ($\times 400$) of a portion of the inflammatory cells in the media. (c) Another hematoxylin-eosin stained section ($\times 400$) showing giant cells among the collection of cells.

Figure 8. Patient 10. A Movat-stained section of the temporal artery showing (a) severe narrowing of the lumen ($\times 100$) and (b) less narrowing ($\times 100$). The amount of adventitial fibrous tissue is considerable. Inflammatory cells are present in the intima, media, and adventitia. The dark staining in the medial wall in part b represents calcific deposits.

Figure 9. Patient 13. (a) A Movat-stained section of the temporal artery ($\times 40$) with severe luminal narrowing. The lumen in all sections in temporal arteritis tends to be in the more central portion of the artery and not on the periphery, as it is in typical atherosclerosis. (b) A close-up hematoxylin-eosin-stained section ($\times 400$) shows several giant cells in the outer intima adjacent to the media. These cells are lined up perpendicular to the smooth muscle cells in the media.

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Table 3. Reported studies of patients with temporal arteritis confirmed by biopsy of the temporal artery

First author, year of publication	Biopsy- proven		M/F	Age range (mean)	Length of study (yr)	Follow- up (yr)	Deaths (n)	Deaths attributed to TA	Median			Received cortico- steroids (n)	Maximal length of treatment (mo)	Patients that relapsed (n)	Tem- poral artery photo- micro- scopy	Giant cell histol- ogy (n)
	Patients (n)	TA (n)							Mean ESR values (mm/hr)	hemo- globin values (g/dl)	Median platelet count (mm ³)					
Birkhead, 1957	55	55	28/27	60-83	6	-	-	-	-	-	-	21	15	-	0	-
Huston, 1978	42	38	9/33	56-92 (75)	25	19	21	1	96	11.5	-	17	77	11	0	-
Graham, 1981	90	90	26/64	55-88	10	11	32	3	81	-	-	55	84	18	0	-
Nordborg, 1989	284	284	-	-	10	1	82	7	-	-	-	-	-	-	0	-
Achkar, 1994	535	175	190/345	31-93 (71.7)	3	-	-	-	63	-	-	-	-	-	0	-
Lie, 1995	72	67	21/51	54-96 (69)	25	-	-	-	96	-	-	-	-	-	0	-
Salvarani, 1995	125	115	22/103	50-80+	42	-	-	-	-	-	-	-	-	-	0	-
Gonzalez-Gay, 1998	239	239	106/133	50+ (73.5)	21	-	-	-	94	-	-	64	239	-	0	-
Cid, 1998	200	200	59/141	57-92 (74.5)	16	-	-	-	82.7	12.2	337,000	28	-	-	0	-
Duhaut, 1998	400	207	118/282	50+ (74)	6	-	-	-	-	-	-	-	-	-	0	-
Duhaut, 1999	292	207	50/157	50+ (74.5)	6	36	38	1	88	15.6	424,000	20	36	-	0	-
Brack, 1999	148	74	16/58	50+ (72)	36	-	-	-	-	-	-	18	-	-	0	-
Boye, 1999	756	722	-	-	28	-	-	-	-	-	-	-	-	-	0	-
Gonzalez-Gay, 2000	190	161	79/82	50-80+ (75)	18	-	-	-	93	11.8	412,816	42	-	-	0	-
Gonzalez-Gay, 2001	190	161	79/82	50-80+ (75)	18	-	-	-	93	11.8	412,816	-	-	-	0	-
Liozon, 2001	174	147	38/109	- (75)	22	-	-	-	90	11.3	419,000	58	-	-	0	-
Ray-Chaudhuri, 2002	11	9	3/8	65-90 (75)	-	-	-	-	66	-	-	5	1.5	-	0	7
Hall, 2003	181	74	-	-	11	2	-	-	-	-	-	-	-	-	0	-
Salvarani, 2004	173	151	36/137	50-80+	50	6.8	-	-	-	-	-	-	-	-	0	-
Gonzalez-Gay, 2005	240	240	110/130	50-80+ (74)	23	-	-	-	93	11.7	397,000	21	-	-	0	-
Larsson, 2006	49	49	0/49	50-69 (64.1)	9	3	-	-	-	-	-	-	-	-	0	-
Gonzalez-Gay, 2007	255	255	116/139	55-94 (75)	25	-	-	-	-	-	-	89	-	-	0	-
Narvaez, 2007	78	57	28/50	56-89 (74.6)	18	-	-	-	94.4	11.4	340,000	16	1	-	0	-
Makkuni, 2008	30	30	10/20	- (77.5)	6	-	-	-	78.5	-	-	12	-	-	+	-
Gonzalez-Gay, 2009	287	287	132/155	50-80+	27	-	-	-	-	-	-	138	-	-	0	-
Zhou, 2009	107	26	-	-	19	-	-	-	-	-	-	-	-	-	+	26
Martinez-Lado, 2011	207	174	80/94	(74.2)	14	3.5	0	0	92.7	11.7	392,000	36	44.1	71	0	-
Wavick, 2011	3001	459	-	-	9	-	-	-	81	-	391,000	-	-	-	0	-
Ninan, 2011	225	225	62/163	(78.2)	14	5.5	71	12	-	-	-	-	-	-	0	-

ESR indicates erythrocyte sedimentation rate; F, female; M, male; TA, temporal arteritis; -, no information.

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