Research Article

Serum Magnesium Levels in Hospitalised and Outpatient Populations: A Cross-Sectional Observational Study.

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ABSTRACT

Background: Magnesium is the second most abundant intracellular cation and an essential cofactor for over 600 enzymatic reactions. Dysmagnesaemia, particularly hypomagnesaemia, is a frequently underrecognised electrolyte abnormality in hospitalised patients despite its significant clinical consequences including neuromuscular dysfunction, cardiac arrhythmias, and impaired glucose metabolism.

Objectives: To determine the prevalence and demographic distribution of serum magnesium dysregulation among inpatients (IP) and outpatients (OP), and to assess its association with age group, sex, and care setting.

Methods: A retrospective cross-sectional study was conducted over 12 months (January–December 2019) at a tertiary care hospital. Of 649 samples requisitioned, 646 with valid results were analysed. Serum magnesium was measured by automated colorimetric assay. Reference range: 1.7–2.3 mg/dL. Statistical analyses included descriptive statistics, independent samples t-tests, and chi-square tests.

Results: The mean serum magnesium was 1.88 ± 0.41 mg/dL (range: 0.45–4.25). Of 646 patients, 491 (76.0%) were normomagnesaemic, 129 (20.0%) were deficient, and 26 (4.0%) had hypermagnesaemia. Inpatients had significantly lower levels than outpatients (1.85 vs 1.98 mg/dL; $p = 0.0008$) with 2.65× higher deficiency prevalence (23.3% vs 8.8%). Children (<20 yr) had the highest deficiency rate (31.2%); the elderly (>60 yr) also had high deficiency (26.8%) and hypermagnesaemia (7.0%). Age group was significantly associated with magnesium status ($\chi^2 = 24.03$; $p = 0.0005$). Sex differences were not statistically significant ($p = 0.175$).

Conclusion: Hypomagnesaemia affects 1 in 5 hospitalised patients and is disproportionately high in children, the elderly, and inpatients. Routine serum magnesium screening should be incorporated into standard inpatient biochemical assessment, particularly for high-risk populations.

Keywords: *serum magnesium; hypomagnesaemia; hypermagnesaemia; dysmagnesaemia;*

1. INTRODUCTION

Magnesium (Mg^{2+}) is the fourth most abundant cation in the body and the second most plentiful intracellular cation after potassium. It is an obligatory cofactor for over 600 enzymatic reactions encompassing adenosine triphosphate (ATP) synthesis, glycolysis, oxidative phosphorylation, DNA and RNA replication, and protein biosynthesis [1]. Beyond these biochemical roles, magnesium is indispensable for neuromuscular transmission, cardiac rhythm regulation, bone mineralisation, and insulin-receptor signalling. Magnesium stabilises the resting neuromuscular membrane potential and physiologically antagonises calcium influx at the motor end-plate; its deficiency lowers the depolarisation threshold, producing neuromuscular irritability, tremor, tetany, and seizures. In cardiac tissue, magnesium regulates inward-rectifier potassium channels and the slow calcium current, so hypomagnesaemia prolongs the cardiac action potential and predisposes to QTc prolongation, ventricular ectopy, and torsades de pointes, particularly when coexisting with hypokalaemia. In glucose homeostasis, magnesium is a required cofactor for tyrosine kinase activity at the insulin receptor; hypomagnesaemia impairs receptor

autophosphorylation and downstream GLUT4 translocation, contributing to insulin resistance and a bidirectional relationship with type 2 diabetes mellitus [1].

The normal serum magnesium concentration in adults ranges between 1.7 and 2.3 mg/dL (0.70–0.95 mmol/L). Hypomagnesaemia is estimated to affect 10–20% of general hospitalised patients, rising to 60–65% in critically ill individuals [2, 3]. It commonly perpetuates hypokalaemia and hypocalcaemia, and is associated with ventricular arrhythmias, neuromuscular irritability, seizures, and increased all-cause mortality [4, 5]. Hypermagnesaemia, though less common, occurs predominantly in patients with impaired renal clearance and can cause life-threatening cardiovascular depression.

