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BSR Guidelines

The 2026 British Society for Rheumatology guideline for the treatment of polymyalgia rheumatica

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Abstract

Polymyalgia rheumatica (PMR) is a common inflammatory rheumatic condition in older adults. Glucocorticoids (GCs) remain the cornerstone of treatment, but many people with PMR relapse during tapering and are exposed to prolonged GC therapy with associated toxicity. There is therefore a need for evidence-based strategies that optimize disease control while minimizing GC-related harm. This guideline was developed in line with the British Society for Rheumatology's Creating Clinical Guidelines Protocol (v6.0), using GRADE and AGREE II frameworks, and supersedes previously published 2009 BSR/BHPR PMR guidelines. Several key recommendations have been included in this guideline update. These include

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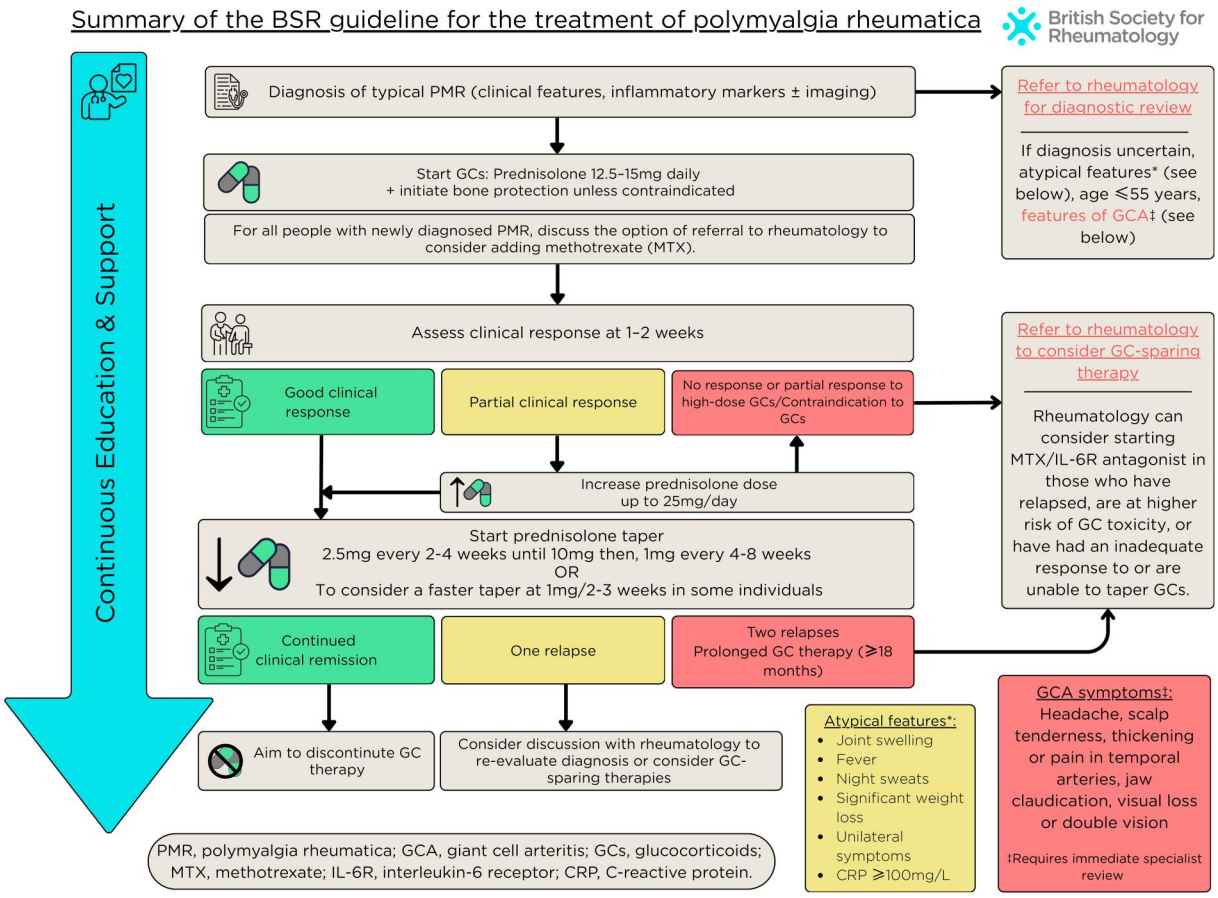
when to discuss or refer people with PMR to rheumatology; initiation of GCs and tapering with options for a faster GC taper; consideration of early methotrexate; IL-6 receptor antagonist use in people with PMR who are unable to taper GCs or have had an inadequate response; and bone protection in all people with PMR. Furthermore, the guideline emphasizes the importance of ongoing patient education and counselling, monitoring for glucocorticoid toxicity and suggestions for when to refer people with PMR to secondary care. This guideline aims to support clinicians in delivering safe, effective and individualized care for people with PMR, while promoting judicious use of GCs and timely introduction of GC-sparing therapies where appropriate.

Graphical abstract



When to refer a person with PMR to rheumatology. PMR: polymyalgia rheumatica; RF: rheumatoid factor; anti-CCP: anti-citrullinated peptide antibody; CRP: C-reactive protein.

Keywords polymyalgia rheumatica, rheumatic diseases, DMARDs, therapies, health services and practice

Scope and purpose

Background to disease

Polymyalgia rheumatica (PMR) is an inflammatory rheumatic disease that affects ∼1% of the UK population. The lifetime prevalence is 2.4% in females and 1.7% in males [1, 2]. PMR is most commonly diagnosed in people over the age of 50; the peak incidence is between the seventh and eighth decade [3]. A general approach to the assessment of people with PMR was outlined in the 2015 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) recommendations for the management of PMR [4].

PMR typically presents as pain and stiffness affecting both shoulder girdles and often also the neck and the hip girdles. The onset of symptoms may be subacute over days to weeks, or more insidious. Pain and stiffness are usually worse in the early hours of the morning and improve in the afternoon. These symptoms may cause difficulty with daily activities such as dressing, combing hair, rising from a seated position or climbing stairs. Fatigue is also frequently reported. Laboratory tests typically reveal elevation of inflammatory markers [both C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR)] above the patient's baseline value, although in some patients with a low baseline, this may still be within the laboratory reference range. Patients may also have a mild anaemia that is typically normocytic and normochromic.

Imaging tests may be done to rule out mimicking diseases, whereas positive identification of PMR on imaging would require a modality that can reveal inflammation around tendons, ligaments and joint capsules, such as musculoskeletal ultrasound,

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magnetic resonance imaging (MRI) or 18-fluorodeoxyglucose positron emission tomography. However, these imaging tests require specialist expertise for interpretation and are not usually necessary in clinically typical PMR. Various diagnostic or classification criteria have been proposed for PMR [5–9] but none have been validated and should not be used for clinical diagnosis.

The impact of untreated PMR on daily function and quality of life can be profound; therefore, prompt diagnosis and treatment is important. The clinical diagnosis of PMR is based on symptoms, physical examination and a panel of standard laboratory tests, with a directed search for other diseases that may mimic PMR, depending on the clinical presentation. The differential diagnosis is broad due to the diffuse nature of the symptoms associated with PMR. These include serious diseases (e.g. malignancy, infection, myositis, vasculitis), glucocorticoid-responsive diseases for which other treatments are available [e.g. inflammatory arthritis including rheumatoid arthritis (RA), spondyloarthritis and crystal arthritis], and other diseases that may cause pain and stiffness in a similar distribution (e.g. cervical spinal stenosis, shoulder osteoarthritis, frozen shoulder, rotator cuff disease with or without hip osteoarthritis, or fibromyalgia).

Approximately 5–10% of patients with PMR may later develop giant cell arteritis (GCA) and therefore ongoing vigilance for this important complication is essential [10]. Many patients with GCA also report symptoms of PMR associated with their disease, either at GCA onset or at GCA relapse, and this requires management of both conditions concurrently [11].

In the UK, most patients with PMR are diagnosed and managed in primary care, although some patients are diagnosed in secondary care or referred for a specialist opinion during treatment. The mainstay of PMR treatment is glucocorticoids (GC), also called corticosteroids or 'steroid' therapy, typically oral prednisolone at a starting dose of 15 mg daily. The symptoms of PMR typically respond dramatically to these moderate GC doses; lack of significant response after one week should prompt reconsideration of the diagnosis. However, in some cases, the initial prednisolone dose needs to be increased up to a maximum of 25 mg daily to achieve acceptable symptom response. Once the symptoms have been controlled, the dose is tapered gradually over 9–12 months; ∼50% of patients who follow a standard taper do not relapse and are able to stop treatment without needing to escalate the GC dose [12, 13]. If the taper is successful and they have not relapsed by 6–12 weeks after stopping all treatment, then the prognosis is good and most patients will not have a recurrence of disease [2].

The other 50% of people with PMR follow a relapsing course, with one or more relapses necessitating GC dose escalation. This multiple relapsing pattern can result in very prolonged GC therapy. In a meta-analysis, 51% of patients were still taking GCs after 2 years and 25% after 5 years from PMR diagnosis [13]. These patients are likely to experience significant toxicity from long-term GC therapy due to the high cumulative GC dose [14]. Patients with multiple relapses or prolonged GC therapy have historically been prescribed off-label disease-modifying antirheumatic drugs (DMARDs), such as methotrexate (MTX), as GC-sparing agents [15]. More recently, the biologic interleukin-6 receptor (IL-6R) antagonist, sarilumab, has been licensed for PMR, and is beginning to be prescribed in the United States of America (USA) but at the time of writing it is not commissioned by the UK National Health Service (NHS). Clinical trials of various other agents are also in process.

Need for guideline

The last British Society for Rheumatology (BSR) guideline for the management of PMR was published in 2009 [16]. EULAR/ACR recommendations were published in 2015 [17]. Since then, further evidence about treatment of PMR has emerged. In this guideline, we focus on the treatment of people with confirmed PMR.

Objectives of this guideline

This guideline provides up-to-date, evidence-based and expert recommendations to support health professionals in the clinical management of people with PMR.

Target audience

This guideline is intended for health professionals who are involved in the care of people with PMR, including general practitioners, rheumatologists, nurses, pharmacists, physiotherapists and other allied health professionals. It may also be informative for patient groups and patient support organizations. People with PMR are advised to consult a licensed healthcare professional before making any decisions about their disease or its treatment.

The areas the guideline does not cover

This guideline does not cover the diagnosis of PMR, the diagnosis or management of GCA, or immune checkpoint inhibitor-induced PMR. Guidelines for these are published elsewhere and can be found here [18, 19].

Stakeholder involvement

These recommendations were commissioned by the BSR Guidelines Steering Group (GSG). The guideline was developed by a multidisciplinary guideline working group (GWG) co-chaired by M.Y. (rheumatologist) and S.L.M. (rheumatologist) and coordinated by T.T. (rheumatology trainee) as the guideline fellow. The GWG included two other rheumatologists (S.S., J.D.R.), a rheumatology clinical nurse specialist (C.B.), four general practitioners (H.T., S.E., P.M., D.W.), three physiotherapists (N.S., S.B., A.V.O.), two pharmacists (C.J., E.H.) and three experts by experience of PMR (J.M., S.L., K.M.). J.M. is also a representative from the charity PMRGCAuk. The systematic literature review was led by researchers with evidence synthesis expertise (D.A.vdW., L.F., N.C.).

Rigour of development

This guideline was developed in line with the British Society for Rheumatology's Creating Guidelines Protocol (v6.0), using GRADE and AGREE II framework to ensure methodological rigour and transparency [20].

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Scope of the guideline

The guideline scope was published in February 2024 [21]. Eighteen key clinical PICO (patient, intervention, comparator, outcome) questions were developed by the GWG to guide the systematic literature review process. A further PICO question addressing long-term GC outcomes was developed; in recognition of the limited randomized evidence in this area, this was informed by observational studies.

Searches

The full methodology and evidence tables are provided in Supplementary Data S1 and S2–S4. The literature search was informed by the guideline scope and registered in advance (PROSPERO: CRD42024586393).

