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Review Article

Effects of montelukast on allergic rhinitis in children: a systematic review and meta-analysis

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Abstract: Objective: To systematically evaluate the efficacy and safety of montelukast in the treatment of allergic rhinitis (AR) in children through a meta-analysis. Methods: Pubmed, Embase, and Cochrane Library were retrieved for randomized controlled trials and cohort studies on montelukast for pediatric AR from databases inception to June 2025. Two researchers independently screened the literature, extracted data, and evaluated the risk of bias. Meta-analysis was conducted using RevMan5.3. Effect size was expressed as weighted mean difference (WMD) or odds ratio (OR) with 95% confidence interval (CI). Result: A total of 14 studies were included. Those treated with non-montelukast regimens were set as Group A, and those treated with montelukast were set as Group B. Meta-analysis showed that the total effective rate in group B was significantly higher than that in group A [*OR*=0.32 (0.20, 0.50), *P*<0.001]. The reduction in the Total Nasal Symptom Score (TNSS) was significantly greater in group B [*WMD*=0.83 (0.13, 1.54), *P*=0.02]. There was no significant difference in the incidence of adverse reactions between the two groups [*OR*=1.00 (0.71, 1.39), *P*=0.98]. In addition, no significant differences were observed between the two groups in immunoglobulin E (IgE) levels and the percentage of eosinophils (EOS). Conclusion: Montelukast improves the total effective rate and clinical symptoms of pediatric AR with a favorable safety profile.

Keywords: Montelukast, children, allergic rhinitis, meta-analysis

Introduction

Allergic rhinitis (AR) is a type I hypersensitivity reaction mediated by immunoglobulin E (IgE), involving immune-active cells and cytokines in genetically susceptible individuals upon exposure to allergens. Its main clinical manifestations include nasal itching, sneezing, clear nasal discharge, and nasal congestion [1]. Children are the primary affected population of AR. A phase III clinical study from the International Children's Allergy Center reported that AR typically begins early in life, with a prevalence exceeding 5% in 3-year-olds, 8.5% in 6-7-year-olds, and rising to 14.6% in 13-14-year-olds [2]. In recent years, environmental changes and lifestyle shifts have contributed to a global increase in childhood AR [3].

Although AR in children is not life-threatening, its symptoms are recurrent. Without timely and standardized treatment, complications such as adenoid hypertrophy, secretory otitis media, and obstructive sleep apnea-hypopnea syndrome may develop [4]. Current treatment of AR primarily relies on pharmacotherapy, including antihistamines, intranasal corticosteroids, and leukotriene receptor antagonists (LTRA). Leukotrienes are key inflammatory mediators in the pathogenesis of AR; thus, antagonizing leukotriene receptors has become an important therapeutic strategy [5].

Montelukast, a highly selective LTRA, is regarded as a safe and effective anti-inflammatory agent and has been recommended for a range of inflammation-related respiratory diseases [6]. Numerous experimental and clinical studies have confirmed the efficacy of montelukast in the treatment of AR. However, given the diversity of treatment options for pediatric AR and individual variability in therapeutic response, the efficacy and safety of montelukast in children remain to be fully clarified. Systematic reviews and meta-analyses, as advanced forms

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Table 1. Search strategy (PubMed)

Steps	
#1	((((Montelukast[Supplementary Concept]) OR (MK 0476[Title/Abstract])) OR (Singular[Title/Abstract])) OR (MTLU[Title/Abstract])) OR (montelukast sodium[Title/Abstract])
#2	(((Rhinitis, Allergic[MeSH Terms]) OR (Allergic Rhinitis[Title/Abstract])) OR (Rhinitis, Allergic[Title/Abstract])) OR (Allergic Rhinitis[Title/Abstract])
#3	(((((((Child[MeSH Terms]) OR (Adolescent[MeSH Terms])) OR (Children[Title/Abstract])) OR (Adolescents[Title/Abstract])) OR (Adolescence[Title/Abstract])) OR (Youths[Title/Abstract])) OR (Teens[Title/Abstract])) OR (Teenagers[Title/Abstract])
#4	#1 AND #2 AND #3

of evidence-based medicine, can synthesize existing data and provide reliable basis for clinical decision-making. Therefore, this study aims to systematically evaluate the efficacy and safety of montelukast in the treatment of pediatric AR through a comprehensive literature search, critical appraisal, and meta-analysis.

Methods

Register

This study was registered in PROSPERO (CRD-420251107444).

Eligibility criteria

Inclusion criteria: (1) Study design: Randomized controlled trials (RCTs) or cohort studies; (2) Population: Children (≤18 years) diagnosed with AR without severe comorbidities; (3) Intervention: Montelukast alone or in combination with other drugs, with no restriction on dosage or treatment course (defined as Group B); (4) Comparator: Placebo, conventional treatment, or other active drugs (defined as Group A); (5) Outcomes: At least one primary outcome measure reported.

