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CASE REPORT

A Case of Eosinophilic Granulomatosis with Polyangiitis Emerging During Benralizumab Therapy: Successful Management Through a Switch to Mepolizumab Therapy

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Background: Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare systemic vasculitis characterized by eosinophilic inflammation and involvement of small- to medium-sized vessels. Given its heterogeneous clinical presentation, early diagnosis and appropriate management are critical. Although systemic corticosteroids remain the mainstay of therapy, concerns regarding their long-term adverse effects underscore the growing need for alternative treatment strategies.

Case Presentation: A 70-year-old man with a medical history of sinusitis, eosinophilic pneumonia, and bronchial asthma was referred for assessment of pleural effusion, which resolved spontaneously at the time of presentation. The patient was observed without intervention, while continuing asthma management. Approximately 6 months later, the patient experienced exacerbation of asthma and recurrence of pleural effusion. Thoracentesis was performed for symptom relief and benralizumab (30 mg) was initiated, resulting in temporary clinical improvement. However, after the third dose, the patient developed abdominal pain, peripheral neuropathy, and a skin rash. Laboratory studies revealed peripheral eosinophilia and elevated levels of rheumatoid factor (RF), myeloperoxidase–anti-neutrophil cytoplasmic antibody (MPO–ANCA), and total immunoglobulin E (IgE). A second skin biopsy revealed granulomatous inflammation, confirming the diagnosis of EGPA. Mepolizumab treatment (300 mg) was initiated, resulting in clinical resolution.

Conclusion: This case highlights that EGPA may develop during anti-IL-5 therapy. Close monitoring of clinical signs, along with biomarkers such as eosinophil count, RF, MPO–ANCA, and IgE, is essential for timely diagnosis. Furthermore, successful disease control by switching to mepolizumab monotherapy suggests the potential for managing EGPA using appropriate biologic regimens to minimize corticosteroid-related adverse effects. Further accumulation of similar cases and larger studies is warranted to inform future clinical practice.

Keywords: eosinophilic granulomatosis with polyangiitis, asthma, benralizumab, mepolizumab

Introduction

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare systemic small- to medium-sized vessel vasculitis, with an estimated prevalence of approximately 30 cases per million individuals.[1–4] The clinical course typically progresses through three distinct phases: a prodromal phase characterized by bronchial asthma or allergic rhinosinusitis; an eosinophilic phase with peripheral blood eosinophilia and organ infiltration; and a final vasculitic phase involving multi-organ damage. Clinical presentations vary widely, including mononeuritis multiplex, palpable purpura, and life-threatening complications such as myocarditis or gastrointestinal perforation. Diagnosis is established based on a combination of clinical criteria, laboratory findings (eg, myeloperoxidase-anti-neutrophil cytoplasmic antibody (MPO-ANCA) positivity in approximately 40% of cases), and histopathological evidence of necrotizing vasculitis or eosinophilic granulomas.[2–4]

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The management of EGPA traditionally relies on high-dose corticosteroids, often combined with immunosuppressive agents such as cyclophosphamide or azathioprine for severe cases.[2–4] However, the long-term morbidity associated with steroid use has necessitated the development of steroid-sparing alternatives. Mepolizumab, an anti-IL–5 monoclonal antibody, was approved in Japan in 2018 for refractory EGPA, demonstrating significant efficacy in reducing glucocorticoid requirements.[5] Similarly, benralizumab, an anti-IL-5 receptor monoclonal antibody, has shown non-inferiority to mepolizumab in achieving remission (MANDARA study) and was recently approved for EGPA in Japan in December 2024.[6]

Nevertheless, de novo EGPA development or exacerbation has been reported following the administration of anti-IL-5 or anti-IL-5 receptor antibodies for severe asthma.[7–17] These rare reports often describe cases where respiratory symptoms are well-controlled while systemic vasculitis emerges, a phenomenon sometimes attributed to the "unmasking" of underlying EGPA as systemic steroids are tapered, or to a failure of biologic agents to fully suppress the vasculitic endotype. Despite these observations, direct evidence regarding the emergence of EGPA during biologic therapy and the therapeutic impact of switching between biologic agents remains limited. Here, we report a case of EGPA that developed during benralizumab treatment, with subsequent symptom improvement following a switch to mepolizumab.

