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CASE SERIES

Severe Dry Cough Followed by Eosinophilia Without Pulmonary Infiltrates After Dupilumab Treatment in Patients with Severe Asthma: A Series of Five Cases

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Purpose: Dupilumab is known to cause transient, asymptomatic eosinophilia, but in rare cases it can present as eosinophilic pneumonia accompanied by cough. This study reports a distinct manifestation of dupilumab-related eosinophilia presenting as a severe dry cough despite the absence of radiological or clinical evidence of eosinophilic pneumonia.

Patients and Methods: A retrospective analysis of clinical data from patients with severe eosinophilic asthma (SEA) treated with dupilumab at the Guangzhou Institute of Respiratory Disease between August 2020 and February 2025 was conducted. Data on clinical manifestations, objective indicators and subjective questionnaires were collected at four time-points: before dupilumab initiation, preceding dupilumab-related cough, during the cough episode, and after cough resolution.

Results: Five patients treated with dupilumab (three males and two females with a mean age of 65.6 ± 9.2 years) developed a severe dry cough and marked eosinophilia (1,130–4,050 cells/μL) without radiologic or clinical evidence of eosinophilic pneumonia. The cough began after one to eleven dupilumab courses. Despite the increase in eosinophils, all patients achieved their personal best FEV1 versus baseline. The symptoms resolved with a short course of systemic corticosteroids (in four patients) or antitussive therapy alone (in one patient).

Conclusion: Dupilumab-related eosinophilia can manifest as a severe, steroid-responsive coughing despite the absence of parenchymal eosinophilic disease.

Keywords: severe eosinophilic asthma, dupilumab, adverse effect, severe dry cough

Introduction

Dupilumab, a humanized anti-interleukin-4 (IL-4) monoclonal antibody, has been shown to be effective in the treatment of severe eosinophilic asthma (SEA), atopic dermatitis (AD), chronic rhhinitis with nasal polyps (CRSwNP), eosinophilic esophagitis (EoE) and prurigo nodularis.[1] Dupilumab has a good safety profile and no serious adverse events have been reported. The most common adverse reactions include temporary injection site reactions, ocular surface reactions, nasopharyngitis, oral herpes, headache and other herpes complex virus infections.[2] Transient eosinophilia (>1% incidence) is also common and usually asymptomatic. However, there have been reports of rare cases of serious eosinophilic complications of severe hypereosinophilic syndrome (HES), eosinophilic granulomatosis with polyangiitis (EGPA), or eosinophilic pneumonia after dupilumab treatment.[3]

Eosinophilia, defined as a peripheral blood eosinophil count of ≥500 cells/ul, was observed more frequently in patients with eosinophilic airway inflammation than in healthy individuals.[4] This inflammation is present in 30–50% of

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patients with chronic cough,[5] particularly those with asthma, non-asthmatic eosinophilic bronchitis (NAEB) and upper airway cough syndrome(UAS). The mechanism of cough accompanied by eosinophilia should be addressed in terms of type-2 immunity-mediated inflammatory networks,[6] which respond well to corticosteroids or anti-IL-5 biologics (eg, mepolizumab). However, the cough severity of some patients with asthma did not improve after specific attenuation of eosinophils, indicating that other potential mechanisms, such as mast cell neuronal interactions,[7] might be involved.

Cases of dupilumab-related eosinophilia causing cough have been reported only sporadically. Some cases were associated with eosinophilic pneumonia,[8] while in others, cough was merely listed as an adverse event without further detail.[9,10] Here, we present five patients who developed a new-onset severe dry cough concomitant with dupilumab-related eosinophilia in the absence of eosinophilic pneumonia or any other identifiable cause. It raises awareness among clinicians about this potential adverse reaction in patients treated with dupilumab.

Materials and Methods

Patients

This study retrospectively analyzed adults with severe asthma who started dupilumab at the Guangzhou Institute of Respiratory Disease between August 2020 and September 2025. Severe asthma was defined as disease that remained uncontrolled despite daily high-dose inhaled corticosteroids (ICS) plus at least one additional controller medication (long-acting β2-agonist (LABA), long-acting muscarinic antagonist (LAMA), leukotriene-receptor antagonist, or systemic glucocorticoid), after excluding or optimizing reversible factors such as ongoing allergen exposure, poor adherence and incorrect inhaler technique. Our study was approved by the Ethics Committee of the First Affiliated Hospital of Guangzhou Medical University (Ethics registration number ES-2023-114-01), which also authorized the publication of case details. Written informed consent was obtained from all patients for their information to be published.

