BMJ Best Practice

Polymyalgia rheumatica

Straight to the point of care

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Summary

Polymyalgia rheumatica (PMR) is an inflammatory rheumatologic syndrome. Affected patients describe difficulty rising from seated or prone positions, significant shoulder and hip girdle stiffness, varying degrees of muscle tenderness, shoulder/hip bursitis, and/or oligoarthritis.

More common in women.

About 15% to 20% of patients with PMR have giant cell arteritis (GCA); 40% to 60% of GCA patients have PMR.

Diagnosis is made via history and with supportive laboratory tests indicating an elevated ESR or CRP.

Rapid improvement often occurs within 24 to 72 hours with low-dose prednisone.

Definition

Polymyalgia rheumatica (PMR) is an inflammatory rheumatologic syndrome that manifests as pain and morning stiffness involving the neck, shoulder girdle, and/or pelvic girdle in individuals older than age 50 years. Peripheral musculoskeletal involvement may also be present.[1] PMR occurs either as an isolated condition or associated with giant cell arteritis.

Epidemiology

The incidence of polymyalgia rheumatica (PMR) increases with age, typically occurring in those older than 50 years and rarely in younger individuals. In a population-based study conducted in Minnesota between 2000 and 2014, the age- and sex-adjusted annual incidence of PMR was 63.9/100,000 population aged ≥50 years.[2] The mean age at diagnosis was 74 years, and 64% of patients were women.[2]

Incidence appears highest in those of northern European descent.[3] PMR occurs in nonwhite populations, but the epidemiology has not been well studied in these populations. The incidence of PMR appears to be increasing both in the US and in Europe. A geographical gradient has been observed in Europe, decreasing from north to south: the highest incidence rates have been reported in Norway and the lowest rates in Italy, Spain, and Israel.[4] [5] Cyclic fluctuations in the incidence of PMR have been observed, and there does not appear to be an increase in mortality associated with PMR.[2] [6][7]

Etiology

The exact cause of polymyalgia rheumatica (PMR) is unknown. There have been conflicting observations regarding seasonal variation on the incidence of PMR. These observations have suggested a role of infectious agents such as adenovirus, respiratory syncytial virus, parainfluenza virus type I, parvovirus B19, *Mycoplasma pneumoniae* , and *Chlamydia pneumoniae* .[4] [8] Although association studies may suggest an infectious agent as a possible etiology, no study has yet established this fact. A role for genetic factors has been suggested based on observed ethnic differences and familial cases. There is an association between PMR and the HLA-DRB1*04 and -DRB1*01 alleles, and with the genetic polymorphisms for ICAM-1, tumor necrosis factor-alpha, and interleukin (IL)-1 receptor antagonist.[4] [9] These factors may influence the susceptibility to PMR. A potential hormonal role has been suggested by the observation of adrenal gland hypofunction in untreated patients. Inappropriately normal cortisol levels and low levels of dehydroepiandrosterone have been observed.[10]

Pathophysiology

The pathogenesis of PMR remains unclear. Given the association of PMR with giant cell arteritis (GCA), it is thought that mechanisms similar to those contributing to GCA may be involved.[11] Although vasculitis is present in GCA, vasculitis involving vessels to the neck, shoulder girdle, and hip girdle has not been found. Increased interleukin (IL)-6 in the serum and in temporal artery biopsy specimens has been observed in both PMR and GCA patients, suggesting an inflammatory role for IL-6. Moreover, IL-6 promoter polymorphisms have been suggested to affect the clinical expression of PMR and GCA.[4] Study results suggest that both PMR and GCA patients share similar in situ macrophage-derived cytokine mRNA profiles. GCA-recruited T cells in vasculitic lesions were found to secrete IL-2 and interferon (IFN)-gamma; however, in PMR there was no IFN-gamma expression, suggesting a role for IFN-gamma in the progression to vasculitis.[12] [13] A decline in immunosuppressive T-regulatory lymphocytes and an increase in proinflammatory T-helper 17 cells has been observed in a study of PMR patients compared with age-matched controls, suggesting an imbalance in immunologic regulation.[14] Decreased numbers of B cells and a redistribution of B-cell subsets have been shown in newly diagnosed PMR patients compared with controls, suggesting a regulatory role for B cells in the pathogenesis of PMR.[15] Significant declines in muscle glutamate and prostaglandin E2 but not in muscle lactate or blood flow with successful corticosteroid treatment in a study of PMR patients suggested that tissue ischemia may not be the underlying cause of PMR muscle symptoms.[16]

Case history

Case history #1

A 58-year-old woman presents with a 2-week history of fatigue, anorexia, fevers, and bilateral pain and stiffness in the shoulder and hip girdles. These symptoms are worse at night. Upon awakening in the morning, she feels as if she has a bad flu. She reports difficulty getting out of bed in the morning due to stiffness. Her wrists and finger joints are also painful and swollen.

Other presentations

Less common peripheral musculoskeletal manifestations include a monoarthritis or polyarthritis, pitting edema of the hand, carpal tunnel syndrome and tenosynovitis.[1] A second presentation is the presence of the characteristic neck, shoulder girdle, and hip girdle symptoms associated with normal serum markers of systemic inflammation (i.e., ESR or CRP).

Approach

Diagnosis is primarily based on history.

History

Polymyalgia rheumatica (PMR) typically occurs in people aged ≥50 years and is more common in women.[2] [9] Patients complain of acute onset of pain and stiffness in the neck, shoulder, and/or hip girdle region that is worse in the morning. The stiffness may be so severe that getting out of bed is difficult. Stiffness improves to a varying extent throughout the day. Constitutional symptoms such as low-grade fever, weight loss, asthenia, malaise, anorexia, night sweats, and depression may be present. The patient may report asymmetrical joint pain, carpal tunnel syndrome symptoms, and swelling of the hands and feet. It is also important to review the history for symptoms suggestive of giant cell arteritis (GCA), such as unilateral temporal or occipital headaches, scalp tenderness, jaw claudication, or visual symptoms.[9]

Response to corticosteroids

A rapid response to corticosteroids, within 24 to 72 hours of treatment (some criteria provide for 4-7 days), can be very helpful in supporting a diagnosis of PMR and in distinguishing it from other inflammatory disorders.[3] [13] [17] An incomplete response to low-dose corticosteroids does not rule out the diagnosis.[18]

Physical examination

Patients may exhibit a limited range of active movement of the shoulders and hips due to pain and stiffness. The patient may also have difficulty rising from a chair or the examination table. These symptoms may result from muscle tenderness of the shoulder and hip regions, subacromial bursitis of the shoulders, trochanteric bursitis of the hips, and occasionally an oligoarthritis of the peripheral joints. Sometimes swelling of the fingers and dorsal surfaces of the hands and feet occur as a result of tenosynovitis.[4] Pertinent negative findings of other organ systems should be noted, including the absence of oral ulcers, rashes, cardiopulmonary abnormalities, abdominal abnormalities, and focal neurologic findings. Patients should be assessed for signs of GCA: temporal and occipital artery thickening, nodules, and tenderness, as well as scalp tenderness.

