Anarthritic Rheumatoid Arthritis and the Continuing Dilemma of Polymyalgia Rheumatica: Case Report

Arthur E. Brawer

Department of Internal Medicine, Division of Rheumatology, Monmouth Medical Center, Long Branch, New Jersey, USA

Corresponding Author: Arthur E. Brawer
arthurbrawer@optimum.net

ABSTRACT

A 71 year-old female presented with classical symptoms of polymyalgia rheumatica (PMR), accompanied by multiple other clinical phenomena highly suggestive of giant-cell arteritis (GCA). The latter diagnosis could not be confirmed by temporal artery biopsy, and nine months later her PMR evolved into seronegative rheumatoid arthritis (SNRA). Corticosteroid (CSs) treatment was avoided by utilizing traditional disease modifying anti-rheumatic drugs (DMARD’s), thereby sparing this patient from a multitude of potentially devastating CSs-induced side effects. This case reinforces a recently published clinical study emphasizing the need for reassessment of prevailing knowledge and treatment recommendations in PMR.

Keywords: polymyalgia rheumatica; rheumatoid arthritis; giant-cell arteritis; corticosteroids

1. INTRODUCTION

Polymyalgia rheumatica (PMR) is an inflammatory syndrome in older individuals, with initial clinical features of acute pain and stiffness in the shoulder and pelvic girdles. Traditional decades-old concepts regarding (a) differential diagnoses in PMR patients, (b) overlapping features of giant-cell arteritis (GCA), and (c) routine corticosteroid (CSs) treatment [1,2] have recently been challenged by data suggesting that most patients with PMR eventually evolve into seronegative rheumatoid arthritis (SNRA) and should not be treated with CSs at the time of their initial PMR presentation [3].

2. **CASE REPORT**

A 71 year-old Caucasian female presented with three months of pain and stiffness in her shoulders, upper arms, hips, buttocks and thighs. These symptoms were accompanied by two hours of morning stiffness, fatigue, night sweats, fever to 101.5, loss of appetite, ten pound weight loss, jaw claudication, temporal headaches, and transient visual disturbances. Her only medication was longstanding thyroid replacement, which was appropriately dosed. Physical examination was notable for normal blood pressure, normal heart sounds, normal peripheral pulses, diminished pulsations in both temporal arteries without tenderness or nodularity, and limitation of motion in her shoulders and hips with pain on motion. The only abnormality in a multitude of laboratory tests was an elevated Westergren sedimentation rate (ESR) of 67. Ophthalmologic evaluation was normal, and a temporal artery biopsy was normal. Corticosteroids (CSs) were withheld, and she was treated solely with hydroxychloroquine (HCQ) 400mg daily. After four months there was complete resolution of weight loss, fever, night sweats, headaches, jaw claudication, visual disturbances and elevated ESR, accompanied by dramatic improvement (but not resolution) of visual disturbances and elevated ESR, accompanied by dramatic improvement (but not resolution) of fatigue, morning stiffness and musculoskeletal complaints. Two months later, while still taking HCQ, all presenting symptoms and signs recurred (including headaches, night sweats, fever, visual disturbances, etc.), accompanied by pain and swelling and limited motion in her hands, wrists, knees and ankles. Rheumatoid factor and anti-nuclear antibody tests remained negative. The addition of oral methotrexate (MTX) 20mg per week for the next five months did not afford any consistent improvement in her overall condition. Fourteen months after disease onset HCQ and MTX were discontinued, and treatment was begun with intramuscular gold in the form of Myochrysine (MYO) 50mg per week. After four months there was complete resolution of fever, night sweats, headaches, jaw claudication and visual disturbances, accompanied by ninety percent improvement in her SNRA. Indefinite treatment with MYO continues at three week intervals without side effects.

3. **DISCUSSION**

Mystery has always surrounded the syndrome of PMR, in part due to the breadth of medical conditions that can present with such phenomena and in part due to the decades-old practice of giving CSs and seeing what is left over. There is only one peer-reviewed published study that has reported on the natural course of PMR without CSs treatment [3]. It suggests that most patients with PMR evolve their disease process into SNRA, which in turn can be successfully treated without ever implementing CSs. And yet recent reviews by investigators studying PMR have chosen to ignore this evidence [4,5,6]. Instead, these researchers not only perpetuate traditional concepts of CSs treatment in PMR, but they also speculate on atypical temporal artery biopsy findings in PMR to explain why it not infrequently overlaps with classical biopsy proven GCA. It may well be verified that pure PMR (i.e., PMR that does not evolve into SNRA) is accompanied by limited subclinical large vessel inflammation that does not become transmural, thereby precluding the development of classical clinical and pathological GCA [6]. My patient did not manifest any temporal artery biopsy abnormalities, and she exemplifies the pitfalls of utilizing classical symptomatology for definitive diagnosis of both PMR and GCA. More specifically, it has been reported that night sweats, fever, weight loss, headaches and visual disturbances are not discriminating features that help distinguish between PMR, GCA, and SNRA, and that true PMR is infrequent in the absence of biopsy proven GCA [3].

The question to be asked, therefore, is whether it is reasonable to continue to perpetuate the self-serving advice of multiple prior publications that promote the unbridled use of CSs in PMR. As this case illustrates, there are many facets of PMR that warrant reappraisal via the implementation of additional prospective studies devoid of CSs treatment. A recent report that treatment of PMR with modest doses of CSs is devoid of devastating long-term side effects [7] is not universally accepted [8], and does little to clarify the confusion. There exists a long list of DMARD’s that can be tested, including IL-6 inhibitors [9], but since this latter medication is now utilized for its CSs sparing effect in biopsy proven GCA [10], such studies need to be properly designed.

4. **CONCLUSION**

This case further illustrates that traditional concepts of PMR are in need of reassessment, including the diagnostic trial of CSs. The confusion encompassing PMR can likely be clarified by comingling other current investigative tools (e.g., ultrasound, cytokine profiles, etc.) with additional prospective studies that utilize a variety of DMARD’s in the absence of CSs treatment.
REFERENCES