An information specialist (N.C.) designed and performed searches across eight databases (MEDLINE, EMBASE, EMCARE, CINAHL Plus, AMED, PsycINFO, Pedro and Cochrane CENTRAL) without language restrictions, covering the period between inception and 20 May 2024 (updated on 14 January 2026) for RCTs (stage 1) and 4 November 2024 for observational studies (stage 2). Full search details are provided in Supplementary Data S1.

Screening and selection

The GWG agreed eligibility criteria for randomized controlled trials (RCTs) and observational studies for the systematic literature review. Observational studies were included if they included at least 100 participants and were prospective clinical cohort studies, retrospective studies using registry data, or electronic health record studies. Other observational study types including cross-sectional studies, case-control designs, case series and other publication types were excluded, as well as full papers in any language other than English without an English translation. Further detail of inclusion and exclusion criteria and PRISMA flow diagrams are provided in Supplementary Data S1 and S5. All titles, abstracts and full texts were screened by L.F. or N.C. and T.T., with disagreements resolved by D.A.vdW. At both stages 1 and 2, two reviewers (T.T. and L.F. or N.C.) independently screened the titles first, followed by the abstracts and full texts. A third reviewer (D.A.vdW.) resolved any disagreements between the reviewer's selection decision.

Data extraction and risk of bias assessment

A standardized data extraction form was developed, piloted and used to collect data for analysis. Data were collected on country, study design, characteristics of the study population (including disease duration at screening, clinical symptoms, drug history related to GCs and/or DMARDs, and the presence of comorbidities), intervention characteristics and efficacy and safety outcomes. Cochrane risk of bias tool [22] was used to assess risk of bias for RCTs and controlled clinical trials. For observational studies, we used an adapted version of the QUIPS tool [23]. One reviewer undertook the data extraction and risk of bias assessment, which was independently checked by a second for correctness and consistency.

Quality of supporting evidence

Evidence tables were prepared for each guideline question (Supplementary Data S2–S4), and the quality of evidence for each outcome was summarized using the GRADE [24]. Each outcome was graded where A, B and C represent high, moderate and low/very low quality of evidence.

Strength of recommendation

A strength of recommendation rating of 1 (strong) or 2 (weak) was given to each recommendation statement, accounting for the risks and benefits. A strong recommendation is one in which the benefits clearly outweigh the risks whilst a weak recommendation is one in which the recommendation may be conditional, the risks and benefits are closely balanced or uncertain based on the current evidence.

Strength of agreement

Each recommendation was discussed and refined across a series of eight virtual GWG meetings. Each recommendation was subject to voting by all GWG members, with strength of agreement (SoA) scores using a scale of 1 (no agreement) to 100 (complete agreement). The mean SoA score for each recommendation is presented as a percentage. If any GWG member gave a score of $<$80% agreement to any single recommendation, then wording was altered until the 80% minimum agreement had been reached.

Plan for review

This guideline should be reviewed and updated in five years.

The guideline

This guideline is split into two sections: the overarching principles and evidence-based recommendations for the treatment of people with PMR. Each recommendation statement is accompanied by the strength of recommendation (1 or 2), the quality of supporting evidence (A, B or C) and the mean SoA score (as a percentage).

Overarching principles

The GWG agreed upon nine overarching principles (Table 1) which reflect current best practice according to expert consensus.

Overarching principle 1	Decisions about the clinical management of PMR should be shared between the treating clinician and the person with PMR, incorporating individual preferences and clinical risk profile. (GRADE 1C, SoA 96%)
Overarching principle 2	Care for people with PMR should be individualized: adjusted to disease course, comorbidities and treatment side effects. (GRADE 1C, SoA 97%)

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Overarching principles by their nature are not based on the evidence synthesis from the systematic literature review but define the context of good care and best practice at the time of writing.

The management of PMR should be person-centred, aiming to improve quality of life via treatment of symptoms, while seeking to manage and mitigate any treatment-related harms that may occur over the short or long term. Partnership between the person with PMR and their treating clinician(s) is essential to enable shared decision-making and an individualized approach to care.

Overarching principle 3	The clinical management of people with PMR across their disease course should be delivered by individuals within a clinical team with appropriate knowledge and experience, striving for continuity of care where possible. (GRADE 1C, SoA 95%)
Overarching principle 4	People with PMR should be managed in primary care with support from secondary care where clinically indicated by clinical complexity, diagnostic uncertainty, or suboptimal treatment response. (GRADE 1C, SoA 95%)
Overarching principle 5	Consider discussing with rheumatology after any relapse of PMR using established liaison pathways like NHS Advice and Guidance. In the event of a second relapse, refer to rheumatology. (GRADE 1C, SoA 96%)

This includes decisions such as tapering the GC dose, escalating if needed in the event of relapse, and education and counselling on sick day rules. As the clinical course is so variable, and because comorbidities may add clinical complexity to the wide range of adverse effects that may be caused by long-term GCs, clinical care needs to be individualized to the needs of the patient.

At present, in the UK, PMR is most often managed in primary care and may involve general practitioners (GPs), advanced nurse practitioners, pharmacists and physiotherapists. Oral GC therapy is generally prescribed in primary care. Because tapering occurs over a prolonged period of time, continuity of care and good interprofessional communication can help to ensure a consistent plan.

Referral to secondary care

Patients are most often referred to rheumatology to confirm or refute the diagnosis of PMR, and primary care teams are therefore encouraged to refer patients who have atypical features. This is because long-term GC monotherapy can cause significant harm and for this reason it is important to be sure that the diagnosis is correct.

Rheumatologists can also have an important role in advising on ongoing management, including initiation of GC-sparing therapies where appropriate, as well as advising on treatment-related toxicities including bone protection. Once patients have been stable on GC-sparing therapies for a period of time, prescribing and monitoring in primary care under shared care arrangements may be considered. In between scheduled

Table 1 Overarching principles for the management of PMR.

Overarching principle 1	Decisions about the clinical management of PMR should be shared between the treating clinician and the person with PMR, incorporating individual preferences and clinical risk profile. (GRADE 1C, SoA 96%)
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Overarching principle 5	Consider discussing with rheumatology after any relapse of PMR using established liaison pathways like NHS Advice and Guidance. In the event of a second relapse, refer to rheumatology. (GRADE 1C, SoA 96%)
Overarching principle 6	People with PMR receiving glucocorticoid therapy should be reviewed periodically according to need by a healthcare professional. (GRADE 1C, SoA 91%)
Overarching principle 7	All people with PMR must receive clear and accessible education about their condition and management plan, including potential benefits, risks and side effects of treatments. (GRADE 1C, SoA 96%)
Overarching principle 8	People with PMR should have access to educational material and support including information on self-care such as diet, physical activity and pacing. (GRADE 1C, SoA 97%)
Overarching principle 9	People with PMR should be supported to recognize and report anything that might relate to relapses of PMR, treatment toxicities or indicate other conditions including GCA. (GRADE 1C, SoA 96%)

PMR: polymyalgia rheumatica; SoA: strength of agreement; NHS: National Health Service; GCA: giant cell arteritis.

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consultations, people with PMR who are under the care of a rheumatology department can also access advice, education and support from experienced clinical nurse specialists via enquiry lines and similar services.

Historically, most of the evidence for the benefit of GC-sparing therapy has been derived from clinical trials of people with new-onset PMR, as this also allows investigators the opportunity to simultaneously verify the diagnosis of PMR. Yet, if around half of patients with new-onset PMR will be able to taper and stop GCs without having a single relapse, it has been argued that initiating GC-sparing therapy at disease onset is not necessary, unless reliable risk factors for prolonged therapy or relapsing course could be identified prospectively to challenge this assertion. Observational studies have attempted to address this. In a registry study of 26 102 people with PMR from rheumatology practices across the USA [15], female patients and those with a higher number of comorbidities were more likely to continue to be prescribed GCs for >1 year of follow-up. A previous meta-analysis had found that prior evidence regarding risk factors for relapse or prolonged therapy was inconsistent [13]; this likely relates to the small size of studies published at that time. Therefore, as it has been difficult to identify at diagnosis which people with PMR are likely to follow a relapsing course *vs* an uncomplicated course, the common practice in UK primary care has been to wait until multiple relapses have occurred before referring to secondary care. By this point, however, it can be challenging for secondary care clinicians to offer useful solutions for several reasons. First, the window of opportunity for early intervention may have passed. Evidence from the PMR-MODE [25] trial suggests that MTX is most effective when introduced early in newly diagnosed disease, and GC-dependency may become established with repeated relapses and prolonged exposure. Second, cumulative GC toxicity will already have accrued, which may limit the potential benefit of GC-sparing strategies. Third, people with PMR may have developed comorbidities or complications of long-term GC use that constrain future treatment options.

In this guideline update, the GWG spent some time explicitly outlining when to involve rheumatology. Patients who follow an uncomplicated course without relapse or treatment toxicity might not need to see a rheumatologist, but if specialist intervention is needed, it is likely to be of greatest value to the patients if referral occurs early, at the first or second relapse. Given the digital channels now available for quick specialist advice (e.g. NHS Advice and Guidance) we have suggested that it would be reasonable to discuss the patient at the first relapse, with the potential to convert to a formal referral to specialist care if deemed appropriate. It was agreed that all people with PMR should be referred to rheumatology after two relapses, as these patients are at higher risk of subsequent relapses. This principle is intended to apply to all NHS patients. It should not be viewed as precluding an earlier specialist appointment if that is available locally.

Since the publication of the last guideline, further evidence has accumulated for the benefit of GC-sparing therapies in both new-onset and relapsing PMR, allowing patients to taper GC dose faster and thus reduce their overall GC exposure. It has also been increasingly recognized that the long-term effects of cumulative GC therapy can persist for a long time; for example, a persistent elevation of fragility fracture risk [26]. In this guideline, we present the evidence for each class of GC-sparing agent separately in order to assist clinicians and patients in making treatment decisions.

For further information on comorbidities that may be exacerbated by GCs, please see current guidance; for example, from NHS Specialist Pharmacy Service: https://sps.nhs.uk/monitorings/corticosteroids-monitoring/

Overarching principle 6	People with PMR receiving glucocorticoid therapy should be reviewed periodically according to need by a healthcare professional. (GRADE 1C, SoA 91%)
Overarching principle 7	All people with PMR must receive clear and accessible education about their condition and management plan, including potential benefits, risks and side effects of treatments. (GRADE 1C, SoA 96%)
Overarching principle 8	People with PMR should have access to educational material and support including information on self-care such as diet, physical activity and pacing. (GRADE 1C, SoA 97%)
Overarching principle 9	People with PMR should be supported to recognize and report anything that might relate to relapses of PMR, treatment toxicities or indicate other conditions including GCA. (GRADE 1C, SoA 96%)

Ongoing monitoring, education and support for self-management is fundamental to safe and effective PMR care. In this context, monitoring refers to monitoring of both the disease and the adverse effects of treatment, in order to balance the 'good' and 'bad' effects of GC therapy and find the lowest dose that adequately controls symptoms [27]. Monitoring for the disease includes assessing whether PMR disease activity (musculoskeletal inflammation) remains adequately controlled, and whether GCA has developed. Various definitions of disease activity, remission and relapse have been proposed for clinical trials, and it remains undefined how to apply these to clinical practice [28]. This is an important research agenda for PMR. Furthermore, symptoms or signs of GCA, especially if there are new visual symptoms, require same-day assessment by a specialist. For more detail, please refer to the national GCA guidelines [18] and local GCA diagnostic pathways.

Monitoring treatment-related adverse effects

Monitoring for adverse effects of DMARDs and other GC-sparing agents is relatively straightforward, and up-to-date guidance exists [29]. Monitoring of adverse effects of GC treatment, on the other hand, is clinically complex because the various side effects of GCs occur very commonly when prescribed over the long-

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term, and may affect disparate organ systems, frequently interacting with patient comorbidities. For example, osteoporosis, sarcopenia, falls and fractures, obesity, diabetes, cardiovascular disease, cataracts, glaucoma and neuropsychiatric adverse effects such as insomnia, anxiety, irritability or aggression, depression, suicidal ideation or psychosis. These toxicities and adverse effects may combine to produce a 'Cushingoid' syndrome, but the pattern of adverse effects can vary from patient to patient.