Lab tests

Elevated serum erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP) levels are supportive in the setting of characteristic history and examination findings.[4] [11] Normal values in the setting of a characteristic history and examination findings do not rule out the diagnosis, as rarely PMR can present with normal markers of systemic inflammation. Normal ESR and/or CRP in patients with PMR may indicate a milder subset of disease or identification of patients earlier in their disease course.[19]

As hypothyroidism can occasionally present with arthralgias and stiffness, thyroid function tests should be included in the workup of suspected PMR. As some myeloproliferative diseases may also present similarly to PMR with symptoms of fatigue, bony pain, and an elevated ESR, checking CBC and serum protein electrophoresis is also recommended as part of PMR evaluation. Rheumatoid factor and anti-cyclic citrullinated peptide antibodies are ordered if late-onset rheumatoid arthritis is suspected.

In patients in whom shoulder and hip pain predominate, myositis should be considered in the differential diagnosis and creatine phosphokinase (CPK) level should be checked. As rheumatoid arthritis (RA)

can more commonly present with shoulder joint stiffness in older individuals, the presence of an inflammatory arthritis involving the finger joints (particularly the PIP and MCP joints) is an indication to check rheumatoid factor and anti-cyclic citrullinated peptide antibody titers; significantly elevated titers of these antibodies increases the likelihood of early rheumatoid arthritis.[20]

Imaging

Ultrasound and magnetic resonance imaging (MRI) can be useful in distinguishing a bursitis, synovitis, or tenosynovitis in the shoulders and hips.[4] [11] These tests may be helpful in diagnosing PMR with less characteristic clinical signs and symptoms. Subdeltoid bursitis, bicipital tenosynovitis, glenohumeral synovitis, and/or hip synovitis are characteristic of PMR.[11] Trochanteric bursitis is present in many patients with PMR.[22] Interspinous bursitis may be detected on MRI.[23] The presence of symmetric shoulder and/or hip arthritis in the absence of a bursitis or tenosynovitis would be more suggestive of a systemic arthritis such as rheumatoid arthritis.

Increased uptake of fluorodeoxyglucose F-18 on PET scans can be helpful in identifying inflammation in the joints, bursae, and tenosynovial tissues in the shoulders and large vessels that may be helpful in making a diagnosis of PMR.[24] [25] Although this information may be supportive in diagnosing PMR, it remains unclear if such scanning can predict relapses.[17]

History and exam

Key diagnostic factors

shoulder/hip girdle stiffness (common)

- The typical presentation is morning stiffness for >1 hour in the shoulder girdle in 70% to 95% of patients and/or pelvic girdle pain in 50% to 70% of patients.[5]

shoulder/hip girdle pain (common)

- The typical presentation is inflammatory pain for >1 hour in the morning in the shoulder girdle in 70% to 95% of patients and/or pelvic girdle pain in 50% to 70% of patients.[5]

rapid response to corticosteroids (common)

- Response to a corticosteroid therapeutic trial is typically dramatic and rapid, usually occurring within 24 to 72 hours. This can be a helpful diagnostic finding (and is a criterion in one guideline[27]) that can distinguish PMR from other inflammatory conditions.[3] [13] [17]

Other diagnostic factors

acute onset (common)

- The onset is generally acute, but patients often delay several weeks in seeking treatment.[26]

low-grade fever (common)

- Part of constitutional symptoms present in one third of patients.[4] [26]

anorexia (common)

- Constitutional symptoms such as loss of appetite are common and may occur in approximately one third of patients.[4] [26]

weight loss (common)

- Up to one third of patients report weight loss as part of their presentation.[4] [26]

malaise (common)

- Many patients report a feeling of general malaise.[4] [26]

depression (common)

- Some patients report low mood or depression.[4] [26]

asthenia (common)

- Patients may report unaccustomed physical weakness or lack of energy.[4] [26]

oligoarticular arthritis (common)

- Most patients present with both oligoarticular and transitory manifestations at disease onset. The joints most frequently involved are the wrist, metacarpophalangeal, and knee joints.[18]

Risk factors

Strong

age ≥50 years

- PMR is uncommon in young patients.[9]

giant cell arteritis (GCA)

- About 15% to 20% of patients with PMR have GCA, whereas 40% to 60% of those with GCA have PMR.[4]

Weak

female sex

- The incidence is higher in women. In one study, the incidence in women was 72.7/100,000 population aged ≥50 years, compared with 55.6/100,000 population aged ≥50 years in men.[2]

Tests

1st test to order

Test	Result
erythrocyte sedimentation rate (ESR) • Normal values of ESR vary with age. ESR is elevated in 91.5% of patients at diagnosis but is not as sensitive as a CRP, which is elevated in 98.9%. An elevated ESR at diagnosis is associated with a higher risk of more than 1 relapse.[28] [29] Abnormal levels are more common in males.[30] • Normal ESR in patients with PMR may indicate a milder subset of disease or identification of patients earlier in their disease course.[19]	**elevated**
C-reactive protein (CRP) • A sensitive indicator of disease activity, which is elevated in 98.9% of patients diagnosed with PMR.[28] [31] [32] Unlike the ESR, the pretreatment levels of the CRP are not prognostic. However, persistently elevated levels of CRP despite treatment are associated with higher rates of relapse.[29] [33] • Normal CRP in patients with PMR may indicate a milder subset of disease or identification of patients earlier in their disease course.[19]	**elevated**
CBC • As some myeloproliferative diseases may also present similarly to PMR with symptoms of fatigue and bony pain and an elevated ESR, checking a CBC and serum protein electrophoresis is also recommended as part of PMR evaluation.	**variable**

