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Phase Ib/II Study of Preliminary Efficacy, Safety and Pharmacodynamics of MG-K10, a Humanised Monoclonal Antibody Targeting IL-4Rα, in Adult Chinese Patients With Asthma

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ABSTRACT

Background: MG-K10 is a long-acting, humanised monoclonal antibody against interleukin-4 receptor alpha (IL-4Rα), which inhibits IL-4 and IL-13-mediated signalling to reduce type 2 inflammation in asthma.

Objective: This Phase Ib/II study aimed to evaluate the preliminary efficacy, safety and pharmacodynamic characteristics of MG-K10 in Chinese patients with asthma.

Methods: This study included an initial phase Ib to evaluate safety and tolerability, followed by a phase II study in which eligible patients with moderate-to-severe asthma were randomised 1:1:1 to receive MG-K10 300 mg every 2 weeks (Q2W), MG-K10 300 mg every 4 weeks (Q4W), or a matched placebo (2 mL) Q2W subcutaneously for 24 weeks. The primary endpoint was the absolute change from baseline in the prebronchodilator forced expiratory volume in 1 s (FEV_1) at week 12. Secondary efficacy endpoints, including asthma control, the rate of severe exacerbations and safety, were assessed. This trial is registered with ClinicalTrials.gov (NCT05382910).

Results: A total of 64, 60 and 63 patients were randomised to the MG-K10 Q2W, MG-K10 Q4W and placebo groups respectively. At week 12, the least squares mean improvements in prebronchodilator FEV_1 were significantly greater in both MG-K10 groups than in the placebo group [Q2W vs. placebo: 0.35 L (95% CI, 0.208–0.490), Q4W vs. placebo: 0.30 L (95% CI, 0.156 to 0.441), both $p < 0.0001$]. Greater FEV_1 improvements were observed in patients with baseline blood eosinophils $\geq 0.3 \times 10^9$/L. The incidence of adverse events was similar across groups [MG-K10 300 mg, Q2W (79.7%), MG-K10 300 mg Q4W (85.0%) and placebo groups (79.4%)]. MG-K10 was safe and well-tolerated, and consistent with the known safety signals.

Conclusions: MG-K10 was superior to placebo in improving lung function, enhancing asthma control and reducing severe exacerbations in patients with asthma. The once-every-4-week regimen offers extended dosing intervals that may enhance medication adherence.

Mo Xian and Xu Shi contributed equally to this study and share first authorship.

Key Messages

- Interleukin-4 receptor (IL-4Rα) is a key target for the treatment of type 2 inflammatory diseases.
- MG-K10 is an innovative, humanised monoclonal antibody that targets the same as Dupilumab.
- It allows long dosing intervals owing to its prolonged half-life, which is expected to improve patient adherence.

1 | Introduction

Bronchial asthma (asthma) is a common chronic respiratory disease characterised by airway hyperresponsiveness, variable airway obstruction and mucosal oedema of the central airway. It is estimated that about 334 million people worldwide suffer from asthma, with a rising prevalence that has emerged as a significant global public health concern [1, 2]. In China, the prevalence of asthma among people aged 20 years and above was 4.2%, with a total of 45.7 million patients [3]. However, owing to individual heterogeneity, variability in asthma severity and inconsistent adherence to standardised treatment, the overall asthma control rate is only 28.5% [2]. The burden associated with poor asthma control, including increased treatment costs and reduced quality of life, remains an urgent clinical need. Available maintenance-and-reliever therapy with inhaled corticosteroids (ICS) plus long-acting β2 agonists (LABAs) provides effective long-term control for most patients with asthma [4]. However, about 5%–10% of patients experience symptomatic disease despite taking high-dose combination therapies [5]. Furthermore, long-term use of ICS at medium-to-high doses is associated with systemic side effects, such as infection, adrenal suppression and metabolic disorders [6, 7].

Type 2 (T2) inflammation is the common pathophysiological foundation for various inflammatory diseases and plays a crucial role in the onset and progression of asthma [8]. Up-regulation of cytokines, including interleukin-4 (IL-4), IL-5 and IL-13, is considered a key inflammatory pathology of T2-mediated asthma [1, 9]. Notably, T2 inflammatory biomarkers, such as total blood immunoglobulin E (IgE), blood or sputum eosinophils and fractional exhaled nitric oxide (FeNO), can help identify the asthma phenotypes and their quantification facilitates the optimisation of therapeutic strategies to improve patient outcomes [9–12]. Currently, four types of biologics targeting IL-5 or its receptor alpha subunit (IL-5Rα), IL-4Rα, IgE and thymic stromal lymphopoietin (TSLP) have been approved as add-on therapies for severe asthma [13, 14]. Dupilumab, a fully human monoclonal antibody, exerts its therapeutic effects by selectively binding to the IL-4Rα subunit, thereby inhibiting IL-4/IL-13-mediated signalling and suppressing T2 inflammatory responses in asthma [15]. Numerous clinical trials [16–19] have demonstrated that dupilumab significantly improves the forced expiratory volume in 1 s (FEV_1) and reduces the rate of severe asthma exacerbations. Based on strong evidence, dupilumab has been approved for the maintenance treatment of asthma in paediatric patients aged 6–11 years, adolescents and adults.

MG-K10 is an innovative, long-acting, anti-IL-4Rα humanised monoclonal antibody developed by Hunan Mabgeek Biotech Co. Ltd. By binding to IL-4Rα, MG-K10 blocks the signal transduction induced by IL-4 and IL-13, and alleviates T2-type inflammatory symptoms by inhibiting the activation of signal transducer and activator of transcription 6 (STAT6) [20]. Notably, MG-K10 harbours a genetic modification that confers an approximately 10-fold higher affinity for the neonatal Fc receptor (FcRn) compared with dupilumab. Pharmacokinetic results from a previous phase I study indicated that the half-life and area under the concentration-time curve ($AUC_{0\text{-}t}$) of MG-K10 after a single dose were approximately twice that of dupilumab at the same dose [21], which supports the feasibility of extended dosing intervals. Here, we conducted a phase Ib/II trial of MG-K10 in patients with asthma to evaluate its preliminary efficacy, safety and pharmacodynamic (PD) characteristics, including changes in T2 inflammatory biomarkers.

2 | Methods

2.1 | Study Design

This was a two-part, multicentre trial (NCT05382910), comprising a phase Ib safety and tolerability assessment of MG-K10 humanised monoclonal antibody injection (300 mg, 2 mL) followed by a phase II study to evaluate its preliminary efficacy in patients with moderate-to-severe asthma. This phase Ib/II study was conducted from July 5, 2022, to July 10, 2024, at 32 sites across China (A list of sites is provided in Table S1). The phase Ib portion was designed to assess the safety and tolerability of multiple doses of MG-K10 in patients with asthma. All subjects received 300 mg of MG-K10 via subcutaneous injection every 2 weeks (Q2W), with a total of 6 doses. The phase II part was initiated after review and discussion between the sponsor and investigators, following evaluation of safety data up to 4 weeks after the last subject was enrolled in Phase Ib. In the phase II part, eligible patients were randomised at a 1:1:1 ratio into three groups: MG-K10 300 mg Q2W, MG-K10 300 mg every 4 weeks (Q4W), or placebo (2 mL) Q2W for 24 weeks.

This study was conducted per the principles of the Declaration of Helsinki and the Good Clinical Practice (GCP) of the International Council for Harmonization (ICH). All study-related documents were approved by the institutional ethics committee at each trial site. All patients provided written informed consent before participating in any study procedure.

