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Concurrent tropical pulmonary eosinophilia and allergic bronchopulmonary aspergillosis in asthma: a prospective observational study from a filariasis-endemic region

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Abstract

Tropical pulmonary eosinophilia (TPE) and allergic bronchopulmonary aspergillosis (ABPA) share cough, wheeze, eosinophilia, and marked immunoglobulin (Ig) E elevation and may coexist in patients with asthma in filariasis-endemic settings. We prospectively characterized this overlap phenotype at a tertiary respiratory center in eastern India. Eligible patients older than 10 years had a documented prior physician diagnosis of asthma and, at presentation, an absolute eosinophil count (AEC) >1000 cells/μL and total IgE >1000 IU/mL; all underwent evaluation for both TPE and ABPA. These laboratory thresholds were study-entry criteria and were not substituted for disease-specific diagnostic definitions. TPE attribution required a compatible clinic-serological profile and a study-defined post-diethylcarbamazine (DEC) haematological response of at least 50% reduction in AEC. For the present analysis, ABPA was classified according to the revised 2024 International Society for Human and Animal Mycology criteria using prospectively collected clinical, immunological, and imaging variables. In TPE-compatible patients, DEC was given before ABPA-directed therapy to preserve interpretation of the eosinophilic response. Among 312 prospectively evaluated patients, 132 met the study definitions for TPE-ABPA overlap, 77 for TPE alone, and 103 for ABPA alone. In the overlap cohort, mean age was 36.2±15.9 years; median baseline AEC was 6071 cells/μL [interquartile range (IQR) 3793-11,000] and median total IgE was 7641 IU/mL (IQR 4525-13,621). *Aspergillus fumigatus*-specific IgE and IgG were positive in all 132; the filarial antigen was positive in 128 (97.0%), and there was qualitative filarial IgG in all 132. The dominant recorded high-resolution computed tomography pattern was normal in 73 (55.3%), while bronchiectasis was recorded in 42 (31.8%). All overlap patients received DEC and fulfilled the study-defined ≥50% AEC response criterion. ABPA-directed treatment comprised systemic corticosteroids alone in 71 (53.8%), itraconazole alone in 41 (31.1%), and both in 20 (15.2%). Concurrent TPE and ABPA should be considered in endemic regions when asthma is accompanied by disproportionate eosinophilia and IgE elevation; recognition of one eosinophilic disorder should not prematurely terminate evaluation for the other.

Key words: allergic bronchopulmonary aspergillosis, tropical pulmonary eosinophilia, eosinophilia, filariasis, *Aspergillus fumigatus*.

Introduction

Pulmonary eosinophilia comprises a heterogeneous group of infectious, allergic, inflammatory, and neoplastic disorders. In tropical regions, the differential diagnosis is particularly broad because helminth-associated pulmonary disease and allergic airway disease may occur in the same clinical setting. Tropical pulmonary eosinophilia (TPE) is an occult manifestation of lymphatic filariasis attributed to an exaggerated host immune response to antigens of Wuchereria bancrofti or Brugia malayi. It is classically associated with nocturnal cough and dyspnoea, marked peripheral eosinophilia, very high serum immunoglobulin E (IgE), and clinical and haematological response to diethylcarbamazine (DEC) [1-5].

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity disorder related to airway colonisation by Aspergillus fumigatus. It most often complicates asthma and cystic fibrosis, although the revised International Society for Human and Animal Mycology (ISHAM) framework also recognises chronic obstructive pulmonary disease, bronchiectasis, and a compatible clinico-radiological presentation as relevant predisposing contexts. The 2024 revised ISHAM criteria require A. fumigatus sensitisation and a serum total IgE concentration of at least 500 IU/mL, together with at least two additional components comprising A. fumigatus-specific IgG, blood eosinophilia, or characteristic imaging findings [6,7].

