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An Enigmatic Case of Giant Cell Arteritis

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Abstract

Fatigue is commonly encountered in clinical settings with a myriad of possible etiologies that can range from benign to life-threatening illnesses. In this report, we discuss a case of a 60-year-old female with a history of B12 deficiency, hypertension, and treated hepatitis C who presented with persistent fatigue, diffuse myalgias, night sweats, and reduced appetite. Her initial workup revealed a low-grade fever (101.9 F), slightly elevated erythrocyte sedimentation rate (ESR, 29 mm/hr) and C-Reactive Protein (CRP, 7.6 mg/L). Labs and imaging were otherwise unremarkable. At follow-up, her symptoms persisted, and new-onset shortness of breath was noted. Transthoracic Echocardiography (TTE) showed moderate pericardial effusion with no tamponade, and whole body PET-CT showed avid aortitis and first order vessel vasculitis. The patient was diagnosed with giant cell arteritis (GCA) and began treatment with prednisone and tocilizumab. The pharmacologic treatment led to symptom improvement and reduced pericardial effusion. This case illustrates the diagnostic challenges associated with nonspecific symptoms like fatigue, especially when inflammatory markers are only mildly elevated. While ESR and CRP are widely utilized to evaluate for inflammatory conditions, they lack specificity and may be misleading in atypical presentations of GCA. We aim to detail the diagnostic workup for fatigue, emphasizing the role that inflammatory markers may have. Understanding the correlation between inflammatory processes and chronic fatigue can aid in accurate diagnosis, tailored treatment strategies, and improved long-term outcomes for patients.

Keywords: *Internal medicine, rheumatology, cardiology, vasculitis*

1. INTRODUCTION

Fatigue burdens many patients—significantly impacting quality of life — and its characterization is oftentimes a complex pursuit in clinical practice [1]. There are various organ systems and that may be implicated in presentations of persistent fatigue including rheumatological, cardiopulmonary, musculoskeletal, gastrointestinal, endocrine, and nervous systems [2]. Conditions such as fibromyalgia, sleep apnea, chronic obstructive pulmonary disease, somatic symptom disorder, and systemic lupus erythematous may all present with the same principal complaint of fatigue [2]. In many cases, initial workup of fatigue includes performing a complete history and physical exam while maintaining a broad differential [2]. Laboratory analysis usually includes complete blood counts with differential, fasting serum glucose, thyroid stimulating hormone, and a complete metabolic panel [2]. ESR and CRP are also commonly obtained to hone in on diagnoses which may point to inflammatory, possibly autoimmune, causes [2-3]. However, ESR and CRP are not specific markers for the inclusion or exclusion of common rheumatological diseases such as lupus, polymyalgia rheumatica, and various vasculitides from the differential diagnosis [3]. Indeed, infectious and neoplastic etiologies may also present with elevation of the aforementioned inflammatory markers [3].

Our case report discusses a rather atypical presentation of giant cell arteritis which highlights the need for practitioners to maintain a broad differential and consider vasculitis in patients with ongoing systemic symptoms and unrevealing initial evaluations. Our hope is to shed light on the detection of GCA in the clinical setting which may lead to much improved outcomes and quality of life for affected patients. *An adaptation of this article was submitted as an abstract for poster presentation at the ACP Arizona Annual Scientific Meeting to be held on Nov 14-15, 2025.*

2. Case

A 60-year-old female with a past medical history of B12 deficiency, hypertension, and treated hepatitis C presented to clinic for ongoing fatigue. When initially seen, her symptoms were constant fatigue, whole body myalgias, night sweats, and decreased appetite. Her temperature was 101.9, and her physical exam was normal. Erythrocyte Sedimentation Rate (ESR) was 29, and C-Reactive Protein (CRP) was 7.6. Chest x-ray was negative. At one month follow-up, she reported ongoing fatigue and shortness of breath with no headaches, scalp tenderness, or visual symptoms. A repeat EKG was unremarkable. Given suspicion for a vascular etiology, transthoracic Echocardiogram (TTE) was performed which revealed moderate pericardial effusion without signs of tamponade. Moreover, whole body PET-CT showed avid aortitis and first order vessel vasculitis. A diagnosis of Giant Cell Arteritis (GCA) was reached. Her symptoms of fatigue and decreased appetite both improved after starting prednisone and tocilizumab. Repeat TTE showed a reduction in pericardial effusion.

