

# Polymyalgia Rheumatica and Giant Cell Arteritis: Rapid Evidence Review

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Polymyalgia rheumatica and giant cell arteritis are inflammatory conditions that occur predominantly in people 50 years and older, with peak incidence at 70 to 75 years of age. Polymyalgia rheumatica is more common and typically presents with constitutional symptoms, proximal muscle pain, and elevated inflammatory markers. Diagnosis of polymyalgia rheumatica is clinical, consisting of at least two weeks of proximal muscle pain, constitutional symptoms, and elevated erythrocyte sedimentation rate or C-reactive protein. Treatment of polymyalgia rheumatica includes moderate-dose glucocorticoids with a prolonged taper. Giant cell arteritis, also known as temporal arteritis, usually presents with new-onset headache, visual disturbances or changes, constitutional symptoms, scalp tenderness, and temporal artery symptoms. Inflammatory markers are markedly elevated. Temporal arterial biopsy should be used for diagnosis. However, color duplex ultrasonography, magnetic resonance imaging, and fluorodeoxyglucose positron emission tomography may be helpful when biopsy is negative or unavailable. All patients with suspected giant cell arteritis should receive empiric high-dose glucocorticoids because the condition may lead to blindness if untreated. Tocilizumab is approved by the U.S. Food and Drug Administration for giant cell arteritis and should be considered in addition to glucocorticoids for initial therapy. Polymyalgia rheumatica and giant cell arteritis respond quickly to appropriate dosing of glucocorticoids but typically require prolonged treatment and have high rates of relapse; therefore, monitoring for glucocorticoid-related adverse effects and symptoms of relapse is necessary. Methotrexate may be considered as an adjunct to glucocorticoids in patients with polymyalgia rheumatica or giant cell arteritis who are at high risk of relapse. (*Am Fam Physician*. 2022;106(4):420-426. Copyright © 2022 American Academy of Family Physicians.)

**Polymyalgia rheumatica** (PMR) and giant cell arteritis (GCA) are related inflammatory conditions, which typically affect people 50 years and older. This article provides a summary and review of the best available patient-oriented evidence for each condition.

## Epidemiology

- Incidences of PMR and GCA vary among people 50 years and older depending on geographic location. The incidence per 100,000 people 50 years and older ranges from 2 (Korea) to 113 (Norway) for PMR and 1 (Turkey) to 44 (Iceland) for GCA.<sup>1</sup>
- Approximately 20% of patients with PMR have GCA, and about 50% of patients with GCA have PMR.<sup>2,3</sup>
- Risk factors for PMR and GCA include female sex, Northern European ancestry, and age.<sup>4-6</sup>

**CME** This clinical content conforms to AAFP criteria for CME. See CME Quiz on page 373.

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- The age of onset for both conditions is typically 50 years and older, with peak incidence at 70 to 75 years of age.<sup>2,3</sup>

## Polymyalgia Rheumatica

### DIAGNOSIS

- With no definitive diagnostic test for PMR, the diagnosis is clinical. The typical clinical presentation is shown in *Table 1*.<sup>3,7-10</sup>
- The differential diagnosis of PMR is summarized in *Table 2*.<sup>7,9,11,12</sup>

### History and Physical Examination

- The patient history reveals predominantly symmetrical shoulder and thigh stiffness, which may cause difficulties in rising from a seated position and raising arms overhead.<sup>3</sup>
- Up to 50% of patients report constitutional symptoms such as fever, fatigue, night sweats, or weight loss.<sup>3</sup>
- Physical examination may show restricted active range of motion and weakness or tenderness in the hip and shoulder muscles.

### Laboratory Studies

- An erythrocyte sedimentation rate (ESR) of more than 30 to 40 mm per hour or elevated C-reactive protein (CRP)