Any patient who has been taking ≥5 mg prednisolone for four weeks or more should be considered to be at risk of adrenal insufficiency. Adrenal insufficiency can also last for a year or more after steroids have been stopped. Therefore, all patients currently or recently being treated with prednisolone for PMR should be considered at risk of adrenal insufficiency. In practice, this means that the 'steroid sick day rules' should be applied if there is any significant intercurrent illness [30]. Adrenal insufficiency and glucocorticoid-withdrawal symptoms become more likely the longer a patient has taken GCs, the more often the dose has been escalated, and where GCs are also taken via other routes, such as inhaled or intranasal formulations. Even in the absence of adrenal insufficiency, patients tapering GCs can experience withdrawal symptoms, including arthralgia and fatigue, that can be very difficult to distinguish from true inflammatory relapses of PMR. Rheumatology input for these patients is advisable. This is one reason why this guideline recommends referral for all PMR patients after 18 months or more of GC therapy. Another reason for referral of all patients after 18 months is that chronic inflammatory rheumatic diseases, including GCA or RA, can occur concurrently with PMR, but be partially masked by prednisolone therapy: rheumatology input can be useful for identifying these.

Adequate education is vital to fully inform people with PMR about the potential impact of GC therapy on their health and enable them to report adverse effects if they occur. Regular monitoring for GC-related adverse effects allows clinicians to make fully informed recommendations with regard to GC dose tapering and/or the need for initiation of GC-sparing therapies.

The frequency of monitoring needs to be individualized to the patient, but the GWG suggest that an annual review, as for other inflammatory rheumatic diseases, should be the minimum, with additional checks during the year if needed (Table 2). During the first year after diagnosis, most patients will need a review by a member of the clinical team at least every three months; faster steroid tapers, such as six-month tapers, will require more frequent reviews.

Much of the monitoring of people with PMR is directed by the long-term adverse effects of GC therapy. Many of these monitoring activities already occur in primary care as part of annual long-term condition and medication reviews. How these are delivered and which healthcare professionals deliver them may vary depending on local service provision as well as the needs of the individual person. Checklists, templates and 'pathways' can help to ensure that activities are not missed or unnecessarily duplicated, especially if patients are also under secondary care. Practices may choose to combine some of these monitoring activities into PMR-specific annual reviews, or to 'unbundle' them into existing pathways. Table 2 is intended as a starting point for local discussions by GP practices, or groups of GP practices, as to how monitoring activities might be delivered for the local context.

Education and resources for people with PMR

Monitoring must be combined with patient education, which is an ongoing process throughout the disease course. Education should empower people with PMR to understand the nature of their disease, its expected course including recognizing relapse and symptoms of possible GCA, and the purpose and risks of GC therapy. Monitoring of the disease and its treatment is expected to be more effective when the patient is informed and educated about key aspects of the disease and its treatment, and so education may be needed with regard to each of the items in the monitoring table (Table 2) if it is relevant to the individual person. This is potentially a large amount of information, and it may not be possible to deliver it all within standard consultations where there are also clinical decisions to be made.

Education of patients with PMR is a broad-ranging topic that has not received much formal research. Informal education can occur at every contact with healthcare professionals according to the needs of the individual person. However, of concern, studies have repeatedly shown that many people living with PMR experience GC tapering as a difficult journey and perceive a need for greater education and awareness of the disease amongst healthcare professionals [31, 32]. A recent patient and public engagement project [33], conducted collaboratively by the patient charity PMRGCAuk, clinicians and researchers, sought to explore unmet needs in PMR and suggested various areas where education could be beneficial, such as help in navigating the system, and aspects of self-care such as balancing rest and activity, practical advice on exercise, and aspects of managing the GC taper.

Individuals with PMR should have a clear plan as to what action they should take in the event of relapse of PMR symptoms, appearance of new symptoms suggesting other diseases (e.g. GCA or RA) or treatment toxicities. People with PMR should also be aware of positive action they themselves can take in their lives; for example, optimising diet, physical activity, exercise and psychological wellbeing.

Monitoring, education and support could be effectively delivered by health professionals including doctors, nurses, pharmacists and physiotherapists in primary care, complemented by the clinical teams in secondary care and/or peer educators in voluntary organizations where appropriate. The multidisciplinary team can be invaluable for this, providing these individuals are adequately trained and supported; more complex patients may benefit from education by rheumatology advanced practitioners within secondary care. Voluntary organizations such as PMRGCAuk also play a key role in providing peer support via written materials, local in-person groups and online resources (Table 3).

Physical activity and exercise in PMR

The physical and mental health benefits of exercise in older adults are well established, and active and healthy aging is promoted internationally [34]. Exercise-induced antioxidant effects

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Table 2 Suggested outline of education, counselling and monitoring in PMR, including potential adverse effects of glucocorticoid therapy.

	Suggested intervals (to be adapted according to the needs of the individual)
Education and counselling[a]	
PMR disease course and management	At diagnosis and revisited regularly or when relevant
Written or digital resource about PMR[a]	At diagnosis and revisited regularly or when relevant
Glucocorticoid toxicity (*see below*)	At diagnosis and at least every year
Steroid counselling including sick day rules and Steroid Emergency Card	At initiation of glucocorticoid therapy, and subsequently at annual medication reviews
Relapses in PMR with a clear plan in the event of a relapse	At diagnosis and revisited regularly or when relevant
GCA symptom awareness	At diagnosis, at every relapse and revisited regularly or when relevant
Resources for people with PMR (PMRGCAuk charity, Arthritis UK, online support groups etc.) (see Table 3)	At diagnosis and revisited regularly or when relevant
Glucocorticoid toxicity monitoring	
Bone health (review of risk factors for poor bone health, DEXA scan and need for bone protection therapy)	At diagnosis, and repeated at least every 3 years, or after any fragility fracture, or after cessation of glucocorticoids Advice on dental checks should be given if starting bisphosphonates
Gastroprotection (review for reflux symptoms, review need for proton pump inhibitor or similar)	At initiation of glucocorticoid therapy and every 6–12 months or at annual medication reviews
HbA1c and review of antidiabetic therapy if taking	At diagnosis and annually or 3 months following an increase of glucocorticoid dose
Cardiovascular risk (smoking cessation, blood pressure monitoring, lipid profile, QRISK score)	At diagnosis and then every year
Eye checks at opticians	Advise patient to self-refer ∼2 months after starting glucocorticoids, then every year
Weight management (if relevant) and dietary advice	At diagnosis and then at least every year
Screening for mental health disturbance, insomnia and fatigue	At diagnosis and then at least every year
Education about infection risk and vaccinations	At diagnosis and then at least every year Vaccinations as per JCVI eligibility

PMR: polymyalgia rheumatica; GCA: giant cell arteritis; DEXA: dual energy X-ray absorptiometry; HbA1c: haemoglobin A1c; JCVI: Joint Committee on Vaccination and Immunisation.

[a]Education and counselling are ongoing processes that should be initiated at diagnosis and continued over time according to patient need, treatment changes and disease activity.

associated with improved health outcomes have been demonstrated in healthy individuals, regardless of exercise dose or health status [35].

Although there is a lack of evidence for PMR-specific exercises, research from other musculoskeletal conditions is likely to be generalizable to people with PMR. Participation in exercise is strongly endorsed by UK non-pharmacological management guidelines for RA and osteoarthritis [36, 37], with evidence supporting the safety of even higher-intensity resistance training in appropriately selected individuals [38–40].

Exercise has long been promoted as having a role in managing arthritis symptoms and enhancing cardiorespiratory fitness [39, 41]. Reducing pain and stiffness [40, 42, 43], maintaining flexibility and managing fatigue [40, 44] are of potential relevance to people with PMR. Furthermore, in osteoarthritis, RA and fibromyalgia, reduction in anxiety and depression has also been demonstrated after exercise protocols [45], also relevant in PMR given the high prevalence of these symptoms in PMR [46].

Exercise research in RA additionally suggests potential cardiovascular benefits [47, 48], which may help to mitigate some of the cardiovascular risk associated with long-term GC therapy. Broader evidence indicates that aerobic exercise may improve insulin sensitivity and metabolic health, weight management and blood pressure, outcomes that are pertinent in the context of long-term GC use.

Recent debate about exercise has focused attention towards changes to physical function and body composition in early PMR but also promoting holistic management [49, 50]. Exercise that improves muscle strength is likely to improve both physical function and balance, thereby reducing falls risk [51], as has been demonstrated in healthy community-dwelling older adults [52]. Additionally, strengthening larger muscle groups, via resistance and weight-bearing exercises, could help counteract GC-induced myopathy, reduce sarcopenia and contribute to improved bone health, thus mitigating some long-term GC effects. In PMR, the highest rate of fragility fracture is during the first year of treatment, but the excess risk of fragility fracture compared with non-PMR comparators persists for at least 5 years [26]; therefore, muscle strengthening and review of fracture risk factors (including falls) should be initiated soon after diagnosis.

Overall, the evidence for exercise specifically in PMR is lacking, but there are many significant benefits of both exercise and

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Table 3 Resources for people with PMR.

Organization	Resources available	Website links[a]
PMRGCAuk PMR-GCA Scotland	Charity offering support, information, guidance and online and local support groups	https://pmrgca.org.uk/ https://pmrgcascotland.com/
HealthUnlocked	PMR online forum	https://healthunlocked.com/pmrgcauk
Arthritis UK NHS NHS Inform	Information about living with PMR	https://www.arthritis-uk.org/information-and-support/understanding-arthritis/conditions/polymyalgia-rheumatica-pmr/ https://www.nhs.uk/conditions/polymyalgia-rheumatica/ https://www.nhsinform.scot/illnesses-and-conditions/muscle-bone-and-joints/conditions-that-can-affect-multiple-parts-of-the-body/polymyalgia-rheumatica/
Society for Endocrinology	Patient information on sick day rules and steroids	https://www.endocrinology.org/media/4169/ai-and-exogenous-steroids_patient-information-sheet.pdf
PMRGCAuk, University of Keele and University of Leeds	A self-help guide for maintaining movement, activity and exercise for people with PMR	https://pmrgca.org.uk/wp-content/uploads/2024/01/Exercise-in-PMR-booklet.pdf

[a]Links correct at time of publication; search by organization name if links are unavailable.

physical activity across a wide spectrum of rheumatic diseases. As such, we recommend practitioners to consider the potential benefits of exercise in the overall holistic management of people with PMR and further research in this area should be prioritized.

Various forms of exercise can help mitigate the effects of PMR and the effects of long-term GCs. Table 4 outlines recommended exercises and their potential benefits in people with PMR. Physiotherapists can provide people with PMR with individualized advice on exercise and stretches.

Recommendations

Each evidence-based recommendation statement (Table 5) is accompanied by the strength of recommendation (1 or 2), the quality of supporting evidence (A, B or C) and the mean SoA score (as a percentage).

Table 4 Exercises and their potential benefits in people with PMR.

Exercise	Potential benefit
Aerobic	Increase insulin sensitivity and metabolic health, weight management, reduces blood pressure and reduces cardiovascular risk
Resistance and strength	Counteracts GC-induced myopathy, reduces sarcopenia and improves bone health, improves insulin sensitivity and metabolic health, improves joint stability, reduces falls risk
Balance	Reduces the risk of falls, especially important in those at higher risk of fractures
Stretching	Maintains/recovers reduced range of movement and flexibility

Glucocorticoids

Recommendation 1: Treat newly diagnosed PMR with 12.5–15 mg prednisolone daily, increasing to a maximum of 25 mg daily if necessary for symptom control. After symptoms have been controlled for 2–4 weeks, begin to taper the dose. (GRADE 2C, SoA 94%)

The evidence base considered in formulating this recommendation included six RCTs [53–58] enrolling a total of 253 participants; all six trials recruited fewer than 50 participants per treatment arm. However, these RCTs did not directly address GC starting dose; instead, they compared different GC types (e.g. prednisolone, deflazacort and methylprednisolone) or formulations (e.g. immediate-release, modified-release). No meaningful differences were identified between the GC types or formulations compared, at the doses used in the trials, at outcomes measured at 4–12 weeks, though there were no consistent definitions of response or remission.