Elderly patients are particularly susceptible to magnesium deficiency due to diminished intestinal absorption, impaired renal conservation, polypharmacy, and multiple comorbidities including type 2 diabetes mellitus [6, 7]. Similarly, hypomagnesaemia in paediatric patients may result from acute gastrointestinal losses, malnutrition, or pharmacological exposures. Type 2 diabetes is strongly associated with hypomagnesaemia via renal magnesium wasting and impaired tubular reabsorption [8].

Despite this clinical significance, serum magnesium is frequently omitted from routine biochemical panels, leading to systematic underdiagnosis. Published data from Indian tertiary care hospitals on the epidemiology of dysmagnesaemia remain limited. This study characterises the prevalence and demographic distribution of serum magnesium dysregulation among 649 samples over a full calendar year at a tertiary care teaching hospital. Published data from Indian tertiary care hospitals on the epidemiology of dysmagnesaemia remain limited. Comparing magnesium status across inpatient and outpatient settings is clinically relevant because it distinguishes magnesium derangement attributable to acute illness, intravenous therapy, and hospital-acquired wasting from that present at baseline in the community, helping to identify the care setting and patient subgroups that would benefit most from targeted screening. This study characterises the prevalence and demographic distribution of serum magnesium dysregulation among 649 samples over a full calendar year at a tertiary care teaching hospital, and examines its association with age, sex, and care setting.

2. MATERIALS & METHODS

2.1 Study Design and Setting

A retrospective cross-sectional observational study was conducted in the Department of Biochemistry of a tertiary care teaching hospital in India. All serum magnesium assay results recorded in the laboratory information system (LIS) between 1 January 2019 and 28 December 2019 were eligible for inclusion.

2.2 Study Population and Sampling

A total of 649 samples were requisitioned during the study period. Three samples yielded non-reportable results (recorded as '–' in the LIS, attributable to haemolysis, sample insufficiency, or instrument failure) and were excluded. The final analytic cohort comprised 646 samples with valid numeric serum magnesium results. Both inpatients (IP) and outpatients (OP) from all clinical departments were included. Data were anonymised prior to analysis.

2.3 Laboratory Methods

Serum magnesium was measured using the Xylidyl Blue colorimetric method on a fully automated Beckman Coulter AU480 chemistry analyser (Beckman Coulter Inc., Brea, CA, USA). Serum magnesium was measured using the Xylidyl Blue colorimetric method on a fully automated biochemistry analyser (AU480, Beckman Coulter Inc., Brea, CA, USA) Internal quality control and external quality assurance procedures were maintained throughout the study period. Results were reported in mg/dL.

2.4 Classification and Stratification

Magnesium status was defined as: **Hypomagnesaemia:** <1.7 mg/dL; **Normomagnesaemia:** 1.7–2.3 mg/dL; **Hypermagnesaemia:** >2.3 mg/dL. Age groups: paediatric (<20 yr), young adult (20–40 yr), middle-aged (41–60 yr), elderly (>60 yr).

2.5 Statistical Analysis

Data were analysed using Statistical Package for the Social Sciences (SPSS) software, version 26.0 (IBM Corp., Armonk, NY, USA).

Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Differences in mean serum magnesium levels between two groups were compared using the independent samples t-test. Associations between categorical variables were analysed using the chi-square (χ^2) test. One-way analysis of variance (ANOVA) was performed to evaluate monthly variation in mean serum magnesium levels, followed by Tukey's Honestly Significant Difference (HSD) post-hoc test for pairwise comparisons when the overall ANOVA was statistically significant. A p-value of <0.05 was considered statistically significant.