2.2 | Study Population

Both phases of the study included male or female patients, aged 18–75 years (inclusive) with a body weight ≤ 90 kg, who had received a confirmed diagnosis of asthma for at least 12 months based on the Global Initiative for Asthma (GINA) 2021 guideline. For the phase II, eligible patients additionally met the following key criteria: [1] treatment with medium-to-high-dose inhaled corticosteroids (ICS) (fluticasone propionate ≥ 250 μg twice daily, or equipotent inhaled glucocorticoid daily dosage to a maximum of 2000 μg/day of fluticasone propionate or equivalent) plus an additional controller [e.g., a long-acting beta2-agonists (LABA), or leukotriene receptor antagonists (LTRA), or sustained release theophylline] for at

least two consecutive months before screening; [2] prebronchodilator FEV_1 was 80% or less of the normal predicted value before randomisation; [3] a 5-item Asthma Control Questionnaire (ACQ-5) score of ≥1.5 during the screening and run-in periods; [4] a positive bronchodilator test (BDT) (defined as airway reversibility of ≥12%, and an absolute increase in FEV_1 ≥200 mL after the administration of albuterol/salbutamol); [5] a history of at least one asthma exacerbation in the year before screening that required systemic glucocorticoid (oral or parenteral) treatment, or hospitalisation/emergency care for an asthma exacerbation. Patients were excluded if they: were diagnosed with chronic obstructive pulmonary disease (COPD) or other lung diseases that may impair pulmonary function; had a severe asthma exacerbation resulting in treatment with a systemic glucocorticoid (oral or parenteral) for worsening asthma at least once, or hospitalisation or emergency treatment from 1 month prior to the administration; treated with systemic glucocorticoids within 1 month before study agent administration. Details of the full eligibility criteria are presented in Table S2. Our inclusion/exclusion criteria were designed to balance rigorous mitigation of immune-related risks to maximise participant safety with the feasibility of conducting the study, thereby preserving the integrity and generalisability of the pharmacokinetic (PK), pharmacodynamic (PD) and clinical efficacy data generated.

2.3 | Interventions and Procedures

In phase Ib, patients received MG-K10 at a dose of 300 mg subcutaneously Q2W for 12 weeks, followed by an 8-week safety follow-up. The phase II portion of the study was initiated based on a safety assessment conducted by the investigator and sponsor, which took place 4 weeks following the last patient enrolment in phase Ib. For phase II, patients first underwent a 1-week screening period, after which they entered a 4-week run-in period. At run-in, patients were required to be on a stable dose of their conventional therapy as background treatment. Eligible patients with moderate-to-severe asthma were then randomised in a 1:1:1 ratio, stratified by prebronchodilator FEV_1 (50%–80% vs. <50%) to receive 24 weeks of subcutaneous treatment with either MG-K10 300 mg Q2W, MG-K10 300 mg Q4W, or a matched-volume placebo (2 mL) Q2W. Stratified randomisation was implemented via an interactive web response system (IWRS). All patients, investigators and involved study personnel remained blinded to the treatment assignments throughout the entire phase II period.

2.4 | Endpoints and Assessments

The primary endpoint of phase Ib was to assess the safety and tolerability of multiple doses of MG-K10 in patients with asthma.

For phase II, the efficacy of MG-K10 was evaluated in patients with moderate-to-severe asthma in terms of lung function, asthma exacerbations and symptom control. The primary efficacy endpoint was defined as the absolute change from baseline in the FEV_1 before bronchodilator use at week 12. Secondary efficacy endpoints were as follows: changes from baseline in prebronchodilator FEV_1, morning/evening peak expiratory flow (PEF) and ACQ-5 score (ranging from 0 to 6, with higher scores indicating less control) at scheduled study visits; annualised rate of severe asthma exacerbations; annualised rate of loss of asthma control (LOAC) events; the time-to-first severe exacerbation and LOAC (detailed definitions of severe exacerbation and LOAC are available in the Supporting Appendix).

Safety was assessed by monitoring the incidence and severity of adverse events (AEs), routine measurements of vital signs, physical examinations, laboratory examinations (including haematology, blood biochemistry, urinalysis, pregnancy testing and infectious disease screening) and 12-lead electrocardiography (ECG). The severity of treatment-emergent adverse events (TEAEs) and treatment-related adverse events (TRAEs) was graded using the Common Terminology Criteria for Adverse Events (CTCAE, Version 5.0). AEs were coded according to the Medical Dictionary for Regulatory Activities (MedDRA) (version 27.0).

Pharmacodynamic (PD) analysis was performed by measuring changes from baseline in levels of the type 2 inflammatory biomarker associated with asthma, including serum thymus and activation-regulated chemokine (TARC), serum total immunoglobulin E (IgE) and FeNO. Blood samples (3 mL each) for assays were taken on days 1, 29, 57, 85, 113, 141, 169, 197 (only up to this time point in FeNO) and 225.

2.5 | Statistical Analysis

All randomised patients who received the study treatment, based on the intent-to-treat (ITT) principle, served as the full analysis set (FAS) for this study. The patients' demographic, clinical characteristics and efficacy outcomes were analysed in the FAS population. The per-protocol analysis set (PPS) was a subset of the FAS, analysing the patients who met the protocol and had good compliance. The safety analysis set (SS) included all FAS patients who received at least one dose of the investigational drug. PD was analysed in all randomised patients who received the study treatment and had at least one postbaseline assessable PD data.

Continuous data were expressed as mean ± standard deviation (SD) or median (interquartile range/IQR). Categorical data were presented as counts and percentages. For the primary efficacy endpoint and continuous secondary endpoint variables, analyses were performed in accordance with the study's treatment strategy, and the mixed-effects model for repeated measures (MMRM) was used to estimate the between-group differences, expressed as least-squares mean (LSM) differences, along with their corresponding 95% confidence interval (CI). The model included the endpoint variable described above as the dependent variable, with group, stratification factor and visit as fixed effects, and treatment-by-visit interaction accounted for, with patients as a random effect and baseline measurement as a covariate. Missing data resulting from early patient withdrawal (either before or after treatment initiation) were assumed to be missing at random (MAR). The annualised rates of severe exacerbation events and LOAC events were analysed using a negative binomial regression model. The time to the first severe asthma exacerbation event and LOAC event was analysed using