Data Collection

Data were collected at four predefined time-points: before dupilumab initiation (baseline before dupilumab), before dupilumab-related cough onset (before cough), during the cough episodes (cough), and after cough resolution (after cough). At each time-point we recorded prior medication regimens and measured blood eosinophil counts (BEC), total immunoglobulin E (tIgE), fractional exhaled nitric oxide (FeNO, single-breath on-line chemiluminescence, 50 mL/s plateau, ppb) and forced expiratory volume in one second (FEV1). Sputum eosinophils (SEC) were quantified after induction with 3–4% saline, dithiothreitol processing, filtration, centrifugation and cytospin differential staining. Patients also completed a 0–100 mm visual-analogue cough scale (VAS, 0 = no cough, 100 = worst imaginable) and the 5-item Asthma Control Test (ACT, 5–25, higher = better control).

Results

A total of 156 patients received dupilumab for more than three months and were included in the cohort. Of these patients, 68 developed eosinophilia (peripheral blood eosinophil count of ≥500 cells/ul), including five patients who experienced severe dry cough coincided with eosinophilia. These five patients (3 males, 2 females, mean age 65.6±9.2 years) constitute the study cohort. Baseline demographics and clinical characteristics are summarized in Table 1. Individual serial measurements are presented in Figure 1, and imaging data are shown in Figure 2.

Patient 1

A 71-year-old man presented with a seven-year history of recurrent cough, expectoration, and wheezing. Despite receiving high-dose of ICS (Beclometasone, 400 ug/day) +LABA+LAMA and montelukast (10 mg/day), he requiring oral corticosteroids (OCS, prednisone 5–20 mg/day) and experienced recurrent acute exacerbations need hospitalization. He was diagnosed with allergic late-onset SEA, CRSwNP and chronic obstructive pulmonary disease (COPD).The highest historical BEC before biologic therapy was 2020 cells/ul (22.3%), FeNO level of 248 ppb and tIgE of 2343 IU/mL (dust mite allergen-specific immunoglobulin E (sIgE) was positive). Due to persistently poor symptom control and frequent exacerbations, he underwent 12 courses of omalizumab, followed by two courses of mepolizumab, neither

Table 1 Baseline Patient Demographics

	All	N1	N2	N3	N4	N5
Age (years)	65.6±9.2	71	65	59	55	78
Sex (M/F)	N/A	M	M	F	F	M
Diagnosis	N/A	SEA	SEA	SEA	SEA	SEA
Comorbidities	N/A	CRSwNP,COPD	CRS	CRS,AD	AR,CRSwNP	CRSwNP,COPD
Atopy	N/A	Dust mite	–	–	Dust mite	–
Biologics	N/A	Oma→Mepo→Dupi	Mepo→Dupi	Dupi→Mepo→Dupi +Mepo	Dupi	Dupi
Total treatment duration with dupilumab (Course)	16±9	2	16	27	12	21
Onset of cough after dupilumab initiation (Course)	5±4	1	8	3	4	11
Onset of cough after dupilumab injection (Day)	14±10	30	8	13	4	14
Dupilumab Continued (Y/N)	N/A	No	Yes	Yes	No	Yes
Treatment for cough	N/A	Intravenous methylprednisolone 40 mg for 3 days	Intravenous methylprednisolone 40 mg for 1 day	Oral cough medicine for 7 days	Oral prednisone 15 mg/day for 3 days	Oral prednisone 20 mg/day for 3 days
Clinical outcome	N/A	Remission	Remission	Remission	Remission	Remission
Steroid use						
Before dupilumab initiation	N/A	Beclometasone 400 ug/day + Prednisone 5–20 mg/day	Beclometasone 400 ug/day + Prednisone 10 mg/day	Fluticasone 500 ug/day + Prednisone 10 mg/day	Budesonide 800 ug/day	Budesonide 800 ug/day + Prednisone 5–10 mg/day
During dupilumab treatment	N/A	Beclometasone 400 ug/day	Beclometasone 400 ug/day	Fluticasone 500 ug/day	Budesonide 800 ug/day	Budesonide 800 ug/day
At the time of eosinophilia and cough incidence	N/A	Beclometasone 400 ug/day	Beclometasone 400 ug/day	Fluticasone 500 ug/day	Budesonide 800 ug/day	Budesonide 800 ug/day
After cough	N/A	Beclometasone 400 ug/day + Prednisone 5-20mg	Beclometasone 400 ug/day	Fluticasone 500 ug/day	Budesonide 800 ug/day	Budesonide 800 ug/day

