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Original Research Article

Serum Magnesium Levels in Asthmatic Children and its Correlation with Severity of Asthma – A Hospital-based Cross-sectional Study

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Abstract

Background: Magnesium modulates airway smooth-muscle tone and inflammation, yet the relationship between routine serum magnesium levels and pediatric asthma severity and control remains insufficiently defined.

Objectives: To determine the levels of serum magnesium among asthmatic children; and to correlate levels of serum magnesium with severity of asthma.

Methods: This single-centre, hospital-based analytical cross-sectional study was conducted in the Department of Paediatrics, Melmaruvathur Adhiparasakthi Institute of Medical Sciences & Research (September 2023–March 2024) among children aged 5–18 years with GINA-defined asthma after informed consent/assent. Data were analysed using SPSS v27.

Results: In 200 asthmatic children (mean age 10.8 ± 3.6 y; 58% male; 62% urban), control was suboptimal (ACT/C-ACT 19.2 ± 4.6; 46% uncontrolled) with mean FEV_1 82.2 ± 14.6% (36% <80%). By GINA, 48% were mild, 37% moderate, 15% severe. Mean serum magnesium was 1.86 ± 0.24 mg/dL (median 1.85; IQR 1.72–1.99); 21.5% had hypomagnesemia. Magnesium fell with increasing severity (mild 1.92; moderate 1.83; severe 1.74 mg/dL), with significant pairwise differences ($p=0.021$; $p<0.001$; $p=0.038$) and rising hypomagnesemia (10.4%→25.7%→46.7%). Magnesium correlated with FEV_1 % predicted ($r=0.32$) and ACT/C-ACT ($r=0.28$), both $p<0.001$. Adjusted analyses showed lower odds of moderate–severe asthma per 0.1 mg/dL higher magnesium (OR 0.85; 95% CI 0.76–0.95) and fewer frequent exacerbations (OR 0.88; 95% CI 0.79–0.98). ROC performance for moderate–severe disease was fair (AUC 0.71; cutoff ≤1.80 mg/dL; sensitivity 68%, specificity 66%).

Conclusion: Lower serum magnesium was independently associated with poorer asthma status – lower FEV_1, worse control, and higher odds of moderate–severe disease – supporting its value as a simple, supportive biomarker alongside guideline-based assessment.

Keywords: Asthma, Child, Magnesium, Forced Expiratory Volume, Disease Severity.

Introduction

Asthma remains one of the most prevalent chronic non-communicable diseases in childhood, contributing substantially to symptoms, impaired quality of life, missed school days, and health-care utilization worldwide.[1] Contemporary estimates indicate hundreds of millions of people are affected globally, with a rising burden across regions and heterogeneous prevalence between and within countries, underscoring the need for context-specific data in pediatric populations.[2] In India, the Global Burden of Disease analyses suggest a very large national caseload (34.3 million; accounting for 13.1% of the global burden) and substantial years lived with disability (27.9%),

highlighting pediatric asthma as an enduring public health priority.[3] Current management frameworks emphasize a stepwise approach to achieve and maintain control, with severity defined by the level of treatment required, and with regular review of adherence, inhaler technique, risk factors, and comorbidities.[4-6] Despite these advances, many children remain poorly controlled due to environmental exposures such as second-hand tobacco smoke and indoor pollutants, which are consistently associated with greater asthma morbidity.[7, 8] Against this background, there is growing interest in readily measurable biomarkers that could complement clinical indices and lung function to refine risk stratification and guide preventive strategies in resource-constrained settings.[4]

Magnesium is a divalent cation with multiple airway-relevant actions, including modulation of calcium influx, relaxation of bronchial smooth muscle, and stabilization of mast cells, collectively suggesting potential benefits in bronchoconstrictive disorders.[9] The clinical plausibility of magnesium in asthma is reinforced by evidence that intravenous magnesium sulfate, used as an adjunct in moderate-to-severe exacerbations unresponsive to initial therapy, reduces hospital admissions and improves flow measures in children, although trial sizes are modest and heterogeneity persists.[10] Beyond acute care, observational studies in non-exacerbation settings have linked higher dietary magnesium intake with better lung function and reduced airway hyper-reactivity, suggesting that chronic magnesium status may be related to asthma control and physiology.[11] However, the relevance of circulating (serum) magnesium – an inexpensive and widely available laboratory test – to day-to-day pediatric asthma status remains uncertain, as prior studies vary in sample size, timing (stable vs acute), biospecimen (serum vs intracellular), and control for confounders such as adiposity and smoke exposure.[7, 9] Accordingly, the objective of the present study was to determine the levels of serum magnesium among asthmatic children; and to correlate levels of serum magnesium with severity of asthma.