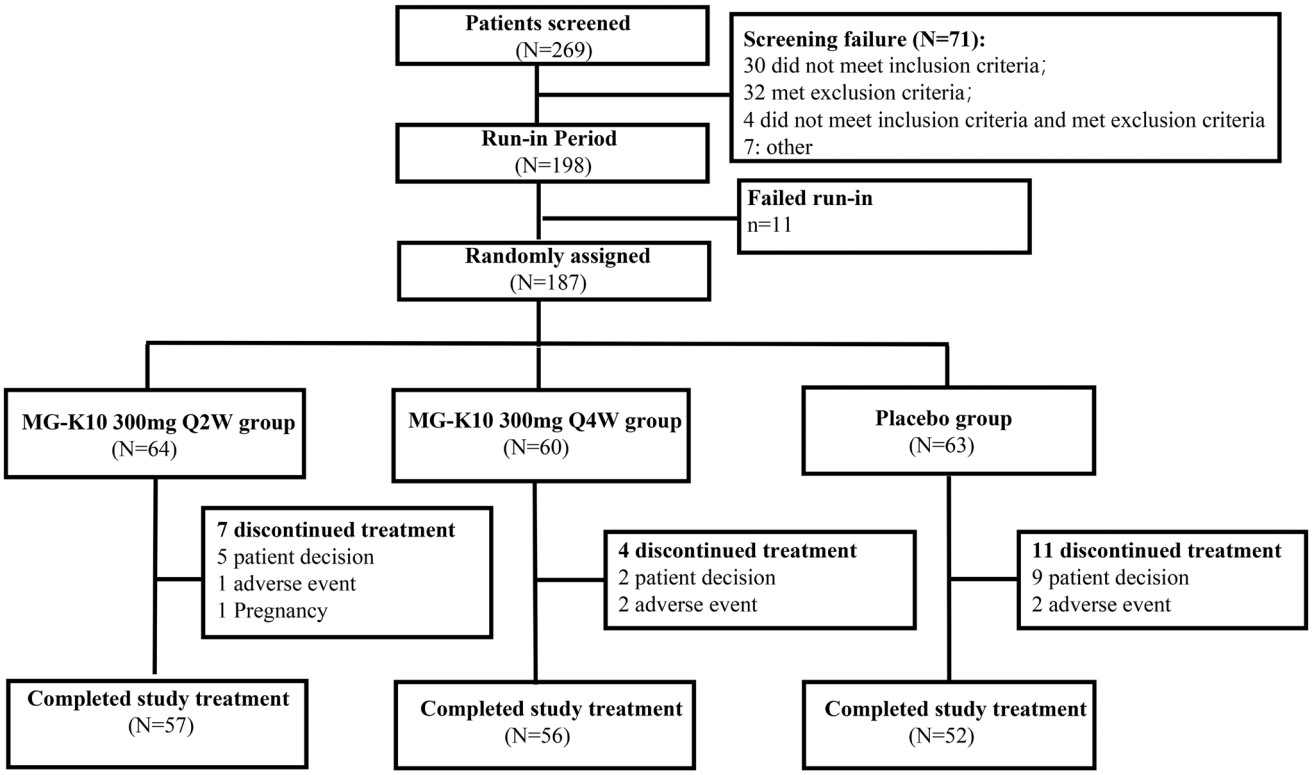


FIGURE 1 | Patient disposition.

a stratified Cox proportional-hazards model, and the differences between the groups were tested using the log-rank test, with results presented as hazard ratio (HR) and 95% CI. PK parameters were estimated using the noncompartmental technique in Phoenix WinNolin software (version 8.3). Subgroup analyses of efficacy endpoints were performed based on prespecified categories of eosinophil count ($<0.30\times10^9$/L, $\geq0.30\times10^9$/L). All statistical analyses were conducted using SAS (version 9.4) (SAS Institute Inc., Cary, NC).

3 | Results

3.1 | Patients' Disposition and Baseline Characteristics

A total of 193 patients were enrolled between July 2022 and July 2024, including six in phase Ib and 187 in phase II. For the phase Ib part, the mean (±SD) FEV1 of the six eligible adults with asthma at baseline was 1.92 ± 0.74 L, with 63.68% ± 19.89% of the predicted value. As the primary outcome of Phase Ib, multiple doses of MG-K10 were safe and well-tolerated in patients with asthma. Prebronchodilator FEV_1 (absolute and percentage changes from baseline) increased continuously throughout the 12-week treatment period. In phase II, 269 patients were screened, and 198 entered the run-in period. Among them, 187 eligible adult patients with moderate-to-severe asthma were successfully enrolled and were subsequently randomly allocated to receive MG-K10 (300 mg, Q2W) ($n=64$), MG-K10 (300 mg, Q4W) ($n=60$), or placebo (2 mL, Q2W) ($n=63$). Out of them, 165 patients completed the study. Trial discontinuation rate was higher in the placebo group ($n=11$, 17.5%) than in the MG-K10 300 mg Q2W (10.9%) and 300 mg Q4W (6.7%) groups, and was most commonly due to the patient's decision to withdraw (Figure 1). The mean age (±SD) of the patients was 55.1 (±10.6) years, and 85 (45.5%) of them were male. The mean asthma duration was 12.33 (±11.76) years. All patients had experienced at least one asthma exacerbation in the preceding year. All enrolled patients were treated with medium-to-high dose ICS (medium dose: 162 vs. high dose: 25) at baseline. Baseline blood eosinophil counts and biomarker levels, including FeNO, serum IgE and serum TARC, were also summarised among the three groups. As shown in Table 1, the three groups were balanced in terms of baseline and clinical characteristics.

3.2 | Efficacy

For the primary efficacy endpoint, the LS mean change from baseline in prebronchodilator FEV_1 at week 12 was 0.407 (SE: 0.051) L in the MG-K10 300 mg Q2W group, 0.357 (0.053) L in the MG-K10 300 mg Q4W group and 0.058 (0.051) L in the placebo group. Treatment with MG-K10 (300 mg, Q2W) or MG-K10 (300 mg, Q4W) increased the FEV_1 before bronchodilator use, as compared with placebo [LS mean difference vs. placebo, 0.35 L (95% CI, 0.208, 0.490) and 0.30 L (95% CI, 0.156, 0.441), respectively, for both $p<0.0001$]. In addition, the LS mean percent change (%) in prebronchodilator FEV_1 at week 12 was significantly improved with MG-K10 300 mg Q2W (LS mean difference: 21.07%, 95% CI: 8.453–33.686, $p=0.0012$) and Q4W regimens (LS mean difference: 16.62%, 95% CI: 3.801–29.43, $p=0.0113$) compared with placebo (Table 2). In the subgroup analyses stratified by baseline blood eosinophil count, both MG-K10 dose groups showed a benefit over placebo in the change

TABLE 1 | Baseline demographic and clinical characteristics (Full Analysis Set, FAS).

Characteristics	MG-K10, 300 mg, Q2W ($N=64$)	MG-K10, 300 mg, Q4W ($N=60$)	Placebo ($N=63$)	Total ($N=187$)
Age (year), (Mean ± SD)	53.7 ± 10.8	56.6 ± 10.5	55.2 ± 10.6	55.1 ± 10.6
Gender, [*n* (%)]				
Male	26 (40.6)	33 (55.0)	26 (41.3)	85 (45.5)
Female	38 (59.4)	27 (45.0)	37 (58.7)	102 (54.5)
Ethnicity, [*n* (%)]				
Han	61 (95.3)	58 (96.7)	59 (93.7%)	178 (95.2)
Hui	1 (1.6)	1 (1.7)	1 (1.6)	3 (1.6)
Man	0	1 (1.7)	1 (1.6)	2 (1.1)
Mongolian	1 (1.6)	0	1 (1.6)	2 (1.1)
Uygur	1 (1.6)	0	0	1 (0.5)
Yao	0	0	1 (1.6)	1 (0.5)
Body weight (kg) (Mean ± SD)	65.03 ± 10.69	64.74 ± 9.77	64.83 ± 9.57	64.87 ± 9.98
BMI (kg/m^2), (Mean ± SD)	24.53 ± 3.49	24.12 ± 2.97	24.42 ± 2.89	24.36 ± 3.12
History of smoking, [*n* (%)]	11 (17.2)	8 (13.3)	11 (17.5)	30 (16.0)
Duration of asthma (years), (Mean ± SD)	11.09 ± 10.28	13.41 ± 13.78	12.57 ± 11.13	12.33 ± 11.76
Number of exacerbations in past year (Mean ± SD)	1.1 ± 0.42	1.1 ± 0.57	1.3 ± 0.94	1.2 ± 0.68
Number of exacerbations in past year, [*n* (%)]				
1	58 (90.6)	55 (91.7)	54 (85.7)	167 (89.3)
2	4 (6.3)	4 (6.7)	7 (11.1)	15 (8.0)
3	2 (3.1)	0	0	2 (1.1)
5	0	1 (1.7)	1 (1.6)	2 (1.1)
7	0	0	1 (1.6)	1 (0.5)
Dose of inhaled glucocorticoids, [*n* (%)]				
Medium	52 (81.3)	53 (88.3)	57 (90.5)	162 (86.6)
High	12 (18.8)	7 (11.7)	6 (9.5)	25 (13.4)
Dual combination therapy, [*n* (%)]	48 (75.0)	46 (76.7)	49 (77.8)	143 (76.5)