TPE and ABPA share several clinical and immunological features, including cough, wheeze, dyspnoea, eosinophilia, and markedly elevated total IgE. In patients with established asthma, coexistence of these disorders may contribute to persistent symptoms and apparent poor asthma control despite prior asthma-directed treatment. Published evidence on concurrent TPE and ABPA remains limited and consists mainly of individual reports and small series describing Aspergillus sensitisation or dual disease [8-10]. Current Global Initiative for Asthma (GINA) guidance emphasises reassessment for alternative diagnoses and comorbid conditions when asthma remains poorly controlled and specifically recognises ABPA within the severe-asthma framework [11]. In a filariasis-endemic region, disproportionate eosinophilia and very high IgE should therefore discourage diagnostic anchoring on asthma, ABPA, or TPE alone. Regional tertiary-care experience has also documented the clinical burden of TPE in Bihar [12].

This study aimed to characterise the demographic, clinical, radiological, immunological, and parasitological phenotype of patients with established asthma who met the study definitions for concurrent TPE and ABPA in a filariasis-endemic region. A secondary objective was to discuss the clinical relevance of recognising both conditions when asthma symptoms persist.

This prospective study was descriptive and was not designed to attribute poor asthma control to the overlap, to estimate its population prevalence, or to assess diagnostic test sensitivity.

Materials and Methods

Study design and setting

This was a single-centre, prospective, hospital-based observational study conducted in the Department of Pulmonary Medicine, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India, from December 2023 to December 2024. Participants were evaluated prospectively, with short-term follow-up after disease-directed therapy where relevant to diagnostic attribution. All screened patients had a physician diagnosis of asthma documented before enrolment. Institutional Ethics Committee approval was obtained at Indira Gandhi Institute of Medical Sciences, Patna (approval no. 26/IEC/IGIMS/2021, dated 23 March 2021). Written informed consent was obtained from adult participants; for those younger than 18 years, written consent was obtained from a parent or legal guardian.

Study entry rationale and participant selection

Eligible participants were older than 10 years, had a documented prior physician diagnosis of asthma, and had both AEC >1000 cells/μL and total IgE >1000 IU/mL at presentation. These prespecified entry thresholds selected patients with marked eosinophilia and IgE elevation; they were not substituted for disease-specific diagnostic criteria. All 312 eligible patients underwent evaluation for both TPE and ABPA, independently of the result for either condition. Of these, 132 met study definitions for TPE–ABPA overlap, 77 for TPE alone, and 103 for ABPA alone. The overlap cohort was the principal analytic population; the other groups were not used for inferential comparisons of asthma control. Active bacterial pneumonia was excluded as a potential confounding infection rather than as an eosinophilic lung disorder: overlapping symptoms and radiological changes could impair diagnostic attribution, and subsequent systemic glucocorticoid treatment could mask or exacerbate untreated infection. Other defined eosinophilic lung diseases, pregnancy, and lactation were exclusion criteria. Active pulmonary tuberculosis and other clinically relevant causes of eosinophilia were excluded before initiation of systemic glucocorticoids.

The entry criteria also excluded patients with AEC 500–1000 cells/μL or total IgE 500–1000 IU/mL, even though ABPA and TPE may warrant evaluation at lower values in clinical practice. Accordingly, the study cannot determine the sensitivity of the entry thresholds or quantify overlap among patients with lower biomarker levels.

Definition of allergic bronchopulmonary aspergillosis

For the present analysis, ABPA was classified according to the revised 2024 ISHAM-ABPA working-group criteria [6] using the clinical, immunological, and imaging variables collected prospectively during the study. Because all participants had a documented prior physician diagnosis of asthma, the predisposing-condition requirement was satisfied. Formal classification required both essential components - A. fumigatus-specific IgE ≥0.35 kUA/L and serum total IgE ≥500 IU/mL - and at least two of the following: positive A. fumigatus-specific IgG, blood eosinophils ≥500 cells/μL (current or historical), or thin-section chest computed tomography findings consistent with ABPA and/or fleeting opacities on chest radiography. The higher IgE and eosinophil thresholds used for study entry were not substituted for the ISHAM diagnostic definition.