Materials and Methods

This was a single centre, hospital-based, analytical cross-sectional study conducted in the outpatient department and/or inpatient wards of the Department of Paediatrics, Melmaruvathur Adhiparasakthi Institute of Medical Sciences and Research, Tamil Nadu, India over a period of six months between September 2023 and March 2024. The study was approved by the Institutional Human Ethics Committee (IHEC) with reference number MAPIMS/IEC/52/2023 (Project No. 340(8)2023) dated 29/08/2023. The parents were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Children 5 to 18 years of age, of both gender, with a diagnosis of asthma (defined per Global Initiative for Asthma (GINA) recommendations for children (5–11 years) and adolescents (≥12 years); severity was classified by the level of treatment required to achieve control, recorded at the time of assessment as – mild (steps 1–2), moderate (step 3), or severe (steps 4–5), after confirming adherence, inhaler technique, and modifiable risk factors; current control was additionally characterized using age-appropriate validated tools (Childhood Asthma Control Test [C-ACT] for 5–11 years; ACT for ≥12 years) and by symptom burden, night waking, activity limitation, and reliever use over the prior 4 weeks) were enrolled. However, children with acute asthma exacerbation within the preceding 4 weeks; current systemic corticosteroid therapy or magnesium supplementation in the past 2 weeks; chronic pulmonary diseases other than asthma (e.g., bronchiectasis, cystic fibrosis, primary ciliary dyskinesia); acute lower respiratory infection in the past 2 weeks; significant comorbidities that may alter magnesium homeostasis (chronic kidney disease, hepatic failure, malabsorption syndromes, uncontrolled thyroid or parathyroid disease); and known metabolic or neuromuscular disorders were excluded.

The sample size was estimated for a single-proportion objective using the formula $N=(Z^2 1-\alpha/2 \times pq)/d^2$, where $Z1-\alpha/2=1.96$ for a 95% confidence level, p is the anticipated prevalence and $q=1-p$, and d is the absolute precision. Based on Somashekar et al.,[12] we assumed a prevalence of 32% (p=0.32, q=0.68). Precision was set at 20% of p (i.e., d=0.064). Substituting these values, the minimum required sample size was rounded off to 200 patients. After obtaining written informed consent from parents/guardians, eligible children were consecutively enrolled, and their demographic profile and baseline clinical details were recorded in a pre-designed structured proforma. Baseline assessment included sociodemographic data (age, sex), anthropometry (height, weight, BMI z-score), detailed asthma history (age at diagnosis, duration, controller step, adherence, and inhaler-technique check), environmental exposures (household tobacco smoke, biomass/indoor pollutants), dietary history suggestive of low magnesium intake, and recent medication use (including systemic corticosteroids, diuretics, aminoglycosides, and proton-pump inhibitors); pulse oximetry at rest was documented, spirometry was performed where feasible in accordance with ATS/ERS standards to obtain FEV_1, FVC, and FEV_1/FVC % predicted using age- and ethnicity-appropriate reference equations, and concomitant serum electrolytes (sodium, potassium, calcium) were measured when clinically indicated. Thereafter, 2 mL of venous

blood was collected under aseptic precautions after an overnight or minimum 6-hour fast, preferably between 08:00 and 10:00 hours to minimize diurnal variation; samples were drawn into plain vacutainers, allowed to clot, and centrifuged at 3,000 rpm for 10 minutes within 60 minutes of collection, with haemolyzed specimens rejected. Total serum magnesium was then estimated on a fully automated analyzer using a xylidyl-blue colorimetric method with two-point calibration and internal bi-level quality controls per laboratory standard operating procedures, in a laboratory participating in external quality assurance. For study purposes, serum magnesium categories were described as hypomagnesemia (<1.7 mg/dL; <0.70 mmol/L), normomagnesemia (1.7–2.4 mg/dL; 0.70–1.00 mmol/L), and hypermagnesemia (>2.4 mg/dL; >1.00 mmol/L), without diagnostic labeling.