Meanwhile, observational data suggests that lower starting doses (<10 mg prednisolone) may be associated with a higher likelihood of incomplete response, and higher doses (>25 mg prednisolone) with increased toxicity without any additional benefit [59–61]. In routinely collected UK data, different starting doses of GC therapy were not associated with any significant difference in cumulative GC dose over the whole disease course [2].

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Table 5 Evidence-based recommendation statements.

Recommendation 1	Treat newly diagnosed PMR with 12.5–15 mg prednisolone daily, increasing to a maximum of 25 mg daily if necessary for symptom control. After symptoms have been controlled for 2–4 weeks, begin to taper the dose. (GRADE 2C, SoA 94%)
Recommendation 2	Prescribe bisphosphonates for people with PMR starting glucocorticoid therapy unless contraindicated. (Graded from external evidence—GRADE 1B, SoA 94%)
Recommendation 3	When PMR disease activity is judged to be sufficiently controlled, taper prednisolone dose by 2.5 mg every 2–4 weeks until reaching 10 mg daily and then by 1 mg every 4–8 weeks. (GRADE 2C, SoA 95%)
Recommendation 4	In people with PMR who are at high risk of glucocorticoid toxicity, consider a faster glucocorticoid taper once disease activity is controlled; for example, reducing by 1 mg every 2–3 weeks, aiming for glucocorticoid discontinuation within 6 months of initial disease control. (GRADE 2B, SoA 92%)
Recommendation 5	If PMR relapses, return to the last effective dose for 2–4 weeks, and then consider slowing the glucocorticoid taper accounting for individual patient factors. (GRADE 1C, SoA 96%)
Recommendation 6	Discuss with people recently diagnosed with PMR the option of referral to rheumatology to consider adding methotrexate to glucocorticoids, with the aim of reducing symptoms and stopping steroids sooner. (GRADE 2B, SoA 93%)
Recommendation 7	Consider adding methotrexate alongside glucocorticoids in people with PMR who have relapsed or are at higher risk of glucocorticoid toxicity. (GRADE 2C, SoA 94%)
Recommendation 8	Consider adding an IL-6R antagonist alongside glucocorticoids in people with PMR who have had an inadequate response to glucocorticoids or are unable to tolerate a taper of their glucocorticoids. (GRADE 2B, SoA 93%)

PMR: polymyalgia rheumatica; SoA: strength of agreement; IL-6R: interleukin-6 receptor.

The lack of trials directly comparing different doses of GCs is likely due to most people with PMR responding rapidly to oral prednisolone within the recommended dose range (within hours or days) and therefore an individualized dose-titration approach is preferable rather than treating all people with the same dose. According to the principle of controlling PMR symptoms at the lowest effective GC dose, and since it is difficult to predict the smallest GC dose that will be effective in people with newly diagnosed PMR, it is recommended to start at the lower end of the range (12.5–15 mg oral prednisolone daily), escalating the dose if necessary at 1–2 weeks if the response is incomplete, but not to >25 mg oral prednisolone daily. Although 15 mg prednisolone daily is the starting dose most commonly used in clinical practice, lower starting doses (e.g. 12.5 mg daily) may be used if there are concerns about GC toxicity; for example, in patients with diabetes, osteoporosis, or glaucoma. In a Japanese study from routinely collected healthcare data, the commonest starting prednisolone dose for PMR was 10 mg daily [62].

According to expert consensus, the higher doses of prednisolone within this range are more often needed in individuals with severe pain and stiffness, markedly impairing physical function; with constitutional symptoms or very high inflammatory markers; and those with larger body weight or habitus. However, since most GC-related toxicities are dose-related, it is also important to consider comorbidities that may place patients at greater risk of GC toxicity. The elimination half-life of prednisolone of 3–4 h generally permits once-daily dosing for PMR. Morning dosing minimizes the risk of insomnia and adrenal suppression. However, for those who have prominent nocturnal symptoms, splitting the dose between morning and evening (e.g. 10 mg morning, 5 mg evening) can sometimes be effective without needing to increase the total daily dose.

Recommendation 2: Prescribe bisphosphonates for people with PMR starting glucocorticoid therapy unless contraindicated. (Graded from external evidence—GRADE 1B, SoA 94%)

Observational studies have confirmed that bone fracture is a common occurrence in patients treated for PMR. A retrospective cohort study reported that by 5 years from diagnosis, 9.5% of individuals with PMR had sustained a fracture (of whom 3.9% had a fractured hip/femur, 3.9% had a symptomatic vertebral fracture and 2.4% had a wrist fracture) [63].

Bisphosphonate therapy, including oral alendronate and risedronate or intravenous zoledronate, is approved for both prevention and treatment of GC-induced osteoporosis, and current National Osteoporosis Guideline Group (NOGG) guidelines [64] clearly recommend initiating bone protection therapy (with bisphosphonate therapy being strongly recommended as the most cost-effective first-line drug option) in any person who will be prescribed 7.5 mg prednisolone daily or more, for 3 months or more, without waiting for the result of a bone density assessment, which should follow later. Given that almost all people with PMR will meet this threshold, the GWG recommends bone protection for all people with PMR without the need for prior FRAX or DEXA-based risk stratification. FRAX-based risk assessment retains an important role in the reassessment of ongoing fracture risk during and after GC treatment. Adequate calcium and vitamin D status should also be ensured in people with PMR commencing GC therapy. As per the NOGG guideline recommendations, calcium intake should be achieved through dietary sources where possible, with calcium supplementation reserved for those unable to meet this through dietary, alongside maintenance of adequate vitamin D status, using supplementation where required.

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The rationale stated in the NOGG guideline for their recommendation is that high rates of bone loss, and an excess fracture risk, are known to occur early after initiation of GCs [65]. Previous large-scale studies, conducted in primary and secondary care, highlighted concerningly low rates of bisphosphonate use in PMR, even though almost all people with confirmed PMR do require at least 7.5 mg prednisolone daily for at least 3 months, and usually far in excess of this [15, 26, 66]. More recently, a large UK primary care study using routinely collected electronic health record data [67] found that 67% of people with PMR were coprescribed bisphosphonates, suggesting a significant improvement in previously reported prescribing rates. However, important disparities in prescribing were identified, with male patients and those in more deprived areas being less likely to receive bisphosphonates.

The same study also provided the first target trial emulation evidence of bisphosphonate effectiveness specifically in people with PMR, demonstrating a significant reduction in fragility fracture risk (number needed to treat of 109 to prevent one fragility fracture over 12 months) [67]. Although the absolute risk reduction is modest, this should be interpreted in the context of a condition affecting older adults with a high background fracture risk, with benefit increasing with longer duration of treatment.

Intravenous zoledronic acid may be preferred where there are concerns about poor absorption of oral bisphosphonates. It is also noted that compliance and persistence with oral bisphosphonate is frequently suboptimal, even amongst patients with proven osteoporosis [68]. Therefore, prescribing should be supported by patient education to enhance their understanding that bone protection can be given to prevent GC-induced osteoporosis and fracture, rather than waiting for osteoporosis to be diagnosed.

Because of the disparity between NOGG guidelines and clinical practice in PMR, it was considered important to make a clear and simple recommendation in this guideline. This recommendation is graded 1B, reflecting moderate quality of evidence extrapolated from the NOGG guidelines and downgraded for indirectness due to mixed GC populations rather than PMR-specific populations and for reliance on surrogate outcomes (bone mineral density, rather than fracture) in many trials. It is acknowledged that bisphosphonates may be contraindicated in some individuals and in this situation, specialist referral for alternative bone protection options should be considered.

Recommendation 3: When PMR disease activity is judged to be sufficiently controlled, taper prednisolone dose by 2.5 mg every 2–4 weeks until reaching 10 mg daily and then by 1 mg every 4–8 weeks. (GRADE 2C, SoA 95%)

Recommendation 4: In people with PMR who are at high risk of glucocorticoid toxicity, consider a faster glucocorticoid taper once disease activity is controlled; for example, reducing by 1 mg every 2–3 weeks, aiming for glucocorticoid discontinuation within 6 months of initial disease control. (GRADE 2B, SoA 92%)

Routinely collected UK primary care data indicate that continuous GC therapy was stopped at a median of 1.31 years from diagnosis [interquartile range (IQR) 0.65–2.6 years] [2]. However, after GC cessation, PMR symptoms may recur, such that the median total time on GC therapy was 1.93 years (IQR: 0.95–4.03 years). Analysis of different gaps between prescriptions suggested that if a patient had received no oral GC prescription for 6 months or more, they were unlikely to need to restart prednisolone. The median cumulative GC dose, over the whole treatment course of PMR, was 4 g (IQR: 2 g–8 g) prednisolone-equivalent dose.

As with starting prednisolone dose, no RCT has compared outcomes of different planned prednisolone monotherapy taper regimens. Some observational studies have attempted to compare clinical outcomes of PMR in relation to different prednisolone doses actually prescribed over time, but in these studies, there was no fixed treatment schedule; instead, doses were adapted (individualized) [62]. Another observational study indicated a significant association between cumulative GC dose in PMR and occurrence of osteoporosis, fragility fracture or hypertension [69]. It is important to state therefore that the taper rate suggested here is a typical provisional prednisolone monotherapy taper regimen that may be planned at the time of initial diagnosis of PMR. It is not meant to imply that all patients will be able to follow this taper schedule without PMR relapse or need for dose escalation. It is important to explain to people with PMR that at diagnosis, it is difficult to predict who will relapse or need prolonged GC treatment.

Data from the PMR-MODE trial provides indirect evidence that faster GC tapering may be feasible in selected people with PMR [25]. Both treatment arms followed a 24-week GC taper, which is notably faster than the conventional 9–12-month taper used in clinical practice, with 46% of the placebo arm achieving GC-free remission at week 52. This suggests the possibility that a subgroup of people with PMR might be being exposed to unnecessarily prolonged GC therapy and its associated toxicity. Our example given in the recommendation statement of reducing by 1 mg every 2–3 weeks mirrors the PMR-MODE tapering schedule, with the aim of GC discontinuation within 6 months. This approach must be balanced against the risk of relapse and should be a shared decision made with the person with PMR. A faster taper may be particularly appropriate for patients who are engaged in their care, where provision for more frequent follow-up is in place or where the risk of GC toxicity is high. For further information on comorbidities that may be exacerbated by GCs, please see current guidance; for example, from NHS Specialist Pharmacy Service: https://sps.nhs.uk/monitorings/corticosteroids-monitoring/.

The assessment of ‘sufficient control of PMR disease activity’ is a subjective clinical assessment based on PMR symptoms, physical examination and laboratory markers of inflammation (if necessary). In practice, sufficient control would encompass an acceptable level of symptom control by the person with PMR, with no features of active PMR on assessment by the treating clinician. The prednisolone taper regimen may need to be modified, as a shared decision between prescriber and patient, if relapse or GC toxicity occurs. The monitoring considerations outlined throughout this guideline apply at all stages of tapering; however, clinicians need to be even more vigilant in the context of an accelerated taper. In particular, inflammatory markers should be checked in symptomatic patients; the

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possibility of adrenal insufficiency should be actively considered at prednisolone doses below 5 mg/day, especially where symptoms recur without a rise in inflammatory markers; and if symptoms recur during a faster taper, the dose should be returned to the last effective dose and the taper rate reconsidered.

In cases where GC withdrawal symptoms make tapering difficult, alternating GC doses for a period of two weeks or more may be helpful. In the event of change in the nature of inflammatory symptoms during the GC taper, it is important to be alert to the possible development of late-onset RA or GCA. Prolonged GC therapy may be complicated by development of adrenal insufficiency, in which case, an extremely slow GC taper may be needed once doses below 5 mg prednisolone daily are reached.