Assessment of tropical pulmonary eosinophilia

TPE was assessed using a clinic-serological framework described in the TPE literature [1-3,13]. Attribution required residence in or exposure to a filariasis-endemic area, compatible respiratory symptoms, marked eosinophilia and elevated total IgE, supportive qualitative filarial serology, exclusion of a more convincing competing cause of eosinophilia, and a haematological response to DEC. For this prospective clinical study, the DEC response criterion was operationally defined as a ≥50% reduction in AEC at reassessment after the 3-week DEC course. In patients with a compatible TPE profile, DEC was intentionally administered before ABPA-directed systemic treatment so that the eosinophilic response could be assessed without corticosteroid-related suppression. All participants nevertheless underwent evaluation for both TPE and ABPA.

Clinical, laboratory, physiological, and imaging assessment

Initial assessment included demographics, respiratory symptoms, physical examination, complete blood count with AEC, serum total IgE, qualitative filarial antigen and filarial IgG testing, stool examination for ova/cysts/parasites, spirometry, chest radiography, and HRCT assessment. Serum total IgE, A. fumigatus-specific IgE, and A. fumigatus-specific IgG were measured by fluoroenzyme immunoassay; A. fumigatus-specific IgE ≥0.35 kUA/L and A. fumigatus-specific IgG ≥27 mgA/L were considered positive/elevated [6]. Filarial antigen was assessed qualitatively by immunochromatography, and filarial IgG was recorded qualitatively. A single dominant HRCT pattern was recorded for this analysis as bronchiectasis, interstitial

abnormality, nodular opacity, or no abnormality. Before systemic glucocorticoids were started, active pulmonary tuberculosis was excluded by sputum acid-fast bacilli smear and cartridge-based nucleic acid amplification testing.

At enrolment, patients in the overlap cohort continued their existing maintenance-and-reliever therapy (MART) with inhaled budesonide 200 µg/formoterol 6 µg, two puffs twice daily as scheduled maintenance treatment. MART was continued during the study. The frequency of additional as-needed inhalations was not systematically recorded. Baseline adherence, prior oral corticosteroid exposure, and other asthma add-on treatments were not systematically tabulated; therefore, the possible effects of background treatment on symptoms and eosinophil counts could not be quantified.

Patients in the overlap cohort were assessed clinically for eosinophilic granulomatosis with polyangiitis (EGPA), including antineutrophil cytoplasmic antibody (ANCA) testing. All 132 were ANCA-negative, and no patient was considered to have EGPA following clinical evaluation. ANCA negativity alone is insufficient to exclude EGPA; the assessment was clinical as well as laboratory-based.

Spirometry was retained as a descriptive clinical measure. Because lung-volume testing was not performed systematically, patterns suggestive of restriction or a mixed ventilatory defect were reported descriptively and were not interpreted as physiologically confirmed restrictive or mixed ventilatory defects. The study did not uniformly re-establish the pre-existing asthma diagnosis by a single spirometric protocol.

Treatment and follow-up

The treatment sequence was intended to preserve diagnostic interpretability. The existing budesonide 200 µg/formoterol 6 µg regimen (two puffs twice daily, with MART) was continued. Patients with a TPE-compatible profile received diethylcarbamazine (DEC) 6 mg/kg/day in divided doses for three weeks [1,3,14,15], followed by reassessment of AEC and symptoms; a ≥50% fall in AEC was the study-defined haematological response. ABPA-directed treatment comprised systemic corticosteroids, itraconazole, or both according to the treating physician's judgement. No biologic treatment was initiated as part of the study protocol. Treatment was not randomly allocated; no comparative efficacy analysis was planned.