Statistical analysis: Data were analyzed using IBM SPSS Statistics v27 (IBM Corp., Armonk, NY). Continuous variables were checked for normality (Shapiro–Wilk, Q–Q plots) and summarized as mean±SD or median (IQR), while categorical variables were presented as n (%). Serum magnesium was compared across GINA severity (mild, moderate, severe) using one-way ANOVA with Bonferroni post-hoc tests (or Kruskal–Wallis with Dunn's adjustment when assumptions were violated). Correlations between magnesium and FEV_1 % predicted and ACT/C-ACT were estimated with Pearson's correlation. Discrimination of magnesium for moderate-to-severe versus mild asthma was assessed by ROC analysis with AUC and 95% CI (DeLong), and the optimal cutoff chosen by Youden's J with corresponding sensitivity/specificity. Multivariable linear (FEV_1 as outcome) and logistic (moderate-to-severe asthma) regressions adjusted for age, sex, BMI z-score, and environmental tobacco smoke; magnesium effects were expressed per 0.1 mg/dL with 95% CIs. Two-tailed α=0.05 was used.

Results

Among 200 asthmatic children (mean age 10.8 ± 3.6 years), 36.0% were 5–8 years, 38.0% were 9–12 years, and 26.0% were 13–18 years; 58.0% were male and 62.0% resided in urban areas. The mean BMI z-score was −0.2 ± 1.1, with 18.0% underweight and 18.0% overweight/obese. Median asthma duration was 3.4 years (IQR 2.0–6.0). Mean ACT/C-ACT was 19.2 ± 4.6 and 46.0% were uncontrolled; mean FEV_1 was 82.2 ± 14.6% predicted and 36.0% had FEV_1 < 80%. By GINA, 48.0% had mild, 37.0% moderate, and 15.0% severe asthma; controller steps were distributed across Step 1–5 as 11.0%, 37.0%, 30.0%, 17.0%, and 5.0%, respectively. Adherence was good in 62.0%, while inhaler technique errors occurred in 29.0%. Environmental tobacco smoke exposure affected 38.0%; biomass/indoor fuel exposure and dampness/mould were 22.0% and 28.0%. Atopy was common (55.0%; allergic rhinitis 44.0%); 41.0% reported ≥1 exacerbation (19.0% ≥2), 24.0% had an emergency visit, and 9.0% were hospitalized in the past year; 92.0% were up to date on immunizations.

Serum magnesium averaged 1.86 ± 0.24 mg/dL (median 1.85, IQR 1.72–1.99), with hypomagnesemia in 21.5%, normomagnesemia in 76.5%, and hypermagnesemia in 2.0% of children. Across asthma severity, mean magnesium declined from 1.92 ± 0.22 mg/dL in mild (hypomagnesemia 10.4%) to 1.83 ± 0.22 mg/dL in moderate (25.7%) and 1.74 ± 0.25 mg/dL in severe (46.7%). Differences were statistically significant (ANOVA with post-hoc) – moderate vs mild $p = 0.021$ and severe vs mild $p < 0.001$; severe vs moderate also differed ($p = 0.038$), indicating a graded increase in hypomagnesemia prevalence and lower mean magnesium with increasing asthma severity. Serum magnesium correlated positively with FEV_1 % predicted (Pearson r=0.32, 95% CI 0.19 to 0.43, p<0.001) and with ACT/C-ACT scores (r=0.28, 95% CI 0.15 to 0.40, p<0.001), indicating that higher magnesium levels were associated with better airflow limitation and symptom control.

In multivariable logistic regression adjusted for age, sex, BMI z-score, and environmental tobacco smoke exposure, each 0.1 mg/dL higher serum magnesium was associated with lower odds of moderate–severe asthma (adjusted OR 0.85, 95% CI 0.76–0.95, p=0.005). A similar model for frequent exacerbations (≥2 in the prior 12 months) showed an adjusted OR of 0.88 per 0.1 mg/dL increase (95% CI 0.79–0.98, p=0.012). In linear regression, serum magnesium was independently associated with higher FEV_1 % predicted (β +8.3 percentage points per 1 mg/dL, p<0.001) after adjusting for the same covariates.

Serum magnesium showed fair discriminatory accuracy for identifying moderate–severe asthma, with an AUC of 0.71 (95% CI 0.64–0.78; p<0.001). An optimal cutoff of ≤1.80 mg/dL yielded 68% sensitivity and 66% specificity, indicating a balanced trade-off between true-positive and true-negative classification at this threshold.