A treat-to-target (T2T) approach aims to adjust and optimize treatment through regular monitoring with the aim of attaining specific treatment targets, usually corresponding to remission or low disease activity at the minimal effective dose of drug. This targeted approach is common amongst many other rheumatic diseases such as RA, spondyloarthritis and systemic lupus erythematosus [70–72]; however, at present this numeric target-driven approach has not yet entered everyday clinical practice in PMR.

Nonetheless, future implementation of a T2T approach could provide structure to tapering decisions. An international task force formed a consensus-based T2T approach in PMR and GCA in 2024 [73]. This recommends monitoring of disease activity as frequently as every 1–4 weeks until remission has been achieved and then increasing the time between monitoring intervals (3–6 monthly) if the person with PMR remains in remission. In practice, a protocolized T2T approach is limited by the absence of validated measures of disease activity in PMR suitable for a T2T approach. This area of practice is still in early development [74].

Recommendation 5: If PMR relapses, return to the last effective dose for 2–4 weeks, and then consider slowing the glucocorticoid taper accounting for individual patient factors. (GRADE 1C, SoA 96%)

There are currently no RCTs that specifically evaluate GC management strategies for relapsing PMR, including how best to adjust the subsequent dose or interval of GC tapering. This likely reflects a major research gap but also the reality that tapering in PMR has become increasingly individualized in clinical practice, influenced by comorbidities, patient preference and the wide heterogeneity in disease trajectories. These factors make a trial-based approach to tapering difficult to evaluate within a traditional RCT framework. Although the evidence base for this recommendation is low quality (C), the GWG judged a strong recommendation to be appropriate. Returning to the last effective GC dose in the event of a PMR relapse is a logical, low-risk intervention consistent with the principle of using the lowest effective GC dose; the potential harms of under-treating a relapse (prolonged symptoms, functional impairment) were considered to outweigh the harms. The strong grading therefore reflects expert consensus and current best practice rather than RCT evidence.

Relapses are common, occurring in up to two-thirds of people with PMR, and each relapse typically prolongs the course of GC therapy [13, 17]. This cumulative exposure contributes to a substantial burden of treatment-related harm, with around 85% of people with PMR experiencing GC-related toxicity [75].

When a relapse is suspected, the person should be clinically assessed and inflammatory markers checked. Alternative explanations for symptom recurrence should always be considered. These include infection, GCA, malignancy and other inflammatory or mechanical conditions. In the context of a normal CRP, clinicians should consider mechanical pain (e.g. osteoarthritis, spinal stenosis), vertebral fracture or osteonecrosis secondary to GC exposure, adrenal insufficiency or complications such as GC withdrawal syndrome.

Relapse can sometimes be difficult to confirm objectively, since inflammatory markers such as CRP or ESR can be suppressed by GC therapy; but on the other hand, inflammatory markers can be useful if they are raised, especially as some people with PMR may become accustomed to chronic PMR symptoms or feel unsure whether they are experiencing a true relapse. When there are new or worsening musculoskeletal symptoms during the taper, clinical acumen is important for distinguishing inflammatory from non-inflammatory causes. After two relapses, specialist referral to rheumatology is recommended.

Disease-modifying antirheumatic drugs (DMARDs)

Methotrexate (MTX)

Recommendation 6: Discuss with people recently diagnosed with PMR the option of referral to rheumatology to consider adding methotrexate to glucocorticoids, with the aim of reducing symptoms and stopping steroids sooner. (GRADE 2B, SoA 93%)

Recommendation 7: Consider adding methotrexate alongside glucocorticoids in people with PMR who have relapsed or are at higher risk of glucocorticoid toxicity. (GRADE 2C, SoA 94%)

The use of MTX in PMR is currently unlicensed in the UK, although in practice it is used off-label. The efficacy of MTX as a GC-sparing drug in PMR has been evaluated across five RCTs [25, 76–79].

In the earlier trials, MTX was administered orally at doses of 7.5–10 mg/week alongside a GC taper [78, 79]. The MTX treated arms demonstrated significantly lower cumulative GC exposure (Ferraccioli *et al.* 1.84 g *vs* 3.2 g, $P < 0.0001$; Caporali *et al.* 2.1 g *vs* 2.97 g, $P = 0.03$) and higher rates of GC-free remission at week 76 in one study {87.5% in the MTX group *vs* 53.3% in the placebo group, risk difference of 34% [95% confidence interval (CI): 11% to 53%, $P = 0.003$]} [79]. However, these studies were small, used low-dose MTX and had high risk of bias. A meta-analysis of the four RCTs of MTX as GC-sparing agent in PMR supports higher remission rates [odds ratio (OR) = 5.699, $P < 0.001$] and lower cumulative GC dose (standard mean difference = −1.636,

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$P = 0.009$), but with no significant difference in rates of relapse (OR = 0.377) [80].

The recently published PMR-MODE trial [25] evaluated higher-dose MTX (25 mg/week) against placebo in newly diagnosed PMR with a rapid 24-week GC taper in both groups (MTX $n = 30$, placebo $n = 28$). GC-free remission at week 52 was higher with MTX than placebo (80% *vs* 46%; risk difference 34%; 1-sided $P = 0.0042$). There was no difference in cumulative GC dose at 52 weeks, although this may be explained by the protocolized nature of the GC taper used. The number of people with PMR who relapsed, the Polymyalgia Rheumatica Activity Score (PMR-AS) and the number of adverse events were also similar between groups. However, MTX favoured lower incidence rate of relapse, faster GC-free remission and improvements in patient-reported outcome measures. These results must be interpreted with caution given that this was a single small trial ($n = 58$). Despite this, the trial demonstrated a large, clinically meaningful effect size for GC-free remission, and showed consistency with the earlier MTX trials.

The 2015 EULAR/ACR guidelines conditionally recommended early consideration of MTX (typically 7.5–10 mg/week) in addition to GCs for those at higher risk of relapse or GC toxicity [17]. The rationale was that any benefit of MTX would be maximized by introducing it early and continuing for at least 12 months. The conditional nature of the recommendation was based on the relatively small evidence base for MTX benefit at the time. The PMR-MODE trial strengthens this rationale by demonstrating that very early introduction of MTX (trial inclusion criteria; PMR treated with GCs for <8 weeks), and used at a higher dose, achieves higher rates of GC-free remission. This may be similar to the established concept in other rheumatic diseases that there is a window of opportunity in early disease in which MTX is most effective. These findings increase confidence that MTX confers clinically meaningful benefit when initiated early in newly diagnosed PMR, supporting and extending the original EULAR/ACR 2015 recommendation and forming the basis of this guideline's recommendation for early rheumatology referral to consider MTX.

MTX dosing and titration in PMR should follow standard rheumatological practice. It is recommended to start at a lower dose (typically 10–15 mg/week) and escalate to 25 mg/week as tolerated, consistent with the PMR-MODE protocol [25]. The prescribing clinician should consider factors that may affect tolerability and safety of MTX such as age, renal function, frailty and concomitant medications, as these may require a more cautious titration or a lower target dose. We would recommend for clinicians considering MTX to consult the 2025 BSR guideline for the prescription and monitoring of conventional synthetic DMARDs (csDMARDs) for detailed guidance on pretreatment evaluation, monitoring schedules and management of adverse effects [29].

When making the decision to start MTX, several considerations should be discussed with the person with PMR. Some people may not wish to take MTX, or MTX may be contraindicated in some individuals, including those with significant renal impairment or advanced liver disease. MTX requires regular monitoring for hepatotoxicity and bone marrow suppression, and these monitoring requirements and potential side effects should be discussed as part of shared decision-making process. MTX may be most appropriate for people at higher risk of GC toxicity due to comorbidities, or those who experience significant GC-related side effects, though it could be considered and offered as an option to all people with newly diagnosed PMR in discussion with their rheumatologist. The optimal duration of MTX in PMR is currently unknown, as no trial has specifically addressed when to stop MTX in people who have achieved remission. In the absence of evidence, this should be an individualized shared decision, acknowledging the risk of relapse on discontinuation.

STERLING-PMR (ISRCTN17828080) is an ongoing phase 3 open-label trial studying the GC-sparing effects of MTX (or leflunomide, if MTX is not tolerated or effective) in people with PMR who have experienced at least one relapse [81].

Interleukin-6 receptor (IL-6R) antagonists

Recommendation 8: Consider adding an IL-6R antagonist alongside glucocorticoids in people with PMR who have had an inadequate response to glucocorticoids or are unable to tolerate a taper of their glucocorticoids. (GRADE 2B, SoA 93%)

Three RCTs—PMR-SPARE (tocilizumab), SEMAPHORE (tocilizumab) and SAPHYR (sarilumab)—demonstrated that IL-6R antagonists are effective GC-sparing agents in PMR, both in new-onset and relapsing disease [82–84].

In the PMR-SPARE trial [82], a phase 2 randomized, double-blind study, patients with new-onset PMR ($n = 36$) received subcutaneous tocilizumab or placebo with an 11-week GC taper. Of 32 total patients, 63.2% (12/19) and 11.8% (2/17) achieved GC-free remission at week 16 (OR 12.9, 95% CI 2.2–73.6, $P = 0.002$) favouring the tocilizumab group. There was a significant reduction in time to first relapse (mean 130 ± 13 and 82 ± 11 days, $P = 0.007$) and a reduction in cumulative GC exposure (727 mg *vs* 935 mg, $P = 0.003$) in the tocilizumab group. These estimates should be treated with caution due to the very small sample size.

The SEMAPHORE trial [83] enrolled 101 GC-dependent (requiring $\geq$10 mg/day) PMR patients who received either 4-weekly intravenous tocilizumab or placebo with a pre-defined GC taper. The primary end point was a composite of PMR-AS <10 and $\leq$5 mg/day of prednisolone or $\geq$10 mg reduction from baseline at week 24. A higher proportion of patients who received tocilizumab achieved the primary end point (67.3% *vs* 31.4%, $P < 0.001$), and more patients achieved GC-free discontinuation at week 24 (49% *vs* 19.6%, $P < 0.001$).

Sarilumab was evaluated in the phase 3 SAPHYR trial ($n = 118$) [84] which recruited people with relapsing PMR. Participants received subcutaneous sarilumab every 2 weeks plus a 14-week GC taper, or placebo plus a 52-week GC taper. Sustained remission at week 52 occurred in 28% of sarilumab patients compared with 10% of placebo ($P = 0.02$), with a lower median cumulative GC dose (777 mg *vs* 2044 mg, $P < 0.001$). There was no difference between the Glucocorticoid Toxicity Index for cumulative disease worsening during the 52-week period.

In all three trials, IL-6R antagonists were used in combination with GCs with the goal of enabling a faster taper and achieving GC-free remission. The GWG recommends IL-6R antagonists to

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be prescribed alongside GCs accordingly. There is currently no evidence about combination therapy of MTX with IL-6R antagonists in PMR.

The safety profile of IL-6R antagonists in PMR is consistent with their use in other rheumatic diseases. The most common adverse events were neutropenia, arthralgia and diarrhoea, with a higher rate of treatment discontinuation in the active drug arms. Serious adverse events were infrequent and not significantly different from placebo, but the sample sizes were small, and longer-term safety data are limited.

A key challenge in monitoring PMR disease activity with IL-6R antagonists must be considered due to the suppression of CRP and other acute phase reactants, which are central to composite disease activity scores such as the PMR-AS. This can affect both the clinical assessment and trial endpoints, as highlighted in SEMAPHORE and SAPHYR, necessitating greater reliance on clinical evaluation and patient-reported outcomes. In clinical practice, suppression of CRP by IL-6R antagonists means that normal acute phase reactants cannot be taken as reassuring evidence of disease remission. Clinicians should be aware that disease relapse, intercurrent infection and development of GCA may all occur in the absence of raised inflammatory markers in people receiving IL-6R antagonists. Monitoring whilst on IL-6R should be primarily guided by careful clinical assessment of symptoms. This has particular implications for the recognition of GCA, where suppression of CRP may mask otherwise typical presentation and a high index of suspicion must be maintained.

