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Impact of Hypomagnesemia at the time of Hospital Admission on the Outcomes of Patients with Critical Care Illness

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KEYWORDS

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ABSTRACT:

Background: Magnesium, often referred to as the 'forgotten electrolyte,' is the fourth most abundant mineral in the body and plays a critical role in cardiovascular, neuromuscular, and renal functions, as well as in maintaining immune system integrity. Magnesium deficiency can complicate patient management in the intensive care unit (ICU).

Objectives: To investigate the impact of hypomagnesemia at the time of admission on patient outcomes in the intensive care units (ICUs); and to examine the correlation between hypomagnesemia and the APACHE II scoring system in predicting outcomes among critically ill patients admitted to the ICU.

Methods: This was a hospital based, prospective observational study (descriptive and non-interventional study) conducted among critically ill patients admitted to the ICU under the Department of General Medicine at the JSS Medical College, Mysuru, Karnataka, India between July 2023 and December 2024.

Results: Hypomagnesemia was observed in 31.3% of patients. It was significantly associated with vomiting ($P < 0.001$), diabetes mellitus ($P = 0.002$), chronic kidney disease ($P < 0.001$), chronic liver disease ($P = 0.007$), alcohol intake ($P = 0.001$), and pallor ($P = 0.016$). Laboratory findings revealed lower haemoglobin, elevated ESR, direct bilirubin, alkaline phosphatase (ALP), blood urea, and procalcitonin levels in the hypomagnesemia group ($P < 0.05$). Electrolyte imbalances (lower sodium, potassium, and chloride) and impaired oxygenation parameters were also noted. Hypomagnesemia correlated negatively with APACHE II scores (rp = -0.484), ICU stay (rp = -0.442), and ventilator days (rp = -0.416). APACHE II scores were superior in predicting mechanical ventilation (AUC = 0.839) and mortality (AUC = 0.843) compared to magnesium levels.

Conclusion: Hypomagnesemia is prevalent in critically ill patients and is associated with worse clinical and biochemical profiles, prolonged ICU stays, and greater ventilator dependency. Routine magnesium monitoring and targeted management strategies may improve patient outcomes.

Introduction

Magnesium, often referred to as the forgotten electrolyte, is the fourth most abundant mineral in the body.(1) Magnesium is a vital electrolyte involved in numerous physiological processes, including cellular metabolism, enzyme activation, protein synthesis, and muscle

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contraction.(2) It plays a critical role in cardiovascular, neuromuscular, and renal functions, as well as in maintaining immune system integrity. Acting as a cofactor for various enzymatic reactions, magnesium is crucial for the synthesis of nucleic acids, mRNA translation, and protein formation.(3, 4) It supports immune functions by promoting lymphocyte proliferation, macrophage activation, granulocyte activity, and has anti-inflammatory properties.(5) Additionally, magnesium is vital for mitochondrial function and energy production, as it enables the Krebs cycle and aerobic metabolism.(6) When magnesium levels are low, energy production can become impaired, forcing a shift toward anaerobic metabolism and contributing to increased serum lactic acid levels, which can complicate critical illnesses.(7)

Hypomagnesemia, defined as a serum magnesium level below the normal reference range (less than 0.85 mmol/L or 2.07 mg/dL or 1.7 mEq/L), is a common electrolyte imbalance observed in critically ill patients, with prevalence estimates ranging from 20% to 60% in intensive care units (ICUs).(8) This imbalance has been associated with higher rates of mechanical ventilation, prolonged ICU stays, and increased mortality. Furthermore, it often coexists with other imbalances, including hypocalcaemia and hypoalbuminemia, and is frequently observed in patients with conditions such as diabetes mellitus and hypertension.(9) While there is a consistent association between hypomagnesemia and poor prognosis in critically ill patients, small sample sizes in prior studies have limited the ability to establish a definitive causal relationship.(10)

Studies suggest that hypomagnesemia in critically ill patients may arise from various factors, including inadequate dietary intake, gastrointestinal and renal losses, redistribution from extracellular to intracellular spaces, and conditions such as sepsis, pancreatitis, and acute kidney injury (AKI).(11) Magnesium deficiency can exacerbate other metabolic abnormalities, increase the risk of cardiac arrhythmias, and impair respiratory muscle function, all of which can complicate patient management in the ICU.(12) Consequently, hypomagnesemia at ICU admission may serve as an early indicator of disease severity and be predictive of outcomes, necessitating timely recognition and management.(13)

The Acute Physiology and Chronic Health Evaluation II (APACHE II) score is a widely used tool for assessing disease severity and predicting outcomes in ICU patients. This scoring system combines physiological measurements, laboratory results, and patient information to generate a numerical score reflecting the likelihood of ICU mortality.(14) While APACHE II is a valuable prognostic tool, its predictive accuracy might be enhanced by incorporating specific biochemical markers such as magnesium levels. The relationship between hypomagnesemia and APACHE II scores has been explored in a limited number of studies, with findings suggesting that low magnesium levels may be associated with higher APACHE II scores and worse clinical outcomes.(15) However, more research is necessary to clarify this association and to determine whether magnesium levels could serve as an adjunctive parameter to refine APACHE II-based prognostication.

Against this background, the objectives of the study were to investigate the impact of hypomagnesemia at the time of admission on patient outcomes in the intensive care units (ICUs); and to examine the correlation between hypomagnesemia and the APACHE II scoring system in predicting outcomes among critically ill patients admitted to the ICU.