Other tests to consider

Test	Result
TSH • TSH may be elevated if hypothyroidism is the cause of symptoms	**normal or elevated in hypothyroidism**
ultrasound • Subdeltoid bursitis, bicipital tenosynovitis, glenohumeral synovitis and/or hip synovitis are characteristic of PMR.[11] Trochanteric bursitis is present in many patients with PMR.[22]	**bursitis, tenosynovitis, synovitis, joint effusions**
MRI • More sensitive than ultrasound, but not routinely done.[4] [22] • Subdeltoid bursitis, bicipital tenosynovitis, glenohumeral synovitis and/or hip synovitis are characteristic of PMR.[11] Trochanteric bursitis is present in many patients with PMR.[22] Interspinous bursitis may be detected on MRI.[23]	**bursitis, joint effusions**
serum protein electrophoresis • As some myeloproliferative diseases may also present similarly to PMR with symptoms of fatigue and bony pain and an elevated ESR, serum protein electrophoresis is also recommended as part of PMR evaluation.	**normal in PMR**
serum creatine phosphokinase • In cases where shoulder and hip pain predominate, myositis should be considered and CPK level should be checked.	**may be elevated in myositis**
rheumatoid factor • Performed if early rheumatoid arthritis is suspected. The presence of an inflammatory arthritis involving the finger joints (particularly the PIP and MCP joints) is an indication to check rheumatoid and anti-cyclic citrullinated peptide antibody titers.[20]	**usually normal**
anti-cyclic citrullinated peptide antibodies • Performed if early rheumatoid arthritis is suspected. The presence of an inflammatory arthritis involving the finger joints (particularly the PIP and MCP joints) is an indication to check rheumatoid and anti-cyclic citrullinated peptide antibody titers.[20]	**normal**

Emerging tests

Test	Result
interleukin (IL)-6 • IL-6 stimulates many acute phase proteins. Elevated IL-6 is characteristic in PMR. Although an elevated level can be a sensitive marker for PMR disease activity and supportive of the diagnosis, it is not yet specific enough to be used as a diagnostic test.Persistently elevated levels of IL-6 despite treatment are associated with higher rates of relapse. [29]	**elevated**
fluorodeoxyglucose F-18 PET scan • Helpful in identifying synovitis, bursitis, and tenosynovitis in the shoulder region or large vessel inflammation to help support the diagnosis.[24] [25] [34] Not part of the standard diagnostic workup at present.	**may show joint inflammation and/or arteritis**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Giant cell arteritis (GCA)	• New-onset unilateral headache, jaw claudication associated with chewing tough foods, diffuse mandibular discomfort, dental discomfort, sinus pain and pressure, and/or tongue pain are associated with GCA. Blindness, diplopia, or blurry vision and an abnormally thickened, tender, erythematous, or nodular temporal artery are also found.[9]	• A positive temporal artery biopsy showing a granulomatous vasculitis confirms the diagnosis of GCA; however, results may be positive in PMR patients without GCA symptoms.
Early rheumatoid arthritis (RA)	• Presentation may be very similar to PMR; however, the absence of a prompt response to low-dose corticosteroids distinguishes the two. The peripheral musculoskeletal symptoms of early RA do not respond rapidly.[4] Some patients with RA will respond to 10 to 20 mg of prednisone.	• Elevated rheumatoid factor and persistently elevated plasma viscosity.[35] Positive anti-CCP antibody assays.[1] Anti-CCP assays were 61.4% sensitive and 100% specific for the diagnosis of late-onset RA.[36] • Repeat x-ray of hands, delayed for months after the initial test, is performed to assess other affected joint damage.[4]
Hypothyroidism	• May show similar signs of muscle and joint pain, weakness in the extremities, and fatigue; however, delayed relaxation of deep tendon reflexes (a rare finding) is strongly suggestive of hypothyroidism.[4]	• An elevated TSH helps to differentiate hypothyroidism.[4] Creatine phosphokinase may also be elevated.
Fibromyalgia	• Compared with PMR, fibromyalgia pain tends to be more widespread and is not associated with shoulder/hip girdle stiffness. The pain does not typically improve with low-dose daily prednisone as is characteristic of PMR.[4] [37]	• The ESR and CRP are normal[37]
Paraneoplastic syndrome	• May present with constitutional symptoms and proximal muscle pain easily	• Tumor screening includes routine baseline tests including a CXR, a CBC,

Condition	Differentiating signs / symptoms	Differentiating tests
	confused with the shoulder and hip girdle stiffness found in PMR. Paraneoplastic syndrome symptoms usually do not respond to low-dose corticosteroid treatment. A thorough tumor workup should be reserved for those unresponsive to low-dose glucocorticoid therapy. Removal of the tumor leads to a resolution of symptoms. [5] • The Lambert-Eaton syndrome, a paraneoplastic condition associated with small-cell lung cancer, may cause proximal weakness that improves later in the day, a feature similar to the symptoms of PMR.	a chemistry panel, and urinalysis. Age-appropriate cancer screening tests should also be performed (i.e., fecal occult blood testing, colonoscopy, mammogram). Additional specialized testing should be directed by the history, the exam findings, and any abnormalities found on the routine CXR and laboratory tests.
Polymyositis	• Symmetrical weakness of shoulder and pelvic girdles.[4]	• Elevated muscle enzyme levels (i.e., creatine phosphokinase) often with a positive antinuclear antibody titer. Characteristic changes in electromyography associated with polymyositis include increased needle insertional activity, spontaneous fibrillations, low-amplitude, short-duration polyphasic motor potentials, and complex repetitive discharges. Diagnosis is confirmed with muscle biopsy, indicating immune cell infiltration and destruction of muscle fibers.[4]
Overuse bursitis/ tendonitis	• No systemic symptoms as in PMR. Typically unilateral shoulder involvement without bilateral involvement.[4]	• A normal ESR helps exclude PMR; however, about 20% of PMR patients have a normal result.[4]
Remitting seronegative symmetric synovitis with pitting edema	• Polyarthritis and bilateral edema of both hands found typically in individuals older than 50 years.[38] [39]	• MRI shows predominantly a tenosynovitis.[4] [39] Serum vascular endothelial growth factor may be useful in diagnosis and monitoring of disease activity, but this is not currently standard practice.[40]

Criteria

There have been a number of similar sets of diagnostic criteria proposed for PMR, none of which has been accepted as a universal standard. These are as follows.

