e-ISSN: 0975-9506, p-ISSN:2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2025; 16(7); 272-277

Original Research Article

A Study on Clinical Presentations, Background Illness and Precipitating Factors of Hypoglycaemia in a Tertiary Care Centre

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Received: 25-06-2024 / Revised: 23-07-2025 / Accepted: 28-08-2025
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Conflict of interest: Nil

Abstract:

Introduction: Hypoglycemia is a common and potentially life-threatening condition encountered in medical wards. It presents with varied clinical symptoms and is influenced by multiple underlying illnesses and precipitating factors. Understanding the clinical spectrum, associated comorbidities, and precipitating causes is crucial for early diagnosis and management.

Aims: Aims to document clinical presentations, identify background illnesses and precipitating factors, and follow the illness course during hospital stay in each hypoglycemia case.

Materials and Methods: This hospital based single Centre study was conducted in the N.R.S. Medical College and Hospital Kolkata (Department of General Medicine Ward from March 2020 to August 2021.This study included 100 patients

Result: Among the 100 patients, the mean age was 48.22 ± 3.65 years, with a male predominance (64%). Common clinical features included cold clammy extremities (41%), convulsions (24%), and loss of consciousness (35%). Drug-induced hypoglycemia was the leading diagnosis (73%), followed by sepsis (19%) and alcohol-induced hypoglycemia (8%). Five patients (5%) died during hospitalization. Significant differences between death and survival groups included higher total leukocyte count, neutrophil percentage, CRP, urea, and urine albumin-creatinine ratio, and lower lymphocyte percentage and cortisol levels in those who died. Imaging showed cerebral edema was more common in deceased patients. No significant differences were found in age, sex, comorbidities, or drug history between groups.

Conclusion: Hypoglycemia in hospitalized patients is associated with diverse clinical presentations and underlying conditions. Elevated inflammatory markers, renal dysfunction, low cortisol levels, and cerebral edema are linked to worse outcomes. Early identification of these factors may improve management and prognosis.

Keywords: Hypoglycemia, Clinical Presentation, Comorbidity, Drug-Induced Hypoglycaemia.

Introduction

Hypoglycemia, defined as plasma glucose below 70 mg/dL, is a serious metabolic condition with significant morbidity and mortality risks [1]. While common in diabetes treatment, it also critically affects non-diabetic patients, especially the critically ill, where it predicts higher in-hospital death rates [2, 3]. Diagnosis rests on Whipple's triad: typical symptoms, confirmed low glucose, and symptom relief after glucose normalization. Symptoms include neurogenic (sweating, tremor, tachycardia, hunger) and neuroglycopenic (weakness, confusion, coma) manifestations, with variability in threshold and presentation—often blunted in elderly and critically ill patients [4]. Causes extend beyond diabetes-related factors like insulin overdose, missed meals, and advanced CKD (eGFR <15 mL/min), to include sepsis, impaired gluconeogenesis, insulinoma, heart failure, bariatric surgery, alcoholism, malnutrition, and endocrine disorders [1,4]. Severe hypoglycemia is linked to a six-fold increase in mortality [5]. Intensive glycemic control (HbA1c ≤7%) reduces microvascular risk but major trials (ACCORD, ADVANCE, VADT) revealed that stricter targets (<6.5%) raise hypoglycemia risk threefold, offsetting benefits and increasing mortality in some cases [6, 7, 8]. Insulin-treated patients face the highest risk: severe hypoglycemia occurs in 35-

42% of T1DM and 16.5% of insulin-treated T2DM patients annually, with substantial healthcare burden due to hospital stays and productivity loss [9, 10]. Given the under-recognition of hypoglycemia and its consequences, this study at NRS MCH aims to characterize its spectrum locally, improving diagnosis, treatment, and outcomes [11]. Aims to document clinical presentations, identify background illnesses and precipitating factors, and follow the illness course during hospital stay in each hypoglycemia case.

Materials and Methods

Study Area: The present work was conducted in the N.R.S. Medical College and Hospital Kolkata (Department of General Medicine Ward.)

Study Period: Study was done from MAACH 2020 to August 2021, through one and half year period.

Sample Size: 100

Sample Design: Newly admitted and already admitted Patients in N.R.S Medical College with hypoglycaemia,

Study Design: It was hospital based single Centre study**.**

Inclusion Criteria: All patients in medical ward who's random measured CBG Is below 70mg/dl irrespective to symptoms and background alchemic status.