In the SAPHYR study, a higher proportion of patients receiving sarilumab achieved scores at or above normative values in patient-reported outcome measures [85]. A greater proportion of patients achieved significantly better scores in the Functional Assessment of Chronic Illness Therapy–Fatigue (FACIT-F), Health Assessment Questionnaire–Disability Index (HAQ-DI) and Short Form-36 Health Survey (SF-36) domains, indicating improved health-related quality of life and physical functioning. However, in SEMAPHORE and PMR-SPARE trials, improvements in quality of life were not consistently observed, and the clinical significance of these changes remains uncertain.

These benefits in patient-reported outcomes likely reflect the direct effects of IL-6R blockade and the reduction in GC exposure. The trial highlights the importance of including patient-reported outcomes as key endpoints in PMR studies, given the limitations of clinical and laboratory assessments in the context of IL-6R inhibition.

Collectively, these studies demonstrate consistent improvement in disease control, relapse prevention, and GC-sparing effect with IL-6R antagonists. Extrapolating from these trial populations, IL-6R antagonists should be considered in people with PMR who have had an inadequate response to GCs or who are unable to tolerate a taper of their GCs. At the time of writing, sarilumab (but not tocilizumab) has received regulatory approval for PMR from both the US Food & Drug Administration (FDA) and European Medicines Agency (EMA). However, it has not been evaluated by the National Institute for Health and Care Excellence (NICE) and is not commissioned by the NHS for PMR in the UK. Whilst sarilumab may be prescribed in some settings, it is not currently available for PMR as a routine treatment option within the NHS. Nonetheless, the evidence supports the consideration of IL-6R antagonists in people with relapsing PMR who are unable to taper GCs or who have had an inadequate response to GCs, with the aims of controlling disease activity and minimizing cumulative GC exposure. There is currently insufficient evidence to support the routine use of IL-6R antagonists as a GC-sparing strategy in newly diagnosed PMR.

Other DMARDs

Non-MTX csDMARDs

Evidence for csDMARDs beyond MTX are very limited and no RCTs have been published in people with isolated PMR.

Hydroxychloroquine has not shown any benefit in relapse rates or cumulative GC dose except in a very low-quality retrospective study [86]. Despite this, it is relatively commonly used in clinical practice, especially in the USA, which may be related to its favourable toxicity profile and low monitoring requirements [15].

Leflunomide as a GC-sparing agent in PMR has been documented in two case series [87, 88] and is sometimes used in clinical practice. More recently, a multicentre observational study from Argentina ($n = 186$) reported comparable effectiveness of leflunomide ($n = 43$) to MTX ($n = 143$) and a faster time to GC discontinuation in those prescribed leflunomide than in those prescribed MTX (median 4.7 months in leflunomide group *vs* median 31.8 months in MTX group) [89]. However, the two treatment groups had important baseline differences, making comparison difficult, and the lack of randomization means the quality of evidence remains very low.

The PMRLEFRCT (NCT03576794) trial, a phase 3 RCT evaluating 20 mg leflunomide against placebo with a 26-week GC taper, is ongoing [90]. The ongoing STERLING-PMR trial (ISRCTN17828080) described above includes leflunomide but only as a second-line agent [81].

One double-blinded RCT from 1986 evaluated azathioprine in a mixed cohort of GCA and PMR but the sub-analysis did not separate the PMR group and the study carried a high risk of bias due to a small sample size and a high attrition rate [91].

Overall, the evidence base for the use of non-MTX csDMARDs in PMR is too scarce to make a formal recommendation at present.

Biological and targeted synthetic DMARDs (b/tsDMARDs)

Tumour necrosis factor (TNF) inhibitors

The evidence base for TNF inhibitors in PMR is very scarce. Two small RCTs have studied the role of TNF inhibitors (infliximab and etanercept) in newly diagnosed PMR [92, 93]. Firstly, Kreiner and colleagues randomized 20 GC-naive PMR patients to receive subcutaneous etanercept 25 mg every other week or placebo. After 14 days of treatment they observed a 24% reduction in PMR-AS in those who received etanercept ($P = 0.011$). Secondly, 51 newly diagnosed patients with PMR ($n = 51$) were randomized to receive intravenous infliximab infusions or placebo, with both groups receiving a 16-week GC taper. There were no differences in both primary or secondary outcomes at week 52, including the proportion of patients who were relapse-free, the total number and duration of relapses and the cumulative GC dose.

The quality of evidence of both trials is low, due to small sample size, imprecision and inconsistency. Notably, both trial populations were newly diagnosed PMR and whether this effect would be different in people with PMR who have had an inadequate response to GCs remains unanswered. The negative results from

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these two small trials may be interpreted as evidence against TNF inhibitors in PMR, but adequately powered (phase 3) trials have not been conducted and therefore, no comment can be made as to whether TNF inhibitors are effective in PMR.

B-cell depleting therapies

There is one published proof-of-concept, single-centre RCT evaluating the effect of rituximab on remission in newly diagnosed ($n = 38$) or relapsing PMR ($n = 9$) compared with placebo [94]. Patients received either a single infusion of 1000 mg of rituximab or placebo alongside a 17-week GC taper. Although the primary end point was met, with 48% in the rituximab arm achieving GC-free remission at week 21 ($P = 0.049$), given the sample size and only being a single trial, the quality of supporting evidence is very low. The one-year extension of BRIDGE-PMR [95] did not demonstrate significant durable benefit of rituximab (47% in the rituximab arm *vs* 23% in the placebo arm, $P = 0.12$) in achieving week 52 GC-free remission though cumulative GC exposure was lower in the rituximab group and the numerical differences in GC-free remission appeared greater in newly diagnosed PMR.

There are two further randomized phase 3 trials underway: the REDUCE-PMR-1 (NCT05533125) and REDUCE-PMR-2 (NCT05533164) investigating rituximab in newly diagnosed and relapsing PMR, respectively. At present, the GWG considered that one small phase 2 trial and the known infection risk of B-cell depletion, there is insufficient evidence to recommend rituximab in clinical practice.

Costimulation blockade

The ALORS trial was a proof-of-concept trial and as yet the only RCT to investigate the use of abatacept in PMR [96]. Thirty-four newly diagnosed people with PMR were randomized (abatacept arm $n = 18$, placebo arm $n = 16$) with a 12-week GC taper. Overall, half the patients in the abatacept group achieved a PMR-AS ≤ 10 without GCs at week 12 compared with 22% in the placebo group; this did not reach statistical significance ($P = 0.07$), nor was the study adequately powered to detect this. There was minimal improvement in patient-reported outcomes and no significant difference in cumulative GC exposure. The GWG agreed that there is not enough evidence to recommend the use of costimulation blockade in PMR.

Janus kinase (JAK) inhibitors

The EAST-PMR study was an open-label RCT comparing tofacitinib to GCs in treatment-naive PMR patients ($n = 76$) over 24 weeks [97]. Both groups achieved similar reductions in PMR-AS, with no significant difference in efficacy or serious adverse events between tofacitinib and GCs. The study was limited by its single-centre design and short duration. The BACHELOR study was a double-blind, placebo-controlled, parallel-group RCT evaluating baricitinib in early PMR without concomitant GCs [98]. Baricitinib monotherapy resulted in sustained low disease activity in nearly 80% of participants at 36 weeks compared with 13% in the placebo group, supporting the feasibility of treating PMR with a GC-sparing agent alone. However, the study was small, and further research is needed to confirm long-term safety and efficacy of JAK inhibitors in PMR. These trials provide the first direct RCT evidence, but larger and longer-term studies are required before JAK inhibitors can be considered in the treatment paradigm for PMR.

IL-17 blockers

At the time of submitting the draft guideline for publication, there were no published RCTs investigating the use of an IL-17 antagonist in people with PMR. A phase 2 trial of canakinumab, secukinumab or GCs (NCT01364389) was terminated early due to insufficient clinical response at week 2 for either drug compared with GCs. The recently published REPLENISH study was a multicentre, randomized, placebo-controlled trial which assessed the efficacy and safety of secukinumab (150 mg or 300 mg) compared with placebo, alongside a 24-week GC taper [99]. Sustained remission at week 52 was significantly higher in both doses of secukinumab compared with placebo (300 mg: 41.2% *vs* 20.4%, $P = < 0.001$; 150 mg: 40.6% *vs* 20.4%, $P = < 0.001$) and those who received secukinumab also had significantly lower annual GC doses (1603 mg *vs* 1683 mg *vs* 2093 mg, in secukinumab 300 mg, 150 mg and placebo, respectively). At the time of submitting the draft guideline, the full results of REPLENISH study had not been published and therefore we were not able to include the study in our evidence appraisal.

Timing of DMARDs

Most available studies in PMR focus on the efficacy of DMARDs as described above for GC-sparing and relapse prevention, but do not address optimal tapering strategies or drug withdrawal in remission. Relapse rates after withdrawal of biologics are high; for example, after discontinuation of tocilizumab in patients who achieved remission, the majority relapsed within six months, indicating that short treatment durations may be insufficient for sustained remission [84, 100]. Drug persistence data show that a significant proportion of people with PMR remain on long-term GCs (77% at 1 year, 25% at 5 years) [13].

Drug persistence for DMARDs in PMR is low with most people with PMR remaining on GCs rather than transitioning to or persisting with DMARDs. In a large US registry study [15], only 13.1% of patients were prescribed a GC-sparing agent at baseline, and after 12–24 months, most patients (63.8%) remained on GCs, with only 39% ever receiving a DMARD during follow-up. This highlights limited DMARD persistence and a strong reliance on GCs in routine practice.

Data on MTX, the most commonly used csDMARD in PMR, is sparse and there is no consensus on optimal treatment length or tapering strategies. For bDMARDs, particularly IL-6R antagonists (tocilizumab, sarilumab), recent studies demonstrate efficacy in reducing GC exposure and achieving remission [82–84]. However, withdrawal of biologics is frequently associated with relapse [100]. In a multicentre observational study, tocilizumab dose reduction or infusion spacing was more successful than abrupt discontinuation, but even with these strategies, relapse rates remained substantial [101]. There are no robust data on the optimal duration of biologic therapy or standardized tapering protocols. Overall, there is a lack of high-quality evidence to guide DMARD tapering or withdrawal in PMR, and most people remain on GCs for extended periods. International societies, including the ACR and EULAR emphasize the need for further research to define optimal DMARD strategies in PMR [17, 102].

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Applicability and utility

This guideline is a framework to support clinical decision making but, as with any guideline, needs to be individualized to the person.

There is a current lack of clear, usable guidance on standards of care for people with PMR, compounded by the unavailability of any licensed product for PMR in the UK NHS apart from GCs. Various treatments that are commonly used in rheumatology clinics, such as MTX, are being prescribed off-label; due to the previous lack of compelling phase 3 trial evidence, it has historically been challenging for rheumatologists to assert with confidence that secondary care management will improve outcomes for people with PMR, compared with primary care management alone. With the publication of clinical trials in PMR, attitudes are beginning to shift, but there is concern about the destabilizing effect of rapid changes in referral pathways. Evaluation of alternative care pathways for PMR was beyond the scope of the current guideline, but this is a clear gap for future research and innovation. Nonetheless, we acknowledge that many may consider our suggested minimum guidance inadequate: there have been calls for all people with suspected PMR to be referred to rheumatology to confirm or refute the diagnosis, in the same way as is done for GCA or early inflammatory arthritis. However, PMR is not a medical emergency like GCA, and yet untreated PMR can be very disabling: so deferring treatment while waiting for a standard outpatient appointment may be unacceptable to many clinicians and patients.