Statistical analysis

Continuous variables were summarised according to their distribution. Age is presented as mean ± standard deviation (SD) with median and interquartile range (IQR) as supplementary descriptors. Because AEC and total IgE showed marked right-skew, these biomarkers are reported primarily as median (IQR), with mean ± SD provided secondarily for comparability with earlier literature. Categorical variables are presented as number and percentage; Wilson 95% confidence intervals (CI) are provided for selected clinically important proportions. No inferential hypothesis testing or multivariable prediction modelling was undertaken because the study objective was descriptive clinical phenotyping and because AEC and total IgE formed part of the study-entry and diagnostic pathway, making predictor modelling vulnerable to circular inference. The study database was analysed using SPSS version 23.0 (IBM Corp., Armonk, NY, USA), and the descriptive summaries reported here were cross-checked against the master dataset during manuscript preparation.

Results

Participant distribution and clinical characteristics

A total of 312 patients with established asthma were prospectively evaluated for both TPE and ABPA. Of these, 132 (42.3%) met the study definitions for the TPE-ABPA overlap phenotype, 77 (24.7%) were classified as TPE alone, and 103 (33.0%) as ABPA alone. These proportions describe a selected tertiary-care referral spectrum and should not be interpreted as population prevalence or as the frequency of overlap among unselected patients with asthma. The 132-patient overlap cohort constituted the principal analytic cohort. Mean age was 36.2 ± 15.9 years and median age was 32 years (IQR 23-45.3); 95 (72.0%) were male and 76 (57.6%) lived in rural areas. Cough was present in all patients, wheeze in 114 (86.4%), and dyspnoea in 108 (81.8%) (Table 1).

Physiological and imaging findings

Spirometry was predominantly described as showing patterns suggestive of restriction or a mixed ventilatory defect, but these categories remain descriptive because lung volumes were not systematically measured. The dominant recorded HRCT pattern was normal in 73 patients (55.3%; 95% CI 46.8-63.5%), bronchiectasis in 42 (31.8%; 95% CI 24.5-40.2%), interstitial abnormality in 8 (6.1%), and nodular opacity in 9 (6.8%) (Table 2).

Immunological and parasitological findings

Baseline AEC and total IgE were markedly right-skewed. Median AEC was 6071 cells/μL (IQR 3793-11,000), with a mean of 8301 ± 6471 cells/μL; by study-entry definition, all 132 overlap patients had AEC >1000 cells/μL at presentation. Median total IgE was 7641 IU/mL (IQR 4525-13,621), with a mean of 11,154 ± 9606 IU/mL; all 132 had total IgE >1000 IU/mL and 93 (70.5%; 95% CI 62.2-77.6) had total IgE >5000 IU/mL. A. fumigatus-specific IgE was ≥0.35 kUA/L and A. fumigatus-specific IgG ≥27 mgA/L in all 132 patients. Filarial antigen was positive in 128 (97.0%; 95% CI 92.5-98.8) and qualitative filarial IgG in all 132. Stool examination was negative in 58 (43.9%) and identified an organism in 74 (56.1%): Ascaris in 27 (20.5%), Giardia in 13 (9.8%), E. histolytica in 27 (20.5%), and a separately recorded roundworm category in 7 (5.3%). Stool findings were considered in the differential diagnosis of eosinophilia and were not used as evidence for TPE (Table 3).

Treatment exposure

All 132 patients in the overlap cohort received DEC and fulfilled the study-defined criterion of a ≥50% post-DEC reduction in AEC. Because this response formed part of TPE attribution, it should not be interpreted as an independent treatment-efficacy outcome. The scheduled budesonide 200 μg/formoterol 6 μg MART regimen (two puffs twice daily) was continued. All 132 overlap patients were ANCA-negative, and none was diagnosed with EGPA on clinical assessment. ABPA-directed treatment comprised systemic corticosteroids alone in 71 patients (53.8%), itraconazole alone in 41 (31.1%), and both in 20 (15.2%); 91 (68.9%) were exposed to systemic corticosteroids and 61 (46.2%) to itraconazole. No biologic therapy was initiated under the study protocol. Treatment exposures are descriptive, not comparative efficacy results (Table 3).