Abbreviations: AR, allergic rhinitis; AD, atopic dermatitis; COPD, chronic obstructive pulmonary disease; CRS, chronic rhinosinusitis; CRSwNP, chronic rhinosinusitis with nasal polyp; SEA, severe eosinophilic asthma; N, no; N/A, not available; Y, yes. Dupi, dupilumab; Mepo, mepolizumab.

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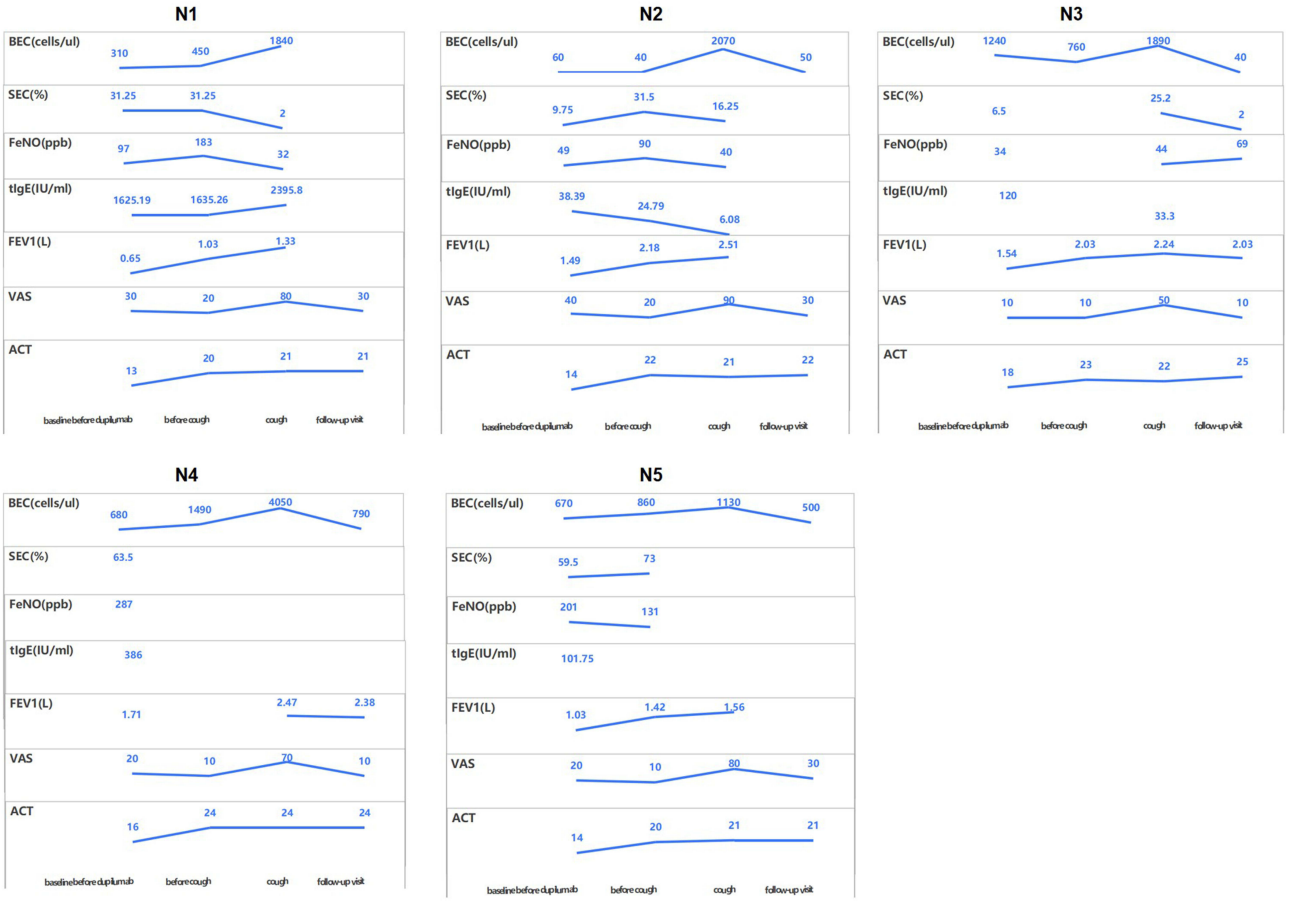

