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CASE REPORT

Early Intervention with Mepolizumab, Corticosteroids, and Intravenous Immunoglobulin for Dupilumab-Triggered Eosinophilic Granulomatosis with Polyangiitis: A Case Report

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Introduction: Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare complication during dupilumab therapy. However, the optimal treatment strategy for dupilumab-triggered EGPA remains unclear, particularly the timing and role of anti-interleukin-5 therapy.

Case Study: A 48-year-old female with severe eosinophilic rhinosinusitis and asthma, increased blood eosinophils (2098/μL) and myeloperoxidase-specific antineutrophil cytoplasmic antibody (MPO-ANCA) (46.1 U/mL) without vasculitic symptoms, developed systemic symptoms, including fever, arthralgia, and peripheral neuropathy immediately after dupilumab administration.

Results: Physical assessment revealed bilateral expiratory wheezes and laboratory tests revealed marked elevations in blood eosinophil (11,889/μL) and MPO-ANCA levels (125.0 U/mL). Other conditions, including parasitic infections, allergic bronchopulmonary aspergillosis, and *FIP1L1-PDGFRA*-positive disease, were excluded. Early intervention with mepolizumab (300 mg), methylprednisolone pulse therapy, and intravenous immunoglobulin (IVIG) was initiated after discontinuing dupilumab, resulting in rapid normalization of blood eosinophil counts and clinical improvement. Residual neuropathy was successfully treated with intravenous immunoglobulin (IVIg). Prednisolone was reduced to 15 mg daily with negative MPO-ANCA one month after treatment initiation.

Conclusion: This case emphasizes the importance of monitoring preexisting MPO-ANCA and blood eosinophils before and during dupilumab therapy. Early intervention with mepolizumab combined with conventional therapy is considered an optimal treatment strategy for dupilumab-triggered EGPA.

Keywords: antibodies, antineutrophil, cytoplasmic, mepolizumab, eosinophils, Churg-Strauss syndrome, dupilumab

Introduction

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare systemic vasculitis characterized by asthma, eosinophilia, and small-vessel vasculitis. Diagnosis can be complex, particularly when overt vasculitic symptoms are absent despite the presence of blood eosinophilia and myeloperoxidase-specific antineutrophil cytoplasmic antibody (MPO-ANCA) positivity.[1] While the 2022 American College of Rheumatology (ACR)/European Alliance of Associations for Rheumatology (EULAR) classification criteria offer guidance, incorporating key clinical features such as obstructive airway disease, significant eosinophilia, neuropathy, and sino-pulmonary manifestations, recognizing atypical presentations remains difficult.[2]

Dupilumab, a monoclonal antibody targeting the interleukin-4/13 (IL-4/13) receptor, has demonstrated significant efficacy in treating severe asthma and chronic rhinosinusitis with nasal polyps.[3] However, recent case reports have presented EGPA development or exacerbation of EGPA following dupilumab administration.[4–8] The mechanisms by which dupilumab may trigger EGPA remains unclear; however, it may involve IL-4/13 signaling blockade leading to

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enhanced IL-5 pathway activation and subsequent eosinophilia.[9] Several cases of dupilumab-associated EGPA have been reported; however, the optimal treatment strategy, particularly the timing and role of anti-IL-5 therapy, has not been established.

Here, we report a case of dupilumab-triggered EGPA successfully treated with early intervention combining mepolizumab, high-dose corticosteroids, and intravenous immunoglobulin (IVIG). This case emphasizes the importance of prompt recognition and a tailored, multi-faceted treatment strategy in managing this condition.

Case Presentation

A 48-year-old female with a history of bronchial asthma, diagnosed seven years prior, which had been well-controlled with fluticasone furoate/vilanterol (200 μ g/day), presented with worsening nasal congestion over the previous three months. She reported no history of smoking or significant comorbidities.