Exclusion criteria: (1) Non-clinical research, such as in vitro experiments and animal experiments; (2) Case reports, reviews, conference abstracts, or other studies lacking original data; (3) Studies with unclear intervention measures, such as montelukast combined with complex therapies where effects cannot be separated; (4) Studies with incomplete data or unextractable data; (5) Studies involving mixed populations without subgroup data for children. (6) Duplicate publications.

Information sources

PubMed, Embase, and Cochrane Library were retrieved for clinical studies on montelukast in the treatment of pediatric AR. Reference lists of relevant articles were also traced to identify additional studies. The search date covered all records from database inception to June 2025.

Search strategy

The search strategy was based on the PICOS principle. Key words included *montelukast*, *allergic rhinitis*, and *Child*. Subject headings were combined with free-text terms, and the strategy was adapted for each database. The specific PubMed search strategy is shown in **Table 1**.

Selection process

Wang and Liu independently screened and cross-checked the literature. Titles and abstracts were first reviewed to exclude literature not meeting the eligibility criteria. Full texts were then assessed against the inclusion and exclusion standards to determine final eligibility. After screening, results were cross verified. Any disagreements were resolved through discussion with Hu.

Data collection process

Wang and Hu independently extracted data using a standardized data-extraction sheet. Extracted information included basic study characteristics (e.g., author, publication year, country, study design, etc.), study details (e.g., sample size, age, gender, administration regimen, course of treatment, and outcome measures, etc.), and methodological quality (e.g.,

design schemes, inclusion and exclusion criteria, and bias-prevention methods, etc.). After extraction, data were cross-checked, and discrepancies were resolved through consultation with a third reviewer.

Outcome measures

Primary outcomes: (1) Total effective rate: the proportion of patients whose symptoms improved, significantly improved, or completely resolved after treatment; (2) Total Nasal Symptom Score (TNSS): changes in TNSS reported in each study. If not directly provided, differences were calculated from pre- and post-treatment data. A greater reduction indicated better symptom improvement; (3) Adverse reactions: the overall incidence of treatment-related adverse events (e.g., headache, gastrointestinal reactions, rash).

Secondary outcomes: Changes in immunoglobulin E (IgE) levels and the percentage of eosinophils (EOS) before and after treatment.

Study risk of bias assessment

Wang and Liu independently evaluated the quality of RCTs and cohort studies using the Cochrane Risk of Bias tool and the Newcastle-Ottawa Scale (NOS), respectively. The Cochrane tool evaluates risk across six domains: selection bias, implementation bias, measurement bias, attrition bias, reporting bias, and other biases. It comprises seven items, each rated as "low risk", "unclear risk", or "high risk". The NOS evaluates observational studies across three domains: selection (4 items), comparability (1 item), and exposure (3 items), totaling 8 items. Scoring adopts a semi-quantitative "star" system: except for the comparability domain (maximum of 2 stars), each item may receive up to 1 star, with each star representing 1 point. The maximum score is 9, and studies scoring ≥6 points are considered as high quality. After completing the quality assessment, results were cross-checked. Any disagreements were resolved through discussion with Hu.

Statistical methods

Quantitative outcomes were analyzed using the weighted mean difference (WMD), and qualitative outcomes using odds ratio (OR). All effect sizes were presented with corresponding 95% confidence intervals (CI).

Statistical analyses were performed using RevMan5.3 software. Heterogeneity was analyzed using the I^2 test. When $I^2 \leq 50\%$ and $P \geq 0.05$, indicating low homogeneity, a fixed-effect model was applied. When $I^2 > 50\%$ or $P < 0.05$, suggesting significant heterogeneity, potential sources were explored from both methodological and clinical perspectives. If the source of heterogeneity could not be identified or eliminated, a random-effect model was used. The robustness of the results was evaluated using sensitivity analysis.

Publication bias was evaluated by funnel plot inspection. Asymmetry of the funnel plot was considered suggestive of potential publication bias.

Results

Study selection

A total of 161 records were retrieved. Thirty-two duplicates were manually removed. A preliminary review of titles and abstracts screened 93 literatures with inconsistent research types and contents, which were excluded. The full texts of remaining 36 articles were retrieved for detailed evaluation. Eight articles were excluded due to unavailable full text. Among the 28 accessible studies, 4 lacked relevant outcome indicators, and 10 had incomplete or unextractable data. Ultimately, 14 studies met the eligibility criteria and were included in the meta-analysis. The study selection process is shown in **Figure 1**.

Study characteristics

The included studies were published between 2004 and 2025, predominantly conducted in China. Participants were children with a mean age of 4-9 years, and a generally balanced gender distribution. Montelukast dosage ranged from 4 to 10 mg, and the treatment course varied from 2 weeks to 16 weeks. Interventions in Group B included montelukast alone or in combination with other drugs, such as loratadine, levobastine, mometasone furoate, or budesonide. Sample sizes ranged from 11 to 200 cases, all of which were of relatively small sample scale. Detailed information is shown in **Table 2**.