Materials and Methods

This was a single centre, hospital based, prospective observational study (descriptive and non-interventional study) conducted among critically ill patients admitted to the intensive care units under the Department of General Medicine at the JSS Medical College, Mysuru, India over a period of 18 months (between July 2023 and December 2024). The study was approved by the Institutional Human Ethics Committee (IHEC) with reference number JSS/MC/PG/2046/79/2023-24 dated 23/06/2023. The participants (and their attenders) were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Enrolment into the study proceeded upon receipt of written informed consent. Patients ≥18 years of age, of both gender, admitted to ICUs with the APACHE II diagnostic criteria were included in the study. However, patients <18 years of age; had received magnesium or calcium supplementation prior to sampling; with history of

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malabsorption syndromes/chronic diarrhoeas; on alcohol intoxication; total parenteral nutrition; and pregnant women were excluded.

For the sample size estimation of a single proportion, we assumed the prevalence of hypomagnesemia among critically ill patients to be 30%. Using a 95% confidence level ($\alpha = 0.05$; $Z = 1.96$) and a relative precision of 20% – yielding an absolute precision d=0.06 – the required sample size was calculated with the formula $n=Z^2p(1-p)/d^2$. Substituting the values (p=0.30, d=0.06), the minimum required sample size was rounded off to 230 patients. We used nonprobability sampling – convenience/purposive sampling technique – complete enumeration of patients in accordance with prespecified inclusion and exclusion criteria. Basic demographic details such as age, gender, and address were recorded for each patient. A detailed clinical history related to the presenting symptoms was taken, including relevant past medical history such as diabetes mellitus, chronic kidney disease (CKD), chronic liver disease (CLD), chronic diarrhoea, and other comorbidities. Information on major surgeries, any history of magnesium or calcium supplementation, and chemotherapy for malignancy was also documented. For female patients, detailed obstetric and menstrual history was noted. Family and personal history, including dietary habits, appetite, and other relevant lifestyle factors, were also obtained. Anthropometric data, including height, weight, and BMI, were recorded for each patient. A thorough general physical and systemic examination was conducted. The clinical diagnosis and specific reason for ICU admission were documented, and the APACHE II score was calculated within the first 24 hours of ICU admission to assess the severity of illness. All patients underwent a Complete Hemogram, Liver Function Test (LFT), Renal Function Test (RFT), and routine urine investigations. Additionally, 4.5 mL of venous blood was collected from each patient within 24 hours of ICU admission. This sample was centrifuged for 10 minutes at 3000 rpm, and the serum obtained was used to measure magnesium levels. Serum magnesium levels were determined using the Xylidyl blue colorimetric method on an IMOLA auto analyser, with levels within the range of 1.5-2.6 mg/dL considered normal. Patients were monitored throughout their ICU stay to record the duration of stay, the need for mechanical ventilation, the duration of mechanical ventilation, and mortality. Follow-up continued until either discharge from the ICU or death.

Statistical analysis: The data obtained was manually entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) v23. All the categorical variables were summarised using frequencies and percentages. Continuous variables were summarized using mean (standard deviation) and/or median (interquartile range) (based on the results of data normality, tested using Kolmogorov–Smirnov test and the Shapiro–Wilk test). To test for statistical significance, Chi square test or Fisher exact test (for categorical variables) and independent t test or Mann Whitney U test (for continuous variables) was used. Statistical significance was considered at p value less than 0.05.

Results

In this cohort of 230 ICU patients, hypomagnesemia was present in 31.3% (72/230). Mean age was similar between groups (~52 years; p=0.768), with no significant differences in age strata or sex distribution. Most presenting symptoms were comparable, except vomiting, which was more frequent in the hypomagnesemia group (41.7% vs 19.0%; p<0.001). Hypomagnesemia showed significant associations with diabetes mellitus (68.1% vs 46.2%; p=0.002), chronic kidney disease (30.6% vs 10.1%; p<0.001), chronic liver disease (8.3% vs 1.3%; p=0.007), thyroid disorders (4.2% vs 0%; p=0.030), and alcohol intake (37.5% vs 17.7%; p=0.001). Other variables – including fever, chest pain, loss of consciousness, cough, breathlessness, burning micturition, loose stools, hypertension, and history of major surgery – did not differ significantly between groups.

Anthropometry and vital signs were comparable between groups (e.g., BMI 28.4±5.7 vs 29.0±7.0 kg/m²; pulse 98.7±21.7 vs 92.6±22.9 bpm, p=0.062). On examination, pallor (38.9% vs 23.4%; p=0.016) and icterus (18.1% vs 2.5%; p=0.012) were more frequent with hypomagnesemia. Laboratory differences favoured lower haemoglobin (9.0±2.4 vs 10.2±3.3 g/dL; p=0.002) and higher ESR (77.6±55.9 vs 55.8±48.0 mm/h; p=0.003) in the hypomagnesemia group. Liver parameters showed higher direct bilirubin (0.7±0.6 vs 0.5±0.4 mg/dL; p=0.009) and alkaline phosphatase (134.6±81.5 vs 113.4±49.5 U/L; p=0.016), while renal

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tests revealed higher blood urea (93.9±42.6 vs 80.8±49.2 mg/dL; p=0.039); other measures, including creatinine, were similar.