Discussion

The central observation of this prospective study is that TPE and ABPA can coexist as a clinically coherent dual pathology in patients with established asthma in a filariasis-endemic region. Marked eosinophilia and very high total IgE served as nonspecific signals prompting evaluation beyond escalation of asthma treatment alone. The overlap phenotype was characterised by prominent cough and wheeze, very marked eosinophilia, extremely high total IgE, universal A. fumigatus sensitisation with elevated specific IgG, and near-universal positivity of qualitative filarial markers. The clinical message is not to screen every patient with

asthma for filariasis, but to retain suspicion for dual eosinophilic pathology when symptoms remain troublesome and the magnitude of eosinophilia or IgE elevation appears disproportionate to ordinary asthma.

This observation is relevant to apparently difficult-to-control asthma. GINA recommends reassessing the diagnosis and searching for comorbidities or alternative explanations when asthma remains poorly controlled, and ABPA is specifically recognised within the severe-asthma framework [11]. In filariasis-endemic regions, TPE may coexist with ABPA and be overlooked if evaluation closes after recognition of only one disorder. Previous reports have described patients in whom persistent eosinophilia or symptoms led to recognition of TPE in the setting of ABPA [9,10], while Chhabra and Dash reported A. fumigatus sensitisation in TPE [8]. In the present study, all participants were evaluated for both disorders, reducing the risk that one diagnosis was sought only after treatment failure of the other. These data do not establish prevalence among unselected patients with asthma, but they support the clinical existence and relevance of dual pathology.

The absence of an asthma-only comparator group and of a uniformly applied, validated longitudinal asthma-control measure prevents attribution of persistent symptoms or poor control specifically to TPE–ABPA coexistence. Although TPE-only and ABPA-only patients were identified within the selected population, they were not analysed as matched asthma-control groups. The present findings therefore characterise a dual diagnostic phenotype but do not show that it causes worse asthma control than either condition alone or asthma without either condition.

Interpretation of the TPE component requires caution. Classic TPE is associated with marked eosinophilia, high total and anti-filarial immunoglobulins, nocturnal respiratory symptoms, and a characteristic response to DEC [1-3,13]. In this study, AEC >1000 cells/μL and total IgE >1000 IU/mL were study-entry criteria that selected a markedly eosinophilic, high-IgE population; they were not used as stand-alone disease-defining criteria for TPE. Filarial antigen and filarial IgG results were qualitative, and stool examination identified other enteric organisms in a substantial proportion of patients. Because anti-filarial antibody assays may cross-react in non-filarial helminth-associated eosinophilic syndromes [16], TPE attribution required the composite clinic-serological profile, exclusion of a more convincing competing cause, and the study-defined ≥50% AEC reduction after DEC rather than seropositivity alone. Persistent lower respiratory tract inflammation and interstitial abnormalities have also been

documented after conventional DEC treatment, underscoring that a haematological response does not necessarily imply complete pulmonary resolution [17].

For the present analysis, ABPA was classified using the revised 2024 ISHAM criteria [6] applied to prospectively collected variables. All participants had a documented prior physician diagnosis of asthma, and the mandatory immunological components were applied at the recommended thresholds. A. fumigatus-specific IgE and IgG were measured by fluoroenzyme immunoassay, with cut-offs of ≥0.35 kUA/L and ≥27 mgA/L, respectively. The higher AEC and total IgE thresholds used for study entry were not substituted for the ABPA definition. Importantly, all participants underwent assessment for both ABPA and TPE; the DEC-first sequence in TPE-compatible patients was used to preserve interpretation of the eosinophilic response rather than to determine whether ABPA testing was performed.