(Continues)

TABLE 1 | (Continued)

Characteristics	MG-K10, 300 mg, Q2W (*N* = 64)	MG-K10, 300 mg, Q4W (*N* = 60)	Placebo (*N* = 63)	Total (*N* = 187)
Triple combination therapy, [*n* (%)]	16 (25.0)	14 (23.3)	14 (22.2)	44 (23.5)
Prebronchodilator FEV1, (Mean ± SD)				
Litre (L)	1.56 ± 0.53	1.48 ± 0.56	1.55 ± 0.64	1.53 ± 0.58
Percent of predicted normal value (%)	56.52 ± 15.09	53.75 ± 17.49	55.38 ± 17.10	55.25 ± 16.52
PEF (L/min), (Mean ± SD)				
Daytime PEF	208.67 ± 98.06	209.84 ± 108.03	196.27 ± 97.67	204.91 ± 100.90
Nighttime PEF	207.51 ± 98.13	210.01 ± 111.84	201.05 ± 99.59	206.16 ± 102.73
ACQ-5 score, (Mean ± SD)	2.24 ± 0.70	2.30 ± 0.59	2.37 ± 0.60	2.30 ± 0.63
Asthma symptom score, (Mean ± SD)				
Daytime	1.14 ± 0.91	1.21 ± 0.82	1.45 ± 0.89	1.27 ± 0.88
Nighttime	1.01 ± 0.81	1.13 ± 0.87	1.43 ± 0.98	1.19 ± 0.90
Biomarker levels				
FeNO (ppb)				
Mean (±SD)	38.10 ± 28.92	36.83 ± 29.59	39.74 ± 36.04	38.25 ± 31.55
IgE (ng/mL)				
Mean (±SD)	687.63 ± 941.55	886.87 ± 1078.08	900.02 ± 1261.31	823.11 ± 1099.49
Median (Q1, Q3)	358.39 (175.60, 856.80)	371.93 (194.50, 1276.01)	370.67 (179.26, 1144.04)	361.66 (179.26, 1016.93)
TARC (pg/mL)				
Mean (±SD)	150.29 ± 201.85	125.38 ± 119.73	108.65 ± 103.44	128.27 ± 149.04
Median (Q1, Q3)	70.06 (41.76, 178.03)	68.43 (44.28, 192.18)	63.29 (37.22, 147.29)	68.16 (41.67, 162.94)
Blood eosinophil count (10^9/L)				
Mean (±SD)	0.35 ± 0.34	0.44 ± 0.41	0.33 ± 0.35	0.37 ± 0.37
Median (Q1, Q3)	0.23 (0.13, 0.47)	0.32 (0.14, 0.61)	0.19 (0.11, 0.46)	0.23 (0.12, 0.49)

Note: The scores of this questionnaire range from 0 to 6, with higher scores indicating less asthma control.
Abbreviations: ACQ-5, 5-item Asthma Control Questionnaire; BMI, Body Mass Index; FeNO, Fractional exhaled nitric oxide; IgE, Immunoglobulin E; IQR, Interquartile Range; PEF, Peak expiratory flow; SD, Standard deviation; TARC, Thymus and activation-regulated chemokine.

in prebronchodilator FEV_1 at week 12, and FEV_1 improvement appeared to be greater in patients with higher blood eosinophil levels ($\geq 0.3 \times 10^9$/L) (Table S3).

The benefit of MG-K10 over matched placebo with respect to the LS mean change in prebronchodilator FEV_1 from baseline was significant by the first assessment at week 4 and was sustained throughout the 24-week treatment period ($p < 0.001$) [LS mean difference vs. placebo at week 24, 0.33 L (95% CI, 0.181, 0.475) with MG-K10 (300 mg, Q2W) and 0.34 L (95% CI, 0.188, 0.486) with MG-K10 (300 mg, Q4W)] (Figure 2A and Table 2). Consistent with absolute FEV_1 changes, the LS mean percent change (%) in prebronchodilator FEV_1 at week 24 was significantly greater with MG-K10 300 mg Q2W and Q4W versus placebo, with LS mean differences of 21.28% ($p = 0.0015$) and 21.31% ($p = 0.0017$) respectively (Figure 2B and Table 2). The results of the subgroup analyses based on PPS were consistent with the results in FAS (Table S3).

Regarding the changes from baseline in morning/evening PEF, MG-K10 treatment was superior to placebo at all visits during the 24-week treatment period (Figure 3A,B).

In the overall population, the annualised rate of severe asthma exacerbations over the 24-week intervention period was 0.09 (95% CI, 0.03, 0.31) among patients assigned to MG-K10 300 mg Q2W versus 0.38 (95% CI, 0.20, 0.73) among those assigned to matched placebo [Relative risk (RR) vs. placebo: 0.25 (95% CI, 0.07, 0.91), $p = 0.0357$]. In contrast, among patients who received 300 mg Q4W of MG-K10, the annualised rate was 0.10 (95% CI, 0.03, 0.33) and the RR of this event for the comparison with matched placebo was 0.27 (95% CI, 0.08, 0.97) ($p = 0.0449$). Moreover, in patients receiving 300 mg MG-K10 every 2 or every 4 weeks, the annualised rate of LOAC was lower than that of placebo, with the RR in LOAC event versus placebo of 0.43 (95% CI, 0.23, 0.78) ($p = 0.0060$) and 0.45 (95% CI, 0.25, 0.82) ($p = 0.0097$) respectively (Table 2). In a prespecified subgroup analysis of patients with eosinophils of $\geq 0.3 \times 10^9$/L, patients who received MG-K10 (300 mg) every 2 or 4 weeks had lower annualised rates of severe exacerbation and LOAC than those who received the placebo. The same results trend was observed in patients with eosinophils of $< 0.3 \times 10^9$/L (Table S3). In time-to-event analyses, the risk of having an LOAC event or severe asthma exacerbations was lower in each of the MG-K10 groups than in the placebo group, and a significant difference in the time to the first event ($p < 0.05$).

In the overall population, the ACQ-5 score, a tool for assessing asthma symptoms and control, trended lower from baseline in all three groups over the 24-week treatment period, with greater improvement in the two MG-K10 treatment groups than in the placebo group across different dose regimens (Table 2).

3.3 | Safety

In total, 187 patients were included in the SS, with the incidence of adverse events summarised in Table 3. One hundred and fifty-two (81.3%) patients reported 571 TEAEs throughout the study, with incidences of 79.7% (51/64), 85.0% (51/60) and 79.4% (50/63) in the MG-K10 300 mg Q2W, MG-K10 300 mg Q4W and placebo groups respectively. The incidence of TRAEs was 34.4%, 23.3% and 27.0%, in the MG-K10 300 mg Q2W, MG-K10 300 mg Q4W and placebo groups respectively. TEAEs were mostly mild to moderate in severity, and 18 patients experienced grade ≥ 3 TEAEs, but none of which were considered related to MG-K10. The top three most common TEAEs (≥ 5% of patients) in the MG-K10 300 mg Q2W group were upper respiratory tract infection (26.6%), urinary tract infection (9.4%), nasopharyngitis (7.8%), elevated eosinophils (7.8%), hyperlipidaemia (7.8%) and cough (7.8%). The top three most frequent TEAEs in patients who received MG-K10 300 mg Q4W were upper respiratory tract infection (18.3%), urinary tract infection (13.3%) and neutrophil count elevation (10.0%). In the placebo group, the most frequent TEAEs were upper respiratory tract infection (19.0%), followed by nasopharyngitis (11.1%) and urinary tract infection (9.5%). Overall, 11 (5.9%) patients experienced at least 1 SAE, none of which was related to the investigational drug. Of note, 7 TEAEs ($n = 5$, 2.7%) resulted in patient withdrawal from the study, but none were related to MG-K10. Twelve TEAEs ($n = 9$, 4.8%) resulted in discontinuation or permanent discontinuation. Although the incidence was highest in the patients receiving MG-K10 300 mg Q4W, only 2 TEAEs ($n = 2$) were judged to be drug-related.