Figure 1 Demographics and clinical characteristics of patients at baseline and after dupilumab therapy.
Abbreviations: ACT, asthma control test; BEC, blood eosinophil count; FeNO, fractional exhaled nitric oxide; FEVl, forced expiratory volume in 1 second; SEC:sputum eosinophil count;tIgE, total immunoglobulin E; VAS, visual Analogue Scale.

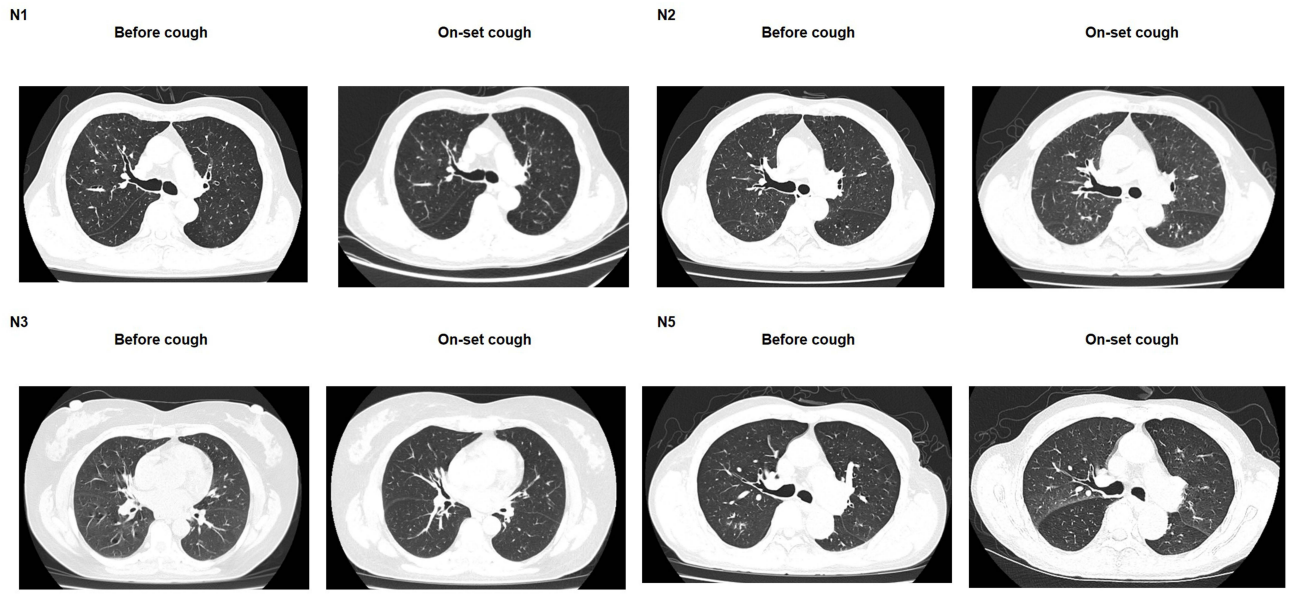

Figure 2 Imaging data of the case series.

treatment improved symptom control or reduced exacerbation frequency. In view of his severe airflow limitation, recurrent exacerbations, and dependence on OCS, dupilumab was initiated in October 2024. Before dupilumab treatment, his BEC was 450 cells/ul (7.2%) with SEC of 31.25%, FeNO of 183 ppb, tIgE of 1635.26 IU/mL and FEV1 was 0.65L (FEV1% pre 31.14%). However, one month after starting dupilumab therapy, he developed a severe dry cough and severe headache without obvious inducement. Computed tomography (CT) scans of head, sinus and chest revealed no significant new lesions. His BEC level reached 1840 cells/μL, which is significantly higher than the BEC level of only 450 cells/μL before dupilumab therapy. Interestingly, his other type 2 inflammation markers were not elevated, with SEC of 2%, FeNO of 32 ppb and tIgE of 2395.8 IU/mL. Meanwhile, his FEV1 did not deteriorate with the increase in eosinophil levels, but instead reached his personal best of 1.33L (FEV1% pre 71%). Celecoxib and compound methoxyphenamine hydrochloride capsules did not significantly relieve headache and cough. After an intravenous injection of methylprednisolone 40 mg for 3 days, his blood eosinophil level returned to the normal range and his symptoms of headache and cough were significantly relieved. After that, the patient unwilling to continue dupilumab treatment. His symptoms were controlled by high-dose of ICS (Beclometasone, 400 ug/day)+LABA+LAMA and OCS (prednisone 5–20 mg/day), and his BEC levels fluctuated between 60 cells/ul (0.5%) and 310 cells/μL (3.3%).