3. Results

3.1 Sample Characteristics

Of 649 total samples, 646 were analysed after excluding 3 non-reportable results. The cohort comprised 391 males (60.5%) and 255 females (39.5%), with a mean age of 46.8 ± 18.3 years (range: 0–94; median: 48). Of 646 patients, 498 (77.1%) were inpatients and 148 (22.9%) outpatients. Data spanned all 12 months of 2019, with April recording the highest sample volume (n = 107).

3.2 Overall Serum Magnesium Statistics

Table 1. Descriptive Statistics of Serum Magnesium Levels — Overall Cohort (N = 646)

Parameter	Value
Total Samples Collected	649
Excluded (Non-reportable)	3
Samples Analysed (n)	646
Mean ± SD (mg/dL)	1.88 ± 0.41
Median (mg/dL)	1.88
Minimum (mg/dL)	0.45
Maximum (mg/dL)	4.25
25th Percentile — Q1 (mg/dL)	1.66
75th Percentile — Q3 (mg/dL)	2.06
IQR (mg/dL)	0.40
Standard Deviation	0.41
Reference Range (Normal)	1.7 – 2.3 mg/dL

*SD = Standard deviation; IQR = Interquartile range.

The mean serum magnesium concentration among the study population was 1.88 ± 0.41 mg/dL, with values ranging from 0.45 to 4.25 mg/dL. The median magnesium concentration was 1.88 mg/dL (IQR: 1.66–2.06 mg/dL).

3.3 Prevalence of Magnesium Status

Table 2. Distribution of Serum Magnesium Status Among Study Patients (N = 646)

Magnesium Status	Criterion (mg/dL)	n	Percentage (%)
Normomagnesaemia	1.7 – 2.3	491	76.0%
Hypomagnesaemia (Deficient)	< 1.7	129	20.0%
Hypermagnesaemia (High)	> 2.3	26	4.0%
Total	—	646	100%

Of 646 patients, 491 (76.0%) were normomagnesaemic, 129 (20.0%) had hypomagnesaemia, and 26 (4.0%) had hypermagnesaemia(Table 2). Hypomagnesaemia was the predominant abnormality, affecting approximately 1 in 5 patients.

3.4 Age-Stratified Analysis

Age group was significantly associated with magnesium status ($\chi^2 = 24.03$, df = 6, p = 0.0005). Paediatric patients (<20 yr; n = 48) had the highest hypomagnesaemia prevalence (31.2%), followed by the elderly (>60 yr; n = 157) at 26.8%. Young adults (20–40 yr) were predominantly normomagnesaemic (83.4%), while the middle-aged group (41–60 yr; largest subgroup, n = 259) had an 18.1% deficiency rate. The elderly also showed the highest hypermagnesaemia prevalence (7.0%). Full data are in Table 3.

Table 3. Age-Stratified Serum Magnesium Levels and Status Distribution

Age Group	n	Mean±SD (mg/dL)	Min	Max	Deficient n (%)	Normal n (%)	High n (%)
< 20 years	48	1.87±0.50	0.89	4.25	15 (31.2%)	29 (60.4%)	4 (8.3%)
20–40 years	181	1.93±0.42	0.49	4.07	25 (13.8%)	151 (83.4%)	5 (2.8%)
41–60 years	259	1.88±0.40	0.45	3.98	47 (18.1%)	206 (79.5%)	6 (2.3%)
> 60 years	157	1.83±0.37	1.04	3.38	42 (26.8%)	104 (66.2%)	11 (7.0%)
Total	646	1.88±0.41	0.45	4.25	129 (20.0%)	491 (76.0%)	26 (4.0%)

$\chi^2 = 24.03$, df = 6, p = 0.0005.Statistical analysis: Chi-square test, $\chi^2 = 24.03$, df = 6, p = 0.0005.

Serum magnesium status differed significantly across age groups ($\chi^2 = 24.03$, p = 0.0005). Hypomagnesaemia was most prevalent among children (31.2%) followed by elderly patients (26.8%), whereas young adults had the highest proportion of normomagnesaemia (83.4%).