Table 1: Baseline Sociodemographic and Clinical Characteristics

Characteristic		Total (N=200)
Age (years), Mean ± SD		10.8 ± 3.6
Age (years), n (%)	5 to 8	72 (36.0)
	9 to 12	76 (38.0)
	13 to 18	52 (26.0)
Sex, n (%)	Male	116 (58.0)
	Female	84 (42.0)
Residence, n (%)	Urban	124 (62.0)
	Rural	76 (38.0)
BMI z-score, Mean ± SD		-0.2 ± 1.1
BMI category, n (%)	Underweight	36 (18.0)
	Normal	128 (64.0)
	Overweight/Obese	36 (18.0)
Asthma duration (years), Median (IQR)		3.4 (2.0–6.0)
ACT/C-ACT score, Mean ± SD		19.2 ± 4.6
Uncontrolled (ACT/C-ACT <20), n (%)		92 (46.0)
FEV1 % predicted, Mean ± SD		82.2 ± 14.6
FEV1 <80% predicted, n (%)		72 (36.0)
GINA severity, n (%)	Mild	96 (48.0)
	Moderate	74 (37.0)
	Severe	30 (15.0)
Controller step, n (%)	Step 1	22 (11.0)
	Step 2	74 (37.0)
	Step 3	60 (30.0)
	Step 4	34 (17.0)
	Step 5	10 (5.0)
Adherence to controller therapy, n (%)	Good (≥80)	124 (62.0)
	Moderate (50–79)	48 (24.0)
	Poor (<50)	28 (14.0)
Environmental tobacco smoke, n (%)		76 (38.0)
Biomass/indoor fuel exposure, n (%)		44 (22.0)
Indoor dampness/mould, n (%)		56 (28.0)
Any atopy, n (%)		110 (55.0)
Atopic conditions, n (%) (numbers are not mutually exclusive)	Allergic rhinitis	88 (44.0)
	Eczema	24 (12.0)
	Food allergy	18 (9.0)
Family history of asthma/atopy, n (%)		80 (40.0)
Number of exacerbations in past 12 months, n (%)	≥1 exacerbation	82 (41.0)
	≥2 exacerbations	38 (19.0)
Emergency/unscheduled visit (past 12 months), n (%)		48 (24.0)
Hospitalization for asthma (past 12 months), n (%)		18 (9.0)
Inhaler technique error present, n (%)		58 (29.0)
Reliever use ≥3 times/week, n (%)		42 (21.0)
Immunizations up to date, n (%)		184 (92.0)

Table 2: Distribution of serum magnesium levels

Measure	Value
Serum Magnesium (mg/dL), Mean ± SD	1.86 ± 0.24
Serum Magnesium (mg/dL), Median (IQR)	1.85 (1.72–1.99)
Hypomagnesemia, n (%)	43 (21.5)
Normomagnesemia, n (%)	153 (76.5)
Hypermagnesemia, n (%)	4 (2.0)

Table 3: Comparison of serum magnesium levels by severity of asthma

Severity	n	Mean ± SD (mg/dL)	Hypomagnesemia n (%)	p-value (ANOVA/post-hoc)
Mild	96	1.92 ± 0.22	10 (10.4%)	Reference
Moderate	74	1.83 ± 0.22	19 (25.7%)	vs Mild p=0.021*
Severe	30	1.74 ± 0.25	14 (46.7%)	vs Mild p<0.001* vs Mod p=0.038*
*Statistically significant at p<0.05				

Table 4: ROC analysis showing AUC of serum magnesium in predicting moderate to severe asthma

Outcome	AUC (95% CI)	Optimal cutoff (mg/dL)	Sensitivity (%)	Specificity (%)	P value
Moderate–Severe	0.71 (0.64–0.78)	≤1.80	68.0	66.0	<0.001*
*Statistically significant at p<0.05					

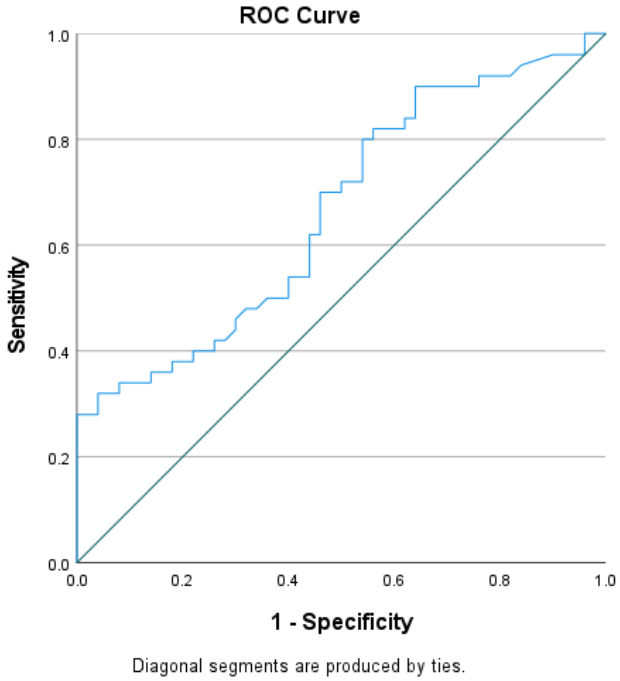

Figure 1: ROC analysis showing AUC of serum magnesium in predicting moderate to severe asthma