Computed tomography (CT) demonstrated diffuse mucosal thickening involving bilateral nasal cavities, maxillary, ethmoid, and sphenoid sinuses, with nasal polyps present in both nasal cavities (Figure 1). Histopathological assessment of the nasal polyps (Figure 2) showed findings consistent with severe eosinophilic rhinosinusitis, including significant eosinophilic infiltration (120 eosinophils per high-power field). The examined tissue was covered by multi-layered ciliated epithelium, with focal superficial erosion. The subepithelial layer showed inflammatory cell infiltration, including plasma cells and lymphocytes, and proliferation of capillaries. No evidence of vasculitis was observed in the examined specimen. Laboratory tests revealed increased peripheral blood eosinophil counts (2098 /μL) and positive MPO-ANCA tests (46.1 U/mL). However, the patient exhibited no symptoms that indicated systemic vasculitis, such as skin lesions, arthritis, or neurological manifestations, and therefore did not meet the diagnostic criteria for EGPA at the time.[2]

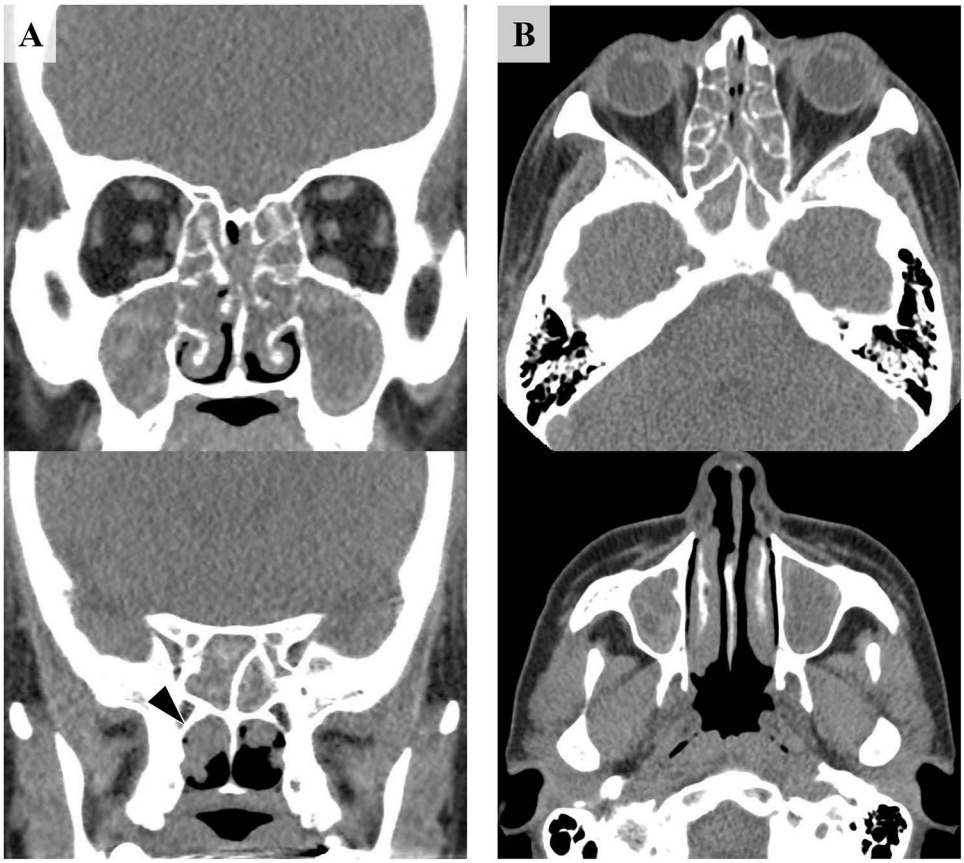

Figure 1 Computed tomography findings of severe eosinophilic rhinosinusitis. Coronal (**A**) and axial (**B**) computed tomography images showing diffuse mucosal thickening in bilateral nasal cavities, maxillary, ethmoid, and sphenoid sinuses with nasal polyps (arrow). The extensive mucosal disease is characteristic of severe eosinophilic rhinosinusitis before dupilumab administration.