Spirometric categories should be interpreted descriptively because lung volumes were not systematically measured; patterns suggestive of restriction or mixed ventilatory impairment were not physiologically confirmed. Asthma was a documented prior physician diagnosis rather than a diagnosis uniformly re-established by a single study spirometry protocol.

The treatment sequence was likewise descriptive rather than comparative. DEC was given first in TPE-compatible patients to document the eosinophilic response before systemic ABPA treatment. ABPA-directed therapy was selected according to the treating physician's clinical judgement: 71 patients received systemic corticosteroids alone, 41 itraconazole alone, and 20 both agents. These data describe treatment exposure only and do not establish superiority of any regimen.

No biologic therapy was initiated under the study protocol. This should not be interpreted as evidence that all patients were ineligible: eligibility for severe-asthma biologic treatment was not formally adjudicated using standardised exacerbation history, symptom-control measures, adherence and treatment optimisation, or maintenance corticosteroid requirements. Before selecting biologic therapy in patients with persistent asthma symptoms, clinical assessment should confirm asthma, review inhaler technique and adherence, and consider treatable comorbidities and alternative diagnoses, including ABPA and, in appropriate epidemiological settings, TPE [11]. This study cannot estimate biologic eligibility or compare biologic with non-biologic management.

Eosinophils contribute to airway inflammation through activation, degranulation, and release of cytotoxic proteins and lipid mediators. These mechanisms are implicated in epithelial injury, mucus hypersecretion, and airway hyperresponsiveness; prolonged eosinophilic inflammation

may contribute to structural airway remodelling. Local eosinophil degranulation in TPE has been reported [5], and persistent pulmonary inflammation after DEC has also been described [17]. These established mechanisms provide biological plausibility for persistent symptoms, but eosinophil activation, tissue injury, airway remodelling, and serial lung-function decline were not directly measured in the current cohort. They must not be presented as outcomes demonstrated by this study.

The study has several strengths, including its prospective design, a relatively large tertiary-care cohort, systematic evaluation of all participants for both TPE and ABPA, application of contemporary ABPA criteria to prospectively collected variables, and a predefined sequential DEC-first pathway for TPE-compatible patients. Limitations include the single-centre referral population and restriction of eligibility to patients with AEC >1000 cells/μL and total IgE >1000 IU/mL, which introduce selection and spectrum bias; asthma based on a prior physician diagnosis rather than uniform study re-confirmation; prior treatment before tertiary referral; use of a study-defined ≥50% post-DEC AEC reduction that has not been externally validated as a TPE response threshold; qualitative filarial testing; the presence of other enteric organisms on stool examination; limited HRCT phenotyping because only a single dominant pattern was recorded; and absence of systematic lung-volume measurements. Formal asthma-control categories were not assigned using a validated GINA symptom-control instrument. ABPA treatment was non-randomised, and corticosteroid and itraconazole exposure should not be interpreted as comparative efficacy data.

Additional limitations include the absence of an asthma-only comparator group, lack of systematically recorded as-needed MART inhalations, baseline adherence, prior oral corticosteroid exposure or other asthma add-on medication use, absence of formal biologic-eligibility assessment, and lack of validated longitudinal asthma-control measurements. EGPA was excluded clinically, and the 132 overlap patients were ANCA-negative; nevertheless, negative ANCA results in isolation cannot exclude EGPA. Patients with lower AEC or total IgE values were outside the sampling frame, while previous systemic corticosteroid treatment or prior DEC therapy could potentially lower eosinophil values and influence case ascertainment. A haematological response to DEC does not demonstrate complete resolution of pulmonary inflammation.