TABLE 2

### Differential Diagnosis for Polymyalgia Rheumatica and Giant Cell Arteritis

Polymyalgia rheumatica	Giant cell arteritis
<b>Musculoskeletal</b>	
Adhesive capsulitis	Dental pain/conditions
Calcium pyrophosphate deposition disease	Temporomandibular disorders
Gout	
Osteoarthritis	
Rotator cuff pathology	
Spondyloarthropathy	
Subacromial bursitis	
<b>Autoimmune</b>	
Dermatomyositis	Optic neuritis
Polymyositis	Polyarteritis nodosa
Rheumatoid arthritis	Small/medium vessel vasculitis
Systemic lupus erythematosus	
Vasculitis	
<b>Neurologic</b>	
Parkinson disease	Cerebrovascular accident/retinal artery occlusion
	Migraine
	Trigeminal neuralgia
<b>Infectious</b>	
Endocarditis	Acute otitis media/externa
Hepatitis B or C	Endocarditis
HIV	Herpes zoster
Parvovirus B19	Meningitis/encephalitis
Tuberculosis	Sinusitis
<b>Malignant</b>	
Lung cancer paraneoplastic syndrome	Central mass effect
Lymphoma	Lymphoma
Multiple myeloma	Multiple myeloma
Prostate cancer	
<b>Endocrine</b>	
Hyperparathyroidism	Hypothyroidism
Thyroid disorders	
<b>Psychiatric</b>	
Depression	Depression
Fibromyalgia	
<b>Iatrogenic</b>	
Statin-induced myopathy	Drug-induced vasculitis

Information from references 7, 9, 11, and 12.

levels are present in approximately 90% of patients with PMR.<sup>13,14</sup>

- Complete blood count often shows associated normochromic anemia and thrombocytosis.<sup>4</sup> Liver function tests may show mildly elevated alkaline phosphatase.<sup>15</sup>
- Muscle enzyme levels, including creatinine kinase, are normal in patients with PMR.<sup>16</sup>
- Positive antinuclear antibodies, antineutrophil cytoplasmic antibodies, rheumatoid factor, and anti-citrullinated peptide antibodies are not found in patients with PMR and indicate an alternative diagnosis if present.<sup>7</sup>

#### Imaging

- Ultrasonography of the shoulder shows subdeltoid bursitis in up to 79% of patients with PMR and may be considered as an adjunct diagnostic study.<sup>17,18</sup>
- If clinically indicated, chest radiography may be considered because lung cancer paraneoplastic syndrome can mimic PMR.<sup>14</sup> An exhaustive evaluation for underlying occult malignancy or infection is not recommended.<sup>4</sup>

TABLE 1

### Typical Clinical Presentation of Polymyalgia Rheumatica

#### Patient demographics

Age 50 years and older

#### Symptoms

Acute onset of bilateral pain in the shoulder or pelvic girdle

Associated morning stiffness lasting longer than 45 minutes

Constitutional symptoms such as fever, fatigue, night sweats, or weight loss (present in up to 50% of patients)

#### Physical examination

Decreased active and passive range of motion in the affected joints

#### Laboratory findings

Evidence of acute phase reaction (e.g., elevated inflammatory markers)

#### Ultrasound findings

Subdeltoid bursitis, biceps tenosynovitis, or glenohumeral synovitis

#### Duration of symptoms

Rapid onset of symptoms lasting longer than two weeks

#### Response to treatment

Rapid response to appropriate glucocorticoid therapy (typically within two to four weeks)

Information from references 3 and 7-10.

## POLYMYALGIA RHEUMATICA AND GIANT CELL ARTERITIS

- Plain radiography of the involved joints may show concomitant degenerative joint disease but does not add diagnostic value.<sup>7</sup>

### TREATMENT

- *Table 3* includes consensus treatment recommendations for PMR.<sup>7,8,19-21</sup>

#### Medical Management

- The consensus primary treatment of PMR is oral glucocorticoid therapy, starting at 12.5 to 25 mg per day of prednisone (the minimum effective dose) or the equivalent. Once clinical improvement is achieved, typically in two to four weeks, an initial taper to 10 mg per day over four to eight weeks is recommended. Remission is likely if the patient remains clinically stable at 10 mg per day, and glucocorticoid dose may be slowly tapered by 1 mg every four weeks until discontinuation.<sup>7,8,14,19,22</sup>
- If there is a relapse or flare-up, the glucocorticoid dose should be increased by 10% to 20% or to the dose before

relapse. Once the patient is stable on the new dose, the glucocorticoid dose should be tapered over four to eight weeks to where it was when the relapse occurred.<sup>8</sup>