In 230 ICU patients, those with hypomagnesemia showed significantly lower electrolytes—sodium (130.5±12.6 vs 136.5±4.0 mmol/L; p<0.001), potassium (4.1±0.9 vs 4.4±0.8 mmol/L; p=0.007), and chloride (97.7±15.7 vs 101.8±6.2 mmol/L; p=0.005)—and markedly higher procalcitonin (15.9±15.2 vs 2.3±3.2 ng/mL; p=0.011). Urinary pus cells were more frequent with hypomagnesemia (44.4% vs 22.2%; p=0.001). On ABG, hypomagnesemia was associated with lower PO_2 (69.8±39.6 vs 96.6±44.3 mmHg; p<0.001), lower SaO_2 (84.6±16.9% vs 89.2±14.5%; p=0.033), higher lactate (1.8±1.3 vs 1.5±0.5 mmol/L; p=0.040), and a lower anion gap (7.6±5.6 vs 10.1±4.9; p=0.001), while pH, PCO_2, and HCO_3 were similar. Chest X-ray and ECG patterns did not differ significantly between groups.

Among 230 ICU patients, those with hypomagnesemia (n=72) had higher illness severity; APACHE II scores were higher (25.5±22.3 vs 19.9±12.1; p=0.023), ICU stays were longer (8.2±4.2 vs 5.9±1.3 days; p<0.001), the need for mechanical ventilation was greater (45.8% vs 27.8%; p=0.007), and ventilator days were longer (9.3±6.6 vs 4.9±1.0; p=0.003). Mortality was numerically higher with hypomagnesemia (58.3% vs 51.9%) but not statistically significant (p=0.364).

Serum magnesium levels showed significant, moderate inverse correlations with key outcomes: higher severity by APACHE II ($r = -0.484$, $p < 0.001$), longer ICU stay (days) ($r = -0.442$, $p < 0.001$), and more ventilator days ($r = -0.416$, $p < 0.001$), indicating that lower magnesium was associated with worse clinical course.

ROC analysis showed APACHE II clearly outperformed serum magnesium for both endpoints. For predicting the need for mechanical ventilation, magnesium had modest discrimination (AUC 0.698, cutoff <1.7 mg/dL, sensitivity 67.3%, specificity 61.0%; p<0.001), whereas APACHE II performed excellently (AUC 0.839, cutoff >29.5, sensitivity 92.2%, specificity 77.8%; p<0.001). For mortality, magnesium was non-discriminatory (AUC 0.463, cutoff <1.7 mg/dL, sensitivity 66.1%, specificity 28.3%; p=0.336), while APACHE II again showed strong discrimination (AUC 0.843, cutoff >22.0, sensitivity 82.3%, specificity 72.6%; p<0.001).

Discussion

This study highlights the prevalence, demographic characteristics, and clinical associations of hypomagnesemia among critically ill patients admitted to the ICU. The findings underscore the significance of hypomagnesemia as a potential contributor to morbidity in this population, as well as its associations with certain clinical and biochemical variables. The prevalence of hypomagnesemia in this study was 31.3%, consistent with earlier studies reporting rates ranging from 20% to 60% in critically ill patients.(16, 17) This variability in prevalence across studies may stem from differences in study populations, definitions of hypomagnesemia, or ICU admission criteria. The finding that nearly one-third of patients exhibited low magnesium levels highlights the importance of magnesium monitoring upon ICU admission. The mean age of patients with hypomagnesemia (52.7 years) was comparable to those without (51.9 years), with no statistically significant difference. This is consistent with previous findings suggesting that age alone may not be a determining factor for hypomagnesemia in critically ill populations.(2) Similarly, the gender distribution did not show significant differences between the groups, aligning with research suggesting that hypomagnesemia is not significantly influenced by gender.(18)

Vomiting was significantly more prevalent in patients with hypomagnesemia (41.7%) compared to those without (19.0%). Vomiting contributes to magnesium depletion through gastrointestinal losses, a mechanism well-documented in previous literature (Elin, 1994).(19) This association emphasizes the need to assess magnesium levels in ICU patients presenting with persistent vomiting. Diabetes mellitus was significantly associated with hypomagnesemia, affecting 68.1% of hypomagnesemia patients compared to 46.2% of those without. Hypomagnesemia in diabetic patients may result from osmotic diuresis, insulin resistance, or altered renal handling of magnesium.(20) Studies have demonstrated that magnesium deficiency exacerbates glycaemic control issues, potentially worsening outcomes in critically ill diabetic patients.(21) Chronic kidney disease was notably more prevalent among hypomagnesemia patients (30.6%) compared to those without hypomagnesemia (10.1%). This finding aligns with evidence that magnesium disturbances are common