3.4 | Pharmacokinetics and Pharmacodynamics

In patients who received MG-K10 300 mg Q2W, the serum concentration of MG-K10 gradually ascended with the increase in administration times and reached a steady state level at week 12. In contrast, the MG-K10 concentration fluctuated less in patients receiving MG-K10 300 mg Q4W and stabilised by week 8.

Regarding the median rate of change (%) from baseline in type 2 inflammatory biomarker levels, patients receiving MG-K10 exhibited significant reductions from baseline throughout the 24-week intervention period in serum TARC, serum total IgE and FeNO concentrations, whereas placebo-treated patients showed no meaningful changes from baseline (Figure 4A–C). Specifically, FeNO and serum TARC levels were significantly suppressed from baseline in both the MG-K10 300 mg Q2W and 300 mg Q4W groups throughout the 24-week treatment period, with rapid and near-maximal suppression achieved by week 4 and sustained low levels thereafter (Figure 4A,C). For serum total IgE level, a marked reduction was observed after week 4 of treatment, with the magnitude of reduction progressively increasing over the course of 24 weeks (Figure 4B). Concurrently, analysis of blood eosinophils revealed that the mean percent change from baseline in blood eosinophil levels increased over the 24-week treatment period in all treatment groups; this increment was particularly pronounced in the MG-K10 300 mg Q2W group. Consistent with the mean changes, the median percent change also showed an initial increase followed by a gradual decline in both treatment groups, falling below baseline levels by Week 24 (Figure S1A,B).

4 | Discussion

In this phase Ib/II study, MG-K10, a novel, long-acting, anti-IL-4Rα humanised mAb, demonstrated a favourable safety profile

TABLE 2 | Summary of primary and secondary efficacy endpoints (Full Analysis Set, FAS).

	MG-K10, 300 mg, Q2W (*N* = 64)	MG-K10, 300 mg, Q4W (*N* = 60)	Placebo (*N* = 63)
Primary efficacy endpoint: absolute change in the prebronchodilator FEV_1 from baseline at week 12			
FEV_1 (L) at baseline (Mean ± SD)	1.56 ± 0.53	1.49 ± 0.56	1.55 ± 0.64
LS mean change from baseline in FEV_1 (L) at week 12 (SE)	0.407 (0.051)	0.357 (0.053)	0.058 (0.051)
N (miss)	57 (7)	56 (4)	54 (9)
LS mean change difference vs. placebo (95% CI)	0.35 (0.208, 0.490)	0.30 (0.156, 0.441)	
p value vs. placebo	< 0.0001	< 0.0001	
LS mean percent change from baseline in FEV_1(%) at week 12 (SE)	33.364 (4.579)	28.912 (4.767)	12.294 (4.584)
N (miss)	59 (5)	56 (4)	59 (4)
LS mean change difference vs. placebo (95% CI)	21.07 (8.453,33.686)	16.62 (3.801,29.435)	
p value vs. placebo	0.0012	0.0113	
Secondary efficacy endpoint			
LS mean change from baseline in FEV_1 (L) at week 24 (SE)	0.380 (0.053)	0.389 (0.055)	0.052 (0.054)
LS mean change difference vs. placebo (95% CI)	0.33 (0.181, 0.475)	0.34 (0.188, 0.486)	
p value vs. placebo	< 0.0001	< 0.0001	
LS mean percent change from baseline in FEV_1(%) at week 24 (SE)	32.176 (4.695)	32.208 (4.847)	10.899 (4.762)
LS mean change difference vs. placebo (95% CI)	21.28 (8.249,34.305)	21.31 (8.129,34.488)	
p value vs. placebo	0.0015	0.0017	
Annualised rate of severe asthma exacerbations over 24 weeks			
Estimate (95% CI)	0.09 (0.03, 0.31)	0.10 (0.03, 0.33)	0.38 (0.20, 0.73)
Relative risk (RR) vs. placebo (95% CI)	0.25 (0.07, 0.91)	0.27 (0.08, 0.97)	
p value vs. placebo	0.0357	0.0449	
Annualised rate of LOAC over 24 weeks			
Estimate (95% CI)	1.32 (0.78, 2.24)	1.39 (0.82, 2.37)	3.09 (1.94, 4.92)
Relative risk (RR) vs. placebo (95% CI)	0.43 (0.23, 0.78)	0.45 (0.25, 0.82)	
p value vs. placebo	0.0060	0.0097	
LS mean change from baseline in daytime PEF at week 12 (SE)	44.168 (7.053)	33.519 (7.217)	15.030 (7.196)
LS mean change difference vs. placebo (95% CI)	29.14 (9.447, 48.830)	18.49 (−1.427, 38.406)	
p value vs. placebo	0.0039	0.0686	
LS mean change from baseline in nighttime PEF at week 12 (SE)	42.470 (7.091)	27.918 (7.211)	9.629 (7.205)

(Continues)

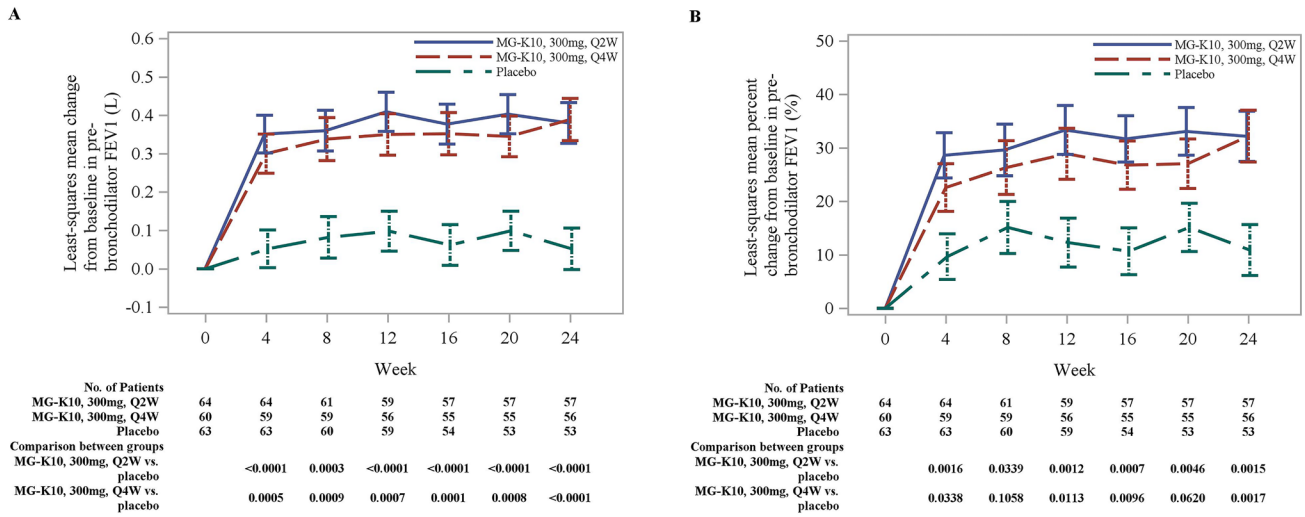
TABLE 2 | (Continued)

