Revista Colombiana de
REUMATOLOGÍA
www.elsevier.es/rcreuma

Review Article

Polymyalgia Rheumatica☆

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ARTICLE INFO

Article history:
Received 15 April 2014
Accepted 16 April 2015

Keywords:
Polymyalgia rheumatica
Giant cell arteritis
Glucocorticoids
Pain
Myalgia

ABSTRACT

Polymyalgia rheumatica is an inflammatory disease common in the geriatric population. The clinical profile is characterized by pain, mainly in the shoulder girdle, hip and cervical region. The diagnosis of this disease is clinical, and should be made to rule out another diseases such as late onset rheumatoid arthritis and spondyloarthritis. Although no standardized classification criteria have been accepted, although there are some, yet to be evaluated, pro-visional classification criteria that have recently been developed by the American College of Rheumatology and European League Against Rheumatism. The polymyalgia rheumatic is associated with up to 30% of patients with giant cell arteritis. The main treatment is low-dose glucocorticoids, with which patients have rapid symptomatic improvement.

Polimialgia reumática

Palabras clave:
Polimialgia reumática
Arteritis de células gigantes
Corticosteroides
Dolor
Mialgia

RESUMEN

La polimialgia reumática es una enfermedad inflamatoria crónica común en la población geriátrica. Su cuadro clínico se caracteriza por dolor en la cintura escapular, región cervical y caderas, asociado frecuentemente a rigidez de estas áreas articulares posterior a periodos de reposo. El diagnóstico de esta patología es clínico y debe hacerse posterior a descartar otras entidades como artritis reumatoide o espondiloartropatía de aparición tardía. Hasta el momento, no hay criterios de clasificación estandarizados y aceptados, por lo que recientemente se desarrollaron unos criterios provisionales por parte del Colegio Americano de Reumatología y de la Liga Europea Contra el Reumatismo. La polimialgia reumática

☆Please cite this article as: García Arias RL, Martín Gutiérrez J, Díaz MC, Fernández-Ávila DG. Polimialgia reumática. Rev Colomb Reumatol. 2015. http://dx.doi.org/10.1016/j.rcreu.2015.04.002
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se asocia hasta en un 30% de los pacientes con arteritis de células gigantes. El principal tratamiento es con dosis bajas de glucocorticoides, con lo cual los pacientes presentan rápida mejoría sintomática.

Materials and Methods

A literature search was conducted using the following databases: Medline, Cochrane collaboration and CRD Data base. The search was restricted to the English and Spanish languages, topic review articles, systematic reviews and original articles that have been published until March 2014. The mesh terms used were the following: "polymyalgia rheumatica" [Mesh], "glucocorticoids" [Mesh], "Myalgia" [Mesh]. The Boolean operators AND/OR were used for each of the terms. 171 articles were obtained, of which 53 were chosen by consensus of the group of authors, considering that these articles provided information about the topics needed to carry out the present review. Based on the obtained information, it was made a narrative review about the diagnosis and treatment of polymyalgia rheumatica (PMR) (Fig. 1).

Epidemiology

PMR is a typical disease of elderly patients. It occurs almost exclusively in people over 50 years and the average age of onset is 73 years.[1] Its prevalence is estimated at 700/100,000 inhabitants older than 50 years.[2] Seven hundred and eleven thousand people in the United State have the disease and the incidence rate is 58.7 people per 100,000 people/year.[3] Its incidence increases with age and varies according to the geographic region studied, finding a higher incidence in Scandinavian countries and in people descended from Northern Europe.[1,4] During the first year after diagnosis, patients use more the healthcare services and are prone to myocardial infarction, peripheral arterial disease and cerebrovascular events.[5]

PMR is associated with giant cell arteritis in 30% of patients. Clinically, 40-60% of patients with giant cell arteritis have symptoms of PMR at the time of diagnosis.[6] PMR and giant cell arteritis have many similarities (age of onset, prevalence in women, similar geographical distribution and, in both, documentation of a chronic inflammatory state), suggesting that the two entities could represent different types of the same disease.

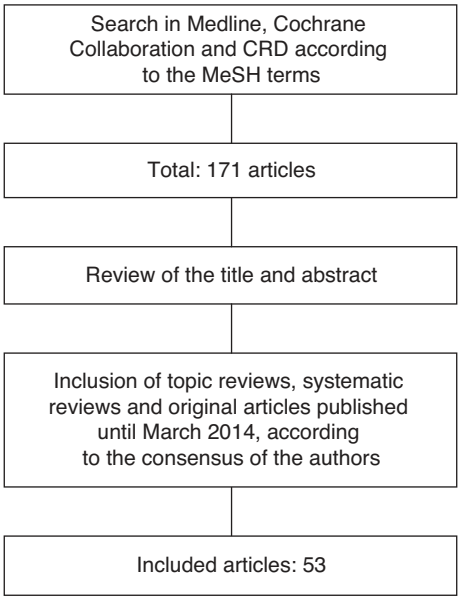

Figure 1 – Flowchart for selection of articles.