3.5 Sex-Based Analysis

Males (n = 391) had a mean magnesium of 1.90 ± 0.40 mg/dL and females (n = 255) 1.85 ± 0.42

mg/dL. Deficiency rates were comparable: males 19.2%, females 21.2%. No statistically significant sex difference was found (t = 1.358, p = 0.175; χ^2 = 1.138, p = 0.566). See Table 4.

Table 4. Comparison of Serum Magnesium Levels Between Males and Females

Sex	n (%)	Mean ± SD (mg/dL)	Median (mg/dL)	Deficient n (%)	Normal n (%)	High n (%)
Male	391 (60.5%)	1.90 ± 0.40	1.88	75 (19.2%)	298 (76.2%)	18 (4.6%)
Female	255 (39.5%)	1.85 ± 0.42	1.87	54 (21.2%)	193 (75.7%)	8 (3.1%)
Statistical test	—	t=1.358, p=0.175	—	χ^2=1.138, p=0.566	—	

NS = Not significant (p ≥ 0.05)

*Independent samples t-test; †Chi-square test.

Result:

Although males had a slightly higher mean serum magnesium concentration than females, the difference was not statistically significant (t = 1.358, p = 0.175). Similarly, magnesium status distribution did not differ significantly between sexes (χ^2 = 1.138, p = 0.566).

3.6 Inpatient vs Outpatient Analysis

Inpatients (n = 498) had significantly lower mean magnesium than outpatients (n = 148): 1.85 ± 0.41 vs 1.98 ± 0.40 mg/dL (t = −3.372, p = 0.0008). Inpatient deficiency prevalence was 2.65× that of outpatients (23.3% vs 8.8%). Chi-square test confirmed highly significant association (χ^2 = 15.17, p = 0.0005). Table 5 shows full data.

Result: Inpatients had significantly lower serum magnesium concentrations than outpatients (1.85 ± 0.41 vs. 1.98 ± 0.40 mg/dL; p = 0.0008). Hypomagnesaemia was 2.65 times more common among inpatients (23.3% vs. 8.8%), indicating a significant association between care setting and magnesium status (χ^2 = 15.17, p = 0.0005).

Table 5. Serum Magnesium Levels and Status by Patient Type

Patient Type	n (%)	Mean ± SD (mg/dL)	Median	Deficient (%)	Normal (%)	High (%)
Inpatient (IP)	498 (77.1%)	1.85 ± 0.41	1.85	23.3%	72.7%	4.0%
Outpatient (OP)	148 (22.9%)	1.98 ± 0.40	1.95	8.8%	87.2%	4.1%
Test	—	t=−3.372, p=0.0008**	—	χ^2=15.17	p=0.0005**	**

** *p < 0.001 — Highly significant*

*Independent samples t-test; †Chi-square test.

3.7 Monthly Distribution

April recorded the highest test volume (n = 107) and highest mean magnesium (1.956 mg/dL; SD = 0.28). A declining trend was observed from July through November, with the lowest means in October (1.762 mg/dL) and November (1.765 mg/dL). Monthly data are in Table 6.

Table 6. Monthly Distribution of Serum Magnesium Tests and Mean Values

Month	n	Mean (mg/dL)	SD
January	60	1.924	0.435
February	40	1.943	0.362
March	49	1.914	0.489
April	107	1.956	0.282
May	57	1.908	0.400
June	50	1.951	0.442
July	62	1.800	0.308
August	53	1.841	0.355
September	44	1.843	0.389
October	48	1.762	0.554
November	54	1.765	0.379
December	22	1.888	0.633
Overall	646	1.880	0.408

One-Way ANOVA Summary

Table 7: One-way ANOVA summary of monthly serum magnesium levels

Source of Variation	Sum of Squares (SS)	Degrees of Freedom (df)	Mean Square (MS)	F-statistic	p-value
Between Groups	3.256	11	0.296	1.848	0.045*
Within Groups	101.405	634	0.16	—	—
Total	104.661	645	—	—	—

April recorded the highest test volume (n = 107) and the highest mean serum magnesium (1.956 ± 0.282 mg/dL). A declining trend was observed from July through November, with the lowest means recorded in October (1.762 ± 0.554 mg/dL) and November (1.765 ± 0.379 mg/dL). One-way ANOVA confirmed that these monthly variations were statistically significant (F = 1.848, p = 0.045).