	MG-K10, 300 mg, Q2W (*N* = 64)	MG-K10, 300 mg, Q4W (*N* = 60)	Placebo (*N* = 63)
LS mean change difference vs. placebo (95% CI)	32.84 (13.121, 52.562)	18.29 (−1.602, 38.181)	
p value vs. placebo	0.0012	0.0713	
LS mean change from baseline in daytime PEF at week 24 (SE)	45.195 (7.739)	32.178 (7.920)	9.609 (8.001)
LS mean change difference vs. placebo (95% CI)	35.59 (13.788, 57.385)	22.57 (0.524, 44.615)	
p value vs. placebo	0.0015	0.0449	
LS mean change from baseline in nighttime PEF at week 24 (SE)	43.012 (7.699)	33.724 (7.842)	3.779 (7.968)
LS mean change difference vs. placebo (95% CI)	39.23 (17.574, 60.893)	29.95 (8.084, 51.807)	
p value vs. placebo	0.0005	0.0076	
LS mean change from baseline in ACQ-5 score at week 12 (SE)	0.992 (0.096)	−1.040 (0.098)	−0.823 (0.096)
LS mean change difference vs. placebo (95% CI)	0.17 (−0.435, 0.097)	−0.22 (−0.486, 0.051)	
p value vs. placebo	0.2113	0.1117	
LS mean change from baseline in ACQ-5 score at week 24 (SE)	−1.109 (0.107)	−1.257 (0.108)	−0.875 (0.109)
LS mean change difference vs. placebo (95% CI)	−0.23 (−0.534, 0.066)	−0.38 (−0.684, −0.080)	
p value vs. placebo	0.1262	0.0135	

Abbreviations: ACQ-5, 5-item Asthma Control Questionnaire; FEV1, Forced expiratory volume in 1 seconds; LOAC, loss of asthma control; LS, Least squares; PEF, Peak expiratory flow; SD, Standard deviation; SE, Standard error.

and outperformed placebo in multiple outcomes for patients with moderate-to-severe asthma. Across both 300 mg Q2W and 300 mg Q4W dosing regimens, MG-K10 produced statistically significant improvements in lung function, clinical symptoms and asthma control, alongside a marked reduction in the annualised rate of severe asthma exacerbations over a 24-week treatment period. These clinical benefits were evidenced by the achievement of the primary endpoints and supported by all the ranked secondary endpoints throughout the study.

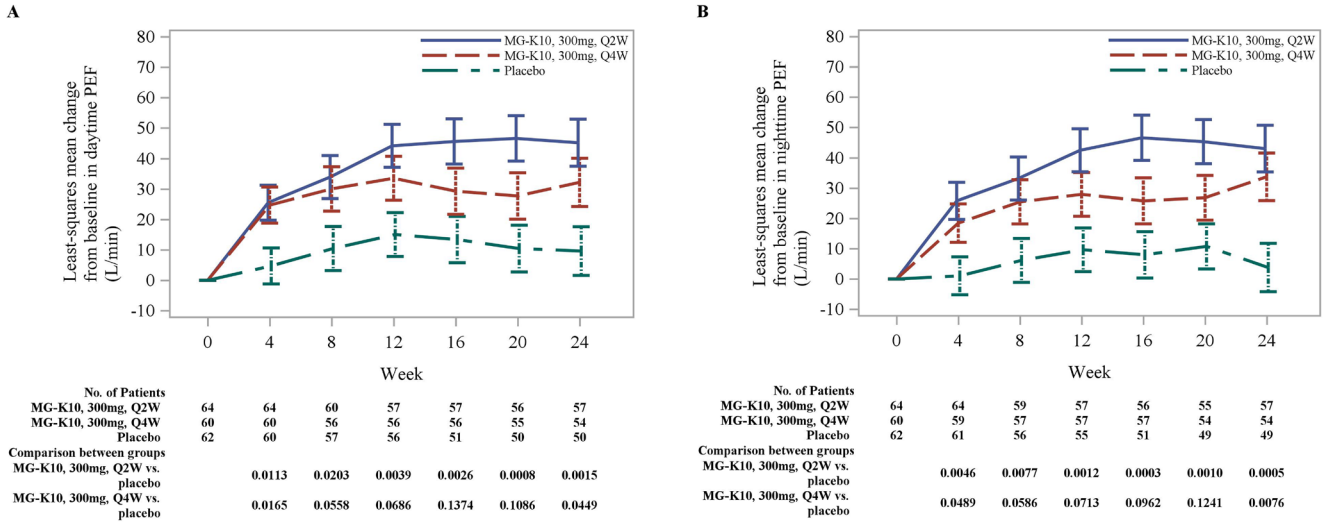
FEV_1 is a crucial measurement to determine the progression of pulmonary function. Concerning the primary efficacy endpoint, patients treated with MG-K10 exhibited a significant improvement in prebronchodilator FEV_1 from baseline at week 12, with the LS mean increase of 0.41 L in the 300 mg Q2W group (vs. matched placebo, 0.35 L, $p < 0.001$) and 0.36 L in the 300 mg Q4W group (vs. matched placebo, 0.30 L, $p < 0.001$). This magnitude of FEV_1 improvement compares favourably with data from the phase III trial of dupilumab [16], an approved IL-4Rα-targeted therapy, where patients treated with 300 mg Q2W dupilumab showed a 0.34 L increase in FEV_1 from baseline at week 12 (vs. placebo: 0.13 L, $p < 0.001$). Notably, the engineered FcRn-binding site mutation in MG-K10 greatly prolonged its half-life, allowing for an extended 4-week dosing interval [20]. Importantly, the 300 mg Q4W regimen achieved efficacy comparable to the Q2W regimen in this study. Consistent with findings of studies of dupilumab [16, 18], patients with baseline blood eosinophil counts $\geq 0.3 \times 10^9$/L experienced greater FEV_1 improvements with MG-K10 treatment. Assessment of FEV_1 and morning/evening PEF over time revealed that both MG-K10 dosing regimens produced statistically significant differences versus placebo as early as week 4, indicating a rapid onset of action with MG-K10, which was sustained over the 24-week treatment period. Enhancing the management of asthma exacerbations would be expected to further lower asthma morbidity and potential mortality [22]. In our overall study population, both MG-K10 dosing regimens significantly reduced the annualised rate and risk of severe exacerbation or LOAC relative to placebo, with greater treatment effects observed as the baseline eosinophil level increased. These trends are also consistent with the results of previous pivotal studies of dupilumab [16, 23]. The ACQ-5 is a validated patient-reported outcome (PRO) tool for assessing asthma symptoms and control. In this study, ACQ-5 scores trended lower than baseline over 24 weeks, with greater improvements in MG-K10 groups than placebo, though not statistically significant. In contrast, the dupilumab QUEST study showed statistically significant ACQ-5 improvements at weeks 12 [16]. Given the high variability of PROs like ACQ-5, a longer



No. of Patients							
MG-K10, 300mg, Q2W	64	64	61	59	57	57	57
MG-K10, 300mg, Q4W	60	59	59	56	55	55	56
Placebo	63	63	60	59	54	53	53
Comparison between groups							
MG-K10, 300mg, Q2W vs. placebo		<0.0001	0.0003	<0.0001	<0.0001	<0.0001	<0.0001
MG-K10, 300mg, Q4W vs. placebo		0.0005	0.0009	0.0007	0.0001	0.0008	<0.0001

No. of Patients							
MG-K10, 300mg, Q2W	64	64	61	59	57	57	57
MG-K10, 300mg, Q4W	60	59	59	56	55	55	56
Placebo	63	63	60	59	54	53	53
Comparison between groups							
MG-K10, 300mg, Q2W vs. placebo		0.0016	0.0339	0.0012	0.0007	0.0046	0.0015
MG-K10, 300mg, Q4W vs. placebo		0.0338	0.1058	0.0113	0.0096	0.0620	0.0017

FIGURE 2 | Improvement in FEV_1 in L (A) and percentage change (%) (B) from baseline to week 24 (Full Analysis Set, FAS). Error bars indicate SE.