Exclusion: Patients develop hypoglycaemia (CDG<70 mg /dl) admitted in ITU, CCU, Surgical patients pregnant Lady, Patients who have not given informed consent to participate in this research

Study Tool: Patients of hypoglycaemia require a complete work up to find a potentially treatable disease or precipitating factors.

A. Co-morbidity checklist L Biochemical clinical

1. Diabetes mellitus
2. Hypertension
3. Chronic kidney disease
4. Others

Statistical Analysis: Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages.

Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data. Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

Results

Table 1: Association between Demographic parameters: outcome

	Parameters	Death n (%)	Good Outcome n (%)	Total n (%)	p-value
Age Group (years)	35-40	0 (0%)	5 (5.3%)	5 (5%)	0.469
	41-45	1 (20%)	16 (16.8%)	17 (17%)	
	46-50	1 (20%)	46 (48.4%)	47 (47%)	
	51-55	3 (60%)	28 (29.5%)	31 (31%)	
	Total	5 (100%)	95 (100%)	100 (100%)	
Sex	Female	2 (40%)	34 (35.8%)	36 (36%)	0.8483
	Male	3 (60%)	61 (64.2%)	64 (64%)	
	Total	5 (100%)	95 (100%)	100 (100%)	

Table 2: Association between Clinical Spectrum: outcome

Clinical Spectrum	Death n (%)	Good Outcome n (%)	Total n (%)	p-value
Cold clammy extremities with sweating	2 (40%)	39 (41.1%)	41 (41%)	0.9637
Convulsion	1 (20%)	23 (24.2%)	24 (24%)	
Loss of consciousness	2 (40%)	33 (34.7%)	35 (35%)	
Total	5 (100%)	95 (100%)	100 (100%)	

Table 3: Association between Drug History: outcome

Drug History	Death n (%)	Good Outcome n (%)	Total n (%)	p-value
Chloroquine	0 (0.0%)	14 (14.7%)	14 (14.0%)	0.9055
Glimepiride	0 (0.0%)	12 (12.6%)	12 (12.0%)	
Levofloxacin	0 (0.0%)	6 (6.3%)	6 (6.0%)	
Linagliptin	0 (0.0%)	4 (4.2%)	4 (4.0%)	
Misadjustment between meal and pre-meal regular insulin	1 (20.0%)	12 (12.6%)	13 (13.0%)	
NIL	2 (40.0%)	18 (18.9%)	20 (20.0%)	
NSAIDs	0 (0.0%)	5 (5.3%)	5 (5.0%)	
Propranolol	0 (0.0%)	2 (2.1%)	2 (2.0%)	
Regular insulin	1 (20.0%)	9 (9.5%)	10 (10.0%)	
Several hematemesis after binge alcohol intake	0 (0.0%)	4 (4.2%)	4 (4.0%)	
Stop medications for heart failure last 2 days	1 (20.0%)	9 (9.5%)	10 (10.0%)	
Total	5 (100.0%)	95 (100.0%)	100 (100.0%)	

Table 4: Association between Clinical, Laboratory, Imaging Findings, Diagnosis, and Patient Outcome

Variable	Category	Death n (%)	Good Outcome n (%)	Total n (%)	p-value
Blood Culture	No growth	5 (100.0%)	95 (100.0%)	100 (100%)	
Urine Culture	No growth	4 (80.0%)	82 (86.3%)	86 (86.0%)	0.6915
	Plenty of pus cells in urine	1 (20.0%)	13 (13.7%)	14 (14.0%)	
USG	B/L CMD Completely lost	1 (20.0%)	3 (3.2%)	4 (4.0%)	0.5224
	B/L CMD Partially lost	0 (0.0%)	7 (7.4%)	7 (7.0%)	
	B/L Mild pleural effusion	0 (0.0%)	4 (4.2%)	4 (4.0%)	
	Hepatic abscess	0 (0.0%)	2 (2.1%)	2 (2.0%)	
	Hepatic echotexture lost (Cirrhosis)	1 (20.0%)	14 (14.7%)	15 (15.0%)	
	WNL	3 (60.0%)	65 (68.4%)	68 (68.0%)	
NCCT Brain	Normal	4 (80.0%)	58 (100.0%)	62 (98.4%)	0.0005
	Not available	1 (20.0%)	0 (0.0%)	1 (1.6%)	
MRI Brain	Cerebral edema	3 (60.0%)	11 (19.6%)	14 (23.0%)	0.001
	Normal	0 (0.0%)	42 (75.0%)	42 (68.9%)	
	Not available	2 (40.0%)	3 (5.4%)	5 (8.2%)	
Diagnosis	Alcohol induced hypoglycemia	0 (0.0%)	8 (8.4%)	8 (8.0%)	0.0533
	Drug induced hypoglycemia	2 (40.0%)	71 (74.7%)	73 (73.0%)	
	Sepsis	3 (60.0%)	16 (16.8%)	19 (19.0%)	