- The addition of methotrexate may be warranted in patients at high risk of relapse (e.g., females, those with high baseline ESR), glucocorticoid-related adverse effects, or comorbidities that are likely to be exacerbated by glucocorticoids (e.g., diabetes mellitus, osteoporosis).<sup>7,8,19</sup> Multiple high-quality randomized controlled trials have shown that the early addition of methotrexate therapy is associated with lower relapse rates and lower cumulative glucocorticoid dose. The dosage of methotrexate used in these trials ranged from 7.5 to 10 mg per week.<sup>23-25</sup>
- One small but high-quality randomized controlled trial of intramuscular methylprednisolone, 120 mg administered every three weeks, demonstrated comparable remission rates but lower cumulative glucocorticoid dose and less weight gain compared with standard oral therapy.<sup>26</sup> Therefore, intramuscular methylprednisolone may be considered as an

**TABLE 3**

### Treatment of Polymyalgia Rheumatica and Giant Cell Arteritis

Treatment	Polymyalgia rheumatica	Giant cell arteritis
Initial therapy	Oral glucocorticoid, 12.5 to 25 mg per day of prednisone (minimum effective dose) or the equivalent, until clinical improvement is achieved, typically two to four weeks	Oral glucocorticoid, 40 to 60 mg per day of prednisone or the equivalent, plus subcutaneous tocilizumab (Actemra) weekly for one to two months until stable
Taper	Taper to 10 mg per day over four to eight weeks, then decrease by 1 mg every four weeks until discontinuation	Taper prednisone or equivalent dose by 10% to 20% per month while monitoring C-reactive protein level and erythrocyte sedimentation rate When dose is < 10 mg daily, taper by 1 mg per month until discontinuation, individualizing duration to the patient, typically over one to five years
Remission	Patient is considered in remission once clinically stable at 10 mg per day	Patient is considered in remission once clinically stable at 20 mg per day
Treatment of relapse	Reassess diagnosis Increase prednisone dose or equivalent by 10% to 20% or to dose before relapse Reattempt taper gradually over four to eight weeks	Mild flare-up: increase prednisone dose or equivalent by 10% to 20% Severe flare-up: repeat initial therapy and consider adding glucocorticoid-sparing drugs (e.g., tocilizumab, methotrexate, abatacept [Orencia])
Additional considerations	Consider oral methotrexate, 7.5 to 10 mg per week, in patients at high risk of relapse, with glucocorticoid-related adverse effects, or with comorbidities likely to be exacerbated by glucocorticoids Consider intramuscular methylprednisolone, 120 mg administered every three weeks, in patients at high risk of glucocorticoid-related adverse effects	Consider methotrexate with a glucocorticoid or glucocorticoid alone

Information from references 7, 8, and 19-21.

alternative to oral prednisone equivalents in patients at high risk of glucocorticoid-related adverse effects.<sup>8,19</sup>

- Infliximab (Remicade) and etanercept (Enbrel) have not shown benefit in the treatment of PMR.<sup>27,28</sup>

*Follow-up and Monitoring*

- Patients should be assessed for response to therapy, glucocorticoid-related adverse effects, relapse, and development of GCA.<sup>14</sup>
- Dose-tapering schedules for patients with PMR should be individualized based on monitoring of disease activity, laboratory markers, and adverse events.<sup>19</sup>
- An alternative diagnosis should be considered in patients who do not respond to glucocorticoid therapy or who have recurrent relapses.<sup>19</sup>
- Assessment of bone mineral density with dual-energy x-ray absorptiometry and treatment of osteoporosis should be considered in patients at high risk of fracture (e.g., those 65 years and older or with a history of fracture) while undergoing glucocorticoid therapy.<sup>14,29</sup>
- Rheumatology consultation should be considered for patients who respond poorly to initial treatment, are at high risk of relapse or treatment-induced adverse effects, or have contraindications to glucocorticoid therapy.<sup>19</sup>

*Lifestyle and Alternative Therapies*

- An individualized exercise program to maintain muscle mass and prevent falls, especially in older or frail patients with PMR, is recommended based on expert opinion.<sup>7,19</sup>

- The use of herbal preparations for the treatment of PMR is not recommended given a paucity of supporting evidence and unclear adverse effect profiles.<sup>19</sup>