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in CKD due to altered renal excretion and retention mechanisms.(22, 23) Hypomagnesemia in CKD may exacerbate the risk of cardiovascular complications, which are already heightened in critically ill patients. Chronic liver disease was significantly associated with hypomagnesemia, occurring in 8.3% of patients with hypomagnesemia compared to 1.3% without. Magnesium deficiency in CLD may arise from malnutrition, reduced intestinal absorption, or increased renal losses. These factors can impair metabolic and cardiovascular stability in critically ill patients.(24) Alcohol intake was significantly more common among hypomagnesemia patients (37.5%) compared to those without (17.7%). Chronic alcohol consumption contributes to magnesium depletion through renal losses, reduced dietary intake, and malabsorption.(25, 26) Alcohol-induced hypomagnesemia can exacerbate complications such as arrhythmias and neurologic deficits in critically ill patients. Pallor and icterus were significantly associated with hypomagnesemia, with 38.9% of hypomagnesemia patients exhibiting pallor and 18.1% exhibiting icterus. The finding also corroborates with the lower mean haemoglobin levels reported among patients with hypomagnesemia in the present study. These findings may reflect underlying systemic diseases or nutritional deficiencies that commonly coexist with hypomagnesemia in critically ill patients.(16) The elevated erythrocyte sedimentation rate (ESR) levels in the hypomagnesemia group (77.6 mm/h vs. 55.8 mm/h) may indicate heightened systemic inflammation, which is consistent with the pro-inflammatory milieu seen in magnesium deficiency.(27) Monocyte counts showed a marginally higher trend in the hypomagnesemia group, suggesting an immune-modulatory role of magnesium. Elevated ESR and monocyte counts support previous studies linking hypomagnesemia with systemic inflammation and immune dysfunction.(28)

Direct bilirubin and ALP levels were significantly elevated in the hypomagnesemia group suggesting a possible association with hepatic dysfunction. Magnesium plays a role in liver enzyme regulation, and hypomagnesemia may exacerbate hepatic stress or injury.(29) Blood urea levels were significantly higher in the hypomagnesemia group (93.9 mg/dL vs. 80.8 mg/dL), while serum creatinine levels were not significantly different. Elevated blood urea may indicate impaired renal clearance or increased protein catabolism associated with critical illness and magnesium deficiency. Previous research has shown that magnesium plays a role in glomerular function and tubular reabsorption, with deficiency potentially altering renal hemodynamics.(30) Hypomagnesemia was significantly associated with lower serum sodium, potassium, and chloride levels. These disturbances can exacerbate metabolic instability in critically ill patients – magnesium deficiency may impair sodium-potassium ATPase activity, influencing sodium homeostasis;(31) magnesium is a cofactor for potassium transport and its deficiency can lead to intracellular potassium depletion;(20) and hypochloraemia may reflect disruptions in acid-base balance and renal handling of chloride linked to magnesium status. Procalcitonin levels were markedly higher in the hypomagnesemia group indicating a potential link with sepsis or systemic inflammation. Elevated procalcitonin levels are a marker of infection, and magnesium's role in modulating the immune response may be crucial in this context.(32)

The significant presence of pus cells in urine among hypomagnesemia patients highlights an association with urinary tract infections or inflammation. Magnesium’s role in maintaining epithelial integrity and modulating inflammatory responses may explain this finding.(33) Hypomagnesemia was associated with impaired oxygenation, as evidenced by significantly lower partial pressure of oxygen (PO2) and arterial oxygen saturation (SaO2). Magnesium influences vascular tone and microcirculation, with deficiency contributing to tissue hypoxia.(34) Additionally, the anion gap was lower, and lactate levels were slightly elevated, indicating metabolic stress. Elevated lactate and reduced anion gap may indicate altered cellular respiration and acid-base balance due to magnesium depletion.(27)

The study found that patients with hypomagnesemia had significantly higher APACHE II scores, longer ICU stays, and increased need for mechanical ventilation. These findings align with existing literature suggesting that magnesium deficiency exacerbates disease severity in critical illnesses through its effects on metabolic, inflammatory, and cardiovascular stability.(35, 36) The moderate negative correlation between magnesium levels and APACHE II scores underscores magnesium's role as an inverse marker of illness severity. Low magnesium levels are known to impair mitochondrial

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function, increase oxidative stress, and contribute to systemic inflammation, which could explain the observed association with higher APACHE II scores.(37) The prolonged ICU stays and ventilator days in patients with hypomagnesemia highlight the adverse effects of magnesium deficiency on recovery. A moderate negative correlation between magnesium levels and ICU duration and ventilator days further supports this relationship. Magnesium's role in maintaining respiratory muscle function, reducing neuromuscular excitability, and modulating systemic inflammation may influence ventilator dependency.(31)

The ROC analysis revealed that magnesium levels predicted the need for mechanical ventilation with moderate accuracy (AUC = 0.698, P<0.001). The identified cutoff of <1.7 mg/dL demonstrated moderate sensitivity (67.3%) and specificity (61.0%). This finding suggests that hypomagnesemia, while not a definitive predictor, serves as a significant risk factor for respiratory compromise. In comparison, APACHE II scores exhibited higher predictive accuracy (AUC = 0.839, P<0.001), with a cutoff score of >29.5 yielding high sensitivity (92.2%) and specificity (77.8%). This underscores the utility of APACHE II as a robust tool for predicting the need for mechanical ventilation while highlighting the adjunctive role of magnesium assessment.(38) The study found no significant predictive value for magnesium levels in determining mortality (AUC = 0.463, P=0.336). While hypomagnesemia was associated with higher disease severity, its direct impact on mortality may be confounded by other factors such as comorbidities and overall critical care interventions. Previous studies have also reported mixed findings on the relationship between hypomagnesemia and mortality, suggesting that its impact may be context-dependent.(39) In contrast, APACHE II scores demonstrated strong predictive accuracy for mortality (AUC = 0.843, P<0.001). A cutoff score of >22.0 provided a sensitivity of 82.3% and specificity of 72.6%, reaffirming its role as a reliable prognostic marker in critically ill patients. These findings align with extensive literature validating APACHE II for assessing mortality risk in ICU settings.(40) Given the significant associations with ICU outcomes, routine monitoring of magnesium levels in critically ill patients is essential. Early identification and correction of hypomagnesemia may reduce the need for mechanical ventilation, shorten ICU stays, and improve recovery rates.(30)