4. DISCUSSION

The present study found hypomagnesaemia in 20.0% (n=129) of 646 patients over 12 months at

a tertiary care teaching hospital in India. This figure aligns with striking precision with the landmark multicentre study by Cheungpasitporn *et al.,* [2], which analysed 65,974 unique hospitalisations at the 2,000-bed Mayo Clinic, Rochester, and identified hypomagnesaemia (serum Mg <1.7 mg/dL) in exactly 20.2% using an identical reference threshold and comparable tertiary care setting. Hypermagnesaemia (Mg ≥2.1 mg/dL) was found in 31.5% in that cohort [2], considerably higher than our 4.0%, likely reflecting higher Mg supplementation use in the US setting; our definition of hypermagnesaemia used a lower threshold (>2.3 mg/dL), making direct comparison of hypermagnesaemia rates methodologically limited. Wong *et al.,* [3], in one of the earliest systematic hospital screening studies (n=621 patients), found hypomagnesaemia in 11.0% using a lower threshold (<1.2 mEq/L, ~1.46 mg/dL), and hypermagnesaemia in 6.9% using >1.05 mEq/L (≥2.54 mg/dL). The lower deficiency rate in their study is attributable directly to the more conservative cut-off, underscoring that threshold selection substantially influences prevalence figures and that our 20.0% at <1.7 mg/dL is methodologically consistent with modern series [2]. From India, Limaye *et al.,* [4] documented a high prevalence of hypomagnesaemia (52%) in 100 critically ill medical patients at a Mumbai tertiary hospital using the same <1.7 mg/dL threshold; the much higher rate compared to our 20.0% reflects the ICU-only population in that study versus our mixed inpatient/outpatient cohort. Our overall mean of 1.88 ± 0.41 mg/dL falls within the normal range but, as demonstrated by the 20.0% deficiency prevalence, masks a clinically significant tail of hypomagnesaemic patients.

The significantly lower mean serum magnesium in inpatients versus outpatients (1.85 vs 1.98 mg/dL; p=0.0008) and the 2.65-fold higher inpatient deficiency rate (23.3% vs 8.8%) represent the most clinically actionable finding of this study. This gradient is corroborated by Cheungpasitporn *et al.,* [5], who in a separate study of 26,020 patients normomagnesaemic at admission found that 28% developed hospital-acquired dysmagnesaemia during their inpatient stay; hospital-acquired hypomagnesaemia independently predicted in-hospital mortality (adjusted OR 1.77; 95% CI 1.31–2.38; p<0.001) after adjustment for comorbidities and medications, establishing the hospital environment - through intravenous fluids, nasogastric suction, and drug-induced renal wasting - as an independent driver of magnesium depletion beyond baseline patient status. Hansen and Bruserud [6], in their comprehensive review of hypomagnesaemia in critically ill patients, documented a stepwise escalation from approximately 10–20% in general medical wards to 20–65% in medical ICUs, with the steepest gradients in patients with sepsis, receiving aminoglycosides, or undergoing continuous renal replacement therapy. Our inpatient rate of 23.3% occupies exactly the expected range for general ward patients documented by Hansen and Bruserud [6], while our outpatient rate of 8.8% is consistent with community-level hypomagnesaemia estimates of 7–12% reported in population-based cohorts cited by Cheungpasitporn *et al.,* [2].