No. of Patients							
MG-K10, 300mg, Q2W	64	64	60	57	57	56	57
MG-K10, 300mg, Q4W	60	60	56	56	56	55	54
Placebo	62	60	57	56	51	50	50
Comparison between groups							
MG-K10, 300mg, Q2W vs. placebo		0.0113	0.0203	0.0039	0.0026	0.0008	0.0015
MG-K10, 300mg, Q4W vs. placebo		0.0165	0.0558	0.0686	0.1374	0.1086	0.0449

No. of Patients							
MG-K10, 300mg, Q2W	64	64	59	57	56	55	57
MG-K10, 300mg, Q4W	60	59	57	57	57	54	54
Placebo	62	61	56	55	51	49	49
Comparison between groups							
MG-K10, 300mg, Q2W vs. placebo		0.0046	0.0077	0.0012	0.0003	0.0010	0.0005
MG-K10, 300mg, Q4W vs. placebo		0.0489	0.0586	0.0713	0.0962	0.1241	0.0076

FIGURE 3 | Diurnal variation in peak expiratory flow (PEF) during the 24-week treatment period (Full Analysis Set, FAS). A. The least-squares mean change from baseline in morning PEF; B. The least-squares mean change from baseline in evening PEF. Error bars indicate SE.

observation period or larger sample size may be needed to detect significant changes.

Overall, the incidence of TEAE was similar among the MG-K10 300 mg, Q2W (79.7%), MG-K10 300 mg Q4W (85.0%) and placebo groups (79.4%); however, the placebo group had the highest proportion of grade ≥ 3 drug-related TEAEs (6.3%). The incidence of TRAE was also similar among the three groups, with the lowest incidence of patients receiving MG-K10 300 mg Q4W (23.3% vs. MG-K10, 300 mg, Q2W: 34.4% and placebo: 27.0%). Viral upper respiratory tract infection (17.6%), upper respiratory tract infection (12.2%) and bronchitis (11.2%) have been reported as common TEAEs of dupilumab (300 mg, Q2W) in a study of patients with moderate-to-severe asthma [16]. These TEAEs were also observed with our investigational drug, and there were no unexpected safety concerns that arose. In addition, all patients with eosinophil counts of ≥ 3000/mm^3 during treatment with dupilumab were reported as TEAEs. Of note, eosinophil elevation was also observed in our study, with incidences of 7.8% in the MG-K10 300 mg Q2W group and 6.7% in the 300 mg Q4W group. This phenomenon is consistent with findings from the Phase III study of dupilumab [24], where the incidence of eosinophilia-related adverse events (eosinophil count increase and eosinophilia) was 14% in the dupilumab 300 mg Q2W group versus 1% in the placebo group over 24 weeks, all of which were isolated laboratory findings without clinical consequences. Given that MG-K10 shares the same mechanism of action as dupilumab, this eosinophil elevation may be attributed to the drug blocking IL-4 and IL-13-mediated eosinophil survival, activation and recruitment to tissues but not IL-5-regulated eosinophil export from the bone marrow [16]. Thus, we speculate that initial MG-K10 treatment may induce a transient increase in blood eosinophil counts, which, similar to dupilumab, does not affect clinical efficacy. Interestingly, injection-site reactions occurred in 18.4% of asthmatic patients aged ≥ 12 years receiving dupilumab 300 mg Q2W, with a higher incidence following the first dose [16]. In our study, only 1 patient (1.7%) in the MG-K10 300 mg Q4W group reported such a reaction. This favourable tolerability is further

TABLE 3 | Overview of adverse events (Safety set, SS).

Events	MG-K10, 300 mg, Q2W (*N* = 64)	MG-K10, 300 mg, Q4W (*N* = 60)	Placebo (*N* = 63)	Total (*N* = 187)
Treatment-emergent adverse event (TEAE), *n* (%)	51 (79.7)	51 (85.0)	50 (79.4)	152 (81.3)
Treatment-related adverse events (TRAEs), *n* (%)	22 (34.4)	14 (23.3)	17 (27.0)	53 (28.3)
Grade ≥ 3 TEAEs	3 (4.7)	9 (15.0)	6 (9.5)	18 (9.6)
Grade ≥ 3 TRAEs	0	0	4 (6.3)	4 (2.1)
Serious adverse events (SAEs), *n* (%)	2 (3.1)	7 (11.7)	2 (3.2)	11 (5.9)
Any TEAEs leading to withdrawal from the study, *n* (%)	1 (1.6)	2 (3.3)	3 (3.2)	5 (2.7)
Any TRAEs leading to withdrawal from the study, *n* (%)	0	0	1 (1.6)	1 (0.5)
Any TEAEs leading to discontinuation or permanent discontinuation of the intervention, *n* (%)	1 (1.6)	5 (8.3)	3 (4.8)	9 (4.8)
Any TRAEs leading to discontinuation or permanent discontinuation of the intervention, *n* (%)	0	2 (3.3)	1 (1.6)	3 (1.6)
TEAEs occurring in ≥ 5% of patients in any group, *n* (%)				
Upper respiratory tract infection	17 (26.6)	11 (18.3)	12 (19.0)	40 (21.4)
Urinary tract infection	6 (9.4)	8 (13.3)	6 (9.5)	20 (10.7)
Nasopharyngitis	5 (7.8)	5 (8.3)	7 (11.1)	17 (9.1)
Elevated eosinophils	5 (7.8)	4 (6.7)	5 (7.9)	14 (7.5)
Cough	5 (7.8)	2 (3.3)	4 (6.3)	11 (5.9)
Hyperlipidaemia	5 (7.8)	2 (3.3)	3 (4.8)	10 (5.3)
Fever	3 (4.7)	3 (5.0)	4 (6.3)	10 (5.3)
Elevated white blood cell count	0	5 (8.3)	4 (6.3)	9 (4.8)
Elevated serum creatine phosphokinase	2 (3.1)	4 (6.7)	3 (4.8)	9 (4.8)
Elevated neutrophil count	0	6 (10.0)	3 (4.8)	9 (4.8)
Hyperuricaemia	3 (4.7)	3 (5.0)	1 (1.6)	7 (3.7)
Itching	4 (6.3)	1 (1.7)	2 (3.2)	7 (3.7)
Elevated alanine aminotransferase	1 (1.6)	3 (5.0)	2 (3.2)	6 (3.2)
Urinary occult blood	1 (1.6)	3 (5.0)	2 (3.2)	6 (3.2)
Elevated monocyte count	0	4 (6.7)	1 (1.6)	5 (2.7)
Proteinuria	0	3 (5.0)	1 (1.6)	4 (2.1)

supported by MG-K10's Phase II atopic dermatitis study, where no significant injection-site reactions were observed [25]. While these observations are encouraging, cross-trial comparisons must be interpreted cautiously due to differences in study populations and designs. The low incidence of injection-site reactions with MG-K10 may be partially attributed to its engineered structure and simplified formulation components, which are designed to optimise product stability and patient tolerability. The ongoing Phase III study (NCT06837922), with a larger sample size and longer treatment duration, will provide a more robust assessment of MG-K10's efficacy and safety profile.