Case Presentation

A 70-year-old man presented with persistent cough and dyspnea. His medical history included pulmonary tuberculosis at the age of 36 years, bronchial asthma at 39 years, eosinophilic pneumonia at 59 years, and chronic sinusitis with anosmia for which he underwent four surgical interventions. For years X–13, he had received intermittent oral corticosteroid therapy for asthma and eosinophilic pneumonia. Beginning in years X–4, he experienced episodes of unexplained cough approximately every 1–2 months, each of which was managed with oral prednisolone (20 mg/day) for a duration of 1–2 weeks. In May of year X–2, a chest abnormality was identified on imaging, and a subsequent CT scan revealed newly developed ground-glass opacities, granular shadows, and interlobular septal thickening in the left lower lobe (Figure 1A). Left-sided pleural effusion was detected in November of the same year (Figure 1B), with peripheral blood eosinophil counts ranging from 5% to 10%. He was referred to our institution on January of year X–1 for further evaluation. At his initial visit, the left pleural effusion had resolved spontaneously (Figure 1C). Asthma management with fluticasone/formoterol (1000 μg/day), tiotropium bromide (5 μg/day), and montelukast (10 mg/day) continued. Aside from pitavastatin calcium hydrate for hyperlipidemia, the patient was not taking any other systemic medications throughout the clinical course.

In October of year X–1, he experienced dyspnea again, and right-sided pleural effusion was observed. The eosinophil count had increased to 1294 cells/μL. Pleural fluid analysis revealed neutrophils (7.5%), eosinophils (55.0%), lymphocytes (12.0%), and macrophages (22.5%), indicating significant eosinophilic predominance. Although systemic glucocorticoid therapy was considered, the presence of pleural effusion and dyspnea alone were insufficient to support the diagnosis of EGPA because no other hallmark features of systemic vasculitis, such as mononeuritis multiplex, palpable purpura, or MPO-ANCA positivity, were present at that time. Consequently, as there were no clinically apparent skin lesions or other indications for tissue biopsy, a skin biopsy was not performed at this stage. The effusion was drained, and benralizumab (30 mg) was initiated to control the asthma symptoms. This led to a reduction in peripheral blood eosinophils to 0 cells/μL and an improvement in the Asthma Control Test (ACT) score from 13 points to 22 points. However, within 1 month, pleural effusion recurred (Figure 1D). Following the third benralizumab dose, the patient developed bilateral lower limb numbness and purpuric skin lesions. Laboratory testing showed peripheral eosinophilia (3792 cells/μL), elevated rheumatoid factor (RF; 495 U/mL), and MPO-ANCA; 7.7 U/mL, raising suspicion for EGPA (Table 1 and Figure 2). The initial skin biopsy performed in December of year X–1 was non-diagnostic for EGPA, revealing no evidence of vasculitis or eosinophilic infiltration. A second skin biopsy was performed in mid-January of year X. Because the biopsy results were still pending and clinical management of his asthma symptoms was required, a fourth dose of benralizumab was administered as scheduled, 8 weeks after the third dose, without waiting for the histopathological findings. At that time, his Birmingham Vasculitis Activity Score (BVAS) was 17, with clinical manifestations including numbness and purpura in the lower extremities, myalgia, and transient hearing loss. The biopsy results later revealed granulomatous inflammation with eosinophilic infiltration of small-caliber blood vessels, confirming the diagnosis of EGPA in February of the year X (Figure 1E). Two weeks after the diagnosis was established, the patient experienced transient hematuria, accompanied by a rise in BVAS to 27 and a decline in the ACT score to 17, and the peripheral blood eosinophil count was 350 cells/μL. Given the development of EGPA during benralizumab therapy,