Etiopathogenesis

The cause of PMR is unknown as well as the reason for its higher frequency among the geriatric population. In the pathogenesis of the disease the studies suggest a genetic basis associated with environmental factors.[7] Genetic polymorphisms related to immune regulation and associated with the risk and severity of the disease include: intercellular adhesion molecule-1, interleukin-6 and interleukin-1 receptor antagonists.[8,9] The association with some antigens, such as HLA DR4 and HLA DRB1, as well as with infectious agents, such as Mycoplasma *pneumoniae*, parvovirus B19 and *Chlamydia pneumoniae*, shows controversial results in the studies and cannot be justified yet in the pathogenesis of the disease.[10]

It has been documented as an important mechanism in the pathophysiology of PMR, the increase in the local production of proinflammatory cytokines, mainly interleukin 6, detected in the muscle interstitium and in the blood of patients with the disease, whose values decrease significantly with the remission of the symptoms after treatment with corticosteroids.[11] Likewise, the process of endocrine-senescence which produces decreased levels of dehydroepiandrosterone or androstenedione and alterations in the hypothalamic-pituitary-gonadal axis with adrenal insufficiency and decreased secretion of cortisol in response to the inflammatory state, have been postulated as important etiopathogenic mechanisms.[12]

Clinical Manifestations

PMR is characterized by pain and stiffness of the shoulder and pelvic girdles and the cervical region. The characteristic clinical profile, in general, of more than one month of dura-

tion, consists of pain in the shoulder girdle, bilateral, predominantly at night, which increases with rest, associated with morning stiffness that makes difficult the performance of daily life activities, and is accompanied by constitutional symptoms such as fatigue, malaise, anorexia, weight loss and fever that may occur in 40-50% of patients.[13]

Given the association between PMR and giant cell arteritis, it is mandatory to identify symptoms such as headache, jaw claudication, scalp tenderness, carotidynia, visual alterations and claudication of the extremities.[14]

On physical examination there can be found stiffness and pain in shoulder girdle with inability of the individual for active elevation of the upper extremities, while passive mobilization is normal. Additionally, it can appear pain on palpation of the muscles without joint swelling, and in 25% of patients it can be seen joint synovitis, which is transient, oligoarticular, of peripheral joints, mainly with involvement of the knees, carpal, and metacarpophalangeal joints, of mild to moderate intensity and without production of erosions or joint destruction.[15]

Diagnosis

The diagnosis of PMR is basically clinical. An exhaustive clinical history and a careful physical examination should be carried out in order to distinguish it from other entities (Table 1).

Table 1 – Differential Diagnosis of Polymyalgia Rheumatica.

Rheumatic diseases
- Rheumatoid arthritis
- Spondyloarthropathies
- Arthritis caused by crystals
- Systemic lupus erythematosus
- Vasculitis
- Inflammatory myopathies

Non-inflammatory musculoskeletal diseases
- Fibromyalgia
- Adhesive capsulitis
- Degenerative joint disease
- Rotator cuff disease

Endocrinopathies
- Thyroid disorders
- Alterations of the parathyroid glands

Infections
- Viral
- Bacterial
- Mycobacterial

Malignant diseases
- Solid, hematological

Miscellaneous
- Parkinsonism
- Depression
- Hypovitaminosis D
- Drug-induced myopathy. Example: statins

Several classification criteria for PMR are mentioned in the literature (Bird, Jones and Hazleman, Chuang and Healey), none of which have been standardized and accepted.[13,16,17] With the aim of getting better classification criteria for PMR, an international initiative was developed by the European League Against Rheumatism and the American College of Rheumatology, entities that developed provisional classification criteria that where published in April, 2012.[18] (Table 2) These criteria have not been validated as diagnostic criteria because they are criteria for classification, useful for distinguishing polymyalgia from other disorders, and their applicability is addressed, primarily, to the field of research. It is reported a sensitivity of 66% and a specificity of 81% to discriminate patients with the disease, compared with control subjects.[19]