**PROGNOSIS**

- Mortality in patients with PMR is comparable to that in an age-matched control group.<sup>2</sup>
- Given the nonerosive nature of PMR-associated synovitis, permanent tissue damage to the involved joints is rare.<sup>30</sup>
- Relapse may occur in up to 50% of patients.<sup>31</sup> Risk factors for relapse and the development of concomitant GCA include female sex, high baseline ESR, initial glucocorticoid dosing of less than 10 mg per day or more than 20 mg per day, and a faster than recommended glucocorticoid taper.<sup>22,32,33</sup>

**Giant Cell Arteritis**

- GCA, also known as temporal arteritis, most commonly affects branches of the external carotid arteries (i.e., temporal and occipital arteries), although it may affect any medium or large artery.<sup>8,11</sup> GCA can result in blindness if untreated.

**DIAGNOSIS**

- The differential diagnosis of GCA is summarized in (Table 2).<sup>7,9,11,12</sup>

*History and Physical Examination*

- Diagnosis of GCA relies on a thorough history focused on vascular, ocular, musculoskeletal, and neurologic examinations.

• Classic clinical findings of GCA include new-onset headache, visual disturbances or changes, constitutional symptoms (e.g., weight loss, fatigue, anorexia), scalp tenderness, and temporal artery symptoms (i.e., enlargement, loss of pulses, nodularity, or tenderness).<sup>8,11,20</sup>

• Additional clinical findings include limb claudication, stroke or transient ischemic attack, dysphagia, peripheral pulse disturbances, or aortic aneurysm (Table 4).<sup>8,11,20</sup>

*Laboratory Testing*

- Temporal artery biopsy can confirm the diagnosis and should be performed in all patients with suspected GCA (Table 5).<sup>20</sup>
- ESR, CRP, complete blood count, and comprehensive metabolic panel should

**TABLE 4**

**Clinical Findings in Giant Cell Arteritis**

Classical presentation	Sensitivity (%)	Specificity (%)	LR+	LR-
Scalp tenderness	41	98	21	0.60
Jaw/tongue claudication	39	98	20	0.62
Temporal artery physical findings*	57	97	19	0.44
New-onset headache	65	82	3.6	0.43
Visual disturbance or changes	28	89	2.5	0.81
<b>Additional clinical findings†</b>				
Aortic aneurysm	Stroke or transient ischemic attack			
Limb claudication	Systemic symptoms: weight loss, fever, fatigue, anorexia			
Peripheral pulse disturbances				

LR- = negative likelihood ratio; LR+ = positive likelihood ratio; .

\*—Enlargement, loss of pulses, nodularity, or tenderness.

†—Sensitivity and specificity of these findings are not well defined in the literature.

Information from references 8, 11, and 20.

**SORT: KEY RECOMMENDATIONS FOR PRACTICE**

Clinical recommendation	Evidence rating	Comments
Patients with PMR should be treated with the minimum effective dose of oral glucocorticoids with individualized duration. <sup>14,19,22</sup>	<b>C</b>	Expert opinion
Early addition of methotrexate to glucocorticoid therapy should be considered in patients with PMR who are at high risk of relapse or glucocorticoid-related adverse events. <sup>7,8,19</sup>	<b>A</b>	Consistent results from multiple randomized controlled trials
Intramuscular methylprednisolone every three weeks should be considered as an alternative to oral glucocorticoids in patients at high risk of adverse effects from glucocorticoids. <sup>8,19</sup>	<b>B</b>	A single small, high-quality randomized controlled trial
Dose-tapering schedules for patients with PMR should be individualized based on monitoring of disease activity, laboratory markers, and adverse events. <sup>19</sup>	<b>C</b>	Expert opinion in the absence of clinical trials
In patients with newly diagnosed GCA, noninvasive vascular imaging (i.e., magnetic resonance imaging or computed tomography angiography) of the neck, chest, abdomen, and pelvis should be performed to evaluate for large vessel involvement. <sup>20</sup>	<b>C</b>	Lower-quality evidence and consensus guidelines
To improve chances of successful remission, patients with newly diagnosed GCA should be treated with high-dose oral glucocorticoids and subcutaneous interleukin-6 antagonist tocilizumab (Actemra) over glucocorticoids alone. <sup>8,20,38,39</sup>	<b>A</b>	Two high-quality randomized controlled trials with consistent results and consensus guidelines
Long-term monitoring is recommended for patients with GCA who are in clinical remission. <sup>20</sup>	<b>C</b>	Evidence showing that monitoring is low risk and there is potential for harm from not monitoring; consensus guidelines

GCA = giant cell arteritis; PMR = polymyalgia rheumatica.