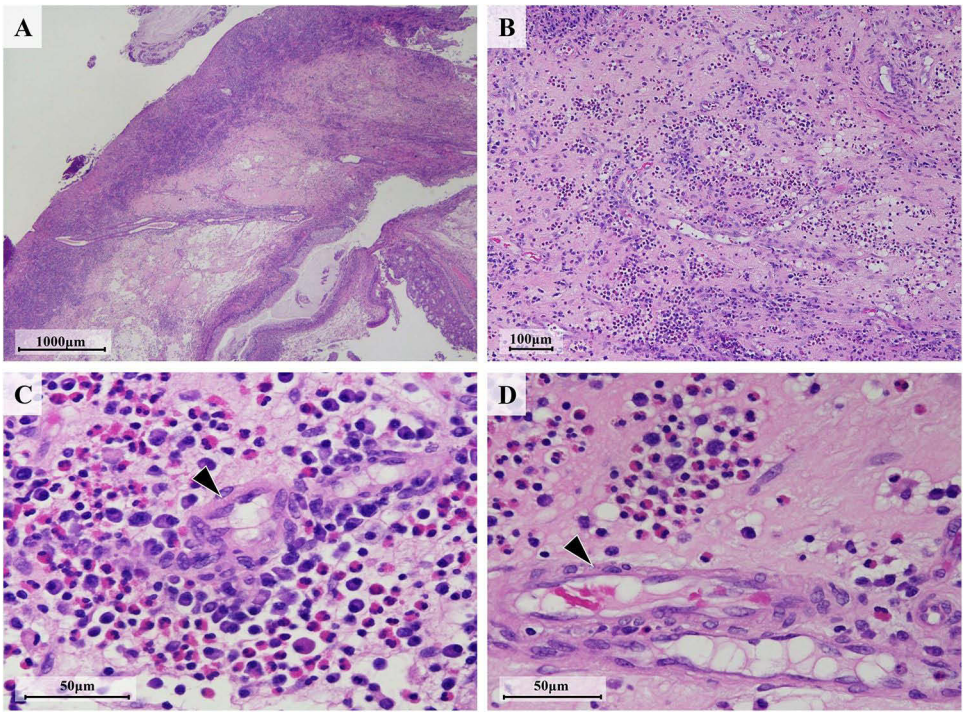

Figure 2 Histopathological Findings of Nasal Polyps. Hematoxylin and eosin stain. (**A**) Low-magnification view demonstrating overall mucosal architecture with marked eosinophilic infiltration in the subepithelial layer. (**B**) Higher magnification view focusing on the subepithelial layer, revealing dense inflammatory cell infiltrate rich in eosinophils, plasma cells, and lymphocytes. (**C** and **D**) High-magnification views of two different capillaries (arrows) demonstrating perivascular inflammatory cells, including numerous eosinophils. The capillary structures appear intact, with no definitive features of necrotizing vasculitis (eg, fibrinoid necrosis, destruction of the vessel wall).

Considering the severity of her eosinophilic rhinosinusitis, treatment with dupilumab was initiated. The day after the first administration, the patient developed systemic symptoms including fever, general fatigue, arthralgia, and bilateral lower limb numbness. Physical examination revealed bilateral expiratory wheezes throughout all lung fields. Laboratory tests two weeks after dupilumab initiation revealed marked elevation in the following: peripheral blood eosinophils 11,889 /μL, MPO-ANCA 125.0 U/mL, immunoglobulin E 120 IU/mL, and C-reactive protein 6.13 mg/dL. Aspergillus-specific IgE and IgG were negative. The renal function tests and urinalysis results were within the respective physiological reference ranges. Parasitic infection was excluded according to the negative stool examination results for ova and parasites, and genetic analysis revealed no *FIP1L1-PDGFRA* gene rearrangement. Chest CT showed only bronchial wall thickening consistent with asthma. Transthoracic echocardiography revealed no abnormalities. Pulmonary function tests revealed normal findings: forced vital capacity (FVC) was 2.77 L (101.1% of predicted), diffusing capacity for carbon monoxide was 19.65 mL/min/mmHg (102.7% of predicted), forced expiratory volume in 1 second (FEV1) was 2.37 L (95.2% of predicted), and FEV1/FVC ratio was 85.6%. Brain magnetic resonance imaging showed no abnormalities. Neurological examination revealed symmetrical, distal sensory deficits in a stocking-glove distribution, consistent with a peripheral neuropathy. Deep tendon reflexes were normal, and there were no pathological reflexes. Nerve conduction studies were performed on the left side, including motor nerve conduction studies of the median, ulnar, and tibial nerves, and sensory nerve conduction studies of the median, ulnar, and sural nerves. F-wave studies were performed on the left median and tibial nerves. All measured parameters were within normal limits. Although these nerve conduction study results did not show evidence of large-fiber involvement, the clinical presentation, particularly the symmetrical stocking-glove sensory deficits, was highly suggestive of small-fiber neuropathy, which is often not detected by standard nerve conduction studies.[10,11] EGPA-related neuropathy has a dual pathogenesis: ischemic damage due to necrotizing vasculitis of the small- and medium-sized vessels supplying the peripheral nerves, and direct nerve damage caused by eosinophil infiltration and the release of toxic granule proteins.[12,13] In this case, while the MPO-ANCA positivity suggests a potential for vasculitic involvement, the absence of large-fiber abnormalities on nerve conduction studies, combined with the clinical presentation, points towards a predominantly small-fiber neuropathy, where eosinophil-mediated

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mechanisms may play a more prominent role. Further evaluation with skin biopsy or quantitative sensory testing could have provided additional evidence for small-fiber involvement, but these tests were not performed in this case.[13,14] The Birmingham Vasculitis Activity Score (BVAS) was 14 at disease onset.

