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CASE REPORT

Myocardial Infarction and Coronary Artery Involvement in Giant Cell Arteritis

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ABSTRACT: *Purpose.* To describe the pathologic findings in an unusual case of giant cell arteritis that presented initially with visual loss and rapidly culminated in myocardial infarction. *Case Report.* After the death of the patient, a complete autopsy was performed, including bilateral enucleation. All specimens, including a temporal artery biopsy completed before the patients death, were processed for routine paraffin histology and initially stained with hematoxylin and eosin. Elastic stains were subsequently used on specimens of temporal and coronary artery. The patient presented with loss of vision in the right eye. The clinical diagnosis was anterior ischemic optic neuropathy, secondary to temporal arteritis. The temporal artery biopsy was positive. Despite high-dose corticosteroid administration, the patient progressed to neurologic impairment, and subsequently to a fatal myocardial infarction. *Discussion.* Previous reports of temporal arteritis with coronary involvement are summarized. Myocardial infarction may be a more common early complication of temporal arteritis than appreciated previously. This important complication can occur despite administration of high-dose corticosteroid therapy. (Optom Vis Sci 1999;76:14–18)

Key Words: giant cell arteritis, granulomatous inflammation, coronary arteries, anterior ischemic optic neuropathy, myocardial infarction

Giant cell arteritis, GCA (temporal arteritis) was first described by Hutchinson[1] in 1890 and later documented by Horton et al.[2] in 1932. Frequently recognized systemic clinical features include malaise, fatigue, weight loss, low-grade fever, scalp tenderness, headache, and jaw claudication. A variety of ophthalmic manifestations have been reported including anterior and posterior ischemic optic neuropathy, central retinal artery occlusion, and ophthalmoplegia. Large artery involvement has been described in these patients, including aortic arch syndrome and aortic dissection.[3] Treatment with corticosteroids has been shown to dramatically alleviate the symptoms associated with GCA.[4] Myocardial infarctions have been thought to be a rare but documented consequence of this disease.[5] We wish to draw attention to the occurrence of myocardial infarction as a result of giant cell arteritis that manifested itself in the coronary arteries.

CASE REPORT

A 75-year-old white male presented with a history of painless loss of vision in the right eye, occurring 3 weeks before his initial examination. Past medical history revealed bilateral calf claudication and hypertension. He also acknowledged joint stiffness for the past 5 years. In the past few months, there was a weight loss of 10 pounds. There were no complaints of headaches, jaw claudication, or temporal tenderness. He had no history of diabetes mellitus or angina.

Eye examination showed a visual acuity of 20/300 OD and 20/40 OS. There was a relative afferent pupillary defect OD with moderate pallor of the right optic disc. A visual field examination revealed a central scotoma OD. His motility examination was completely normal, with the rest of his ocular examination being unremarkable.

Systems evaluation revealed blood pressure was 140/80 mmHg. There was no temporal artery tenderness. There were no neck bruits. The cardiac examination revealed normal sounds and no gallop. A systolic ejection murmur at the left sternal border without radiation was detected. Neurologic examination revealed no abnormalities on admission.

The sedimentation rate (ESR) was 121 mm/h (Westergren), and anemia was also present. A diagnosis of temporal arteritis with anterior ischemic optic neuropathy involving the right eye was made. He was started on prednisone 80 mg daily (p.o.), pending temporal artery biopsy. The rest of his laboratory examination

revealed a hematocrit of 35, a platelet count of 317,000, and a white count of 5,000. Serum electrolytes were normal. The chest X-ray was normal with no evidence of cardiomegaly. The admission ECG was normal.

On day 4, a right temporal artery biopsy was performed and showed findings consistent with temporal arteritis (Fig. 1). His initial hospital course was unremarkable except for elevated blood pressure (186/100) on days 4 and 5, for which he was treated with hydrochlorothiazide and later hydralazine (10 mg bid). On day 7, the patient was observed to be intermittently confused. Neurologic examination by one of us (C.K.) that morning revealed fluent aphasia with paraphasic errors, poor comprehension, poor repetition, and difficulty with naming. The cranial nerve examination was normal. There was a right arm drift with distal weakness. Sensory examination revealed decreased sensation to pinprick on his right arm. Deep tendon reflexes were normal with an up-going right toe. By early evening, the patient's neurologic examination showed disorientation to time, and place, empty speech, frequent paraphasic errors, and bilateral up-going toes. This time, there was a left hemiparesis, with no withdrawal to deep pain. A lumbar puncture revealed no abnormalities. A computed tomographic scan of the brain was read as normal.

