

Pauley J W & Hughes J P. Giant-cell arteritis, or arteritis of the aged.
Brit. Med. J. 2:1562-7, 1960.
[Ipswich Hospitals, England]

Using data from 76 cases collected over six years, the article gave clinical support to the hypothesis that polymyalgia rheumatica and giant-cell (temporal) arteritis were different presentations of the same disease. [The *SCI*⁹ indicates that this paper has been cited in over 195 publications.]

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Eugene Garfield has kindly asked me why I think this article has been frequently cited: chiefly, I suspect, because it gave clinical support to a suggestion I made in the *Lancet* in 1956 that polymyalgia rheumatica (PMR) and giant-cell (temporal) arteritis (GC(T)A) were different presentations of the same disease.¹ This idea was pooh-poohed at the time. Perhaps another reason for the paper's frequent citation is that the series was a large one (76 cases) collected over six years in Ipswich, whose hospitals serve a mixed rural and urban population of 300,000.

The clinical observations in the paper were made possible by careful study of the "natural history" of the disorder and were facilitated by a relatively static community and a good liaison with the patients' general practitioners. In this way relapses of the disease following renewed emotional stress and/or premature reduction of corticosteroids were often seen in one or more of its diverse presentations.

The article was also the first to point out that the disorder was common yet often undiagnosed because only a few of its numerous manifestations were sufficiently recognised. For example, cases were likely to be referred to neurologists, cardiologists, vascular surgeons, gastroenterologists, otologists, dental surgeons, psychiatrists, and geriatricians rather than rheumatologists and ophthalmologists. It was suggested that when elderly people begin to fail mentally or physically, GC(T)A should be one of the first conditions considered—not one of the last.

The broad spectrum of presentation has since been confirmed by many workers. Apart from the prime importance of a patient's history, other helpful physical signs are tenderness and thickening of arteries other than those in the temporal region, for

example, facial, iliac, femoral, and subclavian arteries. If associated bruits over long sections of artery are also present, the likelihood of arteritis is enhanced. (In 1970 I added tenderness of the abdominal aorta to this list.)

Proof that PMR and GC(T)A were manifestations of the same disorder was not published until 1963² and 1965.³ The latter study by B. Hamrin and colleagues demonstrated the characteristic histology in the larger arteries of the muscles of hip and shoulder girdles. Evidence that clinical reports greatly underestimate the incidence of the disorder was provided in a most important article by G. Ostberg,⁴ who found biopsies of aorta and/or temporal artery positive in 1.7 percent of 889 consecutive routine autopsies. Another important article is that by R.G. Klein and coauthors⁵ on large artery involvement in GC(T)A. It emphasizes the risk of the aortic dissection (in poorly monitored cases) with smouldering disease, together with the frequent presentation of claudication.

In a lighter vein, it may be a comfort for frustrated authors to know that the article was rejected by one journal and then by the one that eventually accepted it. Fortunately for me, editors then were more independent of their expert assessors than they are today. I pleaded to my editor that the fact that a cardiological advisor did not like my inclusion of aortic and coronary artery involvement in GC(T)A because he had not personally seen it, or bothered to read about it, was insufficient reason for rejection; I also reminded him that the speed of a convoy was that of the slowest ship and that if assessors' entrenched opinions were always to prevail, much deserving work would be lost.

I am also grateful to the 1960 editor of the *British Medical Journal* for allowing me to include the brief psychosomatic histories that stressed the importance of mourning and loss shortly prior to the onset of GC(T)A, as well as its relevance to the numbers of affected widows and widowers with their now-recognised higher rates of morbidity and mortality. Helping these patients through their unresolved loss and mourning reactions is most important if effective reduction or cessation of corticosteroid dosage is to be achieved without relapse.^{6,7}

Finally, in 1966 Landberg suggested in jest that PMR should be renamed Pauley myalgia rheumatica. I thanked him but felt that the nomenclature was already too complicated!

1. Pauley J W. Letter to editor. (Anarthritic rheumatoid disease.) *Lancet* 271:946, 1956.
2. Alestig K & Barr J. Giant-cell arteritis: a biopsy study of polymyalgia rheumatica, including one case of Takayasu's disease. *Lancet* 1:1228-30, 1963. (Cited 140 times.)
3. Hamrin B, Jonsson N & Landberg T. Arteritis in polymyalgia rheumatica. *Lancet* 1:1193-6, 1965. (Cited 105 times.)
4. Ostberg G. Morphological changes in the large arteries in polymyalgia arteritica. *Acta Pathol. Microbiol. Scand.* (Suppl. 237):6-8, 1973.
5. Klein R G, Hunder G G, Stanson A W & Sheps S G. Large artery involvement in giant cell (temporal) arteritis. *Ann. Intern. Med.* 83:806-12, 1975. (Cited 150 times.)
6. Pauley J W. Coronary ischaemia and occlusion in giant cell (temporal) arteritis. *Acta Med. Scand.* 208:259-63, 1980.
7. ———. Pathological mourning: a key factor in the psychopathogenesis of auto-immune disorders. *Psychother. Psychosom.* 40:181-90, 1983.

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