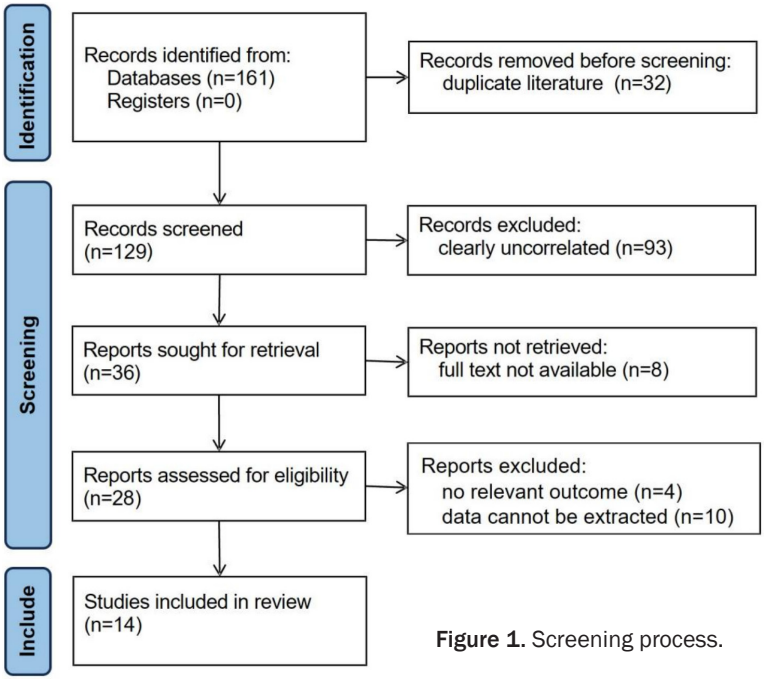

Figure 1. Screening process.

Risk of bias

Ten studies were RCTs. All reported complete data with no evidence of selective reporting. One study did not describe its randomization procedure, resulting in an unclear risk of bias. Three studies did not mention blinding of participants and personnel, and one reported blinding only of investigators. Given the differences in treatment regimens between groups, these were judged to carry a high risk of performance bias. None of the RCTs described the blinding of outcome evaluators, and the corresponding risk of bias was therefore unclear. Details are presented in **Figure 2**.

Four studies were retrospective cohort studies. All demonstrated appropriate selection and grouping of participants, with adequate adjustment for potential confounders. Outcome assessment methods were accurate and reliable, and no loss to follow-up was reported. Follow-up period was at least one month in all studies. Detailed information is provided in **Table 3**.

Meta-analysis results

Total effective rate of treatment: Seven studies [13-17, 19, 20] reported the total effective rate, involving 723 patients (357 in Group A and 366 in Group B). Heterogeneity was low (I^2=17%, *P*=0.30), and a fixed-effect model was used. The pooled results showed that the total effective rate in group A was 79.27% (283/357), and that in Group B was 92.35% (338/366). Group B demonstrated a significantly higher effective rate than Group A [*OR*=0.32 (0.20, 0.50), *P*<0.001] (**Figure 3**).

TNSS: Eight studies [9, 10, 12-14, 17-19] reported changes in TNSS scores, involving 1001 patients (499 in Group A and 502 in Group B). Substantial heterogeneity was observed (I^2=98%, *P*< 0.001), and its source could not be identified; therefore, a random-effects model was applied. The pooled results showed that the reduction in TNSS score was greater in Group B than in Group A [*WMD*=0.83 (0.13, 1.54), *P*=0.02] (**Figure 4**). Sensitivity analysis, performed by sequentially excluding each study, showed no change in the direction of the combined results, supporting the robustness of the findings (**Table 4**).

IgE level: Three studies [7, 8, 14] reported the changes in serum IgE levels, involving 227 patients (112 in Group A and 115 in Group B). Heterogeneity was low (I^2=46%, *P*=0.12), supporting the use of a fixed-effect model. The pooled results demonstrated no significant difference in IgE level alterations between the two groups [*WMD*=45.13 (-0.28, 90.55), *P*=0.05] (**Figure 5**).

EOS percentage: Three studies [7-9] assessed changes in the percentage of EOS, involving 217 patients (108 in Group A and 109 in Group B). Significant heterogeneity was present (I^2=92%, *P*<0.001), with no identifiable source; therefore, a random-effects model was employed. The meta-analysis revealed no significant difference in EOS percentage change between the two groups [*WMD*=8.41 (-9.04, 25.88), *P*=0.35] (**Figure 6**). Sensitivity analysis showed that sequential exclusion of individual studies did not alter the direction of the results, suggesting robustness of the results, as shown in **Table 5**.