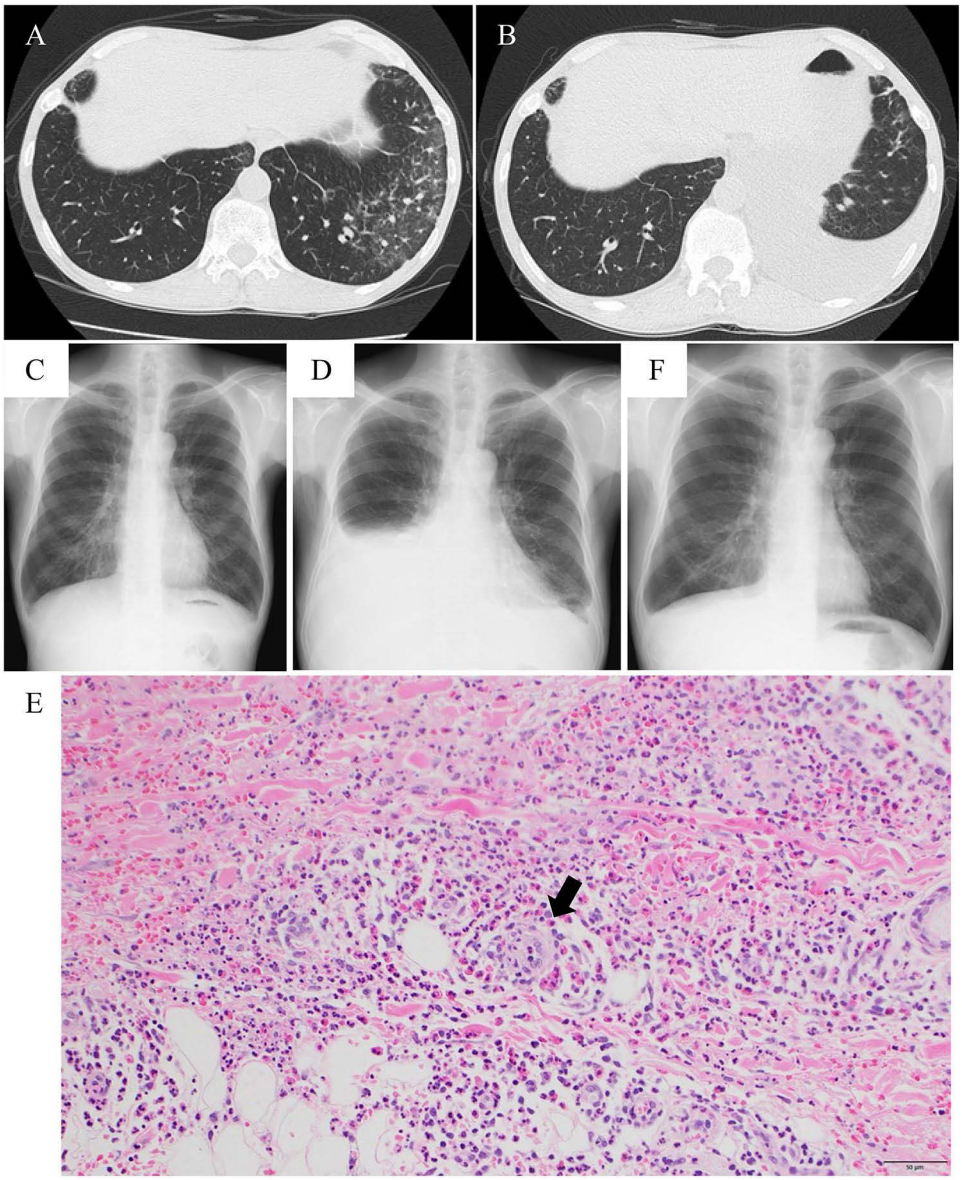

Figure 1 Radiological and histopathological evaluation. (**A**) Chest computed tomography (CT) in May of year X–2 showed new findings in the left lower lobe, including ground-glass opacity, granular infiltrates, and interlobular septal thickening. (**B**) Chest CT in November of year X–2, demonstrating left-sided pleural effusion. (**C**) Chest radiograph at initial presentation to our hospital. (**D**) A chest radiograph was obtained 1 month after the initiation of benralizumab therapy, showing the progression of right-sided pleural effusion. (**E**) Second skin biopsy revealed granulomatous inflammation of small vessels with eosinophilic infiltration. (**F**) Chest radiograph following two doses of mepolizumab.