The laboratory findings are nonspecific and are similar to those of a process of systemic inflammation. The most characteristic is the elevation of the erythrocyte sedimentation rate above 40 mm/h, however, low (≤ 30 mm/h) or normal values have been reported in 6-20% of patients with the disease.[20,21] C-reactive protein is a more sensitive marker of inflammation in this pathology.[22,23] The rheumatoid factor, antinuclear antibodies and anti-cyclic citrullinated peptide antibodies are negative and the diagnosis should be reconsidered if they are positive.[24]

Regarding diagnostic imaging, the conventional radiology of the affected joints is usually normal, which also happens with electromyographic studies. Ultrasonography frequently detects abnormalities of periarticular structures and, according to the new classification criteria, it improves the specificity for the classification of PMR (Table 2), being useful in cases of patients with polymyalgia and normal inflammatory markers.[25]

The MRI shows abnormalities of periarticular structures, such as subdeltoid, subacromial and trochanteric bursitis, as well as synovitis.[26]

The PET scan is an expensive study and it is not routinely used in patients with PMR. It can be used in the evaluation of patients with unexplained or refractory symptoms and suspicion of occult neoplasm or vasculitis.[27]

Table 2 – Provisional Classification Criteria for Polymyalgia Rheumatica.

Clinical criteria
- Morning stiffness >45 minutes 2
- Hip pain or limitation 1
- Negative RF or anti-CCP 2
- Absence of other joint affection 1

Ultrasonographic criteria
- At least one shoulder with subdeltoid bursitis or bicipital tenosynovitis or glenohumeral 1 synovitis and at least one hip with trochanteric bursitis or synovitis
- Both shoulders with subdeltoid bursitis, bicipital tenosynovitis or glenohumeral synovitis 1

Required criteria: age ≥ 50 years, bilateral shoulder pain, increased ESR or CRP

With only clinical criteria a score ≥ 4 has a sensitivity of 68% and a specificity of 78% to differentiate PMR from other disorders

With the combination of clinical and sonographic criteria a score ≥ 5 has a sensitivity of 66% and a specificity of 81% to differentiate PMR from other disorders

The synovial biopsy shows synovitis, to a lesser extent than in rheumatoid arthritis. The biopsy of the temporal artery is not routinely indicated and should be performed only for suspected giant cell arteritis.[6]

Treatment

The treatment of choice for PMR consists of low-dose-glucocorticoids, with which a rapid resolution of symptoms is achieved, usually within 24 to 72 hours, being considered, even, as a therapeutic test to help confirm the diagnosis, given the excellent and rapid response that patients usually have with this type of treatment.

The recommended dose is 15 to 20 mg of prednisolone/day. High doses are rarely necessary unless there is a suspicion of giant cell arteritis.[28] The initial dose of glucocorticoids should be maintained for 2 to 4 weeks, reducing it slowly at a rate of 2.5 mg every 2 to 4 weeks up to a dose of 10 mg/day; this dose is used as maintenance during one month, with subsequent progressive reductions in the dose until it is discontinued.[28,29]

The majority of patients require treatment for 12 to 24 months. Sometimes, after dose reduction, the symptoms reappear, being necessary to increase again the dose until reaching a dose which eliminates the symptoms and, in some cases, long term low doses of glucocorticoids may be needed to prevent these relapses. In mild cases or in patients with multiple comorbidities, intramuscular methylprednisolone at a starting dose of 120 mg every 3 to 4 weeks, during 3 months, followed by a reduction of 20 mg every 2 to 3 months may be considered.[30,31] Along with the clinical response, the markers of inflammation should also be normalized within a few weeks after starting treatment, if it does not occur, other diagnoses should be considered.

The chronic use of glucocorticoids increases the risk of osteoporosis,[32,33] and therefore, the administration of prophylactic treatment for bone mass loss with calcium and vitamin D is indicated in elderly patients, as well as bisphosphonate therapy in high-risk patients.[34,35]

Regarding immunomodulators, their use could be considered in patients with PMR and high risk for developing adverse effects associated with the chronic use of glucocorticoids or in patients with recurrent disease.[36,37]

Methotrexate is the most studied antirheumatic agent in PMR, however, it should be taken into account that this drug has been tested in 3 randomized clinical trials, finding different results; 2 studies demonstrated efficacy[38,39] and in the third study, it was not possible to prove a corticosteroid-sparing effect.[40] Regarding azathioprine, there is only one study