Patient 2

A 65-year-old man presented with a 30-year history of cough, expectoration and recurrent wheezing. Despite maximal therapy with a high-dose of ICS (Beclometasone, 400 ug/day), LABA+LAMA and montelukast (10 mg/day), his symptoms were poor controlled and need for repeated OCS (prednisone 10mg) treatment. He was diagnosed with non-allergic late-onset SEA. Given his poor symptom control and OCS dependence, he started treatment with mepolizumab in December 2023. Prior to biologic therapy, his BEC was 1650 cells/ul (19.1%) with SEC of 39%, FeNO of 69 ppb, tIgE of 29.32 IU/mL (inhalant allergen-sIgE was positive) and FEV1 of 1.58 L (FEV1% pre 56%). However, after four courses of mepolizumab (100 mg, q4w), his asthma symptoms remained only partially controlled with BEC of 60 cells/ul, SEC of 9.75%, FeNO of 49 ppb, tIgE of 38.39 ppb and FEV1 of 1.49L (FEV1% pre 53.14%). In May 2024, his treatment was switched to dupilumab (initially 600 mg, followed by 300 mg, q4w), which led to stabilization of his asthma symptoms. In January 2025, 8 days after completing the 8th course treatment of dupilumab, he developed a severe dry cough without obvious inducement accompanied by BEC of 2070 cells/ul (24.6%), SEC of 16.25%, FeNO of 40 ppb, tIgE of 6.08 IU/mL and FEV1 of 2.51 L (FEV1% pre of 90.11%). It is worth noting that the FEV1 value was his personal best result. The chest CT scan showed no obvious exudative lesions. After excluding infections and heart failure as potential causes of the cough, we considered that the patient's cough might be related to the elevated eosinophil levels associated with dupilumab treatment. The day after receiving an intravenous injection of 40 mg of methylprednisolone, the patient's cough symptoms were significantly reduced and the BEC decreased to 50 cells/ul. The patient continued receiving dupilumab (300 mg q4w) treatment, with blood eosinophil levels fluctuating between 1460 cells/ul (21.2%) and 1690 cells/μL (20.6%). His symptoms remained stable, and there were no further episodes of severe coughing.

Patient 3

A 59-year-old woman presented with a 13-year history of recurrent wheezing. Despite high-does ICS (Budesonide, 800 ug/day), LABA+LAMA, montelukast (10 mg/day) and OCS (prednisone 20 mg/day), her symptoms remained uncontrolled. She was diagnosed with non-allergic, late-onset SEA, CRS, and atopic dermatitis (AD). Her baseline BEC was 1240 cells/ul (18.3%), with a SEC of 6.5%, FeNO of 34 ppb, tIgE of 120 IU/mL and FEV1 of 2.03 L (FEV1% pre 92.5%). Because of her AD, poorly controlled asthma, and adverse effects of OCS, she began dupilumab (600 mg q2w, followed by 300 mg q2w) in 2022. The treatment significantly improved her asthma symptoms and allowed her to discontinue OCS. However, after 13 days of completing the 3th course of treatment, she developed a severe dry cough without obvious trigger and her BEC increased to 1890 cells/ul (25.2%). A chest-X-ray showed no significant exudative lesions. Notably, the elevated eosinophil levels did not worsen her FEV1. After taking oral compound methoxyamine and other cough medicine for seven days, her cough symptoms were improved. She continued to receive dupilumab treatment. However, her BEC level was fluctuating between 1170–1570 cells/ul with monthly tests between July 2022

and March 2023. Meanwhile, she experienced occasional mild coughing episodes. Switching to mepolizumab alone did not control her symptoms effectively. Finally, she was started on combination therapy with dupilumab and mepolizumab. One month after starting the combination therapy, the patient's blood eosinophil levels returned to the normal and her cough symptoms were significantly alleviated.