Conclusions

TPE and ABPA can coexist in patients with previously diagnosed asthma in filariasis-endemic settings. Where symptoms persist alongside disproportionately high eosinophil counts and total IgE levels, recognising one eosinophilic disorder should not automatically terminate evaluation for the other. In this selected cohort, a compatible clinic-serological profile and a ≥50% DEC-associated fall in AEC supported TPE attribution, while ABPA was classified independently using the revised ISHAM criteria. Because there was no asthma-only comparator or standardised longitudinal measure of asthma control, the incremental impact of overlap on asthma control cannot be established. These findings support targeted clinical awareness, not universal filarial screening for asthma. Larger multicentre studies including patients with lower biomarker values, appropriate comparison groups, and longitudinal clinical and physiological outcomes are needed.

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Table 1. Demographic and clinical characteristics of the study-defined TPE–ABPA overlap cohort (n=132)

Characteristic	Category	n (%) or summary
Age, years	Mean ± SD; median (IQR)	36.2 ± 15.9; 32 (23-45.3)
Age group, year	≤20	26 (19.7)
	21-30	35 (26.5)
	31-40	22 (16.7)
	41-50	21 (15.9)
	51-60	17 (12.9)
	>60	11 (8.3)
Sex	Male	95 (72.0)
	Female	37 (28.0)
Residence	Rural	76 (57.6)
	Urban	56 (42.4)
Symptom	Cough	132 (100)
	Wheeze	114 (86.4)
	Dyspnoea	108 (81.8)
	Fever	32 (24.2)
	Chest pain	30 (22.7)
Physical finding	Crackles	19 (14.4)

ABPA, allergic bronchopulmonary aspergillosis; TPE, tropical pulmonary eosinophilia. All participants in the overlap cohort had a prior physician diagnosis of asthma.

Table 2. Spirometric, imaging, and immunological findings in the TPE–ABPA overlap cohort (n=132)

Domain	Variable	Result
Spirometry	Spirometric pattern suggestive of restriction (mild)	65 (49.2%)
	Spirometric pattern suggestive of mixed ventilatory defect	61 (46.2%)
	Moderate obstruction	2 (1.5%)
	Normal spirometry	4 (3.0%)
Dominant recorded HRCT pattern	Normal HRCT	73 (55.3%)
	Bronchiectasis	42 (31.8%)
	Interstitial abnormality	8 (6.1%)
	Nodular opacity	9 (6.8%)
Haematology / immunology	AEC, median (IQR); mean ± SD	6071 (3793-11,000); 8301 ± 6471 cells/μL
	Total IgE, median (IQR); mean ± SD	7641 (4525-13,621); 11,154 ± 9606 IU/mL
	Total IgE >5000 IU/mL	93 (70.5%)
	A. fumigatus-specific IgE ≥0.35 kUA/L	132 (100%)
	A. fumigatus-specific IgG ≥27 mgA/L	132 (100%)

AEC, absolute eosinophil count; HRCT, high-resolution computed tomography; IgE, immunoglobulin E; IgG, immunoglobulin G; IQR, interquartile range. Spirometric categories are descriptive because lung-volume testing was not systematic. HRCT categories denote one dominant recorded pattern. A. fumigatus-specific IgE/IgG were measured by fluoroenzyme immunoassay.

Table 3. Parasitological and treatment findings in the TPE–ABPA overlap cohort (n=132)

Domain	Variable	Result
Filarial markers	Filarial antigen positive (qualitative)	128 (97.0%)
	Filarial IgG positive (qualitative)	132 (100%)
Stool examination	Negative	58 (43.9%)
	Any organism detected	74 (56.1%)
Treatment	Diethylcarbamazine	132 (100%)
	Systemic corticosteroids only	71 (53.8%)
	Itraconazole only	41 (31.1%)
	Both systemic corticosteroids and itraconazole	20 (15.2%)
TPE attribution	Post-DEC AEC reduction ≥50%	132 (100%)

DEC, diethylcarbamazine; TPE, tropical pulmonary eosinophilia. Filarial antigen was tested qualitatively by immunochromatography, and filarial IgG was recorded qualitatively. ABPA-directed treatment categories were mutually exclusive; DEC response formed part of study-defined TPE attribution and is not an independent efficacy outcome.