On day 8, the patient was noted to have rhythmic movements of the left arm, thought to be focal seizures. The patient was given phenytoin (Dilantin). He became diffusely rigid and diaphoretic. His blood pressure remained 150/90. An electrocardiogram revealed acute S-T elevations in the inferior leads. Two hours later, the patient proceeded to ventricular tachycardia, then fibrillation, followed by asystole, and subsequently died.

PATHOLOGY

The temporal artery biopsy demonstrated extensive mononuclear cell infiltration of the intima, media, and adventitia, including epithelioid and multinucleated giant cells (Fig. 1). Numerous disruptions in the internal elastic lamina were also present (Fig. 1, insert). Interestingly, serial sections revealed that the entire length of the biopsy was uniformly affected, and there was no evidence for the existence of "skip lesions" in the specimen.

At autopsy, posterolateral and septal myocardial infarctions were found, which appeared healed but with recent extension. The right coronary artery revealed findings similar to that found in the temporal artery biopsy, including the presence of giant cells at the level of the elastic lamina (Fig. 2). The right coronary artery also exhibited athero-

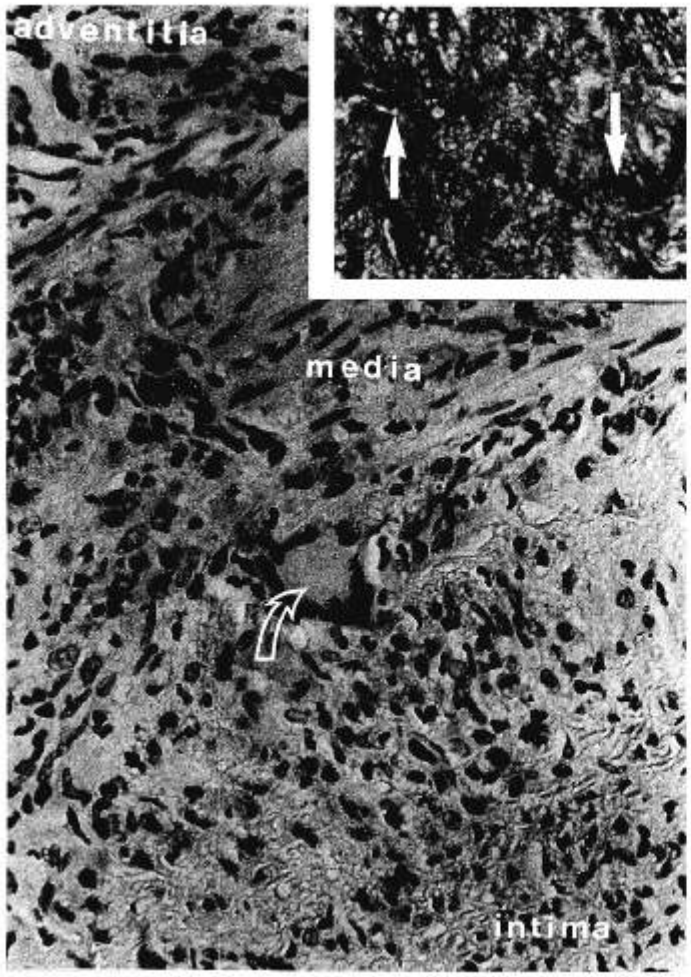

FIGURE 1.
Light micrograph demonstrates inflammatory cell infiltration of temporal artery including multinucleated giant cell (arrow) (501×). Inset: White arrows indicate discontinuity in elastic lamina (750×).