Dupilumab was discontinued due to the rapid development of systemic symptoms and marked eosinophilia, and the patient was diagnosed with dupilumab-triggered EGPA. Initially, oral prednisolone (30 mg daily) was administered for 4 days. As this initial treatment did not result in adequate improvement of her systemic symptoms and eosinophilia, immediate treatment was initiated with methylprednisolone pulse therapy combined with mepolizumab (300 mg subcutaneously), in accordance with the 2021 ACR/Vasculitis Foundation (VF) guidelines recommending mepolizumab for active, non-severe EGPA.[15] Verbal consent for this combination therapy was obtained and documented in the patient's medical record. Oral prednisolone (30 mg daily) was started after the pulse therapy.

This initial intervention resulted in rapid normalization of peripheral blood eosinophil counts and improvement of systemic symptoms. However, despite this initial response, the patient continued to experience residual neuropathic pain in her lower extremities. To address the persistent neurological symptoms, and considering the specific circumstances of this case (drug-induced EGPA, the patient's young age of 48, and the significant impact of pain on her daily life), a decision was made to add IVIG therapy as a single course at a total dose of 2 g/kg divided over five consecutive days, not as repeated cycles. Written informed consent was obtained from the patient for the use of IVIG.

Following the addition of IVIG, the neuropathic pain significantly improved. Prednisolone was successfully reduced to 15 mg daily within one month of initiating treatment, and MPO-ANCA tests were negative. The BVAS decreased to 0, indicating complete remission. The patient's condition remained stable with no systemic symptom recurrence during the one-year follow-up period, and prednisolone was further tapered to 4 mg daily.

Discussion

This case emphasizes several important clinical implications regarding dupilumab-triggered EGPA and its management. First, it illustrates the potential risk of EGPA development in patients with preexisting MPO-ANCA positivity and eosinophilia, even in the absence of distinct vasculitic symptoms. Second, it demonstrates the effectiveness of early mepolizumab intervention in combination with conventional therapy for managing dupilumab-triggered EGPA.

The presence of MPO-ANCA positivity and significant eosinophilia before dupilumab administration may represent important risk factors for EGPA development. The patient included in this case report did not initially meet the diagnostic criteria for EGPA; however, the combination of MPO-ANCA positivity, marked eosinophilia, and respiratory symptoms may have indicated a latent or subclinical disease form. This observation corresponds with previous reports by Olaguibel et al, emphasizing that EGPA initially presents with a prodromal phase characterized by asthma, rhinosinusitis, and blood eosinophilia, with subsequent vasculitic manifestations.[9] Furthermore, Eger et al and others reported that biological therapy may reveal or trigger the progression of underlying EGPA in such patients with preexisting risk factors.[4–8]

The rapid development of EGPA symptoms after dupilumab administration raises questions about the underlying pathophysiological mechanisms. Dupilumab may lead to increased IL-5 pathway activation and subsequent eosinophilia, by blocking IL-4/13 signaling, as revealed by the TRAVERSE study by Wechsler et al.[16] Kushima et al recently reported that patients whose peripheral blood eosinophil counts continued to increase within 6 months of dupilumab initiation had a significantly higher risk of developing EGPA compared with those whose counts peaked and subsequently declined.[17] Furthermore, they showed that patients with eosinophil counts of >1500 cells/μL exhibited a significantly higher risk of developing EGPA. Previous reports have indicated that blood eosinophilia during dupilumab treatment usually peaks within the first few months and is generally asymptomatic.[18–22] However, the dramatic increase in both eosinophil counts and MPO-ANCA levels, accompanied by systemic symptoms in this report, indicates a more complex immune response, potentially involving both eosinophil activation and vasculitic processes. This pattern has also been observed in cases reported by Eger et al, particularly in patients transitioning from anti-IL-5 therapy to dupilumab.[7]

The successful outcome in this case highlights the potential benefit of an early, combined therapeutic approach involving high-dose corticosteroids, mepolizumab, and IVIG for dupilumab-triggered EGPA presenting with non-severe disease but debilitating neuropathy. Previous reports of dupilumab-associated EGPA have shown variable treatment approaches and outcomes. For instance, Tanaka et al described a case requiring high doses of corticosteroids alone,