This study has several limitations. First, its observational design precludes causal inference. Second, as a single-centre investigation, the findings may not generalize to settings with different patient demographics, ICU practices, or healthcare infrastructure. Third, although associations with outcomes were observed, residual confounding is possible; unmeasured factors – such as baseline nutritional status – may have influenced magnesium levels and outcomes. Finally, the heterogeneous case mix of critically ill patients, with varied diagnoses and treatments, may have introduced variability that could dilute effects attributable specifically to hypomagnesemia.

Conclusion

The present study underscores the significant prevalence and clinical implications of hypomagnesemia in critically ill patients admitted to the ICU. Hypomagnesemia was found to be associated with adverse outcomes, including longer ICU stays, increased ventilator dependency, higher APACHE II scores, and significant metabolic disturbances, such as altered sodium, potassium, and chloride levels. These findings highlight the potential role of magnesium as a biomarker of disease severity and its possible influence on critical care management. Despite these associations, the study did not find a significant link between hypomagnesemia and mortality, suggesting that while low magnesium levels correlate with disease severity, other factors may play a more decisive role in determining outcomes. Furthermore, the study demonstrated the predictive value of magnesium levels and APACHE II scores for identifying patients requiring mechanical ventilation, with APACHE II scores showing superior accuracy.

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Table 1: Comparison of demographics, presenting symptoms, comorbidities, and lifestyle factors in ICU patients with and without hypomagnesemia (N = 230)

		Hypomagnesemia			**P value**
		Present N = 72	**Absent N = 158**	**Total N = 230**	
		n (%)	**n (%)**	**n (%)**	
Age (in years), Mean (SD)		52.7 (18.0)	51.9 (18.6)	52.2 (18.4)	0.768
Age (in years)	18 to 30	9 (12.5)	29 (18.4)	38 (16.5)	0.481
	31 to 60	36 (50.0)	69 (43.7)	105 (45.7)	
	>60	27 (37.5)	60 (38.0)	87 (37.8)	

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Gender	Male	40 (55.6)	70 (44.3)	110 (47.8)	0.113
	Female	32 (44.4)	88 (55.7)	120 (52.2)	
Presenting symptoms					
Fever	Present	13 (18.1)	39 (24.7)	52 (22.6)	0.265
	Absent	59 (81.9)	119 (75.3)	178 (77.4)	
Chest pain	Present	4 (5.6)	13 (8.2)	17 (7.4)	0.473
	Absent	68 (94.4)	145 (91.8)	213 (92.6)	
Loss of consciousness	Present	7 (9.7)	25 (15.8)	32 (13.9)	0.215
	Absent	65 (90.3)	133 (84.2)	198 (86.1)	
Poisoning	Present	3 (4.2)	9 (5.7)	12 (5.2)	0.628
	Absent	69 (95.8)	149 (94.3)	218 (94.8)	
Cough	Present	30 (41.7)	69 (43.7)	99 (43.0)	0.776
	Absent	42 (58.3)	89 (56.3)	131 (57.0)	
Breathlessness	Present	34 (47.2)	62 (39.2)	96 (41.7)	0.255
	Absent	38 (52.8)	96 (60.8)	134 (58.3)	
Burning micturition	Present	12 (16.7)	31 (19.6)	43 (18.7)	0.594
	Absent	60 (83.3)	127 (80.4)	187 (81.3)	
Abdominal pain	Present	26 (36.1)	39 (24.7)	65 (28.3)	0.074
	Absent	46 (63.9)	119 (75.3)	165 (71.7)	
Vomiting	Present	30 (41.7)	30 (19.0)	60 (26.1)	<0.001*
	Absent	42 (58.3)	128 (81.0)	170 (73.9)	
Loose stools	Present	10 (13.9)	17 (10.8)	27 (11.7)	0.494
	Absent	62 (86.1)	141 (89.2)	203 (88.3)	
Past history					
Diabetes mellitus	Present	49 (68.1)	73 (46.2)	122 (53.0)	0.002*
	Absent	23 (31.9)	85 (53.8)	108 (47.0)	
Hypertension	Present	20 (27.8)	40 (25.3)	60 (16.1)	0.693
	Absent	52 (72.2)	118 (74.7)	170 (73.9)	
Chronic kidney disease	Present	22 (30.6)	16 (10.1)	38 (16.5)	<0.001*
	Absent	50 (69.4)	142 (89.9)	192 (83.5)	
Chronic liver disease	Present	6 (8.3)	2 (1.3)	8 (3.5)	0.007*
	Absent	66 (91.7)	156 (98.7)	222 (96.5)	
Thyroid disorders	Present	3 (4.2)	0 (0.0)	3 (1.3)	0.030*
	Absent	69 (95.8)	158 (100)	227 (98.7)	
Major surgeries	Present	6 (8.3)	6 (3.8)	12 (5.2)	0.151
	Absent	66 (91.7)	152 (96.2)	218 (94.8)	
Lifestyle factors					
Alcohol intake	Yes	27 (37.5)	28 (17.7)	55 (23.9)	0.001*
	No	45 (62.5)	130 (82.3)	175 (76.1)	