benralizumab was discontinued and mepolizumab was initiated at a dose of 300 mg (the approved dose for EGPA), administered every 4 weeks. This decision was guided by the patient's reluctance to commence systemic corticosteroid therapy owing to concerns about adverse effects and a low peripheral blood eosinophil count. No other new medications were introduced. By the time of the second mepolizumab dose (4 weeks after the first dose), the eosinophil count had decreased to 10 cells/μL, RF had declined to 131.2 U/mL, pleural effusion had diminished (Figure 1F), and the ACT score had improved to 22, thereby avoiding the elective use of systemic corticosteroids. By the fourth dose, the eosinophil count remained at 10 cells/μL, RF had declined further to 19.1 U/mL, the pleural effusion had resolved, and the ACT score had risen to 25. The patient continued treatment with mepolizumab, and over a period exceeding 18 months, no recurrence of clinical symptoms or abnormal laboratory findings was observed except for persistent numbness in the lower extremities.

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Table 1 Laboratory Features of Patients at the Time of Diagnosis of EGPA

Hematology		Biochemistry and Serology			
WBC	9600/μL	AST	20 U/L	IgG	987 mg/dL
Neu	43.50%	ALT	14 U/L	IgG4	240 mg/dL
Ly	9%	γ-GTP	26 U/L	IgE	3260 mg/dL
Mo	7.5%	BUN	16 mg/dL	RF	495 IU/mL
Eos	39.5%	Cre	0.6 mg/dL	PR3-ANCA	2.2 U/mL
RBC	466/μL	Na	137 mEq/L	MPO-ANCA	7.7 U/mL
Hb	13.4 g/dL	K	4.6 mEq/L	sIL-2R	954 U/mL
Hct	40.8%	Cl	99 mEq/L		
Plt	31.7×10^4/μL	CRP	4.8 mg/dL		

Abbreviations: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Cl, chloride; CRE, creatinine; CRP, c-reactive protein; EGPA, eosinophilic granulomatosis with polyangiitis; γ-GTP, gamma-glutamyl transpeptidase; Hb, hemoglobin; Ht, hematocrit; IgE, immunoglobulin E; K, potassium; LD, lactate dehydrogenase; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPO-ANCA, myeloperoxidase-anti-neutrophil cytoplasmic antibody; Na, sodium; Plt, platelet count; RBC, red blood cell count; RF, rheumatoid factor; T-Bil, total bilirubin; TP, total protein; WBC, white blood cell count.

Discussion

Induction therapy for acute EGPA typically necessitates high-dose corticosteroids; however, long-term use is associated with significant morbidity, posing a barrier to optimal management when patients are reluctant to initiate steroids.[2–4] In our case, the patient's avoidance of corticosteroids led to a transition to mepolizumab (300 mg/month). Mepolizumab was, at the time, the sole biologic therapy in Japan to hold regulatory approval for the management of both asthma and EGPA. Remarkably, disease control for EGPA was achieved through mepolizumab monotherapy alone. While this successful management without elective corticosteroids reflects a specific clinical scenario driven by patient preference, it highlights a critical real-world dilemma: balancing the necessity of immediate vasculitis control with the long-term morbidity of corticosteroids. This report provides a rare case suggesting that a steroid-sparing alternative in the management of EGPA might be feasible in a subset of patients already exposed to IL-5 receptor antagonists. There is a pressing need for large-scale clinical research to establish effective, steroid-sparing alternatives in the management of EGPA.

While the MIRRA and MANDARA trials established the efficacy of mepolizumab and benralizumab, respectively, as effective glucocorticoid-sparing treatments for EGPA,[5,6] the development of EGPA during biologic therapy remains a rare but documented challenge.[7] Among the cases summarized in Table 2, our case (case 12) is unique as the only one successfully managed without elective glucocorticoids.[8–17] Among the twelve cases in Table 2, six patients did not receive glucocorticoids at the time of EGPA onset, and the median duration of benralizumab therapy was 9.0 months. And the benralizumab therapy was discontinued in all twelve cases; however, it was resumed in case 5 following the stabilization of EGPA-related symptoms.[11] Potential mechanisms underlying the development of EGPA during benralizumab therapy include the possibility of "primary failure", where the initial response is insufficient to suppress EGPA, as well as the emergence of neutralizing antibodies against benralizumab. While the prevalence of neutralizing antibodies is reported to be 7.12%.[18] In this case, the vasculitis symptoms worsened only 4 months after initiation, having received only four doses. Although the involvement of neutralizing antibodies cannot be definitively excluded, this relatively early onset and the limited number of doses—coupled with the timing of the exacerbation—raise the possibility of primary failure. Furthermore, the dosing regimen employed in this case, which followed the approved schedule for asthma,