An evaluation of criteria for polymyalgia rheumatica[41]

Criteria: must have any 3 factors, or just 1 and a temporal artery biopsy positive for giant cell arteritis

- Age over 65 years
- Bilateral shoulder girdle pain
- More than 1 hour morning stiffness
- Symptom onset <2 weeks
- ESR >40 mm/hour
- Depression/weight loss
- Upper arm tenderness, bilateral.

Prognosis and management of polymyalgia rheumatica[27]

Criteria: all required

- Bilateral shoulder girdle and pelvic girdle pain
- Morning stiffness of any duration
- Greater than 2 months
- ESR >30 mm/hour or CRP protein >6 mg/L
- Other diagnoses excluded
- Rapid improvement with corticosteroids.

Polymyalgia rheumatica: a 10-year epidemiologic and clinical study[42]

Criteria: all required

- Age over 50 years
- Bilateral neck, shoulder girdle, or pelvic girdle pain (need 2 of 3 factors)
- Greater than 30 minutes of morning stiffness
- More than 1 month duration of symptoms
- ESR >40 mm/hour
- Other diagnoses excluded.

Long-term follow-up of polymyalgia rheumatica: evidence for synovitis[43]

Criteria: age and duration required with any 3 of the remaining factors

- Age over 50 years
- Bilateral neck, shoulder girdle, or pelvic girdle pain
- More than 1 hour morning stiffness
- More than 1 month duration of symptoms

- ESR above upper limit of normal
- Other diagnoses excluded
- Rapid improvement with <20 mg of prednisone.

Provisional classification criteria for polymyalgia rheumatica[44]

The lack of definitive classification criteria for PMR presents a problem for patient evaluation in clinical studies and the development of reliable diagnostic criteria. The European League Against Rheumatism and the American College of Rheumatology established a collaborative working group to develop classification criteria for PMR. A set of consensus-based criteria were developed over several years via a multistep process. These criteria were used prospectively comparing new-onset PMR patients with a control group with conditions mimicking patients with PMR. The results were then used to generate a scoring algorithm for the classification of PMR. The criteria (all required) and proposed algorithm are as follows:

- Age 50 years or over
- Bilateral shoulder aching
- Abnormal CRP and/or ESR.

Symptom/finding	Points without ultrasound (0-6)	Points with ultrasound (0-8)
Morning stiffness >45 minutes	2	2
Hip pain/limited range of motion	1	1
Negative rheumatoid factor or anticyclic citrullinated peptide antibody	2	2
No other joint involvement	1	1
Ultrasound 1: at least one shoulder with subdeltoid bursitis, biceps tenosynovitis, and/or glenohumeral synovitis, and at least one hip with synovitis or trochanteric bursitis	NA	1
Ultrasound 2: bilateral subdeltoid bursitis, biceps tenosynovitis, or glenohumeral synovitis	NA	1

A score of ≥4 without ultrasound or a score of ≥5 with ultrasound is classified as PMR.

Scoring algorithm for the classification of PMR

Adapted from Dasgupta B, et al. 2012 Provisional classification criteria for polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Arthritis Rheum. 2012:64:943-954

Approach

The goal of treatment is to establish an inflammation-free and symptom-free state. The patient should ideally return to the prior baseline level of function with a normal ESR and/or CRP.

Corticosteroids

The mainstay of therapy is low-dose corticosteroids; response usually occurs within 24 to 72 hours. The corticosteroid dose is increased until complete symptom resolution and normalization of the ESR and CRP. Failure of response should lead to reevaluation of the diagnosis or to consideration of treatment resistance.

The lowest effective corticosteroid dose is recommended. In general, tapering of the dose should be individualized to the patient. However, it is recommended that once symptoms have resolved and inflammatory markers have normalized, the dose is tapered. Relapses should be treated by increasing the dose to the prerelapse dose, and then slowly tapering the dose over 4 to 8 weeks to the dose at which the relapse occurred.[45]

Although not standard therapy, intramuscular methylprednisolone can be considered for patients who are poorly adherent to treatment.[45] [46]

In all adults over 40 years of age taking prednisone ≥2.5 mg/day for over 3 months prophylaxis of corticosteroid induced osteoporosis (GIOP) is recommended with calcium and vitamin D supplementation.[47] Patients receiving long term corticosteroids should be assessed for fracture risk as soon as possible after starting corticosteroid treatment, and every 1 to 2 years while corticosteroid treatment continues, any additional prophylactic treatment for GIOP should be stratified by the risk of fracture.[47] See Osteoporosis: Management approach.

Concomitant use of nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids should be avoided to minimize the risk of gastrointestinal bleeding.

Withdrawal symptoms including arthralgias, malaise, and fatigue might be confused with recurrent PMR symptoms. Slow tapering of the corticosteroids over a 2- to 3-year period is often required to prevent relapses and symptom exacerbations.

Nonsteroidal anti-inflammatory drugs

NSAIDs are not recommended for the treatment of PMR. They can be considered for short-term use for pain associated with other conditions (e.g., corticosteroid withdrawal symptoms).

Chronic use should be avoided due to serious adverse effects. The risk of gastrointestinal bleeding may be increased due to concurrent corticosteroid use. A proton-pump inhibitor (e.g., omeprazole) should be considered to prevent ulcer development.

Methotrexate

The use of methotrexate should be considered early during the course of treatment on an individual basis. In particular, it should be considered in:[11] [45]

- Individuals at high risk for relapse or prolonged therapy (female sex, high ESR [>40 mm/hour], and peripheral arthritis at diagnosis[48])

- Patients who have relapsed
- Patients who have not had an adequate response to corticosteroids
- Patients for whom prolonged corticosteroid use is associated with significant adverse effects (i.e., osteoporosis, glaucoma, cataracts, diabetes)
- Patients with comorbidities which could be exacerbated by corticosteroid therapy.