*Statistically significant at $p<0.05$
SD, Standard deviation

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Table 2: Anthropometry, vital signs, examination findings, and laboratory parameters in ICU patients with and without hypomagnesemia (N = 230)

		Hypomagnesemia			P value
		Present N = 72	Absent N = 158	Total N = 230	
		Mean (SD)	Mean (SD)	Mean (SD)	
Anthropometry					
Height (in cm)		159.3 (12.5)	159.9 (13.0)	159.7 (12.8)	0.773
Weight (in kgs)		71.5 (12.9)	72.7 (12.8)	72.3 (12.8)	0.506
Body mass index (in kg/m2)		28.4 (5.7)	29.0 (7.0)	28.8 (6.6)	0.553
Patient vitals					
Pulse (in beats per minute)		98.7 (21.7)	92.6 (22.9)	94.5 (22.7)	0.062
Systolic BP (in mmHg)		137.4 (30.0)	138.5 (28.8)	138.2 (29.1)	0.789
Diastolic BP (in mmHg)		82.1 (13.2)	81.0 (12.9)	81.3 (12.9)	0.528
Respiratory rate (per minute)		16.3 (4.7)	16.5 (4.6)	16.5 (4.6)	0.802
General examination findings					
Pallor	Present	28 (38.9)	37 (23.4)	65 (28.3)	0.016*
	Absent	44 (61.1)	121 (76.6)	165 (71.7)	
Icterus	Present	13 (18.1)	4 (2.5)	17 (7.4)	0.012*
	Absent	59 (81.9)	154 (97.5)	213 (92.6)	
Oedema	Present	24 (33.3)	56 (35.4)	80 (34.8)	0.755
	Absent	48 (66.7)	102 (64.6)	150 (65.2)	
Lymphadenopathy	Present	4 (5.6)	6 (3.8)	10 (4.3)	0.544
	Absent	68 (94.4)	152 (96.2)	220 (95.7)	
Laboratory investigations (Complete blood count)					
Haemoglobin		9.0 (2.4)	10.2 (3.3)	9.6 (2.9)	0.002*
Packed cell volume		32.7 (7.3)	34.9 (10.5)	34.2 (9.7)	0.105
Total leucocyte count		12698.1 (4303.9)	12967.6 (4080.4)	12883.2 (4144.1)	0.649
Neutrophils		76.9 (12.8)	75.4 (12.9)	75.9 (12.9)	0.405
Lymphocytes		24.1 (12.3)	23.1 (11.3)	23.4 (11.6)	0.515
Eosinophils		1.1 (2.0)	1.2 (2.0)	1.2 (2.0)	0.605
Monocytes		3.5 (1.6)	3.0 (1.6)	3.2 (1.7)	0.066
Basophils		0.4 (0.4)	0.6 (1.1)	0.5 (0.9)	0.284
Platelets		3.2 (1.8)	3.0 (1.7)	3.1 (1.7)	0.463
ESR		77.6 (55.9)	55.8 (48.0)	62.7 (51.5)	0.003*
Laboratory investigations (Liver function test)					
Total bilirubin		0.9 (1.2)	0.8 (0.7)	0.9 (1.1)	0.326
Direct bilirubin		0.7 (0.6)	0.5 (0.4)	0.6 (0.5)	0.009*
Protein		6.6 (0.9)	6.4 (0.7)	6.4 (0.8)	0.102
Albumin		3.2 (0.6)	3.4 (0.9)	3.4 (0.8)	0.077
AST		40.0 (23.2)	60.0 (127.5)	53.7 (106.7)	0.188
ALT		22.0 (12.9)	39.7 (102.4)	34.2 (85.5)	0.146
ALP		134.6 (81.5)	113.4 (49.5)	120.1 (61.9)	0.016*
Laboratory investigations (Renal function test)					

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Blood urea	93.9 (42.6)	80.8 (49.2)	89.8 (45.1)	0.039*
Serum creatinine	1.6 (1.4)	1.5 (1.0)	1.6 (1.3)	0.853
*Statistically significant at p<0.05 SD, Standard deviation				

Table 3: Electrolytes, procalcitonin, urine analysis, ABG indices, chest radiography, and ECG findings in ICU patients with and without hypomagnesemia (N = 230)