The significant association between age group and magnesium status (χ^2=24.03, p=0.0005) and the 26.8% hypomagnesaemia rate in the elderly (>60 yr; n=157) are directly corroborated by Gautam and Khapunj [7], who in a descriptive cross-sectional study of 384 elderly patients (mean age 70.3±8.1 yr) at a Nepalese tertiary care centre found hypomagnesaemia in 29.0% using a threshold of ≤1.8 mg/dL — closely matching our finding at a near-equivalent cut-off of <1.7 mg/dL. Notably, Gautam and Khapunj [7] reported no sex difference in hypomagnesaemia rates (males 29.4% vs females 28.5%), directly mirroring our finding of no significant sex effect (p=0.175). Malinowska *et al.,* [8], in a dedicated study of 1,068 hospitalised patients aged >65 years in Warsaw, found dysmagnesaemia to be the most prevalent mineral disorder in the elderly, with hypomagnesaemia incidence highest in the geriatrics, oncology, and intensive care divisions (59.0%) and lowest in the 0–18 year age group (9.5%), consistent with the U-shaped age-deficiency curve observed in our data. The bidirectional dysregulation in our elderly cohort - 26.8% hypomagnesaemia

coexisting with 7.0% hypermagnesaemia - is explained by the competing mechanisms documented by Barbagallo *et al.,* [9]: intestinal magnesium absorption declines from approximately 65% in young adults to 40% in the elderly (via reduced TRPM6/TRPM7 transcellular transport), while progressive reductions in glomerular filtration rate simultaneously impair renal magnesium excretion, predisposing to hypermagnesaemia especially in patients receiving magnesium-containing medications. Young adults (20–40 yr) in our study displayed the most favourable profile (13.8% deficient; 83.4% normal), consistent with the lowest hypomagnesaemia rates in young healthy adults without polypharmacy documented across published series [2, 7].

The 31.2% hypomagnesaemia prevalence in our paediatric subgroup (<20 yr; n=48) - the highest of all four age strata in our study - is supported by paediatric data from India. Deshmukh *et al.*, [10], in a prospective study of 80 children admitted to the PICU at BYL Nair Hospital, Mumbai, documented hypomagnesaemia in 70% of critically ill children; deficiency was most prevalent in those receiving aminoglycosides or diuretics, and hypomagnesaemic children had significantly more seizures and cardiovascular instability. Our lower absolute prevalence (31.2%) compared with PICU-exclusive series is expected, given that our paediatric cohort includes ward admissions and not exclusively critically ill patients. The 31.2% rate nonetheless substantially exceeds our overall cohort average of 20.0%, confirming paediatric inpatients as a high-risk subgroup for magnesium depletion driven by acute gastrointestinal losses, nutritional inadequacy, and pharmacological exposures. A recent Indian study by Shekhar et al. [11] (PMID 40322411; Cureus 2025; n=100 ICU patients aged ≥45 yr) found hypomagnesaemia in 24% of critically ill patients, with hypomagnesaemic patients being significantly older (mean 64.8±6.2 vs 59.2±4.9 yr; p<0.001) and showing a strong correlation with concurrent hypocalcaemia (66.7% vs 26.4%; p<0.001), consistent with the well-established mechanistic link between hypomagnesaemia and PTH suppression described by de Baaij *et al.,* [1].

The absence of a significant sex difference in serum magnesium (males 1.90±0.40 vs females 1.85±0.42 mg/dL; t=1.358, p=0.175; χ^2=1.138, p=0.566) is consistent with published data. Gautam and Khapunj [7] reported comparable deficiency rates in elderly males (29.4%) and females (28.5%) with no significant difference, in a study of similar design and population to ours. Malinowska *et al.,* [12], in a retrospective analysis of 20,438 hospitalised patients in Warsaw, explicitly demonstrated that serum magnesium concentration was not gender-dependent (p=NS, Kruskal-Wallis test), with a weak positive correlation with age (Spearman r=0.07; p<0.0001) consistent with the lower mean magnesium in our elderly subgroup (1.83 mg/dL) versus young adults (1.93 mg/dL). Critically, Malinowska *et al.,* [12] found that hypomagnesaemia (at a threshold of 0.75 mmol/L = 1.82 mg/dL) affected 25% of their unselected cohort, and that both hypo- and hypermagnesaemia were independently associated with in-hospital mortality (p<0.0001), with large intra-admission magnesium fluctuations carrying additional mortality risk (p=0.0017). These prognostic data provide direct clinical context for the 20.0% deficiency rate in our cohort, which, like Malinowska *et al.*,'s [12] cohort, would be expected to carry a significantly elevated mortality risk attributable to undetected and uncorrected hypomagnesaemia.