Folic acid is used concomitantly to decrease the risk of methotrexate-associated adverse effects.

Use of methotrexate is recommended for practitioners who are either experienced with its use or in consultation with such experienced practitioners. A baseline CXR is recommended to evaluate for any underlying interstitial lung disease prior to starting treatment. If interstitial lung disease is present, methotrexate is usually not started. Any other significant pulmonary disease may be a relative contraindication to starting methotrexate.

When methotrexate is used as a corticosteroid-sparing agent, it should be continued until the corticosteroids can be tapered without the recurrence of PMR symptoms. There are no definitive guidelines regarding the tapering of methotrexate. However, once the corticosteroids have been successfully tapered, it would be reasonable to discontinue the methotrexate by tapering the dose over approximately 3 months.

Baseline CBC, liver function tests (LFTs), and hepatitis B and C serologies are also recommended prior to initiating methotrexate. Any significant liver test abnormality, hematologic abnormality, history of hepatitis B or C infection, history of ongoing alcohol use, or history of a malignancy is a relative contraindication to methotrexate use.[49] The CBC, creatinine, and LFTs should be checked once per month with each dose increase. Once a stable dose has been established for 6 months without any adverse effects, these levels can be checked every 3 months.

Relapse or disease exacerbation

About 30% to 50% of patients experience relapses unrelated to the corticosteroid dose or taper rate, commonly within 2 years of diagnosis. In these cases, the corticosteroids are increased to a dose that controls symptoms and normalizes the ESR and the CRP. Once the symptoms are controlled, tapering can resume.

Although there is no precise definition for relapse of PMR, the Delphi consensus approach among a panel of rheumatologists identified the following useful parameters for defining PMR relapse and remission:[50]

- morning stiffness
- pain in the neck, shoulders, upper arms, and pelvic girdle
- shoulder pain on active and passive range of motion
- limited shoulder elevation
- hip synovitis
- ESR and CRP
- dose of corticosteroids used for treatment.

Treatment-resistant cases

The daily corticosteroid dose can be increased to twice daily dosing in order to control symptoms and normalize ESR and CRP. Once this occurs, the dose can be tapered.

Tocilizumab has been shown in case reports to be effective in treating PMR. An open-label phase-2a trial studied 10 patients with newly diagnosed PMR (corticosteroids for <1 month) treated with monthly

tocilizumab for 1 year.[51] Corticosteroid-free relapse-free remission was achieved in 9 patients at 6 months, with 1 patient withdrawing; remission was sustained through the 15 months of the study. In a second study, 20 patients not treated with corticosteroids were treated with tocilizumab infusions every 3 months followed by oral prednisone.[52] After 6 months, the PMR activity scores had significantly decreased in all patients, prednisone doses were minimized, and PET-CT imaging showed improvement in inflammatory musculoskeletal changes.

Tocilizumab can be considered for patients who are not responsive to glucocorticoids or when the glucocorticoid dose cannot be tapered because of recurrence. It is also sometimes used for patients at initial presentation if they are unable to tolerate other treatments.[53] [54] Randomized studies examining the use of tocilizumab in isolated PMR are lacking. One meta-analysis suggests tocilizumab may be more effective in combination with glucocorticoids.[55]

Tocilizumab may increase the risk of serious liver injury (e.g., acute liver failure, hepatitis). Measure aminotransferase (ALT) and aspartate aminotransferase (AST) levels before initiation and every 4-8 weeks during the first 6 months of treatment. After the first 6 months, levels can be monitored every 12 weeks. Initiation of treatment is not recommended in patients with ALT or AST higher than 5-times the upper limit of normal. Patients should be advised to seek help immediately if they experience signs and symptoms of liver injury.[56] Although tocilizumab has been approved in the US for use in giant cell arteritis, it is currently not approved for the treatment of isolated PMR.

Leflunomide was also effective for relapsing/refractory PMR in one case series.[57]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
initial presentation		
	1st	**corticosteroid**
	adjunct	**nonsteroidal anti-inflammatory drug (NSAID)**
	2nd	**methotrexate**
	plus	**folic acid**
	3rd	**tocilizumab**

Ongoing		(summary)
treatment-resistant or relapse or disease exacerbation		
	1st	**corticosteroid (increased dose)**
	adjunct	**methotrexate plus folic acid**
	2nd	**tocilizumab or leflunomide**

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

initial presentation

1st **corticosteroid**

Primary options

» prednisone: 12.5 to 25 mg orally once daily initially, then taper to 10 mg once daily over 4-8 weeks, then taper by 1 mg/month thereafter

Secondary options

» methylprednisolone: 120 mg intramuscularly every 3 weeks for 3 months then taper dose gradually, consult specialist for further guidance on dose

» The mainstay of therapy is a low-dose corticosteroid as a single daily dose. Response in this dose range usually occurs within 24 to 72 hours. Failure of response should lead to re-evaluation of the diagnosis or to consideration of treatment resistance.

» Corticosteroids should be continued until symptoms resolve and ESR/CRP normalizes (about 2 to 4 weeks). The minimum effective dose (i.e., 12.5 to 25 mg/day of prednisone or its equivalent) should be given. The dose should then be tapered to 10 mg/day of prednisone (or its equivalent) over 4 to 8 weeks. Tapering can then occur at a rate equivalent to 1 mg/month of prednisone.[45]

» The duration of treatment is typically at least 1 year, and it is often longer, depending upon the patient's response.

» Although not standard therapy, intramuscular methylprednisolone can be considered for patients who are poorly adherent to treatment.[45] [46]

» In all adults ages over 40 years taking prednisone ≥2.5 mg/day for over 3 months prophylaxis of corticosteroid induced osteoporosis (GIOP) is recommended with calcium and vitamin D supplementation.[47] Patients receiving long term corticosteroids should be assessed for fracture risk as soon as possible after starting treatments and every 1-2 years while corticosteroid treatment continues,

Acute

any additional prophylactic treatment for GIOP should be stratified by the risk of fracture.[47] See Osteoporosis: Management approach .

adjunct **nonsteroidal anti-inflammatory drug (NSAID)**

Treatment recommended for SOME patients in selected patient group

Primary options

» naproxen: 250-500 mg orally twice daily when required, maximum 1250 mg/day

OR

» ibuprofen: 400-800 mg orally every 4-6 hours when required, maximum 3200 mg/day

» Concomitant use of NSAIDs and corticosteroids should be avoided whenever possible, to minimize the risk of gastrointestinal bleeding. For patients experiencing peripheral musculoskeletal symptoms during corticosteroid withdrawal NSAIDs may be used for a limited duration. Primary NSAID use for PMR related symptoms is not recommended.