Table 2. Characteristics of the included literature

Study	Country	Sample size		Age		Sex (male/female)	
		Group A	Group B	Group A	Group B	Group A	Group B
Hsieh 2004① [7]	China	20	20	8.05±2.39	8.20±1.96	12/8	13/7
Hsieh 2004② [7]		20		8.05±1.82		11/9	
Chen 2006① [8]	China	20	20	4.53±0.91	4.49±1.09	12/8	11/9
Chen 2006② [8]		20		4.36±0.84		9/11	
Razi 2006 [9]	Türkiye	28	29	-	-	14/14	18/11
Hung 2007① [10]	China	11	11	7.81±1.94	8.45±1.51	6/5	5/6
Hung 2007② [10]		12		9.00±2.96		6/6	
Hung 2007③ [10]		11		9.09±2.03		5/6	
Li 2009 [11]	China	22	22	-	-	14/8	10/12
Mahajan 2012 [12]	India	200	200	-	-	124/76	128/72
Yang 2018 [13]	China	63	63	8.63±2.07	8.57±2.12	34/29	33/30
Wu 2020 [14]	China	32	35	7.23±1.42	7.29±1.56	19/13	19/16
Dai 2022 [15]	China	47	42	7.53±1.7	7.43±2.31	26/21	20/22
Guo 2023 [16]	China	54	60	-	-	34/20	30/30
Mao 2023 [17]	China	51	51	9.03±6.98	8.08±6.91	26/25	27/24
Ghanbari 2024① [18]	Iran	16	13	7.08±4.28	6.61±3.64	8/8	3/10
Ghanbari 2024② [18]		15		8.36±2.83		9/6	
Yang 2025 [19]	China	60	65	7.57±1.70	7.63±1.97	33/27	35/30
Li 2025 [20]	China	50	50	7.00±2.59	7.84±2.42	23/27	25/25

Study	Medication		Montelukast dose	Treatment course	Outcome
	Group A	Group B			
Hsieh 2004① [7]	Cetirizine	Montelukast	5 mg	12w	cde
Hsieh 2004② [7]	Placebo				
Chen 2006① [8]	Cetirizine	Montelukast	4 mg	12w	cde
Chen 2006② [8]	Placebo				
Razi 2006 [9]	Placebo	Montelukast	5 mg	2w	bde
Hung 2007① [10]	Loratadine	Loratadine + Montelukast	5 mg	8w	b
Hung 2007② [10]	Loratadine + Sodium chromate				
Hung 2007③ [10]	Loratadine + Budesonide				
Li 2009 [11]	Placebo + Fexofenadine	Montelukast + Fexofenadine	5-10 mg	16w	e
Mahajan 2012 [12]	Levocetirizine	Montelukast	5 mg	6w	be
Yang 2018 [13]	Yupingfeng Granules	Montelukast	5-4 mg	4w	ae
Wu 2020 [14]	Ketotifen Tablets	Ketotifen Tablets + Montelukast	5 mg	2w	abc
Dai 2022 [15]	Mometasone furoate + Loratadine	Mometasone furoate + Montelukast	5 mg	3m	ae

Guo 2023 [16]	Budesonide + Mometasone furoate	Budesonide + Montelukast	5-10 mg	1m	ae
Mao 2023 [17]	Sublingual immunization	Sublingual Immunization + Montelukast	5 mg	1m	abe
Ghanbari 2024① [18]	Desloratadine (qd)	Montelukast	5 mg	2w	b
Ghanbari 2024② [18]	Desloratadine (bid)				
Yang 2025 [19]	Levocabastine	Levocabastine + Montelukast	-	4w	ae
Li 2025 [20]	Budesonide	Budesonide + Montelukast	4-5 mg	8w	abe

-: not described; qd: quaque die; bid: bis in die; w: weeks; m: months; a: total effective rate of treatment; b: total nasal symptom score; c: immunoglobulin E level; d: eosinophils percentage; e: adverse reactions.