Patient 4

A 55-year-old woman presented with a 3-year history of recurrent cough, wheezing and dyspnoea. Despite receiving high-dose ICS (Budesonide, 800 ug/day), LABA+LAMA and montelukast (10 mg/day), she continued to have a high symptom burden. She was diagnosed with allergic late-onset SEA, allergic rhinitis (AR) and CRSwNP. Her baseline BEC was 680 cells/ul (10.5%), with a SEC of 63.5%, FeNO of 287 ppb, tIgE of 386 IU/mL (inhalant allergen-sIgE was positive) and FEV1 of 1.71 L (FEV1% pre 71.68%). She was started on dupilumab therapy in February 2023 due to her poorly controlled asthma, which was complicated by CRSwNP, markedly elevated FeNO. After two courses of dupilumab treatment, her symptoms stabilized, although BEC mildly elevated to 1490 cells/ul (16.8%). After 4 days of completing 4th course of dupilumab treatment, she experienced a severe dry cough without obvious inducement. Potential causes such as infection and heart failure were excluded. A chest-X-ray revealed no significant exudative lesions. Her BEC peaked at 4050 cells/ul (34.9%). However, her lung function tests showed a personal best FEV1 of 2.47 L (FEV1% pre of 98.64%). After taking oral prednisone (15 mg/day) for three days, her cough symptoms were significantly alleviated. She continued on dupilumab treatment without further corticosteroid therapy. During the subsequent dupilumab treatment, her symptoms remained under control, although a mild increase in blood eosinophils persisted. In September 2024, her BEC was 1110 cells/ul (12.6%).

Patient 5

A 78-year-old man presented with a 4-year history of recurrent cough, expectoration and wheezing. Despite treatment with high-dose ICS (Budesonide, 800 ug/day) +LABA+LAMA, montelukast (10 mg/day) and long-term OCS (prednisone 5–10 mg/day), his asthma symptoms remained poorly controlled. He was diagnosed with non-allergic late-onset SEA, CRSwNP and COPD. His baseline BEC was 670 cells/ul (12.5%), with a SEC of 59.5%, FeNO of 201 ppb, tIgE of 101.75 IU/mL (sIgE was negative) and FEV1 of 1.03 L (FEV1%pre 41.04%). Given his advanced age, the presence of asthma-COPD overlap, and severe airflow limitation, dupilumab therapy was recommended in February 2024. The patient's monthly blood tests showed that his BEC level was mildly elevated, fluctuating between 860 (14.8%) and 1310 (21.5%) cells/μL, but his asthma symptoms remain stable In February 2025, after 14 days of completing 11th course of dupilumab treatment, he experienced a severe dry cough without obvious inducement with an elevated BEC of 1130 cells/ul (22.9%). A chest-X-ray revealed no significant exudative lesions and lung function tests showed a personal best FEV1 of 1.56 L (FEV1% pre of 65%). Despite trying various cough medications, the treatment was ineffective. After taking OCS (prednisone 20 mg/day) for three days, his eosinophil count decreased to 620 cells/ul (14.6%), and his cough symptoms gradually improved, with lung function remaining stable and not worsening. He continued to receive two courses of dupilumab and his asthma symptoms were well controlled without the further need for OCS. His most recent complete blood count in March 2025 showed an eosinophil count of 500 cells/ul (8.7%).

Discussion

This study shows that dupilumab-related eosinophilia can cause severe dry cough despite there being no radiological or clinical evidence of eosinophilic pneumonia. Although peripheral eosinophilia is closely associated with lung function decline, our patients experienced no deterioration and achieved their best FEV1 compared to their pre-dupilumab baseline. The eosinophilic cough did not worsen the underlying condition and responded well to corticosteroids. Continued dupilumab therapy was well tolerated, with no recurrence of cough.