		Hypomagnesemia			P value
		Present N = 72	Absent N = 158	Total N = 230	
		Mean (SD)	Mean (SD)	Mean (SD)	
Electrolytes – Sodium		130.5 (12.6)	136.5 (4.0)	134.6 (8.2)	<0.001*
Electrolytes – Potassium		4.1 (0.9)	4.4 (0.8)	4.3 (0.8)	0.007*
Electrolytes – Chloride		97.7 (15.7)	101.8 (6.2)	100.5 (10.3)	0.005*
RBS		255.9 (107.4)	252.5 (103.8)	253.5 (104.7)	0.816
Procalcitonin		15.9 (15.2)	2.3 (3.2)	4.5 (8.2)	0.011*
Urine routine					
Pus cells	Present	32 (44.4)	35 (22.2)	67 (29.1)	0.001*
	Absent	40 (55.6)	123 (77.8)	163 (70.9)	
Urine sugar	Present	5 (6.9)	15 (9.5)	20 (8.7)	0.525
	Absent	67 (93.1)	143 (90.5)	210 (91.3)	
Urine albumin	Present	24 (33.3)	40 (25.3)	64 (27.8)	0.208
	Absent	48 (66.7)	118 (74.7)	166 (72.2)	
Urine ketones	Present	3 (4.2)	11 (7.0)	14 (6.1)	0.411
	Absent	69 (95.8)	147 (93.0)	216 (93.9)	
Arterial blood gas analysis					
pH		7.4 (0.1)	7.4 (0.1)	7.4 (0.1)	0.844
PCO2		35.8 (14.7)	37.3 (13.3)	37.0 (13.7)	0.385
PO2		69.8 (39.6)	96.6 (44.3)	88.2 (44.6)	<0.001*
HCO3		19.8 (9.3)	20.1 (5.6)	20.0 (7.0)	0.774
SaO2		84.6 (16.9)	89.2 (14.5)	87.8 (15.4)	0.033*
Lactate		1.8 (1.3)	1.5 (0.5)	1.7 (1.1)	0.040*
Anion gap		7.6 (5.6)	10.1 (4.9)	9.3 (5.3)	0.001*
Chest X-ray findings					
X-ray	Normal	62 (86.1)	122 (77.2)	184 (80.0)	0.472
	Consolidation	6 (8.3)	3 (1.9)	9 (3.9)	
	Ground glass opacities	0 (0.0)	17 (10.8)	17 (7.4)	
	Pleural effusion	0 (0.0)	16 (10.1)	16 (7.0)	
	Pulmonary oedema	4 (5.6)	0 (0.0)	4 (1.7)	
Electrocardiography					
ECG	Normal sinus rhythm	12 (16.7)	14 (8.9)	26 (11.3)	0.561
	Anterior wall MI	0 (0.0)	9 (5.7)	9 (3.9)	
	Sinus bradycardia	1 (1.4)	19 (12.0)	20 (8.7)	
	Sinus tachycardia	59 (81.9)	116 (73.4)	175 (76.1)	

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*Statistically significant at p<0.05 SD, Standard deviation; RBS, Random blood sugar

Table 4: APACHE II severity, ICU resource use, and mortality in ICU patients with and without hypomagnesemia (N = 230)

		Hypomagnesemia			P value
		Present N = 72	**Absent N = 158**	**Total N = 230**	
		n (%)	**n (%)**	**n (%)**	
APACHE II, Mean (SD)		25.5 (22.3)	19.9 (12.1)	23.8 (22.2)	0.023*
Duration of ICU stay (in days), Mean (SD)		8.2 (4.2)	5.9 (1.3)	7.1 (2.8)	<0.001*
Need for mechanical ventilation	Yes	33 (45.8)	44 (27.8)	77 (33.5)	0.007*
	No	39 (54.2)	114 (72.2)	153 (66.5)	
Ventilator days, Mean (SD)		9.3 (6.6)	4.9 (1.0)	8.0 (5.9)	0.003*
Mortality	Yes	42 (58.3)	82 (51.9)	124 (53.9)	0.364
	No	30 (41.7)	76 (48.1)	106 (46.1)	
*Statistically significant at p<0.05 SD, Standard deviation					

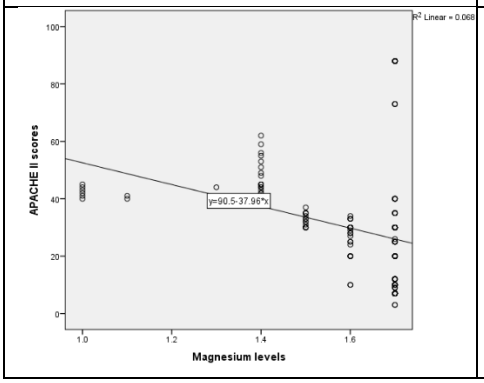
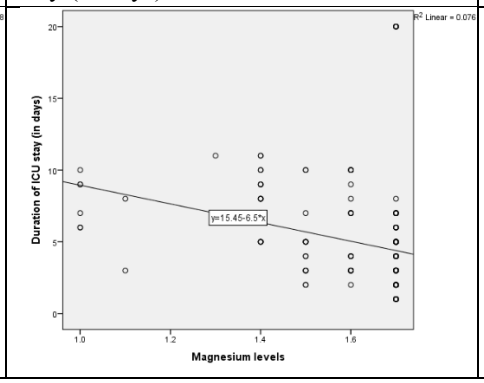
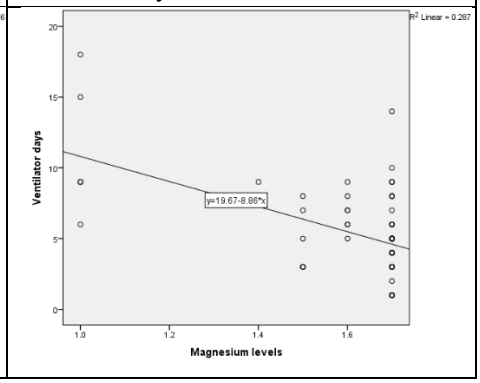
Magnesium levels and APACHE II scores	Magnesium levels and duration of ICU stay (in days)	Magnesium levels and duration of ventilator days
		

Figure 1: Correlation between Serum Magnesium and APACHE II Score, ICU Length of Stay, and Ventilator Days