**A** = consistent, good-quality patient-oriented evidence; **B** = inconsistent or limited-quality patient-oriented evidence; **C** = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to <https://www.aafp.org/afpsort>.

**TABLE 5**

**Recommendations for Performing a Temporal Artery Biopsy**

**Location**

Unilateral study of the most symptomatic side, expanding to the contralateral side if initial biopsy result is negative but strong clinical suspicion remains

**Sizing**

Biopsy should be greater than 1 cm, targeting a 2-cm segment because of concern for missed histologic findings secondary to skip lesions

**Timing**

Should be performed as soon as possible after initiation of therapy, ideally within the first week, but can be extended to within two weeks of starting treatment based on resource availability

*Information from reference 20.*

be obtained at presentation and while monitoring medical management.

- Marked elevation of ESR (40 to 60 mm per hour) or CRP is present in most cases. Disease severity may be reflected in the degree of ESR elevation.<sup>8</sup>
- Measurement of interleukin-6 should be considered when the patient does not have increased ESR or CRP but the index of suspicion is high. Interleukin-6 may be elevated in new and recurrent cases of GCA, returning to baseline on remission.<sup>34</sup>

*Imaging Studies*

- Imaging studies may be indicated if there is strong clinical concern for GCA but temporal artery biopsy results are negative. Characteristic findings on color duplex ultrasonography, magnetic resonance imaging, or fluorodeoxyglucose positron emission tomography can support the diagnosis. Availability and technician experience may limit these modalities.<sup>8,20,35,36</sup>

- In patients with newly diagnosed GCA, noninvasive vascular imaging (i.e., magnetic resonance imaging or computed tomography angiography) of the neck, chest, abdomen, and pelvis should be performed to evaluate for large vessel involvement. Patients with hypertension at the time of GCA diagnosis are at higher risk of thoracic or aortic aneurysm and should be evaluated for large vessel involvement with angiography or ultrasonography.<sup>37</sup>

## TREATMENT

- *Table 3* includes consensus treatment recommendations for GCA.<sup>7,8,19-21</sup>
- Suspected GCA should be treated as a medical emergency, and initiation of treatment should not be delayed for diagnostic studies.<sup>20,21</sup>
- Treatment should be followed by urgent referral to a rheumatologist.<sup>12,20</sup>
- High-dose oral glucocorticoids are the primary treatment for GCA without cranial ischemia, starting at 40 to 60 mg per day of prednisone or equivalent for one to two months, followed by individualized tapering regimens over one to five years.<sup>8,20,21</sup>
- Two high-quality randomized controlled trials showed that adding weekly or every-other-week administration of subcutaneous tocilizumab (Actemra) to a 26-week prednisone taper was superior to prednisone taper plus placebo for achievement of sustained GCA remission.<sup>38,39</sup>
- High-dose intravenous glucocorticoids should be initiated in patients with symptoms of critical cranial ischemia, including visual disturbances.<sup>20</sup>
- Patients undergoing high-dose glucocorticoid therapy require bone-conserving and gastroprotective therapy, including calcium and vitamin D supplementation and consideration of bisphosphonates and proton pump inhibitors.<sup>29</sup>
- Glucocorticoid-sparing therapies such as tocilizumab, methotrexate, abatacept (Orencia), and other biologics can be considered in all patients based on symptom severity, patient goals and preferences, and physician experience.<sup>20</sup>

## PROGNOSIS

- Early recognition and treatment are associated with decreased morbidity.<sup>8,20</sup>
- Although GCA does not affect overall survival, patients with the condition are more likely to die of circulatory diseases than their peers because of the risk of progressive vasculitis involving large vessels elsewhere in the body.<sup>12</sup>
- Morbidity is usually related to adverse effects of glucocorticoids.<sup>21</sup>
- Long-term clinical and laboratory monitoring is associated with decreased frequency and severity of recurrence.<sup>12,20</sup>

This article updates previous articles on this topic by Meskimen, et al.<sup>40</sup>; Epperly, et al.<sup>41</sup>; Unwin, et al.<sup>42</sup>; and Caylor and Perkins.<sup>9</sup>

**Data Sources:** This article was based on literature cited in Essential Evidence Plus, the Cochrane database, and a PubMed search using the Clinical Queries database for the terms polymyalgia rheumatica, giant cell arteritis, and temporal arteritis. Search dates: September and November 2021 and July 2022.