Persaud et al reported using high-dose steroids and rituximab, and Eger et al noted that patients switching from anti-IL-5 therapy required corticosteroid reintroduction and eventual, often delayed, anti-IL-5 reinitiation.[4,5,7] Our approach, initiating mepolizumab within one week of symptom onset alongside methylprednisolone pulse therapy, aligned with the 2021 ACR/VF guidelines for active, non-severe EGPA and addressed the potential for enhanced IL-5 pathway activation following dupilumab's IL-4/13 blockade.[18] This early combined intervention led to rapid clinical and laboratory improvement with relatively swift steroid tapering, contrasting with potentially more protracted courses seen with delayed anti-IL-5 therapy or conventional immunosuppression alone. While corticosteroids were crucial for the initial response, promptly targeting the IL-5 pathway might be especially beneficial when IL-4/13 blockade is a potential trigger for EGPA. Furthermore, as Descamps et al indicated, dual pathway inhibition (IL-4/13 and IL-5) might be relevant in some EGPA contexts, emphasizing careful biologic selection.[23] However, the precise contribution of mepolizumab within the first few weeks remains unclear, particularly regarding the neuropathy, given its typical onset of action, although early effects on asthma control have been reported.[24–27] Despite the initial systemic improvement, the patient experienced persistent, debilitating neuropathic pain suggestive of small-fiber neuropathy. This led to the addition of a single course of IVIG, based on the specific clinical context and evidence supporting IVIG's efficacy in EGPA-related neuropathy.[28,29] The subsequent significant improvement in neuropathic pain and achievement of complete remission support the effectiveness of the overall combined strategy. The rapid normalization of MPO-ANCA, while not used in isolation to guide treatment, further supported the positive clinical trajectory.

This single case report cannot definitively distinguish the individual contributions of corticosteroids, mepolizumab, and IVIG to the final outcome. It remains unknown whether the combination of mepolizumab and corticosteroids alone, given more time, would have eventually resolved the neuropathy. Therefore, we acknowledge the limitation that the necessity of IVIG versus the possibility of overtreatment cannot be definitively determined here. Nevertheless, our observation of a favorable outcome with early mepolizumab initiation aligns with previous reports demonstrating the effectiveness of early mepolizumab in non-dupilumab-triggered EGPA, suggesting that prompt IL-5 blockade, rather than waiting for an inadequate response to conventional therapy, might be beneficial.[30–32] Adding IVIG was considered a reasonable therapeutic option aimed at ensuring the best possible outcome in this specific situation. This case underscores the potential benefits of a tailored, multi-faceted, and early treatment approach for dupilumab-triggered EGPA. Prospective studies are crucial to confirm these observations, delineate the optimal timing and roles of each therapy, and identify patient subgroups most likely to benefit from specific combinations.

Further research is warranted to understand the risk factors for dupilumab-triggered EGPA and to establish optimal monitoring protocols. Analysis of the EudraVigilance database by Olaguibel et al identified 61 cases of EGPA associated with dupilumab therapy, representing 0.46% of all spontaneous case reports for this medication.[9] Together with published case reports, this pharmacovigilance data highlights the importance of continued surveillance and a systematic study of this phenomenon to aid inform clinical practice and improve patient outcomes.

Conclusion

This case underscores the importance of monitoring for EGPA during dupilumab therapy, particularly in MPO-ANCA positive patients. It demonstrates that a combined approach, initially using guideline-concordant mepolizumab and corticosteroids, followed by IVIG for persistent debilitating neuropathy, may lead to successful remission. While attributing improvement solely to mepolizumab is not possible in this multi-therapy context, this case highlights the potential effectiveness of this combined strategy. Further research is required to validate optimal combination therapies, including the precise role of IVIG, for dupilumab-triggered EGPA.

Ethical Approval

Given that this was a single case report, ethics committee approval was not required. Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

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Author Contributions

Shota Kaburaki: Investigation (lead); Resources (equal); Writing–Original Draft (lead). Toru Tanaka: Investigation (equal); Supervision (equal); Writing–Review & Editing (equal). Koichiro Kamio: Investigation (equal); Supervision (equal); Writing–Review & Editing (equal). Yosuke Tanaka: Investigation (equal); Writing–Review & Editing (equal). Kazuo Kasahara: Investigation (equal); Writing–Review & Editing (equal). Masahiro Seike: Investigation (equal); Resources (lead); Supervision (lead); Writing–Original Draft (supporting); Writing–Review & Editing (lead). All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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