Table 6 Future research agenda.[a]

1	**Glucocorticoid treatment strategies:** optimal glucocorticoid tapering strategies to minimize toxicity and management of those who are unable to wean off long-term low-dose glucocorticoids. Development and evaluation of alternative PMR care pathways and models.
2	**DMARD treatment strategies:** head-to-head comparative RCTs of different GC-sparing agents for PMR and strategy trials including treat-to-target approaches and timing of DMARD initiation (early *vs* delayed introduction). Alternative steroid-sparing therapies for the treatment of PMR.
3	**Outcome measures and disease definitions:** development of a unified definition of remission and relapse in PMR, and agreement on the most appropriate outcome measures including patient-reported outcome measures.
4	**Risk stratification and personalized medicine:** predicting who will develop glucocorticoid toxicity or require prolonged glucocorticoid therapy or DMARDs, and the role of imaging in PMR including the optimal modality, timing and management of subclinical large vessel vasculitis.
5	**Non-pharmacological management:** studies of exercise, physical activity and other non-pharmacological interventions (such as diet and nutrition) and their impact on PMR outcomes.

[a]Not in order of priority.
PMR: polymyalgia rheumatica; DMARDs: disease-modifying antirheumatic drug; IL-6R: interleukin-6 receptor; RCT: randomized controlled trial.

We propose that refer-everyone or refer-nobody is a false dichotomy. Alternative, more collaborative models of primary-secondary care collaboration might be developed for appropriate patient subsets, provided they can adequately meet the needs of people with PMR. Health services research studies and collaboration across the primary-secondary care interface will be needed to further evaluate alternative models for clinical care provision for PMR; this is a major area of future research need.

Audit tool

An audit tool has been created from this guideline's recommendations and can be found in Supplementary Data S6.

Research recommendations

There is a distinct lack of high-quality evidence in the management of people with PMR. The GWG has proposed the following research recommendations in Table 6.

Supplementary material

Supplementary material is available at *Rheumatology* online.

Data availability

The data underlying this article are available in the article and in its online supplementary material.

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References

1. Yates M, Graham K, Watts RA, MacGregor AJ. The prevalence of giant cell arteritis and polymyalgia rheumatica in a UK primary care population. BMC Musculoskelet Disord 2016;17:285.
2. Partington RJ, Muller S, Helliwell T, Mallen CD, Abdul Sultan A. Incidence, prevalence and treatment burden of polymyalgia rheumatica in the UK over two decades: a population-based study. Ann Rheum Dis 2018;77:1750–6.
3. Salvarani C, Cantini F, Boiardi L, Hunder GG. Polymyalgia rheumatica. Best Pract Res Clin Rheumatol 2004;18:705–22.
4. Dejaco C, Singh YP, Perel P *et al.*; American College of Rheumatology. 2015 recommendations for the management of polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Arthritis Rheumatol 2015; 67:2569–80.
5. Dasgupta B, Cimmino MA, Kremers HM *et al.* 2012 Provisional classification criteria for polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Arthritis Rheum 2012;64:943–54.
6. Chuang TY, Hunder GG, Ilstrup DM, Kurland LT. Polymyalgia rheumatica: a 10-year epidemiologic and clinical study. Ann Intern Med 1982;97:672–80.
7. Bird HA, Esselinckx W, Dixon AS, Mowat AG, Wood PH. An evaluation of criteria for polymyalgia rheumatica. Ann Rheum Dis 1979;38:434–9.
8. Jones JG, Hazleman BL. Prognosis and management of polymyalgia rheumatica. Ann Rheum Dis 1981;40:1–5.
9. Healey LA. Long-term follow-up of polymyalgia rheumatica: evidence for synovitis. Semin Arthritis Rheum 1984; 13:322–8.
10. Smeeth L, Cook C, Hall AJ. Incidence of diagnosed polymyalgia rheumatica and temporal arteritis in the United Kingdom, 1990-2001. Ann Rheum Dis 2006;65:1093–8.
11. Hemmig AK, Gozzoli D, Werlen L *et al.* Subclinical giant cell arteritis in new onset polymyalgia rheumatica A systematic review and meta-analysis of individual patient data. Semin Arthritis Rheum 2022;55:152017.
12. Espígol-Frigolé G, Dejaco C, Mackie SL *et al.* Polymyalgia rheumatica. Lancet 2023;402:1459–72.
13. Floris A, Piga M, Chessa E *et al.* Long-term glucocorticoid treatment and high relapse rate remain unresolved issues in the real-life management of polymyalgia rheumatica: a systematic literature review and meta-analysis. Clin Rheumatol 2022;41:19–31.
14. Muller S, Hider SL, Singh Sokhal B *et al.* Long-term use of glucocorticoids for polymyalgia rheumatica: follow-up of the PMR Cohort Study. Rheumatol Adv Pract 2022; 6:rkac034.
15. Sattui SE, Xie F, Wan Z *et al.* Treatment of polymyalgia rheumatica by rheumatology providers: analysis from the American College of Rheumatology RISE Registry. Arthritis Care Res (Hoboken) 2024;76:259–64.
16. Dasgupta B, Borg FA, Hassan N *et al.*; BSR and BHPR Standards, Guidelines and Audit Working Group. BSR and BHPR guidelines for the management of polymyalgia rheumatica. Rheumatology (Oxford) 2010;49:186–90.
17. Dejaco C, Singh YP, Perel P *et al.*; American College of Rheumatology. 2015 Recommendations for the management of polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Ann Rheum Dis 2015;74:1799–807.
18. Mackie SL, Dejaco C, Appenzeller S *et al.* British Society for Rheumatology guideline on diagnosis and treatment of giant cell arteritis: executive summary. Rheumatology (Oxford) 2020;59:487–94.
19. Kostine M, Finckh A, Bingham CO *et al.* EULAR points to consider for the diagnosis and management of rheumatic immune-related adverse events due to cancer immunotherapy with checkpoint inhibitors. Ann Rheum Dis 2021; 80:36–48.
20. British Society for Rheumatology. Creating Clinical Guidelines: British Society for Rheumatology Protocol v6.0. 2025. Available from: https://www.rheumatology.org.uk/Portals/0/Documents/Guidelines/BSRGuidelinesProtocol_Guidance_v6.0_FINAL_270126.pdf? ver=h7nsbAhrsLajgtDBJZvRfA%3d%3d
21. Toyoda T, Armitstead Z, Bhide S *et al.* Treatment of polymyalgia rheumatica: British Society for Rheumatology guideline scope. Rheumatol Adv Pract 2024;8:rkae002.
22. Higgins JPT, Altman DG, Gøtzsche PC *et al.*; Cochrane Statistical Methods Group. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ 2011;343:d5928.
23. Hayden JA, Côté P, Bombardier C. Evaluation of the quality of prognosis studies in systematic reviews. Ann Intern Med 2006;144:427–37.
24. Guyatt GH, Oxman AD, Vist GE *et al.*; GRADE Working Group. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ 2008;336:924–6.
25. Bolhuis TE, Kooijman N, Marsman DE, Snijders GF, den Broeder AA, den Broeder N *et al.* Results of a 1-year randomised double-blind placebo-controlled trial with

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methotrexate 25 mg/week in recently diagnosed polymyalgia rheumatica. Ann Rheum Dis 2026;85:761–70.
26. Paskins Z, Whittle R, Sultan AA *et al.* Risk of fracture among patients with polymyalgia rheumatica and giant cell arteritis: a population-based study. BMC Med 2018;16:4.
27. Pofi R, Caratti G, Ray DW, Tomlinson JW. Treating the side effects of exogenous glucocorticoids; can we separate the good from the bad? Endocr Rev 2023;44:975–1011.
28. Bolhuis TE, Bosch P, Falzon L *et al.* Definitions of and instruments for disease activity, remission and relapse in polymyalgia rheumatica: a systematic literature review. Rheumatology (Oxford) 2025;64:455–69.
29. Bechman K, Song K, Abhishek A *et al.* The 2025 British Society for Rheumatology guideline for the prescription and monitoring of conventional synthetic disease-modifying anti-rheumatic drugs. Rheumatology 2026;65: keaf522. https://doi.org/10.1093/rheumatology/keaf522
30. NICE. Adrenal insufficiency: identification and management. 2024. Available from: https://www.nice.org.uk/guidance/ng243/chapter/recommendations
31. Twohig H, Mitchell C, Mallen C, Adebajo A, Mathers N. "I suddenly felt I'd aged": a qualitative study of patient experiences of polymyalgia rheumatica (PMR). Patient Educ Couns 2015;98:645–50.
32. Mackie SL, Arun P, Padmanabhan V, Arjona A, Kullman JA. Patient perspectives on life impact and unmet needs in giant cell arteritis and polymyalgia rheumatica: insights from social media. Rheumatol Adv Pract 2026;10:rkaf140.
33. Mackie SL, Boyce S, Coutin L *et al.* The polymyalgia paradox: addressing north-south inequities in polymyalgia rheumatica using grassroots community connectors in a deprived urban area [version 1; not peer reviewed]. NIHR Open Res 2025. 2025(5:122).
34. World Health Organization. Physical activity. 2024. Available from: https://www.who.int/news-room/fact-sheets/detail/physical-activity
35. de Sousa CV, Sales MM, Rosa TS *et al.* The antioxidant effect of exercise: a systematic review and meta-analysis. Sports Med 2017;47:277–93.
36. NICE. Rheumatoid arthritis in adults: management. 2020. Available from: https://www.nice.org.uk/guidance/ng100/chapter/Recommendations#non-pharmacological-management.
37. NICE. Osteoarthritis in over 16s: diagnosis and management. 2022.
38. Hernández-Hernández MV, Díaz-González F. Role of physical activity in the management and assessment of rheumatoid arthritis patients. Reumatol Clin 2017; 13:214–20.
39. Vliet Vlieland TP, van den Ende CH. Nonpharmacological treatment of rheumatoid arthritis. Curr Opin Rheumatol 2011;23:259–64.
40. Bilberg A, Bjersing J, Börjesson M, Sivertsson J, Mannerkorpi K. High-intensity exercise improves multidimensional fatigue and health-related quality of life in rheumatoid arthritis: a randomized controlled study. Arthritis Res Ther 2025;27:176.
41. Hurkmans E, van der Giesen FJ, Vliet Vlieland TP, Schoones J, Van den Ende EC. Dynamic exercise programs (aerobic capacity and/or muscle strength training) in patients with rheumatoid arthritis. Cochrane Database Syst Rev 2009; 2009:Cd006853.
42. Benatti FB, Pedersen BK. Exercise as an anti-inflammatory therapy for rheumatic diseases-myokine regulation. Nat Rev Rheumatol 2015;11:86–97.
43. Perandini LA, de Sá-Pinto AL, Roschel H *et al.* Exercise as a therapeutic tool to counteract inflammation and clinical symptoms in autoimmune rheumatic diseases. Autoimmun Rev 2012;12:218–24.
44. Rongen-van Dartel SAA, Repping-Wuts H, Flendrie M *et al.* Effect of aerobic exercise training on fatigue in rheumatoid arthritis: a meta-analysis. Arthritis Care Res (Hoboken) 2015;67:1054–62.
45. Kelley GA, Kelley KS, Callahan LF. Brief Report: exercise and anxiety in adults with arthritis and other rheumatic diseases: support for evidential value. Biomed Res Int 2018; 2018:2984671.
46. Vivekanantham A, Blagojevic-Bucknall M, Clarkson K *et al.* How common is depression in patients with polymyalgia rheumatica? Clin Rheumatol 2018;37:1633–8.
47. Hancock AT, Mallen CD, Muller S *et al.* Risk of vascular events in patients with polymyalgia rheumatica. CMAJ 2014;186:E495–501.
48. Warrington KJ, Jarpa EP, Crowson CS *et al.* Increased risk of peripheral arterial disease in polymyalgia rheumatica: a population-based cohort study. Arthritis Res Ther 2009;11:R50.
49. Leung JL, De Ross B, Gianoudis J *et al.* Changes to physical function and body composition during the first 2 years of polymyalgia rheumatica. Rheumatology (Oxford) 2025; 64:5834–43.
50. O'Brien AV, Sattui SE. From symptom amelioration to function modification: is it time to expand the treatment focus in polymyalgia rheumatica? Rheumatology (Oxford). 2025.
51. Dyer SM, Suen J, Kwok WS *et al.* Exercise for falls prevention in aged care: systematic review and trial endpoint meta-analyses. Age Ageing 2023;52:afad217. https://doi.org/10.1093/ageing/afad217
52. Sherrington C, Michaleff ZA, Fairhall N *et al.* Exercise to prevent falls in older adults: an updated systematic review and meta-analysis. Br J Sports Med 2017;51:1750–8.
53. Di Munno O, Imbimbo B, Mazzantini M *et al.* Deflazacort versus methylprednisolone in polymyalgia rheumatica: clinical equivalence and relative antiinflammatory potency of different treatment regimens. J Rheumatol 1995;22:1492–8.
54. Krogsgaard MR, Lund B, Johnsson B. A longterm prospective study of the equipotency between deflazacort and prednisolone in the treatment of patients with polymyalgia rheumatica. J Rheumatol 1995;22:1660–2.
55. Dasgupta B, Dolan AL, Panayi GS, Fernandes L. An initially double-blind controlled 96 week trial of depot methylprednisolone against oral prednisolone in the treatment of polymyalgia rheumatica. Br J Rheumatol 1998;37:189–95.
56. Salvarani C, Cantini F, Olivieri I *et al.* Corticosteroid injections in polymyalgia rheumatica: a double-blind, prospective, randomized, placebo controlled study. J Rheumatol 2000;27:1470–6.
57. Viapiana O, Gatti D, Troplini S *et al.* Prednisone compared to methylprednisolone in the polymyalgia rheumatica treatment. Rheumatol Int 2015;35:735–9.