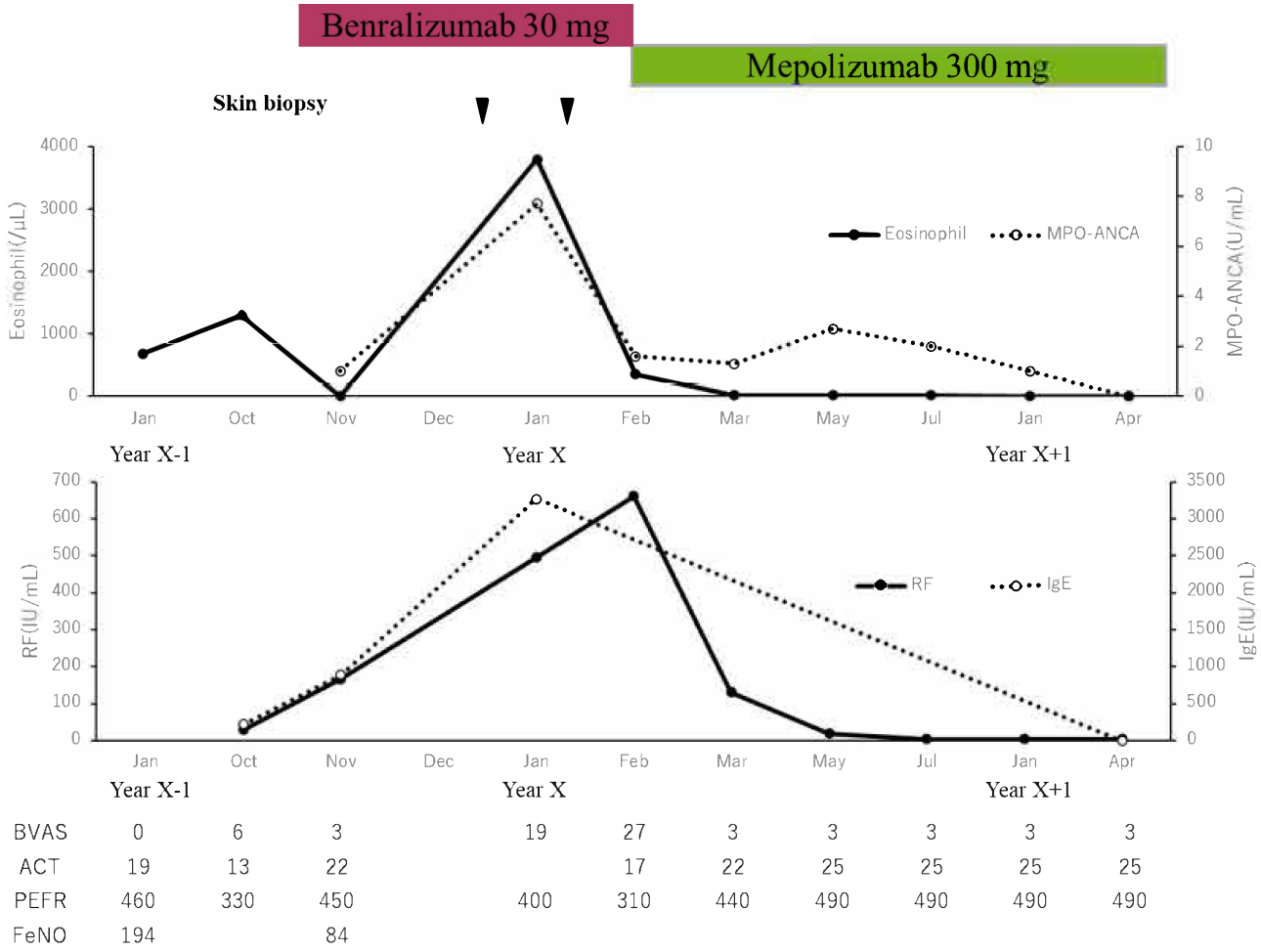

Figure 2 Schematic representation of the clinical course and laboratory trends following initiation of biologics. A timeline summarizing the patient's clinical progression and changes in key laboratory parameters after the initiation of biologic treatment.