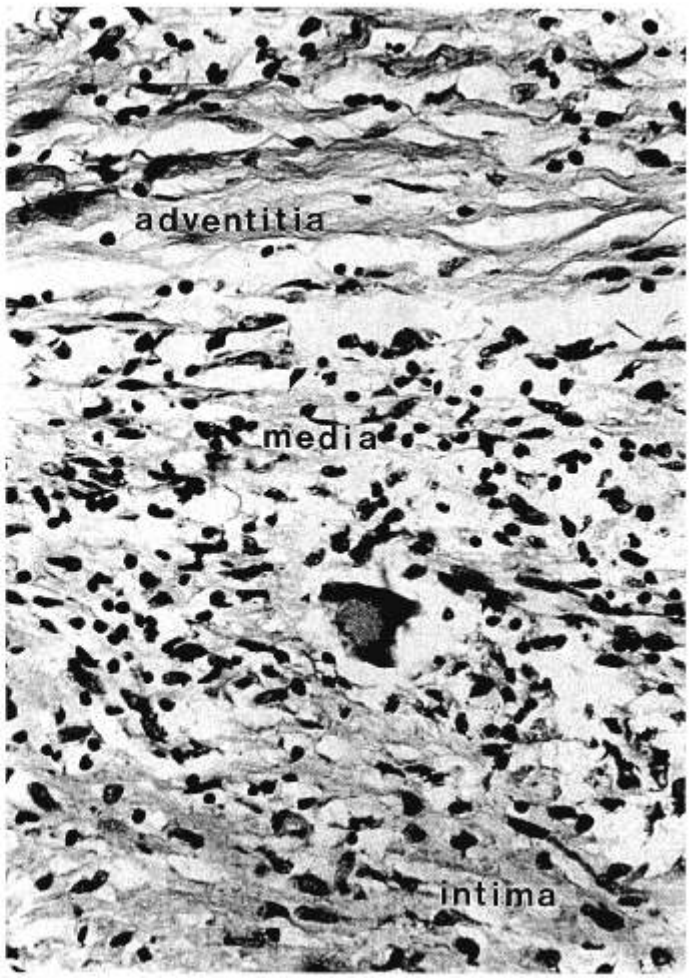

FIGURE 2.
Light micrograph of coronary artery demonstrates inflammatory cell infiltrate including multinucleated giant cell (462×).

sclerotic changes and a large thrombus (Fig. 3). Occlusion of the right coronary artery was 95% at its ostium. Occlusion of the left coronary artery was approximately 80%. The degree of aortic atherosclerosis was graded VI/X.

Additional findings at autopsy were cardiomegaly (500 g) with left ventricular hypertrophy (2.2 cm) and congestive heart failure. Incidental findings included multinodular goiter (120 g), prostatic hypertrophy, diverticulosis coli, and the presence of a small (0.5 cm) gastric leiomyoma.

DISCUSSION

Our patient was a 75-year-old male with giant cell arteritis who was treated with high-dose oral corticosteroids, yet died from a myocardial infarction attributable to coronary artery involvement with arteritis. Spencer and Hoyt[6] reported a case of temporal arteritis that was associated with a myocardial infarction and generalized convulsions. They found granulomatous inflammation of the coronary arteries, but in none of the intracerebral vessels, as was also true for our patient.

In our case, the thrombus and giant cell arteritis were superimposed on atherosclerotic changes in the coronary vessels. Giant cells are diagnostic for temporal arteritis, but can also be present with cholesterol deposits or calcified debris. In the latter, the giant cells would not be accompanied by a florid mononuclear infiltrate, as was present in our case. Martin et al.,[5] however, reported a case in which the temporal arteritis alone resulted in a fatal thrombus of a coronary vessel. In that patient, the coronary vessels were free from atherosclerotic processes, as was the aorta. Despite this, it has been reported that aortic involvement may be quite common in temporal arteritis (11%).[3] This suggests that aortitis, as a secondary phenomenon, may also be culpable for the coronary artery involvement in temporal arteritis.[7] However, a case of temporal arteritis with coronary involvement leading to myocardial infarction and death without histologic involvement of the aorta has been reported.[5]

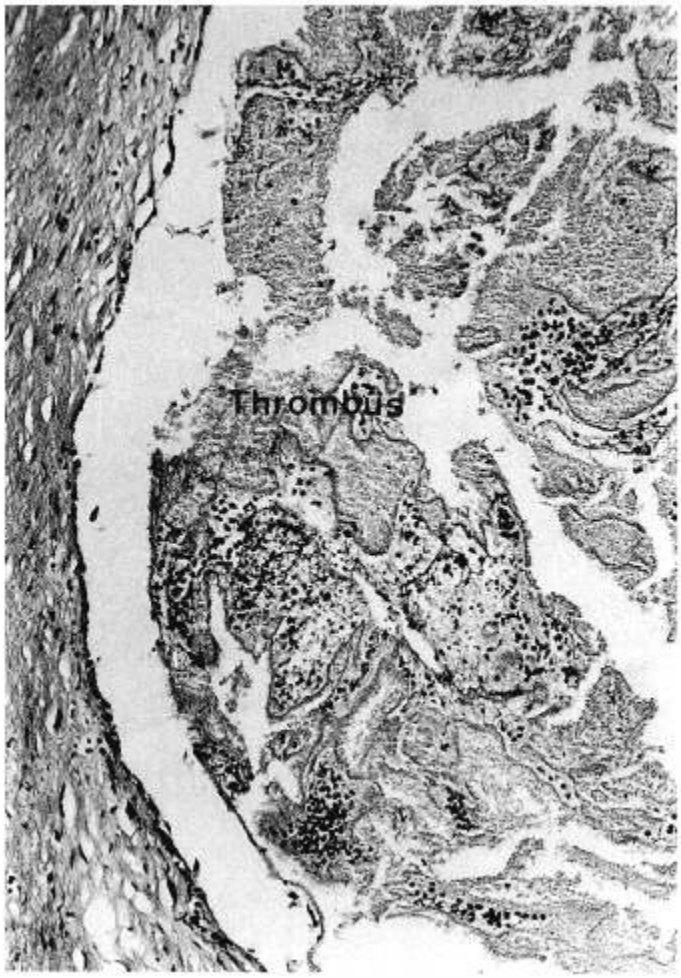

FIGURE 3.
Light micrograph demonstrates thrombus within the lumen of a coronary artery. (189×).