Table 5: Distribution of Mean of Clinical and Laboratory Parameters in Death and Good Outcome: Groups

Variable	Death (N=5)	Good (N=95)	p-value
RBS	42.60 ± 15.11	49.79 ± 12.46	0.2157
HbA1c	8.14 ± 2.92	6.80 ± 1.93	0.1443
Hb	11.12 ± 1.59	11.84 ± 1.05	0.1508
TLC	19398.4 ± 10693.5	11989.6 ± 7487.5	0.0372
Neutrophils (N)	76.80 ± 16.04	63.23 ± 14.53	0.0454
Lymphocytes (L)	17.20 ± 10.83	29.78 ± 12.14	0.0255
CRP	163.88 ± 127.95	58.95 ± 74.03	0.0037
BMI	31.80 ± 4.27	34.92 ± 3.76	0.0746
TG	196.60 ± 22.18	188.62 ± 31.91	0.5831
TCHL	252.92 ± 22.23	242.42 ± 42.58	0.5866
HDL	62.80 ± 13.08	62.27 ± 15.68	0.9415
LDL	150.80 ± 15.12	142.55 ± 34.92	0.6015
Na	135.60 ± 2.97	133.51 ± 6.62	0.4859
K	4.06 ± 0.43	4.66 ± 1.06	0.2123
Urea	67.76 ± 85.89	31.21 ± 29.33	0.0196

Creatinine	1.77 ± 1.19	1.21 ± 0.70	0.1008
TB	1.84 ± 1.99	0.97 ± 2.31	0.4094
SGOT	117.20 ± 183.54	50.89 ± 89.34	0.1316
SGPT	128.40 ± 210.70	58.67 ± 124.84	0.2433
ALP	47.20 ± 18.31	51.05 ± 16.79	0.6194
ALB	3.46 ± 0.98	3.69 ± 0.45	0.2999
GLB	3.64 ± 0.31	3.79 ± 2.03	0.8689
Spot urine ACR	594.60 ± 786.71	138.65 ± 353.19	0.0106
Cortisol Level	6.30 ± 1.73	13.20 ± 7.28	0.0376

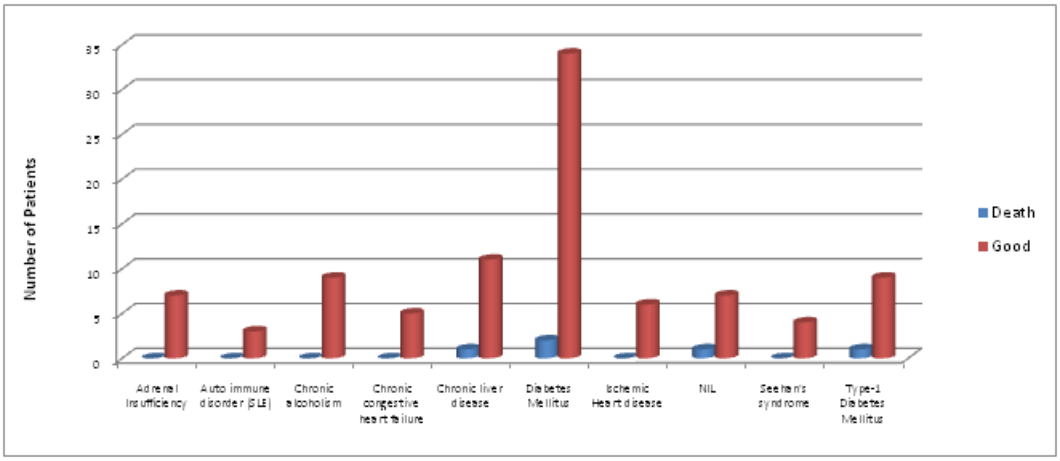

Figure 1: Association between Comorbidity: outcome

Among the patients, the age distribution showed no statistically significant difference between death and good outcome groups (p = 0.469). In the death group (n=5), 0% were aged 35–40 years, 20% were 41–45 years, 20% were 46–50 years, and 60% were 51–55 years. In the good outcome group (n=95), 5.3% were 35–40 years, 16.8% were 41–45 years, 48.4% were 46–50 years, and 29.5% were 51–55 years. Overall, 5% of patients were 35–40 years, 17% were 41–45 years, 47% were 46–50 years, and 31% were 51–55 years.

