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Eosinophilic Granulomatosis With Polyangiitis Associated With Pleural Tuberculosis: A Diagnostic Challenge – A Case Report

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Abstract
Churg-Strauss syndrome (CSS), or eosinophilic granulomatosis with polyangiitis (EGPA), presents a diagnostic challenge when associated with infectious diseases like tuberculosis. We report the case of a 50-year-old man with a history of asthma and sinus polyposis initially diagnosed with right-sided pleural tuberculosis. One month later, he returned with bilateral pleural effusion—notably eosinophilic on the left and lymphocytic on the right—accompanied by dyspnea, chest pain, and peripheral neuropathy. Diagnostic work-up revealed pericardial effusion, leading to a diagnosis of EGPA. This concomitant presentation of pleural tuberculosis and vasculitis raised complex therapeutic considerations. The patient was treated with anti-tuberculosis therapy, alongside corticosteroids and immunosuppressive agents and respiratory physiotherapy, resulting in symptomatic improvement. However, persistent peripheral neuropathy and residual pleural abnormalities were noted at 18 months follow-up. This case underscores the importance of a multidisciplinary approach in managing the rare overlap of EGPA and active tuberculosis.

Keywords: *Churg-Strauss syndrome, Eosinophilic granulomatosis with polyangiitis, Tuberculosis, Asthma, Peripheral neuropathy, Pleural effusion, eosinophilia, vasculitis*

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I. Introduction:

Churg-Strauss syndrome (CSS), also known as eosinophilic granulomatosis with polyangiitis (EGPA), was initially described by Churg and Strauss. Their characterization encompassed patients exhibiting asthma, eosinophilia, granulomatous inflammation, necrotizing systemic vasculitis, and necrotizing glomerulonephritis [1,2].

In 1990, The American College of Rheumatology (ACR) established classification criteria for EGPA. It's important to note that these criteria were designed as "classification criteria" rather than "diagnostic criteria". Recognizing the need to address the limitations of the 1990 ACR criteria, the ACR and the European Alliance of Associations for Rheumatology (EULAR) collaborated on a comprehensive revision of the classification criteria for EGPA, which was finalized in March 2022 [3].

EGPA's prevalence varies across Europe, with reported figures ranging from 10.7 in Paris to 13 in Norway, and 14 in Sweden, while in the USA it stands at 18 per million [4].

II. Case Report:

A 50-year-old male, a former chronic smoker (weaned 2 years prior) and occasional alcohol consumer, presented with a 9-year history of bronchial asthma,3 years of chronic dyspnea, and had undergone two surgeries for nasosinusal polyposis. His current illness began with a dry cough, chest pain, and progressive worsening of dyspnea to stage 2 mMRC caused by a right-sided serofibrinous pleural effusion. A pleural biopsy was performed, which showed granulomatous inflammation with caseous necrosis. Based on this finding, he was diagnosed with pleural tuberculosis and started on standard anti-bacillary therapy.

After one month of anti-bacillary treatment, the patient’s condition deteriorated. His dyspnea worsened to mMRC grade 4, accompanied by night sweats, bilateral chest pain, and a decline in general health. Notably, he developed neurological symptoms characterized by asymmetric paresthesia and "electric shock" sensations in his limbs, predominantly on the left side. Physical examination confirmed a fluid effusion syndrome of the right hemithorax.

A follow-up chest X-ray (figure 1) was performed, which objectified a pleural-type opacity in the right hemithorax (consistent with the known pleural effusion) and blunting of the left costophrenic angle, suggesting

the development of a new, bilateral process. To better characterize these findings, a chest CT scan was performed, confirming bilateral pleural effusion without parenchymal involvement (figure 2).

Bilateral pleural punctures revealed a left-sided eosinophilic pleural effusion (88% eosinophils) and a right-sided serofibrinous pleural effusion.

Laboratory investigations revealed significant hypereosinophilia (1,398/mm³), normocytic normochromic anemia (11.4 g/dL), and an elevated CRP (60 mg/L). Renal function was normal, with a 24-hour proteinuria level below 0.1g/24h, effectively ruling out renal involvement at that time. Fecal parasitological examinations were negative, and the perinuclear antineutrophil cytoplasmic antibody (pANCA) test was negative.

Cardiac echocardiography revealed evidence of diastolic dysfunction and a preserved ejection fraction of 61%. In addition, a moderate, chronic, non-circumferential pericardial effusion was noted.

The patient underwent flexible bronchoscopy with alveolocapillary lavage, bronchial aspirations were negative and bronchial biopsies showed non-specific chronic fibro-inflammatory remodeling.

