OP1. HISTORY OF GIANT CELL ARTERITIS (GCA) AND POLYMYALGIA RHEUMATICA (PMR)
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Whether GCA and PMR existed in antiquity, or are conditions of modern times is unknown. Hutchinson’s report in 1890 of an elderly man with “red streaks” on his forehead appears to be the first detailed case. In 1930, Schmidt discussed a patient with symptoms of GCA but attributed them to an aneurysm. In 1932, Horton et al. reported 2 patients whose temporal artery biopsies showed arteritis. In 1937, these workers summarized 7 cases who manifested systemic symptoms, headache, fever, anemia and jaw claudication. Temporal artery biopsies showed granulomatous inflammation with giant cells. The authors considered this a new vasculitis calling it “temporal arteritis”.

Over the next decade occasional cases were published describing many of the symptoms of GCA. In 1938, Jennings observed a patient with visual loss. In the 1941, Gilmour reported autopsy findings in 3 older patients with inflammation in large vessels that appeared similar to temporal arteritis. He suggested the name “giant cell arteritis”. In 1946, Kilbourne and Wolff, noted involvement of multiple cranial vessels and offered the name “cranial arteritis”. PMR was also described in the late 19th century, by Bruce in 1888. Some physicians (e.g. Copeman, Slocumb, Hamrin) stated that PMR was recognized as a clinical syndrome in the 1930’s or even before, although not written about. However, it wasn’t until the 1940’s and early 1950’s that reports on this condition started to appear under a variety of names such as secondary fibrositis, periartthropathitis, myalgic syndrome of the aged, pseudo-polymyositis rhizomelique, and anarthritic rheumatoid disease. Barber’s article in 1957 in which he coined the term “polymyalgia rheumatica” caught the attention of many, and this name was eventually adopted. In the 1950s, shortly after the discovery of the potent anti-inflammatory properties of cortisone, Birkhead and colleagues showed that PMR responded well to this drug. Later, low doses were found also to be very effective in PMR.

The link between GCA and PMR wasn’t recognized immediately. But, in the 1940’s and 1950’s, several authors noted that some patients with GCA had findings of PMR. The reports by Paulley and Hughes, Alestig and Barr, Hamrin, and others in the 1960’s, led to a wider appreciation of a close connection between PMR and GCA. Modern research on these conditions has increased our understanding substantially. However, much more knowledge is needed and only waits to be discovered by the skilled, inquiring, and dedicated scientist.
OP4. THE EPIDEMIOLOGY OF GIANT CELL ARTERITIS

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The incidence of GCA is highest in Scandinavia and populations of Nordic descent (e.g., Olmsted County, USA). The annual incidence in Scandinavia is 15–35/100,000 persons aged >50 years [1] and in Olmsted County 18.8/100,000 persons aged >50 years [2]. Studies from other regions of Europe report lower incidence rates. Multiple regression analysis suggests there is a significant trend to higher incidence with increasing latitude [1]. Some of the differences may reflect different diagnostic approaches in particular the biopsy rate and intensity of histological search for evidence of vasculitis. Many studies are hospital based and retrospective, and this will underestimate the incidence.

There have been few studies from non-white Caucasian populations. In Southern California a retrospective study from an ophthalmology unit of patients undergoing temporal artery biopsy [3] reported 19/66 white Caucasian patients had a positive biopsy, compared with 1/9 Asian, 0/40 Hispanic and 0/6 African-Americans. Few clinical details are provided and it is possible that referral bias might account for these results. A national survey from Japan reported a prevalence of 1.47/100,000 [4], considerably lower than European figures.

Several long-term studies have reported a steady increase in incidence [1]. In Olmsted County between 1950–54 and 1980–84 there was an increase from 6.7/100,000 to 28.5/100,000 persons aged >50 years. The rate then stabilized and has not risen further [2].


OP5. GCA AND PMR: A GENERAL PRACTITIONER’S PERSPECTIVE

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PMR can be a challenging condition within primary care. Diagnosis can be difficult as it largely depends on having a high index of suspicion supported by history, examination and raised levels of inflammatory markers which of course are not specifically diagnostic for PMR. The incidence of PMR has variously been reported as between 13 and 68 per 100,000 of the population aged over 50 with a peak incidence around age 70. As most general medical practitioners in the UK look after a patient population of around 2000 patients it follows that a new case of PMR does not occur frequently in most GPs clinical practice. General practitioners whose patient population is skewed towards a younger age group may only rarely see a case of PMR and may develop little experience of dealing with the condition.

When PMR presents with classical clinical features of a sudden onset of stiffness and pain in shoulder and pelvic girdles, with tenderness of shoulder muscles and some systemic features of debility, weight loss, tiredness and low-grade fever together with a raised ESR the diagnosis can be fairly straightforward. If the symptoms are not rapidly relieved within a few days with steroid therapy (15mg/day prednisolone) the diagnosis should be re-considered and various laboratory tests should be undertaken to exclude conditions such as multiple myeloma, other malignancy, connective tissue disease, inflammatory arthritis, myopathy, and myositis.

One of the most difficult differential diagnoses is that of rheumatoid arthritis. A polyarticular presentation of RA may occur especially in elderly patients and in the very early stages before there are overt joint signs such as synovitis, may very closely resemble PMR. Generally these patients do not have a rapid response to steroid therapy.

It is important to remember the overlap between PMR and GCA. These two conditions may in fact represent opposite ends of the spectrum of the same disease with PMR at the milder end. All patients suspected of having PMR should be asked about symptoms of headache, jaw claudication and scalp tenderness and if present the patient should be referred immediately for a temporal artery biopsy and treatment with higher dose steroids. If immediate referral is not possible patients should be treated with high dose steroids in primary care to avoid possible blindness.

PMR is a challenging condition in primary care but can be very satisfying to treat as patients often have a very dramatic response to therapy and are most grateful for the relief of their symptoms.

OP6. REVIEW OF DIAGNOSTIC CRITERIA FOR POLYMYALGIA RHEUMATICA/GIANT CELL ARTERITIS

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Polyalgia rheumatica and giant cell arteritis are nowadays normally accepted as opposite ends of the same disease spectrum. Both conditions are probably manifestations of a vasculitic process. Typically patients with eye involvement present to ophthalmologists; patients with polymyalgia to rheumatologists. Perhaps, as a result, separate criteria sets exist for both conditions.

For giant cell arteritis the criteria adopted by the American College of Rheumatology, based on those proposed by Hunder et al. in 1990 [1], remain the gold standard. Three of the following five criteria are required:

(a) age of onset >50 years
(b) new headache
(c) temporal artery abnormality
(d) increased ESR
(e) abnormal artery biopsy

These have replaced earlier criteria proposed by Wilke and colleagues.