» Chronic use should be avoided due to serious adverse effects. The risk of gastrointestinal bleeding may be increased due to concurrent corticosteroid use. A proton-pump inhibitor (e.g., omeprazole) should be considered to prevent ulcer development.

2nd **methotrexate**

Primary options

» methotrexate: 7.5 mg orally once weekly on the same day of each week, increase by 2.5 mg/week increments every 2-3 months if required, maximum 20 mg/week

» Use of methotrexate is recommended for practitioners who are either experienced with its use or in consultation with such experienced practitioners. In particular, it should be considered in: individuals at high risk for relapse or prolonged therapy (female sex, high ESR [>40 mm/hour], and peripheral arthritis at diagnosis); patients who have relapsed; patients who have not had an adequate response to corticosteroids; patients for whom prolonged corticosteroid use is associated with significant adverse effects (i.e., osteoporosis, glaucoma, cataracts, diabetes); patients with comorbidities that could be exacerbated by corticosteroid therapy.[11] [45] [48]

Acute

» When methotrexate is used as a corticosteroid-sparing agent, it should be continued until the corticosteroids can be tapered without the recurrence of PMR symptoms. There are no definitive guidelines regarding the tapering of methotrexate. However, once the corticosteroids have been successfully tapered, it would be reasonable to discontinue the methotrexate by tapering the dose over approximately 3 months.

» A baseline CXR is recommended to evaluate for any underlying interstitial lung disease prior to starting treatment. If interstitial lung disease is present, methotrexate is usually not started. Any other significant pulmonary disease may be a relative contraindication to starting methotrexate.

» Baseline CBC, liver function tests (LFTs), and hepatitis B and C serologies are recommended prior to initiating methotrexate. Any significant liver test abnormality, hematologic abnormality, history of hepatitis B or C infection, history of ongoing alcohol use, or history of a malignancy is a relative contraindication to methotrexate use.[49] The CBC, creatinine, and LFTs should be checked once per month with each dose increase. Once a stable dose has been established for 6 months without any adverse effects, these levels can be checked every 3 months.

» Doses of 7.5 to 10 mg/week have been studied, although higher doses may be needed.[45]

plus **folic acid**

Treatment recommended for ALL patients in selected patient group

Primary options

» folic acid (vitamin B9): 1 mg orally once daily

» Indicated to decrease the risk of methotrexate adverse effects, particularly the risk of oral ulcers and bone marrow suppression.

3rd **tocilizumab**

Primary options

» tocilizumab: consult specialist for guidance on dose

» There have been a few case reports of the use of tocilizumab in patients with contraindications to, or adverse events from corticosteroid treatment.[53] [54] Randomized studies

MANAGEMENT

Acute

examining the use of tocilizumab in isolated PMR are lacking.

» Tocilizumab may increase the risk of serious liver injury (e.g., acute liver failure, hepatitis). Measure aminotransferase (ALT) and aspartate aminotransferase (AST) levels before initiation and every 4-8 weeks during the first 6 months of treatment. After the first 6 months, levels can be monitored every 12 weeks. Initiation of treatment is not recommended in patients with ALT or AST higher than 5-times the upper limit of normal. Patients should be advised to seek help immediately if they experience signs and symptoms of liver injury.[56] Although tocilizumab has been approved in the US for use in giant cell arteritis, it is currently not approved for the treatment of isolated PMR.

Ongoing

treatment-resistant or relapse or disease exacerbation

1st **corticosteroid (increased dose)**

Primary options

» prednisone: consult specialist for guidance on dose

Secondary options

» methylprednisolone: consult specialist for guidance on dose

» Some patients are resistant to corticosteroid therapy, or may have significant PMR symptoms at night. In these patients the corticosteroids can be increased to twice-daily dosing in order to control symptoms and normalize ESR and CRP. Once this occurs, the dose can be tapered.

» About 30% to 50% of patients experience relapses unrelated to the corticosteroid dose or taper rate, commonly within 2 years of diagnosis. In these cases, the corticosteroids are increased to the dose before the relapse occurred, with control of symptoms and normalization of the ESR and the CRP. Once the symptoms are controlled, the dose should be tapered over 4 to 8 weeks to the dose at which the relapse occurred. Tapering the dose at the rate of 1 mg/month prednisone equivalent can then resume if symptoms remain in remission.

» Although there is no precise definition for relapse of PMR, the Delphi consensus approach among a panel of rheumatologists identified the following useful parameters for defining PMR relapse and remission: morning stiffness; pain in the neck, shoulders, upper arms, and pelvic girdle; shoulder pain on active and passive range of motion; limited shoulder elevation; hip synovitis; ESR and CRP; and dose of corticosteroids used for treatment.[50]

» In all adults ages over 40 years taking prednisone ≥2.5 mg/day for over 3 months prophylaxis of corticosteroid induced osteoporosis (GIOP) is recommended with calcium and vitamin D supplementation.[47] Patients receiving long term corticosteroids should be assessed for fracture risk as soon as possible after starting treatment and every 1-2 years while corticosteroid treatment continues, any additional prophylactic treatment for GIOP should be stratified by the risk of fracture.[47] See Osteoporosis: Management approach .

Ongoing

adjunct **methotrexate plus folic acid**

Treatment recommended for SOME patients in selected patient group

Primary options

» methotrexate: 7.5 mg orally once weekly on the same day of each week, increase by 2.5 mg/week increments every 2-3 months if required, maximum 20 mg/week
-and-
» folic acid (vitamin B9): 1 mg orally once daily

» Use of methotrexate is recommended for practitioners who are either experienced with its use or in consultation with such experienced practitioners.