Wadman and Werner[8] suggest that corticosteroid treatment may have contributed to thromboembolism in 4 of their 71 patients. They emphasize that no local angiitis was observed in these four cases. In contrast, our patient had histologically proven coronary arteritis with giant cells. Graham[9] and Auplat et al.[10] separately concluded that steroid therapy was correlated with a higher death rate. Auplat reviewed five cases and found that in three of these five, the myocardial infarction occurred between the third and seventh day of treatment, just as occurred in the present case.[10] Oral corticosteroids (60 to 100 mg/day, p.o.) remain the treatment of choice in cases of giant cell arteritis, although it has been suggested that, because corticosteroids may increase blood coagulability, that anticoagulants should be considered to minimize the risk of thromboembolism.[8]

An epidemiologic study[11] in 1978, found the occurrence of cardiac disease in patients with temporal arteritis to be similar to that in the general population. More recently, it has been suggested that ischemic heart disease is an important presentation of temporal arteritis.[12] Indeed, Graham[9] suggests that the mortality rate specifically attributable to myocardial infarction in temporal arteritis may be higher than previously thought and that the overall mortality rate may also be elevated. Of their 90 patients with biopsy-proven temporal arteritis, 32 died, 13 of whom died of myocardial infarction.

In a literature review on the subject published in 1980, Martin et al.[5] reported being able to find only six reported cases in which coronary artery involvement in giant cell arteritis had been confirmed. In an extensive search of the literature (Table 1), we have been able to document 31 cases of myocardial infarction with confirmed coronary artery involvement in GCA, suggesting that this complication has actually been much more common than previously thought.[13–23]

SUMMARY AND RECOMMENDATION

Early recognition of temporal arteritis is often difficult, in view of the variety of clinical presentations. Its importance has long been established because early treatment with corticosteroids may prevent visual loss associated with temporal arteritis and possibly prevent a fatal myocardial infarction. Furthermore, the possibility of coronary arteritis leading to myocardial ischemia underscores the need for early diagnosis. In our patient, the findings at presentation were limited to pallor of the optic disc, reduced Snellen acuity, and weight loss. This case emphasizes that elderly patients presenting with sudden painless loss of vision should be worked up immediately for the possibility of temporal arteritis and coincident coronary arteritis. Furthermore, accumulating evidence during the past 10 years argues for wider appreciation of the association between GCA and myocardial infarction.

TABLE 1.
Review of reported cases of temporal arteritis with coronary involvement.[a]

Author	Age (yr)	Sex	Temporal Artery Biopsy	ESR	Coronary Artery GCA	Main Cause of Death
Lie et al.[17]	84	M	Positive	No data	Positive	Myocardial infarction
Neri et al.[18]	78	M	Positive	No data	Positive	Myocardial infarction
Hupp et al.[19]	82	F	Positive	88 mm/h	Positive	Myocardial infarction
Paulley[12]	67	F	Positive	93 mm/h	No data	Myocardial infarction
	55	F	Positive	71 mm/h	No data	Myocardial infarction
Save-Soderbergh et al.[20]	73	F	Positive	83 mm/h	Positive	Myocardial infarction
	85	F	Negative	50 mm/h	Positive	Myocardial infarction
Martin et al.[5]	77	F	Positive	85 mm/h	Positive	Myocardial infarction
Morrison and Abitol[14]	67	M	Positive	61 mm/h	Positive	Myocardial infarction
Klein et al.[3]	73	F	No data	98 mm/h	Positive	Myocardial infarction
Ainsworth et al.[15]	85	N.D.	Positive	No data	Positive	Myocardial infarction
Bengtsson and Malmvall[16]	82	F	Negative	50 mm/h	Positive	Myocardial infarction
Harrison and Bevan[21]	65	M	No data	123 mm/h	Positive	Myocardial infarction
Crompton[23]	71	M	Positive	49 mm/h	Positive	? Myocardial infarction
Spencer and Hoyt[6]	77	M	Positive	No data	Positive	Myocardial infarction
Ritama[22]	63	F	Positive	110 mm/h	Positive	Myocardial infarction
Auplat et al.[10]	85	M	Positive	60 mm/h	Positive	Myocardial infarction
Ostberg[13]	N.D.	No data	Positive	No data	Positive	Myocardial infarction
Graham[9] had 12 patients with GCA that later died from myocardial infarction						

[a] GCA, giant cell arteritis.

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