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Case Report

Rotator Cuff Tendon Ruptures and Degeneration as the First Manifestation of Polymyalgia Rheumatica Disease - A Case Report

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Abstract

Polymyalgia Rheumatica (PMR) is a common rheumatic disease of the elderly. Although it is a well-established disease, its causes and pathophysiology remain unclear. In our case report we present an 83-year-old female presented at the emergency department because of fever and diarrhea. Her medical history included a recent orthopedic surgery because of tendons rupture of the rotator cuff. Her blood exams showed increased inflammatory markers and a three-digit ESR. The diagnosis of PMR was set after the exclusion of infectious and other diseases that mimic PMR symptoms. To the best of our knowledge, it is the first time that rotator cuff tendons rupture and degeneration is the first manifestation of PMR disease. Clinicians should be aware of the degeneration of the shoulder and hip extra-articular structures in PMR and they should keep in mind that it can be the first manifestation of the disease.

Keywords: Polymyalgia rheumatica; Rotator cuff denegeration; Tendon rupture

Introduction

Polymyalgia Rheumatica (PMR) is the most common inflammatory rheumatic disease of the elderly [1]. The typical symptoms of the disease are proximal myalgia of the hip and shoulder girdles with morning stiffness that lasts for more than one hour [1]. It is important to discriminate PMR from Giant Cell Arteritis (GCA) because of the significant differences in their prognosis and treatment between them. However PMR and GCA are often overlapping conditions [2]. Actually, to be more specific, approximately 15% of patients with PMR develop GCA, while 40-50% of patients with GCA have associated PMR. The diagnosis of PMR is mainly clinical by excluding other relevant mimicking conditions [3]. The exclusion of an underlying malignancy presenting as PMR-like syndrome is of great importance [3]. Rapid response to relatively low doses of corticosteroids (12.5 mg - 25 mg of prezolon) is considered pathognomonic for the diagnosis. Recommendations for the management of PMR have been proposed [3]. In our case we present an 83-year-old female with rotator cuff tendon rupture and degeneration as the first manifestation of PMR.

Case Presentation

An 83-year-old female with idiopathic hypertension (on amlodipine/balsartan), hyperlipidaemia (on rosuvastatin) and depression (on escitalopram) was presented at the emergency department of our hospital complaining of fever (up to 38.5°C without rigors) and diarrhea (2-3 times per day without abdominal pain, blood or mucus) 6 days ago. She reported fatigue which gradually started a month ago without loss of appetite or weight loss. Furthermore, the patient reported pain in her right shoulder during the last three months. Her orthopaedist recommended a right shoulder MRI which revealed tendon rupture of the supraspinatus and infraspinatus muscles as well as significant tendinopathy of the subscapularis and long head of biceps muscles. Consequently, she underwent an orthopedic surgery and she had the ruptutes repaired with titanium anchors.

The clinical examination did not reveal any pathological findings except for pain in the right shoulder in the passive and active movements. The patient did not report changes in the characters of the pain.

The blood exams revealed a normochromic, normocytic anemia (Hemoglobin: 11.6 g/dl, Hematocrit: 34.9%, MCV: 88.4 fl, MCH: 29.4 pg, MCHC: 33.2 g/dl) and increased inflammatory markers (white blood cells: 11800/μl, erythrocyte sedimentation rate: 110 mm/1 h, C - reactive protein: 19.07 mg/dl, ferritin: 1195.25 ng/ml and haptoglobins 571 mg/dl). Previous laboratory exams showed a gradually decrease in hematocrit values during the last 12 months without any clinically obvious region of hemorrhage. The right shoulder joint paracentesis did not reveal septic arthritis.

The patient was admitted in the Internal Medicine department of our Hospital and an empirical regimen with piperacillin/tazobactam was initiated.