» When methotrexate is used as a corticosteroid-sparing agent, it should be continued until the corticosteroids can be tapered without the recurrence of PMR symptoms. There are no definitive guidelines regarding the tapering of methotrexate. However, once the corticosteroids have been successfully tapered, it would be reasonable to discontinue the methotrexate by tapering the dose over approximately 3 months.

» A baseline CXR is recommended to evaluate for any underlying interstitial lung disease prior to starting treatment. If interstitial lung disease is present, methotrexate is usually not started. Any other significant pulmonary disease may be a relative contraindication to starting methotrexate.

» Baseline CBC, liver function tests, and hepatitis B and C serologies are recommended prior to initiating methotrexate. Any significant liver test abnormality, hematologic abnormality, history of hepatitis B or C infection, history of ongoing alcohol use, or history of a malignancy is a relative contraindication to methotrexate use.[49]

» Doses of 7.5 to 10 mg/week have been studied, although higher doses may be needed.[45]

» Folic acid is indicated to decrease the risk of methotrexate adverse effects, particularly the risk of oral ulcers and bone marrow suppression.

2nd **tocilizumab or leflunomide**

Primary options

Ongoing

» tocilizumab: consult specialist for guidance on dose

OR

» leflunomide: consult specialist for guidance on dose

» Tocilizumab has been shown in case reports to be effective in treating PMR.[51] [52] Tocilizumab can be considered for patients who are not responsive to glucocorticoids or when the glucocorticoid dose cannot be tapered because of recurrence. Randomized studies examining the use of tocilizumab in isolated PMR are lacking. One meta-analysis suggests tocilizumab may be more effective in combination with glucocorticoids.[55]

» Tocilizumab may increase the risk of serious liver injury (e.g., acute liver failure, hepatitis). Measure aminotransferase (ALT) and aspartate aminotransferase (AST) levels before initiation and every 4-8 weeks during the first 6 months of treatment. After the first 6 months, levels can be monitored every 12 weeks. Initiation of treatment is not recommended in patients with ALT or AST higher than 5-times the upper limit of normal. Patients should be advised to seek help immediately if they experience signs and symptoms of liver injury.[56] Although it has been approved in the US for use in giant cell arteritis, it is currently not approved for the treatment of isolated PMR.

» Leflunomide was also effective for relapsing/refractory PMR in one case series.[57]

Emerging

Tumor necrosis factor inhibitors

European League Against Rheumatism/American College of Rheumatology guidelines have strongly recommended against the use of tumor necrosis factor inhibitors, citing absence of evidence for benefit, increased potential adverse effects, and significant cost.[45]

Hydroxychloroquine

Very limited data have suggested benefit with hydroxychloroquine in patients who experience adverse effects to corticosteroids, but this is not standard therapy.[58]

Patient discussions

Patients are to follow up immediately with their rheumatologist or other provider upon developing any recurrent or new visual symptoms, other symptoms suggestive of GCA, worsening of their PMR symptoms, or signs of infections (i.e., fevers, chills, sweats, new cough, and shortness of breath). Patients receiving tocilizumab should seek help immediately if they develop signs and symptoms of liver injury.[56]

Instruct patients to take their corticosteroid medication daily as prescribed. Patients should be advised that signs of corticosteroid withdrawal (i.e., arthralgias, malaise, and fatigue) may occur with tapering, but these symptoms should resolve after a few days.

FOLLOW UP

Monitoring

Monitoring

Patients may be followed on a monthly basis with routine blood ESR and/or CRP levels; however, evidence supporting this is limited. Once symptoms have resolved and the patient is on a stable tapering regimen, follow-up visits can be extended to every 3 months.

Treatment response is typically monitored by history, examination findings, and laboratory levels. This routinely includes evaluation of:

- scalp tenderness
- temporal artery tenderness
- peripheral joint manifestations
- ESR and/or CRP levels.

Signs and symptoms of giant cell arteritis (GCA) should be monitored as well, because GCA may manifest itself when the corticosteroids are tapered.[9]

ESR monitoring

There is conflicting support for ESR monitoring. Some clinicians monitor levels at baseline and every several months to assess treatment response and recurrence. A decline indicates a response.

Patients are seen once a month for the first 3 to 6 months. If the patient is symptom-free with a corticosteroid taper, levels are checked every 2 to 3 months. If there is a history suggesting possible symptom recurrence, the ESR is measured. Patients with an elevated ESR at diagnosis have a higher risk of more than 1 relapse.[28] [29] [48]

Treatment-related complications

Regular follow-up should also include monitoring for treatment-related adverse effects. In patients receiving corticosteroids, monitor for signs of infection, hypertension, muscle weakness, cataract development, glaucoma, and skin changes.

Patients receiving methotrexate need to be assessed for stomatitis, any respiratory changes (new cough, shortness of breath, dyspnea on exertion, new crackles on auscultation) that suggest the development of interstitial lung disease, LFT abnormalities (>2x normal), and myelosuppression. Evaluation for these methotrexate-related adverse effects should occur 1 month after any methotrexate dose increase and every 3 to 4 months in patients on a stable dose. The presence of any of these adverse effects requires further evaluation and either dosage reduction or discontinuation of the methotrexate.

In patients receiving tocilizumab, measure aminotransferase (ALT) and aspartate aminotransferase (AST) levels every 4-8 weeks during the first 6 months of treatment. After the first 6 months, levels can be monitored every 12 weeks.[56]

Ultrasound

The use of ultrasound to monitor the decrease in shoulder joint, shoulder periarticular structure, and hip joint inflammatory changes may be helpful in monitoring the response of PMR to corticosteroids.[64]