Regarding sex distribution, there was no significant difference (p = 0.8483). In the death group, 40% were female and 60% male, while in the good outcome group, females constituted 35.8% and males 64.2%. Overall, females made up 36% and males 64% of the total patients.

The clinical spectrum showed no significant differences between the death and good outcome groups (p = 0.9637). Among patients who died (n=5), 40% presented with cold clammy extremities with sweating compared to 41.1% in the good outcome group (n=95). Convulsions were observed in 20% of the death group and 24.2% of the good outcome group. Loss of consciousness occurred in 40% of patients who died and 34.7% of those with good outcomes. Overall, cold clammy extremities were seen in 41%, convulsions in 24%, and loss of consciousness in 35% of the total 100 patients.

In the death group (n=5), 20% of patients had misadjustment between meal and pre-meal regular insulin, 20% were on regular insulin, and 20% had stopped medications for heart failure in the last 2 days. Additionally, 40% reported no drug history (NIL). In the good outcome group (n=95), 14.7% used chloroquine, 12.6% glimepiride, 6.3% levofloxacin, 4.2% linagliptin, 12.6% had insulin misadjustment, 9.5% were on regular insulin, 9.5% stopped heart failure meds recently, 18.9% reported no drug history, and smaller percentages took NSAIDs (5.3%) or propranolol (2.1%). No statistically significant difference was observed between groups regarding drug history (p = 0.9055).

Blood cultures showed no growth in all patients (100%). Urine culture revealed no growth in 80% of the death group and 86.3% of the good outcome group, with 20% and 13.7% showing plenty of pus cells, respectively (p = 0.6915). Ultrasound (USG) findings included bilateral CMD completely lost in 20% of deaths versus 3.2% in survivors; other findings such as partial CMD loss, mild pleural effusion, hepatic abscess, and cirrhosis were observed mainly in the good outcome group. Normal USG was noted in 60% of deaths and 68.4% of survivors (p = 0.5224). Non-contrast CT (NCCT) brain scans were normal in 80% of deaths and 100% of survivors, with a significant difference between groups (p = 0.0005). MRI brain revealed cerebral edema in 60% of deaths compared to 19.6% of survivors (p = 0.001), with

normal MRI findings in 75% of survivors and none in the death group. Regarding diagnosis, drug-induced hypoglycemia was the most common in both groups (40% deaths, 74.7% survivors), followed by sepsis (60% deaths, 16.8% survivors) and alcohol-induced hypoglycemia seen only in survivors (8.4%). The difference was not statistically significant ($p = 0.0533$). In patients who died, the mean RBS was 42.60 ± 15.11, whereas in survivors it was 49.79 ± 12.46; this difference was not statistically significant ($p = 0.2157$). The mean HbA1c was 8.14 ± 2.92 in the death group and 6.80 ± 1.93 in the good outcome group ($p = 0.1443$). Haemoglobin (Hb) values were 11.12 ± 1.59 versus 11.84 ± 1.05, respectively, without significant difference ($p = 0.1508$). Total leukocyte count (TLC) was significantly higher in the death group (19398.4 ± 10693.5) compared to survivors (11989.6 ± 7487.5, $p = 0.0372$). Neutrophil percentage (N) was also higher in deceased patients (76.80 ± 16.04) than in survivors (63.23 ± 14.53, $p = 0.0454$), while lymphocyte percentage (L) was significantly lower in the death group (17.20 ± 10.83) versus 29.78 ± 12.14 in survivors ($p = 0.0255$). CRP was markedly elevated in patients who died (163.88 ± 127.95) compared to survivors (58.95 ± 74.03, $p = 0.0037$). BMI showed no significant difference (31.80 ± 4.27 vs 34.92 ± 3.76, $p = 0.0746$). Triglycerides (TG), total cholesterol (TCHL), HDL, LDL, sodium (Na), potassium (K), creatinine, total bilirubin (TB), SGOT, SGPT, ALP, albumin (ALB), and globulin (GLB) also showed no significant differences between groups ($p > 0.05$). Urea levels were significantly higher in the death group (67.76 ± 85.89) compared to survivors (31.21 ± 29.33, $p = 0.0196$). Spot urine albumin-to-creatinine ratio (ACR) was elevated in deceased patients (594.60 ± 786.71) versus survivors (138.65 ± 353.19, $p = 0.0106$). Finally, cortisol levels were significantly lower in the death group (6.30 ± 1.73) compared to the good outcome group (13.20 ± 7.28, $p = 0.0376$).