In this series, all patients met the criteria for severe eosinophilic asthma (SEA), which is defined as an elevated peripheral eosinophil count of ≥150 cells/ul, sputum eosinophils of ≥2.5%, or FeNO of ≥20 ppb despite receiving high-dose ICS/LABA/LAMA, montelukast and OCS. Symptoms remained uncontrolled, prompting the initiation of biologic therapy. Patients 1 and 5 started dupilumab treatment due to frequent exacerbations, severe airway limitation and OCS

dependence. Patient 2 switched from mepolizumab to dupilumab due to suboptimal control of symptoms. Patient 3 had concomitant AD. Patient 4 presented with a complicated by CRSwNP and markedly elevated FeNO level of 287 ppb. After one to eleven courses of dupilumab treatment, each patient developed a severe dry cough accompanied by marked eosinophilia (1130–4050 cells/μL).

Transient eosinophilia has been reported in 4.1% to 14% of patients treated with dupilumab in randomized controlled trials[11,12] and at a higher rate in real-world studies (up to 58.7%).[13] However, in some patients, it can persist for more than six months.[14] Our previous study showed that BEC began to rise in the first month after dupilumab treatment started, peaked at 4 to 5 months, declined by 6 months, and had returned to baseline by 12 months.[3] A retrospective Italian multicentre study of 18 patients with severe-asthma found eosinophil rises in 5 patients (27.8%) at 3 months, 4 patients (22.2%) at 6 months and 2 patients (11.1%) at 12 months post-treatment initiation.[14] In the present series, peak BEC elevation was observed at 1, 3 and 4 months in N1, N3 and N4, respectively, and at 8 months in N2 and 11 months in N5 after dupilumab initiation. The potential mechanisms may be related to dupilumab blocking the IL-4/IL-13 pathway, which suppresses the migration of eosinophils from blood to peripheral tissues. Steroid tapering may also exacerbate the increase.

Dupilumab-related eosinophilia is usually asymptomatic or mild and does not affect the efficacy of treatment, discontinuing dupilumab treatment is rarely necessary.[3,10] A post hoc analysis of 11 dupilumab clinical trials revealed that seven out of 4,666 patients treated with dupilumab exhibited clinical symptoms, including six cases of EGPA.[10] Data from the FAERS (FDA Adverse Event Reporting System) showed 218 reports of eosinophilc adverse reactions attributed to dupilumab, the most frequently reported reactions were EGPA and respiratory tract eosinophilic complications (eg, acute and chronic eosinophilic pneumonia, eosinophilic pleural effusion and eosinophilic bronchitis).[15]

Severe dry cough attribute to dupilumab-related eosinophilia is rare. In most patients, dupilumab improves cough by suppressing airway inflammation and reducing mucus plugs.[16] The TRAVERSE extension study recorded an cough incidence of only 0.3 per 100 person-years.[17] In clinical trials tabulations, “cough” is simply listed as an adverse event without further detail.[17,18] When severe or persistent, cough is usually part of eosinophilic pneumonia rather than an isolated symptom.[19] In this series, five patients receiving dupilumab developed severe dry cough without an identifiable trigger, accompanied by significantly increased eosinophil levels (1130–4050 cells/ul). Cough onset occurred as early as one course and ranged from one to eleven courses after initiation. Chest imaging results were essentially normal in all five patients and their severe cough could not be explained by acute asthma attack, infection, heart failure or other potential causes. Paradoxically, other type 2 markers remained stable, in two patients (N1 and N2) FeNO and SEC even declined, while lung function reached personal best levels.

In cases involving cough, peripheral eosinophilia and preserved FEV1, the diagnosis of eosinophilic bronchitis (EB) should be considered. Although EB is classically defined by sputum eosinophilia, some sufferers also have elevated blood eosinophils.[20] EB is a significant cause of cough and is defined as ≥3% sputum eosinophils, normal chest imaging and the absence of variable airflow obstruction or airway hyper-responsiveness (AHR) which are features of asthma.[21] Although both EB and asthma involve airway eosinophilia, EB preserves lung function because the inflammation is confined to the superficial mucosa. It spares the airway smooth muscle and produces no mast-cell-driven bronchial hyper-responsiveness. Therefore, bronchoconstriction and remodeling, which account for the reversible obstruction seen in asthma, are avoided.[22] Our five patients had SEC>3%(range 6.5–63.5%) at baseline of dupilumab treatment. When severe dry cough developed during treatment, blood eosinophil levels increased significantly, changes in SEC were inconsistent: SEC fell markedly in N1 and N4, increased in N2, and was unavailable for N3 and N5. Without histology, we can only speculate that the difference was due to the eosinophilic state of dupilumab-related eosinophilia.