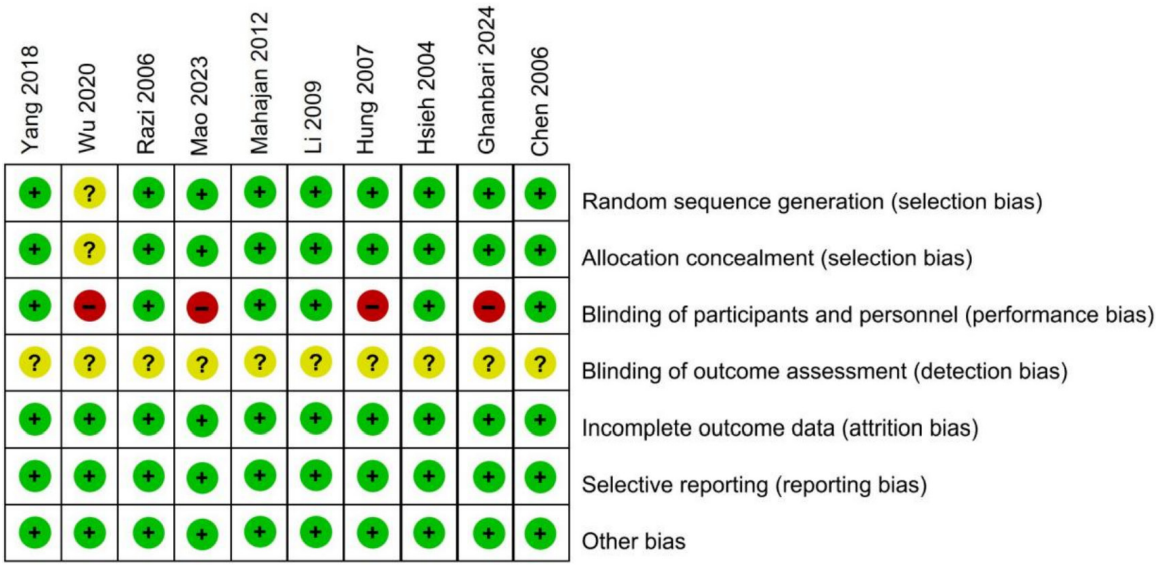

Figure 2. Quality evaluation of randomized controlled trials.

Table 3. Quality evaluation of cohort studies

Study	Selection	Comparability	Exposure	Scores
Dai 2022 [15]	☆☆☆☆	☆	☆☆☆	8
Guo 2023 [16]	☆☆☆☆	☆	☆☆☆	8
Yang 2025 [19]	☆☆☆☆	☆	☆☆☆	8
Li 2025 [20]	☆☆☆☆	☆	☆☆☆	8

☆: indicates 1 point.

Adverse reactions: Ten studies [7-9, 11, 12, 15-17, 19, 20] reported adverse reactions, involving 1191 patients (592 in Group A and 599 in Group B). No significant heterogeneity was observed (I^2=0%, *P*=0.48); therefore, a fixed-effect model was applied. The pooled results showed no significant difference in the incidence of adverse reactions between the two groups [*OR*=1.00 (0.71, 1.39), *P*=0.98] (**Figure 7**).

Reporting bias assessment

Funnel plots were drawn for the total effective rate and adverse reactions, as shown in **Figure 8A**, **8B**. All the included literatures were within the 95% CI; however, their distribution was significantly asymmetrical, suggesting the potential presence of publication bias.

Discussion

At present, there is no definitive cure for AR, largely because its pathogenesis remains incompletely understood [21]. Traditionally, AR has been considered an immune-mediated disorder involving multiple immune cells, cytokines, and inflammatory mediators. A central mechanism is the imbalance between Th1 and Th2 immune responses. Allergens bind to the IgE-FcεRI complex on the surface of mast cells and basophils, leading to release of histamine, leukotrienes, and other inflammatory mediators [22, 23].

Leukotrienes are key inflammatory mediators, comprising leukotriene B4, C4, D4, and E4. The latter three are collectively referred to as cysteine leukotrienes (CysLTs). These mediators exert their biological functions by binding to specific leukotriene receptors (LTRS) on cell membrane. LTRS are categorized into two main types: BLT receptors, activated by leukotriene B4, and CysLT receptors, activated by CysLTs. CysLT receptors belong to the G protein-coupled receptor family and are further classified into CysLT1 and CysLT2 receptors according to their sensitivity to classical antagonists: receptors sensitive to classical antagonists are defined as CysLT1 receptors, and those insensitive are defined as CysLT2 receptors. CysLTs contribute to the pathogenesis of respiratory allergic diseases mainly by binding to CysLT1 receptor, leading to bronchoconstriction, increased vascular permeability, enhanced mucus secretion, and airway remodeling [24, 25].

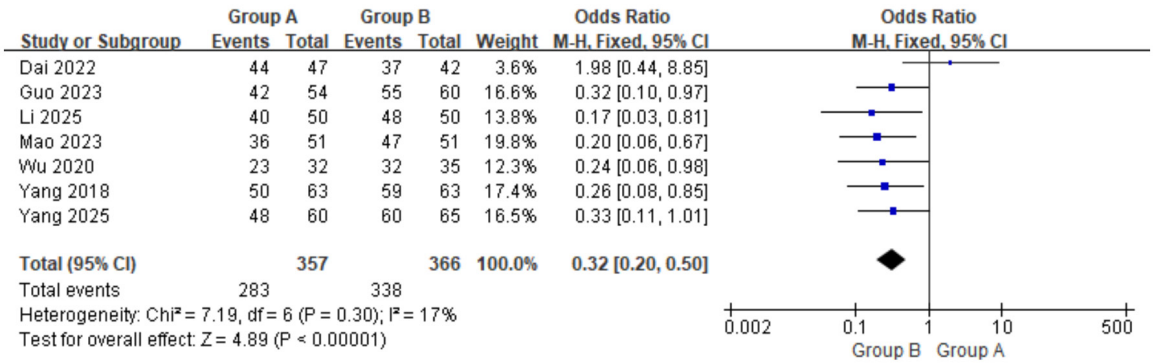

Figure 3. Forest plot analyzing overall treatment efficacy. CI: confidence interval.