appears to have been suboptimal for controlling the systemic vasculitic process. Benralizumab was initiated at 30 mg, with the first three doses administered at 4-week intervals. However, consistent with the asthma protocol, the fourth dose was scheduled 8 weeks after the third dose. It was during this extended 8-week interval that the eosinophil counts increased and vasculitis symptoms exacerbated. In the MANDARA study, which demonstrated the non-inferiority of benralizumab to mepolizumab in EGPA, benralizumab was administered at 30 mg every 4 weeks throughout the study period.[6] These findings suggest that while an 8-week dosing interval is effective for managing asthma, it may be insufficient to maintain disease control for EGPA. This clinical course underscores the complexity of managing EGPA that emerges during anti-IL-5 therapy and provides evidence that the 4-week interval, as utilized in EGPA-specific protocols, is likely essential for preventing systemic flares.

Importantly, the development of EGPA during biologic therapy is not limited to benralizumab. Evidence comparing different biologics suggests no significant differences in clinical parameters—such as age, asthma duration, or the timing of EGPA onset relative to treatment initiation—between patients receiving anti-IL-5 mAbs (including mepolizumab) and those receiving other biologics like omalizumab or dupilumab.[7] This suggests that the emergence of systemic vasculitis despite biologic treatment for asthma is a phenomenon that can occur across different agents, reflecting the potential for these therapies to adequately control the asthma phenotype while failing to suppress the underlying vasculitic process.

The development of EGPA is often accompanied by peripheral eosinophilia and elevated CRP levels, as well as a marked increase in serum total IgE, emergence of RF positivity, and thrombocytosis. When these clinical and laboratory parameters increase concurrently with disease symptoms, the diagnosis of EGPA is relatively

Table 2 Twelve Cases of Newly Diagnosed EGPA Occurring During Benralizumab Treatment

Cases	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
Age (y)	28	58	55	31	55	62	65	79	60	81	65	70
Sex (M/F)	M	F	M	M	M	F	F	M	F	F	F	M
Duration* (m)	9	6	24	7	29	10	20	4	18	6	20	4
Asthma	+	+	+	+	+	+	+	+	+	+	+	+
EP	–	–	–	–	–	+	–	–	+	+	+	+
Sinusitis	+	+	+	–	–	–	+	–	–	–	+	+
EOM	–	–	–	–	–	–	–	–	+	–	–	–
Purpura	–	–	–	–	–	–	–	+	–	–	–	+
OCS[†] (PSL equivalent dose, mg)	–	–	–	10	–	8	–	2	5	0.5	–	–
CRP (mg/dL)	6.7	3.7	NA	NA	10.2	10.3	1.8	11	7.4	4.0	1.8	4.8
MPO-ANCA	Positive	–	Positive	Positive	Positive	–	–	–	NA	–[‡]	–	Positive
Eosinophils (cells/μL)	1310	1410	2800	4600	5460	0	0	6450	31,919	4974	0	3792
RF (U/mL)	NA	NA	NA	–	NA	NA	NA	240	NA	NA	NA	495
Total IgE (IU/mL)	NA	NA	NA	3914	NA	4991	750.2	4841	5140	22615	NA	3260
Treatment												
Glucocorticoids	+	+	+	+	+	+	+	+	+	+	+	–
IVMP	–	–	+	+	–	+	+	–	–	+	–	–
Immunosuppressants	IVCY	AZP	–	–	IVCY	IVCY	IVCY	–	–	IVCY, AZP	IVCY	–
Rituximab	+	–	+	+	+	–	–	–	–	–	–	–
Mepolizumb	–	–	–	–	–	+	–	+	+	+	–	+
Outcome	Improved	Improved	Improved	Improved	Improved	Improved	Improved	Improved	Improved	Improved	Unknown	Improved
References	8	8	9	10	11	12	13	14	15	16	17	Present case

Notes: *Duration of benralizumab treatment, [†]Daily dose of oral corticosteroid, [‡]PR3-ANCA (proteinase 3-myeloperoxidase-anti-neutrophil cytoplasmic antibody) 5.2 IU/mL.