The eosinophil is a fully differentiated granulocyte that migrates from the bloodstream to peripheral tissues after complete maturation in the bone marrow. Eosinophils are usually present in small numbers under normal conditions, but elevated levels of eosinophils in peripheral blood or certain tissues usually indicate an underlying pathological process, such as infectious, inflammatory and allergic processes. Elevated eosinophils in the respiratory system cause a variety of short-term symptoms (such as cough, wheezing, shortness of breath, and fever) and long-term sequelae (such as asthma, bronchiectasis, or even respiratory failure).[23] Eosinophil activation is closely associated to the severity of clinical symptoms.[24] The levels of cell-surface proteins, particularly the expression levels and configuration of Fc-receptors

and integrins, help differentiate between the primed, active, and refractory states of eosinophils.[25] Upon allergen challenge, eosinophils are primed and continuously migrate into the airways and allergen-challenged lung tissue.[25] In SEA, high numbers of eosinophils were detected in the airway wall and lung tissue, presenting in different states including resting, active and degranulated apoptotic cells.[26] The specific activation status of the eosinophilia induced by dupilumab-whether they are refractory, primed or fully activated-remains unclear. Whether alternative mechanisms underlie drug-induced cough without bronchospasm remains to be further explored.[27]

Additionally, blood eosinophil levels are closely associated with lung function. Hong et al showed a significant association between baseline blood eosinophil count and lung function loss in a cohort of 629784 healthy participants.[28] Park et al identified an elevated blood eosinophil count as an independent risk factor for development of obstructive lung disease such as COPD.[29] Mogensen et al showed that elevated blood eosinophil levels are associated with accelerated FEV1 decline in patients with asthma.[30] Interestingly, in our study, dupilumab-related eosinophilia did not appear to impair lung function; instead, it was associated with the best-achieved FEV1. This may be attribute to dupilumab's suppression of type2 inflammation, reduction of mucus plugs, and limitation of airway remodeling.

Currently, there is no established consensus on how to manage dupilumab-related eosinophilia. Asymptomatic patients can continue therapy under regular blood eosinophil monitoring. Mild to moderate complications (eg, eosinophilic pneumonia) are treated with systemic glucocorticoids and dupilumab can be resumed when resolved. Severe or persistent organ damage (eg, EGPA) requires stopping dupilumab, treating the complication and excluding other underlying disorders. The decision on whether to switch an IL-5 inhibitor or combine dual biologics is then reassessed.[31] In our cohort, severe persistent dry cough significantly disrupted patients' daily lives. Short-term glucocorticoids use is effective in rapidly alleviating symptoms. Except for one patient who declined to resume dupilumab, the other four patients did not experience recurrent severe dry cough during the subsequent dupilumab treatment.

Limitation

Our study has several limitations. Firstly, the retrospective design and a small sample size limit the generalisability of our findings. Furthermore, histopathological examination was not performed to confirm the presence of eosinophilic infiltration of the airway mucosa, which may have contributed to the severe dry cough observed. The results may also be confined to particular regions and ethnic populations. Moreover, long-term follow-up data are required to assess disease progression more fully over time.

Conclusion

Dupilumab-induced severe dry cough maybe one of the less commonly recognized adverse reactions. Systemic glucocorticoids can rapidly alleviate the symptom and the occurrence of the adverse reaction does not worsen the underlying disease condition. Clinicians should maintain a high index of suspicion to recognize and effectively manage this phenomenon.

Abbreviations

ACT, asthma control test; AD, atopic dermatitis; AR, allergic rhinitis; BEC, blood eosinophil counts; COPD, chronic obstructive pulmonary disease; CRS, chronic rhinosinusitis; CRSwNP, chronic rhinosinusitis with nasal polyps; CT, Computed tomography; EGPA, eosinophilic granulomatous vasculitis; FeNO, fractional exhaled nitric oxide; FEV1, forced expiratory volume in one second; ICS, inhaled corticosteroids; IgE, immunoglobulin E; LABA, long-acting beta-agonist; LAMA, long-acting antimuscarinic antagonist; OCS, oral corticosteroid; SEA, severe eosinophilic asthma; SEC, sputum eosinophil counts; sIgE, specific immunoglobulin E; tIgE, total immunoglobulin E; VAS, visual analogue scale.

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Disclosure

The authors report no conflicts of interest in this work.

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