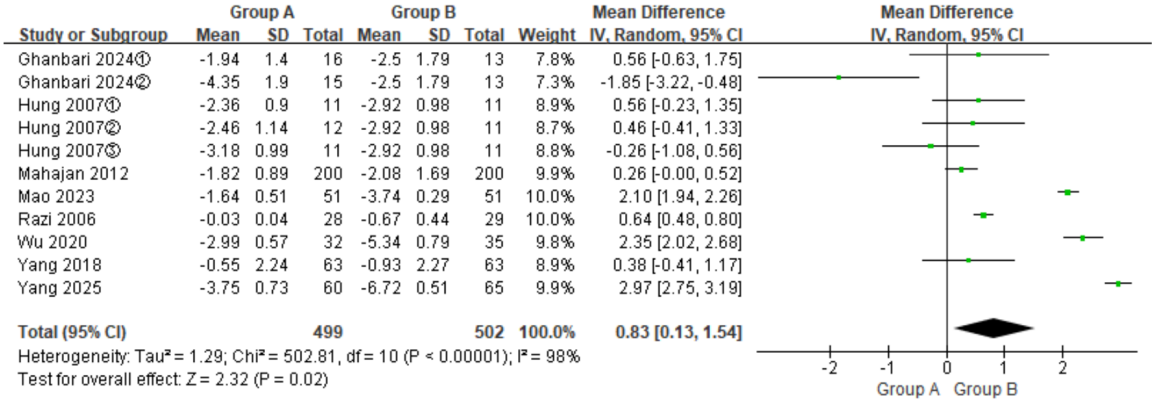

Figure 4. Forest plot analyzing total nasal symptom score (TNSS). CI: confidence interval.

Table 4. Sensitivity analysis of studies reporting TNSS

Exclusion studies	I^2	*WMD*	*P*
Ghanbari 2024① [18]	98%	0.86 (0.12, 1.59)	0.020
Ghanbari 2024② [18]	98%	1.05 (0.33, 1.77)	0.004
Hung 2007① [10]	98%	0.86 (0.12, 1.60)	0.020
Hung 2007② [10]	98%	0.87 (0.13, 1.61)	0.020
Hung 2007③ [10]	98%	0.94 (0.21, 1.67)	0.010
Mahajan 2012 [12]	98%	0.90 (0.17, 1.63)	0.020
Mao 2023 [17]	98%	0.68 (-0.16, 1.51)	0.110
Razi 2006 [9]	97%	0.85 (0.10, 1.61)	0.030
Wu 2020 [14]	98%	0.67 (-0.10, 1.43)	0.090
Yang 2018 [13]	98%	0.88 (0.14, 1.62)	0.020
Yang 2025 [19]	97%	0.62 (-0.04 1.28)	0.070

WMD: weighted mean difference; TNSS: total nasal symptom score.

Montelukast is a highly selective CysLT1 receptor antagonist approved for the treatment of seasonal AR in adults and children ≥2 years, and for perennial AR in adults and children ≥6 months [26]. The prevalence of AR in children is significantly higher than that in adults [27]. However, most pediatric patients cannot accurately express their subjective feelings, and their clinical manifestations are often atypical, leading to the misdiagnosis as common cold. Moreover, high-quality evidence supporting individualized treatment for pediatric AR remains insufficient.

This meta-analysis included 14 studies evaluating the efficacy of montelukast in treating pediatric AR. The results showed that montelukast significantly improved the total effective rate and alleviated nasal symptoms. Mechanistically, montelukast, as a highly selective CysLT1 receptor antagonist, alleviates bronchoconstriction, vascular permeability, and mucus hypersecretion by competitively blocking the binding of cysteinyl leukotriene to their receptors and inhibiting downstream inflammatory pathways. Recent

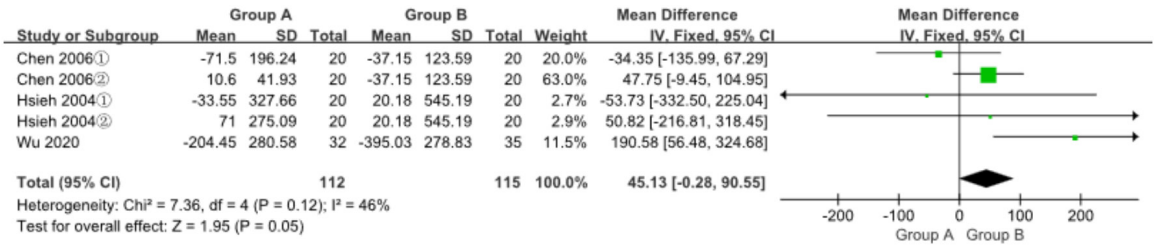

Figure 5. Forest plot analyzing immunoglobulin E (IgE) levels. CI: confidence interval.