3. Discussion

Fatigue is a symptom that perplexes practitioners given that it may encompass a vast array of acute or chronic etiologies. The cause can be identified in about two-thirds of cases [1]. Some patients experience progressive debilitating fatigue with no identifiable cause despite appropriate evaluation [1,4,5]. In many circumstances, the results of laboratory studies and physical exam findings prove to offer little diagnostic clue as to the etiology of chronic fatigue [6]. Assessment includes in-depth history, physical exam, basic laboratory studies, and cancer screening [2]. Initial workup can include complete blood count with differential, chemistries, thyroid stimulating hormone, and creatinine kinase [2]. These were completed and normal in our patient. Inflammatory markers can be obtained when suspecting rheumatologic disorders such as lupus, rheumatoid arthritis, polymyalgia rheumatica, and GCA [7-9]. ESR and CRP elevations may be seen in older female patients, but elevation can also be due to anemia, renal disease, infections, malignancy, and tissue injury [2-3].

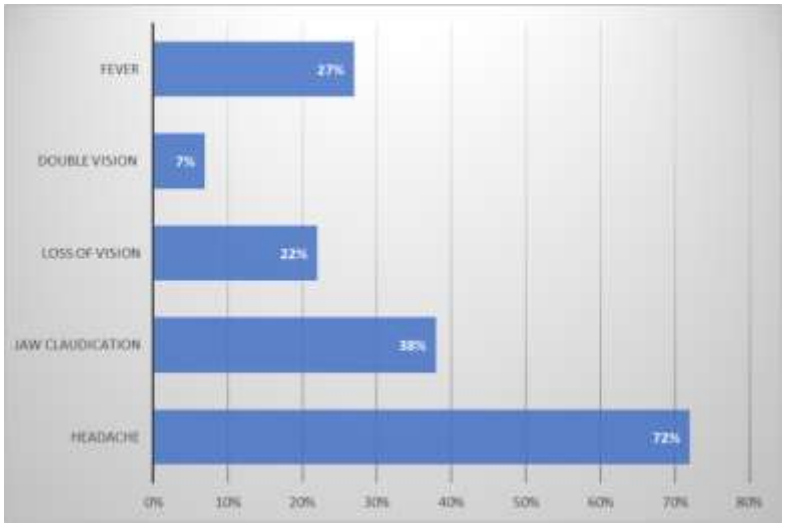

Figure 1. *Diagnostic Sensitivity of the Top 5 Most Common Symptoms of GCA*

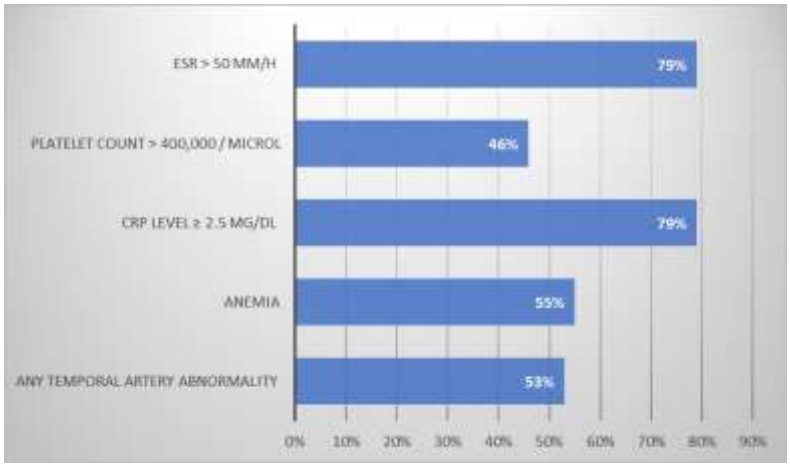

Figure 2. *Diagnostic Sensitivity of the Top 5 Most Common Laboratory and Physical Exam Findings of GCA*