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58. Cutolo M, Hopp M, Liebscher S, Dasgupta B, Buttgereit F. Modified-release prednisone for polymyalgia rheumatica: a multicentre, randomised, active-controlled, double-blind, parallel-group study. RMD Open 2017;3:e000426.
59. Kyle V, Hazleman BL. Treatment of polymyalgia rheumatica and giant cell arteritis. I. Steroid regimens in the first two months. Ann Rheum Dis 1989;48:658–61.
60. Hernández-Rodríguez J, Cid MC, López-Soto A, Espigol-Frigolé G, Bosch X. Treatment of polymyalgia rheumatica: a systematic review. Arch Intern Med 2009;169:1839–50.
61. Duru N, van der Goes MC, Jacobs JWG *et al.* EULAR evidence-based and consensus-based recommendations on the management of medium to high-dose glucocorticoid therapy in rheumatic diseases. Ann Rheum Dis 2013;72:1905–13.
62. Tanaka Y, Takahashi T, Inokuchi S *et al.* Characteristics of patients with polymyalgia rheumatica based on glucocorticoid dose in Japan: a cohort study using routinely collected health data. Mod Rheumatol 2025;35:722–30.
63. Shbeeb I, Challah D, Raheel S, Crowson CS, Matteson EL. Comparable rates of glucocorticoid-associated adverse events in patients with polymyalgia rheumatica and comorbidities in the general population. Arthritis Care Res (Hoboken) 2018;70:643–7.
64. Gregson CL, Armstrong DJ, Bowden J *et al.* UK clinical guideline for the prevention and treatment of osteoporosis. Arch Osteoporos 2022;17:58.
65. van Staa TP, Leufkens HG, Abenhaim L, Zhang B, Cooper C. Oral corticosteroids and fracture risk: relationship to daily and cumulative doses. Rheumatology (Oxford) 2000; 39:1383–9.
66. Sokhal BS, Hider SL, Paskins Z, Mallen CD, Muller S. Fragility fractures and prescriptions of medications for osteoporosis in patients with polymyalgia rheumatica: results from the PMR Cohort Study. Rheumatol Adv Pract 2021; 5:rkab094.
67. Twohig H, Jenkinson D, Bailey J *et al.* Prescribing of medication to prevent glucocorticoid harms in patients with polymyalgia rheumatica: a cross-sectional study and two emulated target trials in the clinical practice research datalink Aurum. Arthritis Rheumatol 2026;78:1731–40.
68. Fatoye F, Smith P, Gebrye T, Yeowell G. Real-world persistence and adherence with oral bisphosphonates for osteoporosis: a systematic review. BMJ Open 2019;9:e027049.
69. Mazzantini M, Torre C, Miccoli M *et al.* Adverse events during longterm low-dose glucocorticoid treatment of polymyalgia rheumatica: a retrospective study. J Rheumatol 2012;39:552–7.
70. van Vollenhoven RF, Mosca M, Bertsias G *et al.* Treat-to-target in systemic lupus erythematosus: recommendations from an international task force. Ann Rheum Dis 2014; 73:958–67.
71. Smolen JS, Breedveld FC, Burmester GR *et al.* Treating rheumatoid arthritis to target: 2014 update of the recommendations of an international task force. Ann Rheum Dis 2016;75:3–15.
72. Smolen JS, Braun J, Dougados M *et al.* Treating spondyloarthritis, including ankylosing spondylitis and psoriatic arthritis, to target: recommendations of an international task force. Ann Rheum Dis 2014;73:6–16.
73. Dejaco C, Kerschbaumer A, Aletaha D *et al.* Treat-to-target recommendations in giant cell arteritis and polymyalgia rheumatica. Ann Rheum Dis 2024;83:48–57.
74. Liew DFL, Owen CE. Treat-to-target in polymyalgia rheumatica and giant cell arteritis: an impending, multifaceted challenge. Rheumatology (Oxford) 2024;63:2044–6.
75. Dejaco C, Brouwer E, Mason JC *et al.* Giant cell arteritis and polymyalgia rheumatica: current challenges and opportunities. Nat Rev Rheumatol 2017;13:578–92.
76. Nazarinia AMU, Moghimi JU, Toussi JU. Efficacy of methotrexate in patients with polymyalgia rheumatica. Koomesh 2013;14:265–70.
77. van der Veen MJ, Dinant HJ, van Booma-Frankfort C, van Albada-Kuipers GA, Bijlsma JW. Can methotrexate be used as a steroid sparing agent in the treatment of polymyalgia rheumatica and giant cell arteritis? Ann Rheum Dis 1996; 55:218–23.
78. Ferraccioli G, Salaffi F, De Vita S, Casatta L, Bartoli E. Methotrexate in polymyalgia rheumatica: preliminary results of an open, randomized study. J Rheumatol 1996; 23:624–8.
79. Caporali R, Cimmino MA, Ferraccioli G *et al.*; Systemic Vasculitis Study Group of the Italian Society for Rheumatology. Prednisone plus methotrexate for polymyalgia rheumatica: a randomized, double-blind, placebo-controlled trial. Ann Intern Med 2004;141:493–500.
80. Song GG, Lee YH. Methotrexate for treating polymyalgia rheumatica: A meta-analysis of randomized controlled trials. Int J Clin Pharmacol Ther 2021;59:366–71.
81. Mackie SL, Hill CL. Steroid-Reducing Options for ReLapsING PMR (STERLING-PMR). 2023.
82. Bonelli M, Radner H, Kerschbaumer A *et al.* Tocilizumab in patients with new onset polymyalgia rheumatica (PMR-SPARE): a phase 2/3 randomised controlled trial. Ann Rheum Dis 2022;81:838–44.
83. Devauchelle-Pensec V, Carvajal-Alegria G, Dernis E *et al.* Effect of Tocilizumab on disease activity in patients with active polymyalgia rheumatica receiving glucocorticoid therapy: a randomized clinical trial. JAMA 2022; 328:1053–62.
84. Spiera RF, Unizony S, Warrington KJ *et al.*; SAPHYR Investigators. Sarilumab for relapse of polymyalgia rheumatica during glucocorticoid taper. N Engl J Med 2023; 389:1263–72.
85. Strand V, Msihid J, Sloane J *et al.* Sarilumab in relapsing polymyalgia rheumatica: patient-reported outcomes from a phase 3, double-blind, randomised controlled trial. Lancet Rheumatol 2025;7:e544–e53.
86. Lee JH, Choi ST, Kim JS *et al.* Clinical characteristics and prognostic factors for relapse in patients with polymyalgia rheumatica (PMR). Rheumatol Int 2013;33:1475–80.
87. Diamantopoulos AP, Hetland H, Myklebust G. Leflunomide as a corticosteroid-sparing agent in giant cell arteritis and polymyalgia rheumatica: a case series. Biomed Res Int 2013;2013:120638.
88. Adizie T, Christidis D, Dharmapaliah C, Borg F, Dasgupta B. Efficacy and tolerability of leflunomide in difficult-to-treat polymyalgia rheumatica and giant cell arteritis: a case series. Int J Clin Pract 2012;66:906–9.

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89. Vinicki JP, Cusa A, Domingo D *et al.* Effectiveness of methotrexate and leflunomide as corticoid-sparing drugs in patients with polymyalgia rheumatica. Rheumatol Adv Pract 2024;8:rkae033.
90. Brouwer E. Treatment With Leflunomide in Patients With Polymyalgia Rheumatica (PMRLEFRCT). 2019. Available from: https://clinicaltrials.gov/study/NCT03576794
91. De Silva M, Hazleman BL. Azathioprine in giant cell arteritis/polymyalgia rheumatica: a double-blind study. Ann Rheum Dis 1986;45:136–8.
92. Kreiner F, Galbo H. Effect of etanercept in polymyalgia rheumatica: a randomized controlled trial. Arthritis Res Ther 2010;12:R176.
93. Salvarani C, Macchioni P, Manzini C *et al.* Infliximab plus prednisone or placebo plus prednisone for the initial treatment of polymyalgia rheumatica: a randomized trial. Ann Intern Med 2007;146:631–9.
94. Marsman DE, den Broeder N, van den Hoogen FHJ, den Broeder AA, van der Maas A. Efficacy of rituximab in patients with polymyalgia rheumatica: a double-blind, randomised, placebo-controlled, proof-of-concept trial. Lancet Rheumatol 2021;3:e758–e66.
95. Bolhuis TE, Marsman DE, den Broeder AA, den Broeder N, van der Maas A. 1-year results of treatment with rituximab in polymyalgia rheumatica: an extension study of a randomised double-blind placebo-controlled trial. Lancet Rheumatol 2023;5:e208–e14.
96. Saraux A, Le Henaff C, Dernis E *et al.* Abatacept in early polymyalgia rheumatica (ALORS): a proof-of-concept, randomised, placebo-controlled, parallel-group trial. Lancet Rheumatol 2023;5:e728–e35.
97. Ma X, Yang F, Wu J *et al.* Efficacy and safety of Tofacitinib in patients with polymyalgia rheumatica (EAST PMR): an open-label randomized controlled trial. PLoS Med 2023; 20:e1004249.
98. Saraux A, Carvajal Alegria G, Dernis E *et al.* Baricitinib in early polymyalgia rheumatica (BACHELOR): a randomised, double-blind, placebo-controlled, parallel-group trial. Lancet Rheumatol 2025;7:e233–e42.
99. Stone JH, Buttgereit F, Saraux A, Schmidt WA, Dejaco C, Spiera R *et al.* Secukinumab in patients with polymyalgia rheumatica: week 52 results of a phase 3, multicentre, randomized, double blind, placebo-controlled trial (REPLENISH). 22nd International Vasculitis Workshop, held in Melbourne, Australia. 2026.
100. Chevet B, Souki A, Nowak E *et al.* Maintenance of remission after Tocilizumab withdrawal in patients with glucocorticoid-dependent polymyalgia rheumatica. Arthritis Rheumatol 2025;77:1416–20.
101. Assaraf M, Chevet B, Wendling D *et al.* Efficacy and management of tocilizumab in polymyalgia rheumatica: results of a multicentre retrospective observational study. Rheumatology (Oxford) 2024;63:2065–73.
102. Boyadzhieva Z, Reisch M, Schneider M, Buttgereit F, Dejaco C. Treatment and prognostic factors in PMR: a systematic literature review informing German, Austrian and Swiss guidelines. Rheumatology (Oxford) 2025; 64:6036–47.

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