Our inpatient deficiency rate of 23.3% stands in stark contrast to the markedly higher rates reported in Indian ICU-exclusive cohorts, illustrating the expected acuity-dependent escalation of magnesium depletion. Limaye *et al.,* [4] reported hypomagnesaemia in 52% of 100 medical ICU patients at TN Medical College, Mumbai, with significant associations with sepsis (p<0.001) and diabetes mellitus (p<0.05), and a higher mortality rate in hypomagnesaemic patients. Gonuguntla *et al.,* [13] (Indian J Crit Care Med 2023; PMID 37214118; n=100 critically ill patients, ≥45 yr, Indian ICU) found hypomagnesaemia in 27% using the <1.7 mg/dL threshold, with hypomagnesaemic patients having significantly higher APACHE II severity

scores and longer ICU stays (8.36±3.62 vs 6.72±3.19 days; p<0.05). Zafar *et al.,* [14] (Int J Appl Basic Med Res 2014; PMID 24600576; n=100 critically ill patients in a Kashmiri ICU) reported hypomagnesaemia in 30% and found a significantly higher mortality in deficient patients (45% vs 17%; p<0.001). The three-level gradient from outpatient (8.8%) to general inpatient (23.3%) to ICU (27–52%) in our study and these Indian series collectively confirms that disease acuity is the principal determinant of hospital-acquired hypomagnesaemia, and that our mixed inpatient cohort falls within the expected general-ward range on this gradient.

The 18.1% deficiency rate in our middle-aged cohort (41–60 yr) is mechanistically linked to the high prevalence of cardiometabolic comorbidities in this age group. Gommers *et al.,* [15] (Eur J Clin Pharmacol 2019; PMID 30581015; n=1,203 geriatric outpatients) demonstrated that the number of concurrent medications was independently and inversely associated with plasma magnesium after multivariate adjustment (p<0.001), with the strongest associations for proton pump inhibitors (mean Mg reduction: −0.06 mmol/L; p<0.001), loop diuretics (−0.04 mmol/L; p<0.001), and thiazides (−0.03 mmol/L; p=0.01). These agents are widely prescribed to middle-aged and elderly Indian inpatients with hypertension, heart failure, and peptic disease. Barbagallo and Dominguez [16] (World J Diabetes 2015; PMID 26322160) documented the bidirectional diabetes-magnesium vicious cycle: type 2 diabetes mellitus reduces serum magnesium via downregulation of TRPM6 channels in the distal convoluted tubule, impairing renal Mg^{2+} reabsorption, while hypomagnesaemia worsens insulin resistance by impairing tyrosine kinase receptor signalling and reducing glucose transporter GLUT4 translocation. This cycle is directly relevant to the 18.1% deficiency in our 41–60 yr group and the 26.8% in the elderly, two age groups with the highest burden of type 2 diabetes mellitus in India.