Discussion

The present study provides new pediatric data linking lower serum magnesium levels with poorer asthma status, reflected by lower FEV_1 % predicted, worse symptom control, and a graded pattern of decline in magnesium across GINA-defined severity categories. This aligns biologically with magnesium's known roles as a calcium antagonist and smooth-muscle relaxant with mast-cell stabilizing properties, which together can reduce bronchial smooth-muscle tone and airway reactivity.[13] Clinical practice already leverages this biology in acute care. Intravenous magnesium sulfate is recommended as an adjunct in moderate–severe exacerbations, with systematic reviews in children suggesting reduced hospital admissions and improved flow measures in emergency settings, although heterogeneity and small study sizes temper certainty.[10]

Our cohort's demographic and clinical profile (school-age predominance, male excess, and substantial urban residence) mirrors contemporary pediatric asthma epidemiology and guideline frameworks that emphasize stepwise, personalized management and regular review of control, adherence, and inhaler technique.[14, 15] The distribution we observed across controller steps (Steps 1–5) and the high rate of suboptimal control (ACT/C-ACT <20 in 46%) are consistent with GINA 2024 guidance,[4] which underscores that severity is assessed by the treatment intensity required to achieve good control, and that persistent symptoms warrant optimization of adherence, inhaler technique, and modifiable risks before stepping up therapy.

The central finding – a monotonic decline in mean serum magnesium from mild to moderate to severe asthma, with hypomagnesemia increasing correspondingly – adds to a mixed literature on

chronic (non-exacerbation) magnesium status in pediatric asthma.[16, 17] Several reports have found lower serum (or intracellular) magnesium among asthmatic children, particularly during attacks, and positive associations between magnesium status and lung function indices, supporting our observed positive correlations with FEV_1 and symptom control scores.

For instance, Hatipoğlu et al.[18] reported lower serum magnesium during acute attacks versus stable asthma and healthy controls, and a recent pediatric series associated hypomagnesemia with higher severity scores and abnormal spirometry.[19] Likewise, studies of intracellular (RBC) magnesium in children with chronic asthma have linked lower levels with reduced lung function.[17] Beyond serum measures, population and interventional data suggest that higher magnesium exposure may benefit airway physiology. A classic population analysis associated higher dietary magnesium intake with better lung function and less airway hyper-reactivity in adults, a signal echoed in later work exploring diet-lung function links and potential modification by adiposity.[9] Meta-analytic evidence in broader asthma populations suggests that oral magnesium supplementation can modestly improve FEV_1 at certain time points, though overall effects are small and inconsistent – findings that fit our observation of only "fair" discriminatory performance for serum magnesium as a single biomarker.[20]

Our multivariable models extend the crude associations by demonstrating that each 0.1 mg/dL increment in serum magnesium was independently associated with lower odds of moderate–severe asthma and with fewer frequent exacerbations, after adjustment for age, sex, BMI z-score, and environmental tobacco smoke exposure – covariates chosen in line with guideline-endorsed risk assessment.[4] Although some pediatric reports have not observed strong links between serum magnesium and chronic severity, differences in timing (stable state versus attack), biospecimen choice (serum vs intracellular), assay methods, diet or supplementation, and control for confounding may explain discordant results across studies.[21] Our adjusted findings therefore support – but do not prove – an independent association between lower magnesium status and worse asthma phenotype.