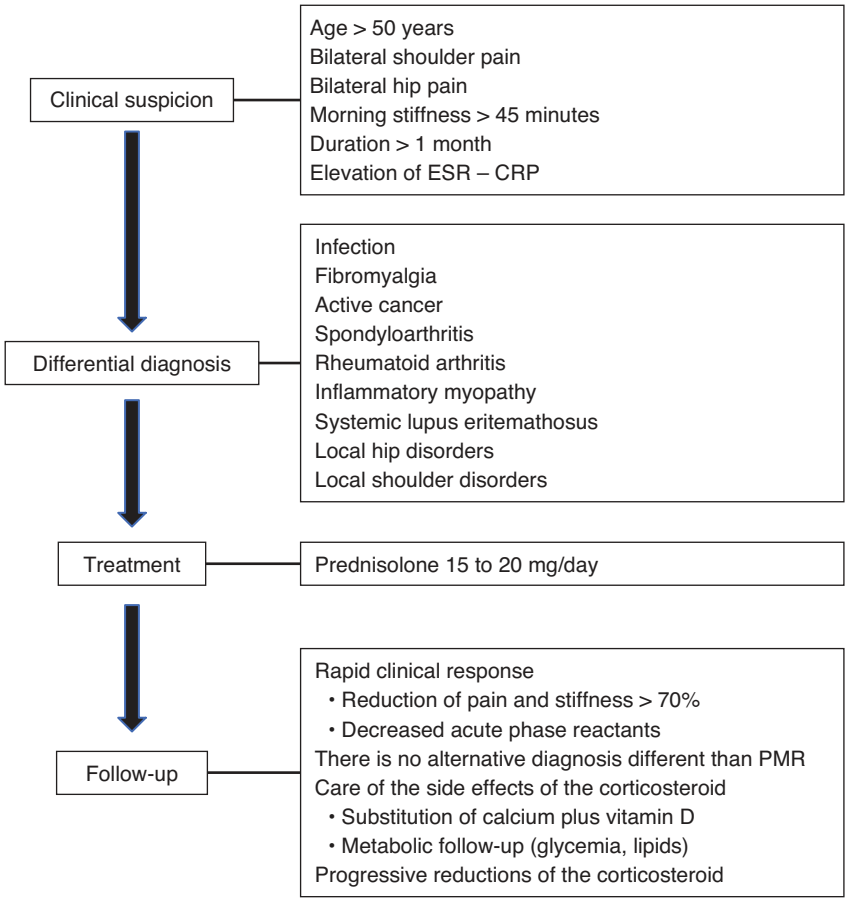

Figure 2 – Diagnostic and therapeutic algorithm in PMR.

available, which was conducted in a randomized, double-blind, placebo-controlled manner, demonstrating a decrease in the dose of prednisolone at the week 52, but with an increase in the frequency of adverse effects related with azathioprine, however, it should be borne in mind that it was a small study (31 patients) and only 65% of the subjects of study completed the follow-up time.[41]

On the other hand, to date there is no evidence that recommends the use of biological therapy, but its use is being investigated in patients with relapses, as is the case of infliximab, which was tested in a randomized multicenter clinical trial, compared with placebo in 40 patients with PMR, who received infliximab and prednisolone vs. placebo and prednisolone, being documented equal relapse rates in both groups.[42] Other therapeutic targets for biological therapies are currently being investigated, as is the case of therapies against IL-1, IL-6 and IL-17.

Prognosis

The quality of life of patients with PMR is usually compromised since the onset of the disease, but it substantially improves when treatment is started.[43,44] The response to treatment is assessed taking into account the inflammation markers and symptomatic improvement. It should be kept in mind that 50% of patients may experience relapses.[45,46] The initial treatment with high doses of steroids or their rapid withdrawal, as well as the female gender and elevated levels of CRP and IL 6 are associated with the risk of relapses.[47,48] On the other hand, the increased risk of peripheral vascular disease generates the possibility of vascular complications,[49] however, the survival of patients with PMR is similar to that of the general population.[50,51]

Conclusion

PMR is an inflammatory disease of unknown cause, very rare in people under 50 years old. The incidence increases with age with a peak in people between 70 and 80 years old, so it should be suspected in all elderly patients with osteomuscular pain in the shoulder or pelvic girdle. There are no specific tests for the disease and the diagnosis is based on the clinical presentation and the usual elevation of acute phase reactants (Fig. 1). Its frequent association with giant cell arteritis should be taken into account. Finally, it should be recalled that the diagnosis of PMR is established by exclusion, i.e., first they should be considered and ruled out other diseases that might show similar symptoms, such as late-onset rheumatoid arthritis and spondyloarthropaties, malignant diseases, and metabolic and infectious diseases. The treatment is based on the use of corticoids, at low doses, with which the clinical improvement is usually rapid and marked (Fig. 2).

Conflict of Interest

The authors declare that they have no conflict of interest.

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