Chest Computed Tomography (CT) scan did not reveal any pathological findings while abdominal CT scan revealed diverticulosis of the sigmoid and descending colon without inflammation. Endoscopic exam of the stomach did not reveal any pathology while coloscopy confirmed the diverticulosis. Mantoux and quantiferon tests were negative. Furthermore, Brucella agglutination tests (Rose-Bengal and Wright) were negative as well. ANA, Ra-test, c-ANCA, p-ANCA, SACE, CEA, a-FP, CA 19-9, protein electrophoresis, HBsAg, anti-HCV, anti-HAV IgM, anti-HIV were normal. The

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diarrhea episodes were discontinued at day 3 of her hospitalization.

The six pairs of blood cultures (aerobic/anaerobic) as well as urine and stools cultures were negative while transthoracic ultrasonography did not reveal valve vegetations. Blood real time - PCR showed the presence of cellulosimicrobium funkei and because the patient remained febrile and despite the sterile cultures the antibiotic regimen was modified to ciprofloxaxin/linezolide on day 5.

On day 7, the patient developed pain in her left shoulder and unilateral pain in left hip during the fever episodes. Biopsy of the temporal artery was obtained without showing giant cell arteritis. In day 12, the patient remained febrile and complained for morning stiffness that lasted more than 1 hour and bilateral pain of the shoulders and hips. The antibiotics were discontinued and due to our suspicion for PMR a low dose of prezolon (25 mg/day) was initiated. The patient remained asymptomatic and afebrile while the inflammatory markers returned within normal ranges.

Discussion

Although PMR is a well-known disease, its etiology and targets of inflammatory damage are not clear. Additionally, its diagnosis is still considered on the basis of clinical findings and elevation of inflammatory markers. Furthermore, as mentioned above, the rapid response to low doses of corticosteroids is considered pathognomonic for the diagnosis.

Recent MRI findings showed the catastrophic effect of PMR on the shoulder and hip joints [4]. In particular, the supraspinatus tendon is significantly thicker in PMR patients compared to patients with rheumatoid arthritis and control patients ($p< 0.05$) while severe rotator cuff tendinopathy is frequent in PMR patients [4]. In another study, MRI findings from the shoulder joints showed that the presence of inflammatory change outside the joint cavity was significant greater in PMR patients compared to rheumatic arthritis patients [3]. Furthermore, MRI showed that hand tenosynovitis, but not joint synovitis, is a frequent finding of PMR patients despite the absence of clinical signs of hand involvement [5]. As a result, it has been proposed that PMR may be a disease of the extra-articular structures [5]. The usage of cheaper techniques such as ultrasonographic evaluation of shoulders and hips and sonoelastography has been proposed for the localization of soft tissue damage in PMR patients [3]. Moreover, it has been shown that ultrasound improves the specificity of PMR diagnosis [3].

As mentioned above, our patient presented with diarrhea and fever. Clinicians should be very careful with PMR patients who report atypical symptoms which cannot be attributed to polymyalgia, such as diarrhea or abdominal pain. In such cases an alternative primary gastrointestinal pathology must be ruled out prior to steroid therapy because it may be detrimental [6]. In addition, a case of PMR patient who complaint for gastrointestinal symptoms and diagnosed with localized mesenteric vasculitis has been reported [7]. In our case, abdominal CT scan did not reveal pathological findings except for an uncomplicated diverticulosis and the inflammatory markers did not decrease with the antibiotic therapy.

Titanium allergy was another condition included in our differential diagnosis. However, the patient's history, clinical presentation and laboratory exams did not make this diagnosis plausible.

To the best of our knowledge, we present the first case of rotator cuff tendons rupture and degeneration as the first manifestation of PMR disease. We believe that PMR pathophysiology processes initiated long before the appearance of the characteristic clinical symptoms of the disease. An indication of that assumption is the gradual decrease in hematocrit that started approximately one year ago, due to the underlying chronic disease.

Conclusion

Clinicians should be aware of the degeneration of the shoulder and hip extra-articular structures in PMR and they should keep in mind that it can be the first manifestation of the disease.

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