Electromyoneurography (ENMG) confirmed an asymmetric, axonal, sensorimotor mononeuritis multiplex affecting all four limbs, predominantly the left median and ulnar nerves, as well as the peroneal and tibial nerves.

A diagnosis of Eosinophilic Granulomatosis with Polyangiitis (EGPA) was established based on the 1990 American College of Rheumatology (ACR) criteria, with the patient meeting four out of six criteria: bronchial asthma, hypereosinophilia, mononeuropathy, and paranasal sinus abnormalities. The diagnostic process was notably complex due to the concurrent presence of pleural tuberculosis.

In collaboration with the Internal Medicine department, a multidisciplinary therapeutic strategy was implemented. Given the unique challenge of managing a systemic vasculitis alongside an active infection, the clinical decision was made to continue the full anti-tuberculosis regimen while simultaneously initiating immunosuppressive therapy for EGPA. Although the patient had a favorable prognosis according to the Five-Factor Score (FFS = 0), the severity of the neurological deficits and serous membrane involvement (bilateral pleural and pericardial effusions) justified an intensive induction regimen. This consisted of high-dose oral corticosteroids (80 mg/day) combined with six pulses of intravenous cyclophosphamide.

Remission was subsequently maintained with oral corticosteroids and azathioprine. Respiratory symptoms were managed with inhaled corticosteroids and bronchodilators, while pregabalin was introduced to address the neuropathic pain. To optimize the functional outcome, the patient underwent intensive respiratory physiotherapy. This pleural rehabilitation was essential to facilitate lung expansion and mitigate the risk of pachypleuritis.

After 18 months of treatment, significant improvements in the patient's respiratory and cardiac symptoms were observed, with persistent peripheral neuropathic manifestations in the lower limbs. A decrease in eosinophil count to 26/mm³ was observed, and the chest x-ray (Figure 3) showed no pleural opacities. ENMG showed improvement in upper limb function, but continued axonal loss in the lower limbs. Overall, the patient had a favorable response to treatment.

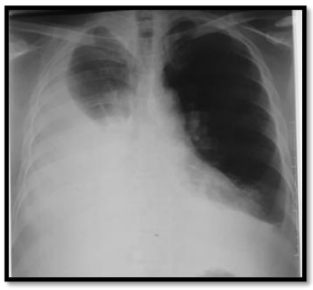

Figure 1: Chest X-ray showing pleural-type opacity in the right hemithorax and blunting of the left costophrenic angle

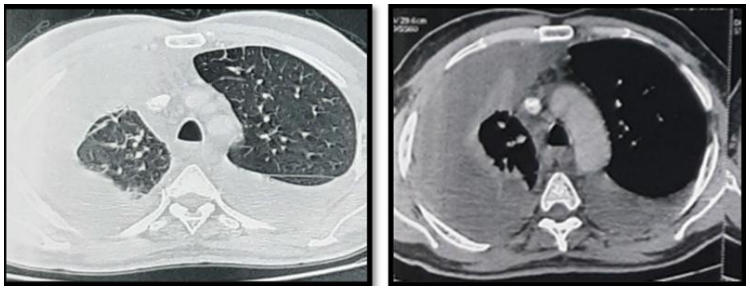

Figure 2: (a) Parenchymal window; (b) Mediastinal window

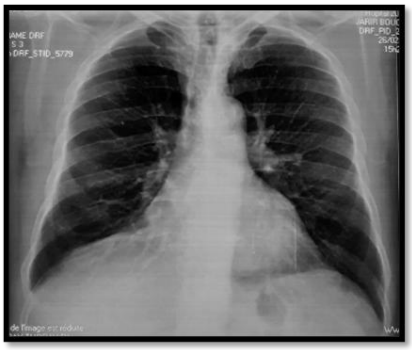

Figure 3: Chest X-ray after 18 Months of treatment

III. Discussion:

Eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg–Strauss syndrome, is a rare systemic vasculitis with heterogeneous immunopathogenesis [5,6]. Although antineutrophil cytoplasmic antibodies (ANCAs), mainly MPO-ANCA, are detected in a subset of patients, EGPA encompasses both vasculitic and eosinophil-driven inflammatory mechanisms, and its triggers are likely multifactorial, involving genetic, environmental, and infectious factors [5,7].

Asthma and chronic sinonasal disease are hallmark features that usually precede systemic manifestations, while peripheral blood eosinophilia remains a key diagnostic clue [5,6]. Pulmonary involvement is frequent and includes transient infiltrates, eosinophilic lung disease, pleural effusions, and pulmonary nodules, whereas cavitary lesions are uncommon [6]. The 2022 ACR/EULAR classification criteria have improved the accuracy of EGPA classification compared with the 1990 ACR criteria and are now recommended for clinical and research use [8].