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## References

1. Sharma A, Mohammad AJ, Turesson C. Incidence and prevalence of giant cell arteritis and polymyalgia rheumatica: a systematic literature review. *Semin Arthritis Rheum.* 2020;50(5):1040-1048.
2. Salvarani C, Gabriel SE, O'Fallon WM, et al. Epidemiology of polymyalgia rheumatica in Olmsted County, Minnesota, 1970-1991. *Arthritis Rheum.* 1995;38(3):369-373.
3. Kermani TA, Warrington KJ. Polymyalgia rheumatica [published correction appears in *Lancet*. 2013;381(9860):28]. *Lancet.* 2013;381(9860):63-72.
4. Salvarani C, Cantini F, Boiardi L, et al. Polymyalgia rheumatica and giant-cell arteritis. *N Engl J Med.* 2002;347(4):261-271.
5. Doran MF, Crowson CS, O'Fallon WM, et al. Trends in the incidence of polymyalgia rheumatica over a 30 year period in Olmsted County, Minnesota, USA. *J Rheumatol.* 2002;29(8):1694-1697.
6. Crowson CS, Matteson EL, Myasoedova E, et al. The lifetime risk of adult-onset rheumatoid arthritis and other inflammatory autoimmune rheumatic diseases. *Arthritis Rheum.* 2011;63(3):633-639.
7. Matteson EL, Dejaco C. Polymyalgia rheumatica. *Ann Intern Med.* 2017;166(9):ITC65-ITC80.
8. Buttgerit F, Dejaco C, Matteson EL, et al. Polymyalgia rheumatica and giant cell arteritis: a systematic review. *JAMA.* 2016;315(22):2442-2458.
9. Caylor TL, Perkins A. Recognition and management of polymyalgia rheumatica and giant cell arteritis. *Am Fam Physician.* 2013;88(10):676-684.