In outcome of died patients, 1 (20.0%) patient had Chronic liver disease, 2 (40.0%) patients had Diabetes Mellitusand 1 (20.0%) patient had Type-1 Diabetes Mellitus.In outcome of Alive patients, 7 (7.4%) patients had Adrenal Insufficiency,3 (3.2%) patients had Auto immune disorder (SLE),9 (9.5%) patients had Chronic alcoholism, 5 (5.3%) patients had Chronic congestive heart failure, 11 (11.6%) patients had Chronic liver disease, 34 (35.8%) patients had Diabetes Mellitus, 6 (6.3%) patients had Ischemic Heart disease, 4 (4.2%) patients had Seehan's syndrome and 9 (9.5%) patients had Type-1 Diabetes Mellitus. Association of Comorbidity with outcome was not statistically significant ($p=0.9375$).

Discussion

This hospital-based single-centre study conducted at N.R.S. Medical College and Hospital, Kolkata, from March 2020 to August 2021 included 100 patients admitted with random blood glucose (RBS) <70 mg/dL, irrespective of symptoms or diabetic status. The mean age was 48.22 ± 3.65 years, with 64% males and 36% females. No significant association was found between age or sex and mortality ($p > 0.05$), which is consistent with Kim et al. [5], who also reported that demographic factors alone are not reliable predictors of hypoglycemia-related death.

Clinically, 41% presented with cold clammy extremities and sweating, 24% with convulsions, and 35% with loss of consciousness. These features showed no significant mortality association, similar to Leese GP et al. [11], who found that symptom severity did not independently predict outcomes in severe hypoglycemia. Drug history revealed 14% on chloroquine and 13% with insulin misadjustment, but no significant association with mortality ($p > 0.05$), which is in agreement with Kagansky N et al. [14].

Laboratory analysis showed that the total leukocyte count (TLC) was significantly higher in deceased patients (19398 ± 10693 vs. 11990 ± 7487; $p = 0.037$), and neutrophil percentage was also elevated (76.8 ± 16.0 vs. 63.2 ± 14.5; $p = 0.045$). Lymphocyte percentage was lower in deaths (17.2 ± 10.8 vs. 29.8 ± 12.1; $p = 0.025$). Similar inflammatory changes were observed by Cryer PE et al. [13], who noted that systemic inflammation could exacerbate hypoglycemia outcomes. C-reactive protein (CRP) was markedly elevated in deaths (163.9 ± 128 vs. 58.9 ± 74; $p = 0.004$), consistent with Zammitt NN et al. [10], who found CRP to be a marker of adverse prognosis. Serum urea and spot urine albumin-creatinine ratio (ACR) were higher in deaths ($p = 0.0196$ and $p = 0.0106$, respectively), in line with findings from Zoungas S et al. [12] linking renal dysfunction to poor hypoglycemia outcomes. Cortisol levels were lower in deceased patients (6.3 ± 1.7 vs. 13.2 ± 7.3; $p = 0.038$), a result comparable to observations by Jørgensen HV et al. [17] implicating inadequate counter-regulatory hormonal response in mortality.

Neuroimaging findings revealed cerebral edema in 60% of deaths versus 19.6% of survivors ($p = 0.001$), and normal NCCT scans were less frequent in deaths (80% vs. 100%; $p = 0.0005$). These results support the conclusions of Suh SW et al. [15] that hypoglycemia-induced neuronal injury and cerebral edema are critical determinants of poor neurological outcome. Overall, our study highlights that while demographic factors, clinical presentation, comorbidities, and medication history may not significantly influence mortality in

hypoglycemia, systemic inflammatory markers, renal function parameters, cortisol levels, and neuroimaging abnormalities are strongly associated with adverse outcomes. These findings echo earlier research [5,10, 12,13,15,17], suggesting that integrating inflammatory, hormonal, and neuroimaging assessments into risk stratification could improve prognosis prediction in hypoglycemia.

Conclusion

This study of 100 patients with hypoglycemia in a tertiary care center found no significant differences in age, sex, clinical presentation, drug history, or comorbidities between patients who died and those with good outcomes. Drug-induced hypoglycemia was the most common cause, followed by sepsis and alcohol-induced hypoglycemia. Significant predictors of mortality included higher total leukocyte count, increased neutrophil percentage, lower lymphocyte percentage, elevated CRP, higher urea and urine albumin-to-creatinine ratio, and notably lower cortisol levels. Imaging findings such as abnormal NCCT and cerebral edema on MRI were also associated with mortality. These findings underscore the importance of comprehensive clinical and biochemical evaluation in hypoglycemic patients to identify those at greater risk and guide timely management.

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