Although ESR and/or CRP are high in GCA, they are not specific for GCA [10]. A 2012 study of 177 patients with GCA confirmed by temporal artery biopsy (TAB), reported that elevated CRP and elevated ESR provided a sensitivity of 86.9% and 84.1%, respectively, for a positive TAB [10]. Only 5% of the patients had an ESR less than 40, like our patient. 4% of patients with GCA had both ESR and CRP in normal range. These data and our patient case emphasize the limitation of ESR/CRP in being definitive markers for GCA. Figures 1 and 2 provide an overview of the diagnostic sensitivities of the most common symptoms, laboratory and physical exam findings in the workup of Giant Cell Arteritis based on a meta-analysis by van der Geest et al. [11]. The results of this meta-analysis underscore the multifaceted, oftentimes, ambiguous ways that GCA may present in patients [11]. Coupled with consistent demographic features, history, imaging and laboratory results, we urge physicians to not overlook conducting a thorough workup when there is an appropriate index of suspicion for giant cell arteritis.

4. Conclusion

Persistent fatigue presents a diagnostic challenge due to its unique presentations and diverse underlying causes. While initial workup typically involves assessing blood counts, chemistries, and thyroid function to identify common etiologies, the complexity of chronic fatigue necessitates a systematic approach. Despite extensive evaluation, a significant proportion of cases may remain idiopathic, highlighting the need for continued research into novel diagnostic biomarkers. Enhancing our understanding of the pathophysiological mechanisms underlying chronic fatigue will improve diagnostic accuracy and optimize patient care.

References

[1] Bates, D. W., Schmitt, W., Buchwald, D., Ware, N. C., Lee, J., Thoyer, E., Kornish, R. J., & Komaroff, A. L. (1993: Prevalence of fatigue and chronic fatigue syndrome in a primary care practice. 153:2759-2765. 10.1001/archinte.1993.00410240067007

[2] Maisel P, Baum E, Donner-Banzhoff N. Fatigue as the Chief Complaint–Epidemiology, Causes, Diagnosis, and Treatment. Dtsch Arztebl Int. 2021; 118(33-34):566-576. doi:10.3238/arztebl.m2021.0192

[3] Bitik B, Mercan R, Tufan A, et al. Differential diagnosis of elevated erythrocyte sedimentation rate and C-reactive protein levels: a rheumatology perspective. Eur J Rheumatol. 2015; 2(4):131-134. doi:10.5152/eurjrheum.2015.0113

[4] Nijrolder, I., van der Windt, D., de Vries, H., & van der Horst, H. (2009: Diagnoses during follow-up of patients presenting with fatigue in primary care. CMAJ: Canadian Medical Association journal = journal de l'Association medicale canadienne. 181:683-687.

[5] Kroenke, K., Wood, D. R., Mangelsdorff, A. D., Meier, N. J., & Powell, J. B. (1988: Chronic fatigue in primary care. Prevalence, patient characteristics, and outcome. JAMA. 260:929-934.

[6] Lane, T. J., Matthews, D. A., & Manu, P. (1990): The low yield of physical examinations and laboratory investigations of patients with chronic fatigue. American journal of the medical sciences, 299, 313-318.

[7] Helfgott, S. M., & Kieval, R. I. (1996: Polymyalgia rheumatica in patients with a normal erythrocyte sedimentation rate. Arthritis and rheumatism. 39:304-307.

[8] Myklebust, G., & Gran, J. T. 1996: A prospective study of 287 patients with polymyalgia rheumatica and temporal arteritis: clinical and laboratory manifestations at onset of disease and at the time of diagnosis. British journal of rheumatology. 35:1161-1168.

[9] Proven, A., Gabriel, S. E., O'Fallon, W. M., & Hunder, G. G. 1999: Polymyalgia rheumatica with low erythrocyte sedimentation rate at diagnosis. Journal of rheumatology. 26:1333-1337.

[10] Kermani, T. A., Schmidt, J., Crowson, C. S., Ytterberg, S. R., Hunder, G. G., Matteson, E. L., & Warrington, K. J. 2012: Utility of erythrocyte sedimentation rate and C-reactive protein for the diagnosis of giant cell arteritis. Seminars in arthritis and rheumatism, 41, 866-871.

[11] Van der Geest KSM, Sandovici M, Brouwer E, Mackie SL. Diagnostic Accuracy of Symptoms, Physical Signs, and Laboratory Tests for Giant Cell Arteritis: A Systematic Review and Meta-analysis. JAMA Intern Med. 2020; 180(10):1295–1304. doi:10.1001/jamaintern med.2020.3050.

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