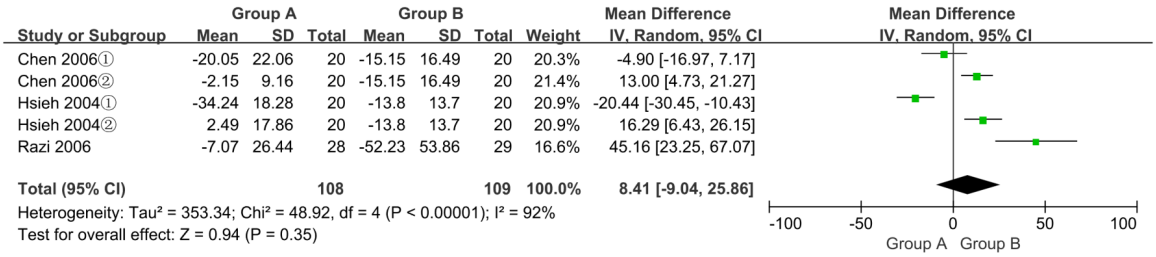

Figure 6. Forest plot analyzing eosinophils (EOS) percentage. CI: confidence interval.

Table 5. Sensitivity analysis of studies reporting EOS percentage

Exclusion studies	I^2	*WMD*	*P*
Chen① [8]	93%	12.05 (-9.48, 33.59)	0.27
Chen② [8]	93%	7.65 (-15.66, 30.95)	0.52
Hsieh① [7]	82%	15.05 (1.04, 29.07)	0.04
Hsieh② [7]	93%	6.65 (-15.23, 28.53)	0.55
Razi [9]	91%	1.14 (-15.88, 18.16)	0.90

WMD: weighted mean difference; EOS: eosinophil.

studies have further demonstrated its anti-inflammatory effects, including inhibition of 5-lipoxygenase activity, reduction of leukotriene synthesis, suppression of NF-κB pathway activation, antagonism of P2Y receptor signaling, and inhibition of EOS adhesion [28-30]. These multi-target mechanisms may underlie its efficacy in AR treatment.

In the pooled analysis of overall treatment efficacy, all included studies administered montelukast in combination with other agents. Notably, for symptom scores, only Razi [9] and Ghanbari [18] investigated montelukast as monotherapy, while the remaining studies assessed combination therapy. The improvement in TNSS in these two monotherapy studies was less pronounced than that in studies using combination therapy. Moreover, in Ghanbari's study [18], montelukast outperformed desloratadine once daily but was less effective than desloratadine twice daily in relieving nasal symptoms. These findings suggest that montelukast combined with antihistamines, immunotherapy, or other treatments provides clear therapeutic effects for pediatric AR, with greater efficacy than monotherapy.

A meta-analysis comparing antihistamines and montelukast in AR reported that montelukast was slightly inferior to antihistamines for overall symptom control but offered greater benefit for nocturnal symptoms; their combination yielded enhanced synergistic effects [31]. Another systematic review on childhood asthma and AR also found montelukast superior to placebo in symptom control, although inhaled corticosteroids achieved better outcomes [32]. In recent years, combination therapy has emerged as a promising approach for AR, achieving therapeutic effects beyond simple additive action through complementary mechanisms.

Safety is key consideration in pediatric pharmacotherapy. The pooled results of this meta-analysis showed no significant difference in the incidence of adverse reactions between the two groups, indicating that montelukast has a favorable safety profile for the treatment of AR in children. Across the included studies, common adverse events included headache, gas-

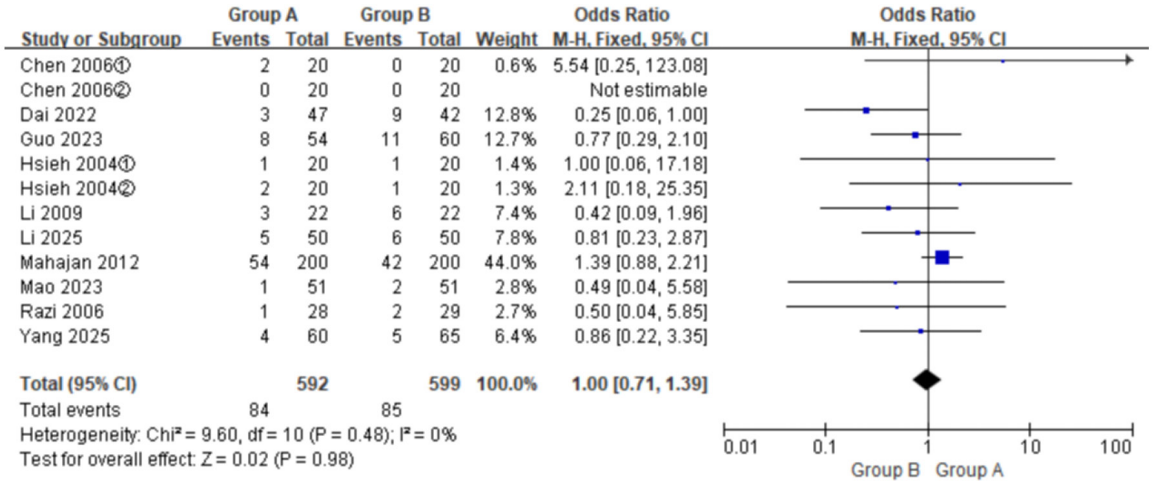

Figure 7. Forest plot analyzing adverse reactions. CI: confidence interval.