The coexistence of EGPA and active tuberculosis is extremely rare, with only a limited number of cases reported in the literature [9]. This association represents a major diagnostic and therapeutic challenge, as tuberculosis may mimic EGPA manifestations and immunosuppressive treatment can worsen underlying mycobacterial infection [9,10]. Furthermore, patients with ANCA-associated vasculitis appear to have an increased risk of tuberculosis, particularly in endemic areas and during the early phase of immunosuppressive therapy [7].

Treatment of EGPA is guided by disease severity and organ involvement. Systemic glucocorticoids remain the cornerstone of therapy, while immunosuppressive agents are indicated in severe or organ-threatening disease [11]. More recently, interleukin-5–targeted biologic therapies, such as mepolizumab, have demonstrated efficacy in inducing and maintaining remission while reducing glucocorticoid exposure [12,13]. Nevertheless, vasculitic complications—especially cardiac and neurological involvement—as well as treatment-related infections remain the main causes of morbidity and mortality [5,13].

This case emphasizes the importance of systematic evaluation for tuberculosis before initiating immunosuppressive therapy in patients with suspected EGPA, particularly in TB-endemic regions, and highlights the need for a multidisciplinary approach to management [7–10].

IV. Conclusion:

This case highlights the diagnostic and therapeutic challenges associated with the management of Churg-Strauss syndrome (CSS), particularly when complicated by co-morbidities such as tuberculosis. The multidisciplinary approach to this patient's care facilitated effective management, resulting in significant symptomatic improvement. However, the persistence of residual neuropathy highlights the need for continued vigilance and comprehensive follow-up. Further research into the pathogenesis and optimal management strategies for CSS, particularly in the context of comorbidities, is warranted to improve patient outcomes and quality of life.

References:

[1]. Mukamal LV, Sodré CT, Prata LB, Nakasato F, Cuzzi T, Ramos-E-Silva M, Et Al. Churg-Strauss Syndrome Or Eosinophilic Granulomatosis With Polyangiitis: Exuberant Classic Clinical Picture Of A Rare Disease. Case Rep Dermatol 2018;10:175-81

[2]. Churg J, Strauss L. Allergic Granulomatosis, Allergic Angiitis, And Periarteritis Nodosa. Am J Pathol 1951;27:277-301.

[3]. Han, Qian Phd[a]; Li, Jie Md[a]; Wang, Jing Md[a]; Zhang, Liming Md[a]; Zhang, Jun Md[a,*]. Application Of The 2022 ACR/EULAR Criteria To Chinese Patients With Previously Diagnosed Eosinophilic Granulomatosis With Polyangiitis: The Concordance Rate Between The 2022 ACR/EULAR Criteria And The 1990 Criteria For EGPA In China. Medicine 103(4):P E37119, January 26, 2024. | DOI: 10.1097/MD.0000000000037119

[4]. Aladdin J Mohammad, An Update On The Epidemiology Of ANCA-Associated Vasculitis, Rheumatology, Volume 59, Issue Supplement_3, May 2020, Pages Iii42–Iii50,

[5]. Comarmond C, Pagnoux C. Eosinophilic Granulomatosis With Polyangiitis (Churg–Strauss). Semin Respir Crit Care Med. 2018;39(4):471–481.
[6]. Sinico RA, Bottero P. Churg–Strauss Angiitis. Best Pract Res Clin Rheumatol. 2009;23:355–366.
[7]. Mohammad AJ. An Update On The Epidemiology Of ANCA-Associated Vasculitis. Rheumatology (Oxford). 2020;59
[8]. Han Q, Li J, Wang J, Zhang L, Zhang J. Application Of The 2022 ACR/EULAR Criteria To EGPA. Medicine (Baltimore). 2024;103:E37119.
[9]. Yamashita H, Et Al. Concomitant Eosinophilic Granulomatosis With Polyangiitis And Active Tuberculosis. Intern Med. 2019;58:2709–2714.
[10]. Chen Y, Et Al. Pulmonary Tuberculosis Mimicking EGPA: Diagnostic Pitfalls. Respir Med Case Rep. 2021;33:101436.
[11]. Ribi C, Et Al. Treatment Of Churg–Strauss Syndrome Without Poor Prognosis Factors. Arthritis Rheum. 2008;58:586–594.
[12]. Samson M, Et Al. Long-Term Outcomes In EGPA. J Autoimmun. 2013;43:60–69.
[13]. Groh M, Et Al. Updated Recommendations For EGPA Management. Nat Rev Rheumatol. 2023;19:476–492.