The clinical consequences of the 20.0% hypomagnesaemia identified in our cohort are underscored by robust outcome data. Upala *et al.,* [17] (QJM 2016; PMID 27016536), in a systematic review and meta-analysis of six studies (n=5,765 ICU patients), found that hypomagnesaemia was independently associated with ICU mortality (pooled OR 1.77; 95% CI 1.29–2.42; p<0.001), longer ICU stay (weighted mean difference +2.25 days; p<0.001), and greater need for mechanical ventilation (pooled OR 1.79; 95% CI 1.28–2.50; p<0.001). Even in non-ICU settings, Malinowska *et al.,* [12] documented an incremental inverse relationship between serum magnesium and in-hospital mortality across 20,438 unselected hospitalised patients (p<0.0001), with large intra-admission Mg fluctuations carrying additional independent mortality risk (p=0.0017). The mechanistic substrate for this mortality burden is comprehensively described by de Baaij *et al.,* [1]: hypomagnesaemia precipitates refractory hypokalaemia through impaired ROMK-channel-mediated potassium reabsorption in the thick ascending limb of Henle's loop; simultaneously suppresses PTH secretion causing refractory hypocalcaemia; and prolongs the cardiac action potential via inhibition of inward-rectifier potassium channels, predisposing to QTc prolongation, torsades de pointes, and ventricular fibrillation — each an independent mortality risk factor. The compound clinical burden of 129 hypomagnesaemic patients (20.0%) in our cohort thus represents a substantial preventable mortality risk that would be entirely addressable with routine magnesium screening and correction.

This study has several limitations. The retrospective single-centre design limits generalisability and precludes causal inference. Absence of concurrent clinical data (primary diagnosis, comorbidities, medication lists, renal function, serum potassium and calcium) prevented multivariate confounding adjustment. The outpatient subgroup (n=148; 22.9%) was smaller than the inpatient cohort, potentially reducing statistical power for OP-specific subanalyses. Serum total magnesium reflects only the extracellular fraction and may not accurately represent intracellular depletion, particularly in critically ill patients; de Baaij *et al.,* [1] and Gonuguntla *et al.,* [13] both acknowledge this limitation in comparable

studies. Three samples (0.46%) were excluded due to non-reportable results, which is unlikely to have introduced material bias. Despite these constraints, the study's 12-month temporal coverage, 99.5% sample capture rate (646/649), and dual inpatient-outpatient comparison provide a robust institutional dataset whose key findings closely parallel international PubMed-indexed evidence [2, 6, 7, 12].

5. CONCLUSION

Hypomagnesaemia affects 20.0% of patients in this 12-month tertiary care cohort — a prevalence concordant with the 20.2% reported by Cheungpasitporn *et al.,* [2] across 65,974 Mayo Clinic admissions, confirming the global consistency of this burden. Inpatients carry 2.65 times the deficiency rate of outpatients (23.3% vs 8.8%; p=0.0008), consistent with the hospital-acquired dysmagnesaemia gradient documented by Cheungpasitporn *et al.,* [5] and Hansen and Bruserud [6]. Paediatric patients (<20 yr: 31.2%) and the elderly (>60 yr: 26.8%) bear the highest burden, corroborated by Indian PICU data [10] and elderly cohort studies [7, 8]. Sex is not a significant predictor, consistent with Gautam and Khapunj [7] and Malinowska *et al.,* [12]. The 4.0% hypermagnesaemia rate and bidirectional elderly dysregulation are consistent with the polypharmacy and declining renal function mechanisms described by Barbagallo *et al.,* [9]. Given the independently established association between hypomagnesaemia and increased in-hospital mortality [5, 12, 17], the low cost and high clinical yield of serum magnesium estimation justify its universal incorporation into standard inpatient biochemical panels, with prioritised surveillance for children, the elderly, patients with diabetes mellitus or chronic kidney disease, and those receiving diuretics, proton pump inhibitors, or aminoglycosides.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

ETHICAL APPROVAL

Ethical approval for this study was obtained from the Vydehi Institutional Ethics Committee (VIEC), Vydehi Institute of Medical Sciences & Research Centre, Bengaluru, India (Approval No. VIEC/2023/APP/007; EC Registration No. ECR/747/Inst/KA/2015/RR-21; Approval Date: 28 June 2023). As this was a retrospective observational study using anonymized laboratory records, the requirement for informed consent was waived by the Institutional Ethics Committee.

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