Abbreviations: AZP, azathiopurine; CRP, c-reactive protein; EGPA, eosinophilic granulomatosis with polyangiitis; EOM, Eosinophilic otitis media; EP, eosinophilic pneumonia; IgE, immunoglobulin E; IVCY, intravenous cyclophosphamide; IVMP, intravenous methylprednisolone; MPO-ANCA, myeloperoxidase-anti-neutrophil cytoplasmic antibody; NA, not applicable; OCS, oral corticosteroid; PSL, prednisolone; RF, rheumatoid factor.

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straightforward. However, as illustrated in the present case, when treatment with IL–5–targeted biologic agents is employed, peripheral eosinophil counts may be suppressed and elevations in other biomarkers may be attenuated, thereby complicating the identification of EGPA onset or exacerbation. In the present case, the peripheral eosinophil count decreased to 0/μL following the initial administration of benralizumab, despite concurrent increases in serum IgE and RF levels. Diagnostic histopathological findings, such as eosinophilic infiltration and granuloma formation, were only observed on the second skin biopsy performed after peripheral blood eosinophils and MPO–ANCA levels began to increase. At the time of transition to mepolizumab therapy, the peripheral eosinophil count had declined; however, the RF level remained elevated. Given that peripheral eosinophil counts decline rapidly after treatment, they may not reliably reflect EGPA disease activity in post-treatment settings. Furthermore, in Table 2, cases 6, 7, and 11 were MPO-ANCA negative, showing zero peripheral eosinophils and histopathology revealing necrotizing vasculitis without eosinophilic infiltration.[12,13,17] These cases suggest that benralizumab may exert a potent "masking effect" by reducing eosinophil counts even in the presence of active vasculitis, potentially complicating the clinical identification of disease flares. Consequently, when IL-5 inhibitors are employed, peripheral eosinophil counts may not reliably reflect disease activity. This case underscores the critical importance of monitoring a comprehensive panel of biomarkers, such as RF and MPO-ANCA, in combination with clinical evaluation to accurately assess EGPA disease activity in post-treatment settings.

Conclusion

The present case illustrates the potential for EGPA to develop during benralizumab therapy for asthma and underscores the importance of comprehensive monitoring at the time of EGPA onset. Importantly, clinical remission and successful management were achieved by switching to mepolizumab (300 mg/month) monotherapy, highlighting the effectiveness of appropriate anti-IL-5 regimens in achieving disease control without conventional corticosteroid therapy. Our findings suggest that systemic vasculitis management requires rigorous monitoring of both clinical manifestations and laboratory markers, such as eosinophil counts, RF, MPO-ANCA, and total IgE levels. Furthermore, the ability to control EGPA using biologics alone raises the possibility of minimizing corticosteroid-related adverse effects. However, large-scale studies and further accumulation of similar cases are warranted to translate these findings into clinical practice.

Abbreviations

ACT, Asthma Control Test; BVAS, Birmingham Vasculitis Activity Score; CRP, C-reactive protein; EGPA, Eosinophilic Granulomatosis with Polyangiitis; IgE, immunoglobulin E; IL-5, Interleukin-5; MPO-ANCA, myeloperoxidase-anti-neutrophil cytoplasmic antibody; RF, rheumatoid factor.

Data Sharing Statement

Data sharing is not applicable to this article, as no datasets were generated or analyzed in this case report.

Ethics Approval and Consent to Participate

In accordance with the guidelines of the Juntendo University Research Ethics Committee (Tokyo, Japan), this case report was exempt from ethical review because it contains no identifiable patient information and poses no foreseeable risk to the patient.

Consent for Publication

Consent for publication was obtained from the patient.

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