In patients with moderate-to-severe asthma, sustained reductions in type 2 inflammatory biomarkers, including serum TARC, FeNO and serum total IgE, were observed over 24 weeks of MG-K10 treatment, providing direct evidence supporting the inhibitory effect of MG-K10 on airway T2-mediated inflammation [26, 27]. FeNO, as a noninvasive biomarker of airway

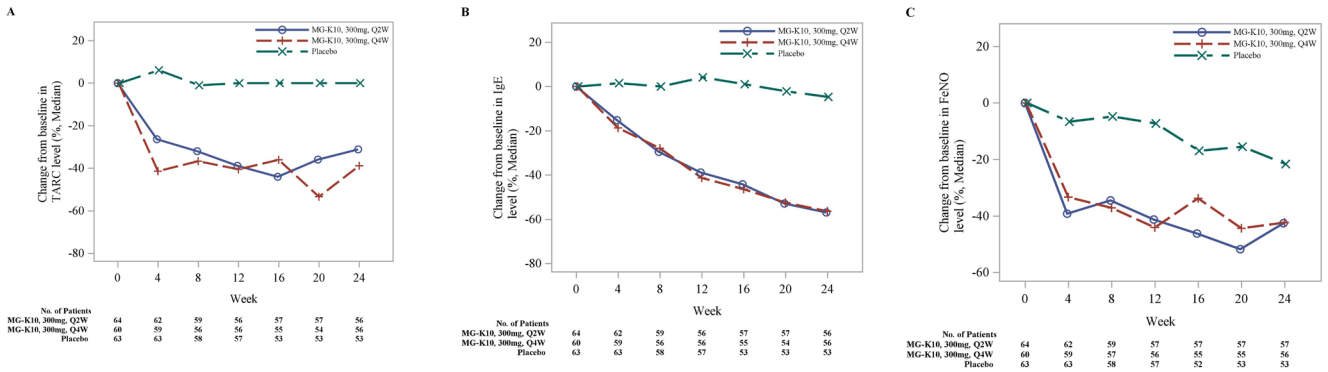


FIGURE 4 | Median change (%) from baseline in biomarker levels of type 2 inflammation in asthma over time. A. Change (%) of serum thymus and activation-regulated chemokine (TARC); B. Change (%) of serum total immunoglobulin E (IgE); C. Change (%) of fractional exhaled nitric oxide (FeNO). Figure S1: Percent change (±SE) from baseline in blood eosinophils. A: Mean percent change (%, Mean); B: Median percent change (%, Median).

inflammation produced by airway epithelial cells in response to IL-13, is a reliable marker of type 2 inflammation; elevated FeNO levels are associated with increased asthma exacerbation risk, corticosteroid insensitivity and serve as a validated predictor of therapeutic response in asthma trials [28, 29]. TARC is a key biomarker of Th2-mediated immunity, with elevated circulating levels in asthma reflecting disease activity and immune dysregulation [30]. Both FeNO and TARC were maximally suppressed by week 4 of MG-K10 therapy and remained suppressed throughout the 24 weeks. Dupilumab, the first anti-IL-4Rα monoclonal antibody approved for asthma, also demonstrated comparable rapid suppression of FeNO levels in prior studies [24, 31]. Notably, a non-head-to-head comparison of the treatment trends indicated that the magnitude of FeNO reduction over 24 weeks in our study was more pronounced than that reported for dupilumab [24], aligning with the robust efficacy of MG-K10 observed in our trial. As an innovative, long-acting anti-IL-4Rα humanised monoclonal antibody, MG-K10 binds to IL-4Rα to block IL-4/IL-13 signalling, thereby inhibiting the differentiation and activation of Th2 cells and ILC2s that drive iNOS-mediated NO production in airway epithelial cells; this dual blockade rapidly suppresses FeNO production and provides a mechanistic basis for its attenuation of airway T2-inflammation, consistent with the sustained reductions in type 2 biomarkers observed in our study [9, 10]. Blood eosinophil levels increased relative to baseline. While IL-4/IL-13 signalling via IL-4Rα drives tissue eosinophil recruitment and activation, an effect blocked by MG-K10 to alleviate airway inflammation and improve FEV. However, MG-K10 does not directly inhibit IL-5, GM-CSF, or IL-3, which regulate bone marrow eosinophil production and maturation. This is a feature shared by all IL-4Rα inhibitors. Consistent with the transient eosinophilia observed with dupilumab [16, 32], the elevation in blood eosinophils with MG-K10 does not compromise clinical efficacy, as blood eosinophils act as a predictive biomarker for treatment selection rather than a dynamic marker of response to anti-IL-4/IL-13 therapy, with efficacy validated by primary and key secondary endpoints including FEV_1 improvement and reduced exacerbation rates.

It should be noted that this study was designed to evaluate the preliminary efficacy of the MG-K10 in patients with moderate-to-severe asthma, thus the results of its two dosing regimens require validation in a large-sample study. A key limitation is the 24-week treatment duration; while the observed efficacy provided a necessary basis for subsequent study, the limited sample size and treatment duration of this Phase II study resulted in insufficient data on the sustained effects of MG-K10, which may have introduced bias in the results regarding the annualised rate of severe exacerbations or events of loss of asthma control. To address these limitations, a confirmatory Phase 3 study (NCT06837922) has been initiated to systematically validate MG-K10's efficacy over a 52-week treatment period (including 8 weeks of follow-up) and further assess its long-term safety and response durability.

5 | Conclusion

In conclusion, the results of this phase Ib/II study confirmed that subcutaneous injection of MG-K10 at a dose of 300 mg, either every 2 weeks or every 4 weeks, significantly improves lung function and asthma symptoms, reduces severe exacerbations and exhibits a favourable safety profile in patients with moderate-to-severe asthma. Notably, the 4-week dosing interval offers advantages of extended dosing cycle, lower treatment costs and improved patient adherence, with comparable efficacy to the 2-week dosing regimen.

Author Contributions

M.X. and X.S. researched the data and wrote the first draft of the manuscript. M.X., X.S., R.L., L.Z., B.Z., B.C., H.S., Z.Y.W., F.W., J.G., Y.C. and J.L. collected the data. W.D., J.L.G., D.Q. and C.H.Z. analysed the data. M.X., X.S., D.Q., C.H.Z. and J.L. interpreted the results. M.X., X.S., D.Q., CH.Z., J.L. and N.S.Z. conceptualised, designed and supervised the study. All authors contributed to and approved the final version of the manuscript before submission.

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Shanghai Mabgeek Therapeutics Co. Ltd. and were involved in concept and design, data analysis and manuscript preparation.

Conflicts of Interest

Wei Dang, Jinlin Guo, Di Qin and Chenghai Zhang are full-time employees of Shanghai Mabgeek Therapeutics Co. Ltd. The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Percent change (±SE) from baseline in blood eosinophils. A: Mean percent change (%, Mean); B: Median percent change (%, Median). **Data S1:** MG-K10V1.1 Study Protocol-Chinese Version. **Data S2:** MG-K10V1.1 Study Protocol-English Version. **Table S1:** List of study sites. **Table S2:** Patients' eligibility criteria. **Table S3:** Subgroup analyses of primary and secondary efficacy endpoints by blood eosinophil count.