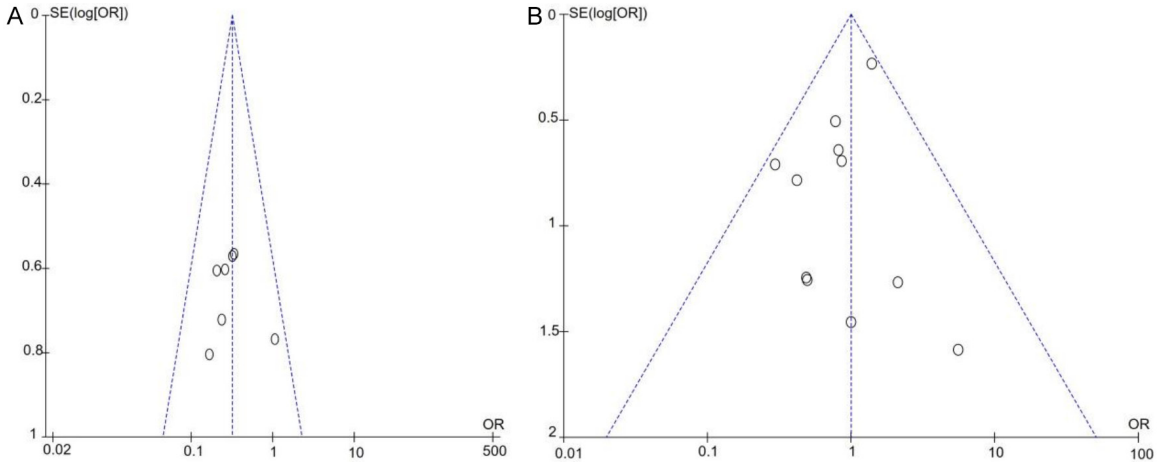

Figure 8. Funnel plot. A: Overall treatment efficacy; B: Adverse reactions; OR: odds ratio; SE(log[OR]): standard error of the logarithm of the odds ratio.

trointestinal reactions, and rash, all with low incidence. Most were mild to moderate and resolved without special treatment. The safety advantage of montelukast supports its clinical value, especially for those who cannot tolerate intranasal corticosteroids or require long-term treatment. Its convenient oral administration also minimizes the discomfort of nasal preparations and may improve adherence.

Although montelukast demonstrated clear benefits in alleviating clinical symptoms, this study did not identify significant effects on IgE levels and EOS percentages. IgE is a core mediator in AR pathogenesis, while EOS is a major effector cell in allergic inflammation. The neutral results on these two indicators suggest that montelukast primarily exerts its action by blocking leukotriene-mediated inflammatory pathways rather than directly regulating IgE production or EOS counts. This is consistent with the pharmacological characteristics of leukotriene receptor antagonists, which mainly inhibit the binding of CysLTs to their receptors, thereby alleviating downstream inflammation rather than modulating upstream immune regulation [33, 34]. Future studies can further explore the effects of montelukast on specific inflammatory cytokines (e.g., IL-4, IL-5, and IL-13) to comprehensively reveal its mechanism of action.

Limitations of this study: (1) Most of the included studies were conducted in China. This geographical concentration may have introduced bias related to genetic background, environmental factors, or local medical practices, po-

tentially limiting the generalizability of the conclusions; (2) The sample sizes of the included studies were relatively small, which may affect the accuracy and reliability of the pooled results; (3) Some studies adopted combination therapy regimens. Variations in dosage and concomitant medications may have influenced outcomes, making it difficult to determine the optimal dose and treatment duration for montelukast monotherapy; (4) Funnel plot revealed an asymmetric distribution, suggesting the possible absence of unpublished studies with negative results, which could lead to an overestimation of the effect size.

Conclusion

The systematic review and meta-analysis suggest that montelukast can increase the overall treatment efficacy for pediatric AR and improve clinical symptoms, with a favorable safety profile. However, its effect on inflammatory markers is limited. In clinical practice, physicians can rationally select montelukast as monotherapy or in combination with other drugs based on the child's specific condition, including symptom severity, comorbidities, and drug tolerance. Given the limitations of the present study, future research should focus on large-sample and long-term RCTs to further validate the efficacy and safety of montelukast and to determine its optimal dosage and treatment course. Additionally, mechanistic studies are warranted to elucidate its anti-inflammatory pathways and to identify optimal combination regimens with other drugs to enhance the therapeutic outcomes in pediatric AR. Furthermore, attention should be directed to the long-term effects of montelukast on growth, development, and the nervous system to provide more comprehensive evidence for its safe clinical use in children.

Disclosure of conflict of interest

None.

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