Complications

Complications	Timeframe	Likelihood
chronic relapsing PMR	**long term**	**medium**
Characterized by frequent exacerbations of symptoms. May require treatment with higher doses of corticosteroids and the addition of a corticosteroid-sparing agent. The course of therapy may be prolonged.		
increased risk of infection secondary to corticosteroids	**long term**	**medium**
Monitoring for infection should be a part of the regular follow-up. The risk can increase if higher doses are needed for prolonged periods of time.		
osteoporosis secondary to corticosteroids	**long term**	**medium**
A recognized complication of long-term corticosteroid use. Prophylaxis with calcium, vitamin D, and a bisphosphonate is indicated for patients taking prednisone >5 to 7.5 mg/day or its equivalent for more than 1 month.		
diabetes mellitus type 2 secondary to corticosteroids	**long term**	**medium**
An increased risk of the development of diabetes mellitus is associated with corticosteroid use.[62] Patients should have a baseline blood glucose and hemoglobin A1C checked. In patients with diabetes, the chronic use of corticosteroids may adversely affect glucose control, necessitating an adjustment of their diabetic treatment regimen.		
giant cell arteritis (GCA)	**long term**	**low**
Patients who have PMR and GCA are at risk for blindness due to inflammation-induced vessel occlusion and ischemia.[4] [9] [12] Prompt treatment with high-dose corticosteroids is indicated to either prevent or limit visual impairment.		
hypertension secondary to corticosteroids	**long term**	**low**
Monitoring for hypertension should be a part of the regular follow-up. Patients should have a baseline blood pressure determination. The risk can increase if higher doses are needed for prolonged periods of time.[4] [62]		
muscle weakness secondary to corticosteroids	**long term**	**low**
Monitoring for muscle weakness should be a part of the regular follow-up. The risk can increase if higher doses are needed for prolonged periods of time.		
cataract development secondary to corticosteroids	**long term**	**low**
Monitoring for cataract development should be a part of the regular follow-up. The risk can increase if higher doses are needed for prolonged periods of time.[4] [62]		

FOLLOW UP

Complications	Timeframe	Likelihood
glaucoma secondary to corticosteroids	**long term**	**low**
Monitoring for glaucoma development should be a part of the regular follow-up. The risk can increase if higher doses are needed for prolonged periods of time.[4] [62]		
skin changes secondary to corticosteroids	**long term**	**low**
Skin thinning with increased bruising found with chronic use of corticosteroids. The risk can increase if higher doses are needed for prolonged periods of time.[4] [62]		
PMR-related vascular events	**variable**	**medium**
Cardiovascular, cerebrovascular, and peripheral vascular event rates were higher in patients with PMR compared with those without from 6 months to 12 years of follow-up. The median 7.8-year risk was increased 2.6-fold. The risk was higher for patients younger than 60 years of age (5.6-fold).[63]		
myelosuppression secondary to methotrexate	**variable**	**low**
In patients on methotrexate, monitoring for myelosuppression (CBC) is recommended. The risk increases with increasing dose. Regular use of folic acid decreases risk.		
oral ulcers secondary to methotrexate	**variable**	**low**
In patients on methotrexate, monitoring for oral ulcers is recommended. The risk increases with increasing dose. Regular use of folic acid decreases risk.		
hepatotoxicity secondary to methotrexate	**variable**	**low**
In patients on methotrexate, monitoring hepatotoxicity (with liver function tests) is recommended. The risk increases with elevated doses. Concurrent alcohol use and nonessential hepatotoxic medications should be avoided.		
interstitial lung disease secondary to methotrexate	**variable**	**low**
A baseline CXR should be obtained prior to initiating dose, and use of another agent should be considered if there is evidence of underlying interstitial lung disease. In patients on methotrexate, clinicians should monitor for interstitial lung disease/alveolitis (new cough, shortness of breath, dyspnea on exertions, new rales on exam, and new interstitial changes on CXR). This is an idiosyncratic reaction, not a function of dose or duration of treatment.		
liver injury secondary to tocilizumab	**variable**	**low**
Tocilizumab may increase the risk of serious liver injury (e.g., acute liver failure, hepatitis). Measure aminotransferase (ALT) and aspartate aminotransferase (AST) levels before initiation and every 4-8 weeks during the first 6 months of treatment. After the first 6 months, levels can be monitored every 12 weeks. Initiation of treatment is not recommended in patients with ALT or AST higher than 5-times the upper limit of normal. Patients should be advised to seek help immediately if they experience signs and symptoms of liver injury.[56]		

Prognosis

Prognosis

The overall prognosis is good. Although response to treatment typically occurs within 24 to 72 hours, relapses or symptom exacerbations are common. Treatment is typically required for 2 to 3 years. Less commonly, there is a chronic relapsing course that may require a longer course of treatment. PMR may also be associated with giant cell arteritis (GCA) and if present, the prognosis and treatment is directly related to GCA.[60] [61]

Increased risk of relapse or prolonged therapy has shown association with female sex, high ESR (>40 mm/hour), and peripheral arthritis at diagnosis.[48]

Diagnostic guidelines

International

2015 recommendations for the management of polymyalgia rheumatica (https://www.eular.org/recommendations-management) [45]

Published by: European League Against Rheumatism; American College of Rheumatology **Last published:** 2015

Treatment guidelines

International

2022 guideline for the prevention and treatment of glucocorticoid induced osteoporosis (https://rheumatology.org/clinical-practice-guidelines) [47]

Published by: American College of Rheumatology **Last published:** 2023

2022 guideline for vaccinations in patients with rheumatic and musculoskeletal disease (https://rheumatology.org/clinical-practice-guidelines) [59]

Published by: American College of Rheumatology **Last published:** 2023

2015 recommendations for the management of polymyalgia rheumatica (https://www.eular.org/recommendations-management) [45]

Published by: European League Against Rheumatism; American College of Rheumatology **Last published:** 2015

Key articles

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Images

Symptom/finding	Points without ultrasound (0-6)	Points with ultrasound (0-8)
Morning stiffness >45 minutes	2	2
Hip pain/limited range of motion	1	1
Negative rheumatoid factor or anticyclic citrullinated peptide antibody	2	2
No other joint involvement	1	1
Ultrasound 1: at least one shoulder with subdeltoid bursitis, biceps tenosynovitis, and/or glenohumeral synovitis, and at least one hip with synovitis or trochanteric bursitis	NA	1
Ultrasound 2: bilateral subdeltoid bursitis, biceps tenosynovitis, or glenohumeral synovitis	NA	1

A score of ≥4 without ultrasound or a score of ≥5 with ultrasound is classified as PMR.

Figure 1: Scoring algorithm for the classification of PMR

Adapted from Dasgupta B, et al. 2012 Provisional classification criteria for polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Arthritis Rheum. 2012:64:943-954

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BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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