Table 5: ROC analysis showing area under the curve of magnesium levels and APACHE II scores in predicting need for mechanical ventilation and mortality among critically ill patients

	AUC (95% CI)	Cutoff	Sensitivity (%)	Specificity (%)	P value
ROC analysis predicting need for mechanical ventilation					
Magnesium levels	0.698 (0.60 to 0.776)	<1.7	67.3	61.0	<0.001*
APACHE II scores	0.839 (0.790 to 0.889)	>29.5	92.2	77.8	<0.001*
ROC analysis predicting mortality					
Magnesium levels	0.463 (0.389 to 0.538)	<1.7	66.1	28.3	0.336
APACHE II scores	0.843 (0.792 to 0.895)	>22.0	82.3	72.6	<0.001*

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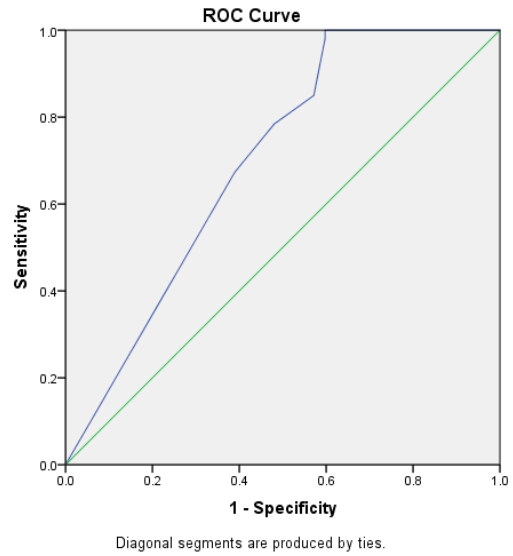
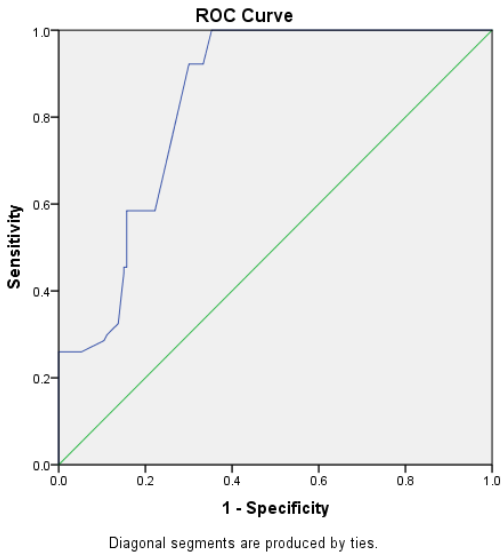
*Statistically significant at p<0.05
AUC, Area under the curve; CI, Confidence interval

ROC analysis showing area under the curve in predicting need for mechanical ventilation among critically ill patients

Magnesium levels	APACHE II scores
	

ROC analysis showing area under the curve in predicting mortality among critically ill patients

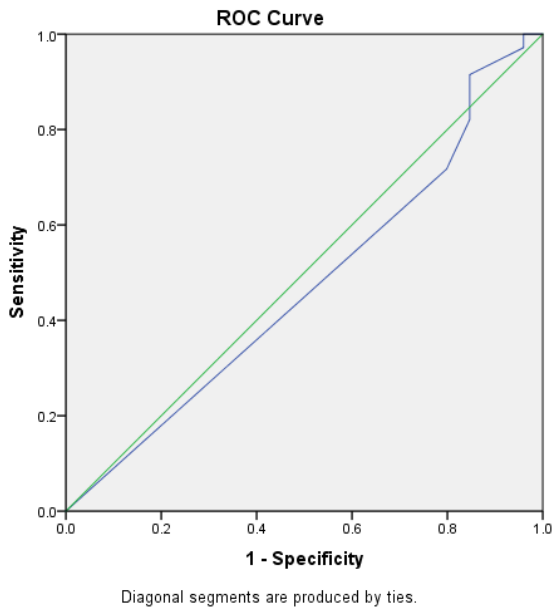
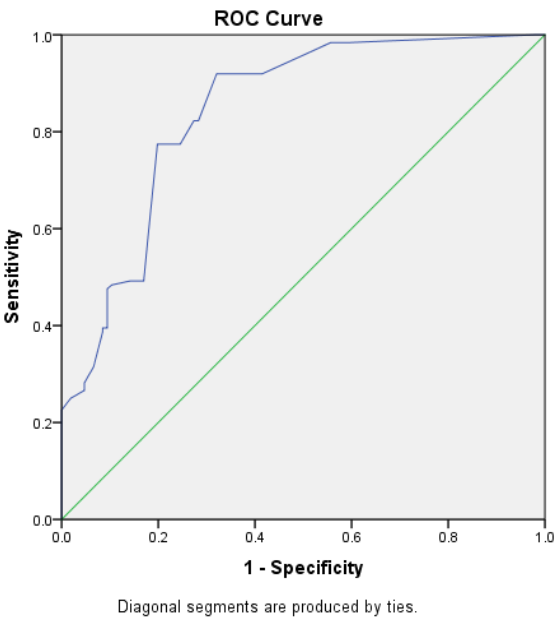
Magnesium levels	APACHE II scores
	

Figure 2: ROC analysis showing area under the curve of magnesium levels and APACHE II scores in predicting need for mechanical ventilation and mortality among critically ill patients