The correlations we observed – $r=0.32$ for FEV_1 % predicted and $r=0.28$ for ACT/C-ACT – are of modest magnitude yet clinically interpretable. Given the multifactorial nature of pediatric asthma (genetics, environment, airway inflammation type, adherence, technique), medium-strength correlations for a single biochemical measure are plausible and parallel effect sizes reported for other nutritional or environmental exposures. The independent linear association between magnesium and FEV_1 in adjusted models (β +8.3 percentage points per 1 mg/dL) strengthens the inference that magnesium status tracks with physiologic airflow limitation beyond confounding by anthropometry or smoke exposure, though causality cannot be inferred from cross-sectional data. In the context of mechanistic and acute-care evidence for magnesium's bronchodilatory potential, these associations provide a coherent narrative across biologic plausibility, acute response, and chronic status.[22]

Environmental factors in our dataset likely intersect with magnesium–asthma relationships. Exposure to environmental tobacco smoke (ETS) was common (38%) and is consistently implicated in worse asthma control and increased risk of wheeze and exacerbations in children; controlling for ETS in multivariable models is therefore essential to isolate magnesium's association. Contemporary syntheses and meta-analyses reaffirm that smoke exposures – both active and passive – contribute to suboptimal control and exacerbation risk, reinforcing the need for smoke-exposure assessment and counselling in routine pediatric asthma care.[23-25]

From a clinical-utility perspective, the ROC analysis (AUC 0.71; optimal cutoff $\leq$1.80 mg/dL with 68% sensitivity and 66% specificity for moderate–severe vs mild asthma) suggests that serum magnesium alone offers only fair discrimination. This is not unexpected; asthma severity reflects a composite of airway inflammation, remodelling, triggers, adherence, and technique, such that any single biomarker will have limited stand-alone predictive value. However, in settings akin to ours – resource-constrained environments with high indoor-pollutant exposure and nontrivial rates of inhaler technique errors – serum magnesium could contribute to a parsimonious risk-stratification panel alongside clinical indices (ACT/C-ACT), spirometry, and environmental risk assessment. Guideline-concordant management would remain anchored in optimizing ICS-containing therapy, adherence, and technique, reserving magnesium primarily for acute care and focusing on upstream determinants (e.g., smoke exposure) to reduce exacerbation risk.[4]

Our findings also prompt consideration of nutritional assessment as part of comprehensive asthma care. While routine supplementation cannot be recommended based on current evidence, selective evaluation of dietary magnesium intake may be reasonable in poorly controlled patients, particularly where diet quality is low or gastrointestinal losses are relevant. Observational signals linking dietary magnesium to lung function, coupled with small interventional effects, suggest

that low magnesium could be one of several modifiable factors affecting control.[9, 20, 26]

This study had several limitations. First, its single-centre, cross-sectional design precludes causal inference and may limit external generalizability beyond similar tertiary-care pediatric settings. Second, although we adjusted for key covariates (age, sex, BMI z-score, and environmental tobacco smoke), residual and unmeasured confounding cannot be excluded – particularly dietary magnesium intake, gastrointestinal/renal losses, pubertal status, vitamin D and calcium balance, and medications that influence magnesium homeostasis (e.g., diuretics, PPIs). Third, we assessed total serum magnesium, which may not fully reflect intracellular or ionized magnesium relevant to airway smooth-muscle physiology; more comprehensive assessment (e.g., RBC/ionized magnesium) was not feasible. Fourth, asthma severity was classified from contemporaneous treatment "controller steps" and current control measures (ACT/C-ACT), introducing potential misclassification related to adherence and inhaler technique; spirometry was not obtainable in all younger children. Fifth, we did not phenotype airway inflammation (eosinophils, FeNO, IgE) or systematically capture indoor air quality metrics beyond proxy exposures (biomass/dampness), limiting mechanistic interpretation. Finally, seasonal variation in allergens and respiratory infections during the six-month enrolment window may have influenced both asthma control and magnesium status, and the study was not powered for extensive subgroup analyses.

Conclusion

In this single-centre cohort of 200 children with asthma, lower serum magnesium was consistently associated with worse disease status. Hypomagnesemia was present in 21.5%, mean magnesium declined from mild to severe asthma (1.92 → 1.74 mg/dL), and magnesium correlated positively with FEV_1 % predicted (r=0.32) and ACT/C-ACT scores (r=0.28). Each 0.1 mg/dL higher magnesium independently reduced the odds of moderate–severe asthma (adjusted OR 0.85; 95% CI 0.76–0.95) and frequent exacerbations (adjusted OR 0.88; 95% CI 0.79–0.98), and serum magnesium showed fair discrimination for moderate–severe disease (AUC 0.71; optimal cutoff ≤1.80 mg/dL: sensitivity 68%, specificity 66%). These findings support serum magnesium as a pragmatic, supportive biomarker linked to airflow limitation, symptom control, and exacerbation burden; while not definitive for risk stratification, magnesium assessment may complement guideline-based evaluation alongside spirometry, symptom scores, and modifiable risk factors.

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