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- Chuang TY, Hunder GG, Ilstrup DM, et al. Polymyalgia rheumatica: a 10-year epidemiologic and clinical study. *Ann Intern Med.* 1982;97(5):672-680.
- Hunder GG, Bloch DA, Michel BA, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum.* 1990;33(8):1122-1128.
- Brekke LK, Fevang BTS, Diamantopoulos AP, et al. Survival and death causes of patients with giant cell arteritis in Western Norway 1972-2012: a retrospective cohort study. *Arthritis Res Ther.* 2019;21(1):154.
- Cantini F, Salvarani C, Olivieri I, et al. Erythrocyte sedimentation rate and C-reactive protein in the evaluation of disease activity and severity in polymyalgia rheumatica: a prospective follow-up study. *Semin Arthritis Rheum.* 2000;30(1):17-24.
- 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(1):186-190.
- Salvarani C, Cantini F, Hunder GG. Polymyalgia rheumatica and giant-cell arteritis. *Lancet.* 2008;372(9634):234-245.
- Kreiner F, Langberg H, Galbo H. Increased muscle interstitial levels of inflammatory cytokines in polymyalgia rheumatica. *Arthritis Rheum.* 2010;62(12):3768-3775.
- Falsetti P, Acciai C, Volpe A, et al. Ultrasonography in early assessment of elderly patients with polymyalgic symptoms: a role in predicting diagnostic outcome? *Scand J Rheumatol.* 2011;40(1):57-63.
- Dasgupta B, Cimmino MA, Maradit-Kremers H, et al. 2012 provisional classification criteria for polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. *Ann Rheum Dis.* 2012;71(4):484-492.
- Dejaco C, Singh YP, Perel P, et al. 2015 recommendations for the management of polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. *Ann Rheum Dis.* 2015;74(10):1799-1807.
- Maz M, Chung SA, Abril A, et al. 2021 American College of Rheumatology/Vasculitis Foundation guideline for the management of giant cell arteritis and Takayasu arteritis. *Arthritis Care Res (Hoboken).* 2021;73(8):1071-1087.
- Hoffman GS, Cid MC, Hellmann DB, et al.; International Network for the Study of Systemic Vasculitides. A multicenter, randomized, double-blind, placebo-controlled trial of adjuvant methotrexate treatment for giant cell arteritis. *Arthritis Rheum.* 2002;46(5):1309-1318.
- Hernández-Rodríguez J, Cid MC, López-Soto A, et al. Treatment of polymyalgia rheumatica: a systematic review. *Arch Intern Med.* 2009;169(20):1839-1850.
- 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(7):493-500.
- Ferraccioli G, Salaffi F, De Vita S, et al. Methotrexate in polymyalgia rheumatica: preliminary results of an open, randomized study. *J Rheumatol.* 1996;23(4):624-628.
- Nazarinia AM, Moghimi J, Toussi J. Efficacy of methotrexate in patients with polymyalgia rheumatica. *Koomesh.* 2013;14(3):265-270.
- Dasgupta B, Dolan AL, Panayi GS, et al. 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(2):189-195.
- 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(9):631-639.
- Kreiner F, Galbo H. Effect of etanercept in polymyalgia rheumatica: a randomized controlled trial. *Arthritis Res Ther.* 2010;12(5):R176.
- Hoes JN, Jacobs JWG, Boers M, et al. EULAR evidence-based recommendations on the management of systemic glucocorticoid therapy in rheumatic diseases. *Ann Rheum Dis.* 2007;66(12):1560-1567.
- Salvarani C, Cantini F, Macchioni P, et al. Distal musculoskeletal manifestations in polymyalgia rheumatica: a prospective followup study. *Arthritis Rheum.* 1998;41(7):1221-1226.
- Dejaco C, Duftner C, Buttgerit F, et al. The spectrum of giant cell arteritis and polymyalgia rheumatica: revisiting the concept of the disease. *Rheumatology (Oxford).* 2017;56(4):506-515.
- Kremers HM, Reinalda MS, Crowson CS, et al. Relapse in a population based cohort of patients with polymyalgia rheumatica. *J Rheumatol.* 2005;32(1):65-73.
- Mackie SL, Hensor EMA, Haugeberg G, et al. Can the prognosis of polymyalgia rheumatica be predicted at disease onset? Results from a 5-year prospective study. *Rheumatology (Oxford).* 2010;49(4):716-722.
- van der Geest KSM, Abdulhad WH, Rutgers A, et al. Serum markers associated with disease activity in giant cell arteritis and polymyalgia rheumatica. *Rheumatology (Oxford).* 2015;54(8):1397-1402.
- Salvarani C, Silingardi M, Ghirarduzzi A, et al. Is duplex ultrasonography useful for the diagnosis of giant-cell arteritis? *Ann Intern Med.* 2002;137(4):232-238.
- Blockmans D, de Ceuninck L, Vanderschueren S, et al. Repetitive 18F-fluorodeoxyglucose positron emission tomography in giant cell arteritis: a prospective study of 35 patients. *Arthritis Rheum.* 2006;55(1):131-137.
- Gonzalez-Gay MA, Garcia-Porrúa C, Piñeiro A, et al. Aortic aneurysm and dissection in patients with biopsy-proven giant cell arteritis from northwestern Spain: a population-based study. *Medicine (Baltimore).* 2004;83(6):335-341.
- Stone JH, Tuckwell K, Dimonaco S, et al. Trial of tocilizumab in giant-cell arteritis. *N Engl J Med.* 2017;377(4):317-328.
- Unizony S, McCulley TJ, Spiera R, et al. Clinical outcomes of patients with giant cell arteritis treated with tocilizumab in real-world clinical practice: decreased incidence of new visual manifestations. *Arthritis Res Ther.* 2021;23(1):8.
- Meskimen S, Cook TD, Blake RL Jr. Management of giant cell arteritis and polymyalgia rheumatica. *Am Fam Physician.* 2000;61(7):2061-2068.
- Epperly TD, Moore KE, Harrover JD. Polymyalgia rheumatica and temporal arteritis. *Am Fam Physician.* 2000;62(4):789-796.
- Unwin B, Williams CM, Gilliland W. Polymyalgia rheumatica and giant cell arteritis. *Am Fam Physician.* 2006;74(9):1547-1554.