Kanem J Med Sci 2025; 19(2): 71-78

ISSN 2006-4772
https://doi.org/10.36020/kjms.2025.1902.001

Original Article

Blood ketone levels in acute paediatric non-diabetic dysglycaemia and associated clinical outcomes

Ruqayyatu A. Imam,[1] Hassan A. Elechi,[2] Asheikh M. Masu,[1] Sajah I. Wala,[1] Hauwa Y. Musa [1] .[2]

[1]Department of Paediatrics, University of Maiduguri Teaching Hospital, Maiduguri, Nigeria.
[2] Department of Paediatrics, College of Medical Sciences, University of Maiduguri, Nigeria.

Correspondence to: Dr Hassan Abdullahi Elechi, Department of Paediatrics, College of Medical Sciences, University of Maiduguri, Nigeria. Email: h3elechi@unimaid.edu.ng. Phone: +2348034318487

Abstract

Background: Children with acute illness are prone to energy dysregulation, marked biochemically as dysglycaemia with or without elevated blood ketone levels. Dysglycaemia has been linked to poor clinical outcomes without recourse to the ketone level. **Objectives** - To determine blood ketone levels among children with Dysglycemia and the association between ketone levels and Clinical outcome. **Methodology**: It was a cross-sectional study carried out at the Emergency Paediatric Unit of the University of Maiduguri Teaching Hospital among Children aged 1 month to 15 years. Children with dysglycaemia were tested for blood ketone levels. Data was analysed using SPSS Version 25. **Results**: Of the 340 subjects, 135 (39.7%) had dysglycaemia, out of which 81 (23.8%) were hyperglycaemic and 54 (15.9%) hypoglycaemic. Fifty-eight (43.0%) of those with dysglycaemia had elevated blood ketone, consisting of 12 (20.7%) in the hyperglycaemic group and 46 (79.3%) in the hypoglycaemic group. Age < 5 years {OR = 34.455 (2.147-550.458), p=0.012}, duration since last meal ≥ 24 hours {OR= 142.92(3.094-6589.604), P=0.011}, severe sepsis {OR=151.26(9.184-2495.760), P= <0.001} and hypoglycaemia, {OR= 50.529 (12.968-196.457) and P = < 0.001 were all independent predictors of elevated blood ketone level. Dysglycaemic subjects with Elevated blood ketone were approximately 3 times more likely to die than those with normal blood ketone {UOR=3.4 (0.983-11.583) and P=0.043}. **Conclusion**: There was a high prevalence of dysglycaemia among acutely ill children. Elevated blood ketone levels among this cohort were associated with a bad outcome. We recommend routine determination of blood ketone level in children identified with dysglycaemia for closer monitoring and prognostication

Keywords: *Blood ketone levels, Clinical outcome, Paediatric acute dysglycaemia*.

Introduction

Acutely ill children are prone to dysregulation in energy metabolism, manifesting biochemically as dysglycaemia with or without elevated blood ketone levels, which is a marker of a switch to an alternative source of energy.[1-3] High blood ketone levels in an ill child with acute hyperglycaemia result from insulin resistance due to the high levels of counter-regulatory hormones. This includes stress hormones such as cortisol, epinephrine and glucagon and may thus be a marker of severity of illness.[4,5] Whereas in hypoglycaemia, low levels of glucose elicit counterregulatory hormones and switch to an alternative source of fuel with ketone body generation. The ketones generated are oxidised in the brain to generate energy and thus are protective to the brain.[6,7] Ketotic hypoglycemia is a normal physiologic response to prolonged fasting and is seen earlier in children due to high glucose utilisation and relatively low glycogen storage.[8] It is, however, seen occasionally in the absence of an appropriate cause to explain the hypoglycaemia, in which case it is called idiopathic ketotic hypoglycaemia. Although the real

Cite this article as: Ruqayyatu A. Imam, Hassan A. Elechi, Asheikh M. Masu, Sajah I. Wala, Hauwa Y. Musa. Blood ketone levels in acute paediatric non-diabetic dysglycaemia and associated clinical outcomes.Kanem J Med Sci 2025; 19(2): 71-78

Received: 15th June 2025 **Accepted: 19th October 2025** **Published: 30th December 2025**

cause of idiopathic ketotic hypoglycaemia is not known, it has been hypothesised that it may be due to failure to maintain hepatic glucose production rather than increased glucose oxidation.[9] Lack of high blood ketone during prolonged hypoglycaemia may suggest endocrine abnormality or inborn errors of lipid metabolism.

Several studies have reported a significant relationship between dysglycaemia and adverse clinical outcomes.[3,10-13] Mortality rates among children with hypoglycaemia from these studies were as high as 61.5 %[12], 43.7%[13], 36.00 %[11] and 20.0 %[3]. Among those with hyperglycaemia were 31.2 %[13], 14.0%[3], 13.0 %[12] and 11.0 %[11,] which, even though they were lower than the rates in hypoglycaemia, were still much higher than observed in children with euglycaemia (25.0 %[12], 5.0 %[11],5.1 %[12] and 3.8 %[3]). Whereas these studies and several others have demonstrated a significant association between dysglycaemia and adverse clinical outcomes, none considered the effect of ketone level, a marker of severity of illness, during dysglycaemia on clinical outcomes.

This study thus determined the serum ketone level among acutely ill children with dysglycaemia and the association of the serum ketone levels with clinical outcome. The result will serve as a novel finding in this aspect of knowledge and a guide for recommendations on the clinical usefulness or otherwise of blood ketone determination among acutely ill children with dysglycaemia.

Material and methods

This was a cross-sectional study carried out from 1st February to September 2020, among children admitted into the Emergency Paediatrics Unit (EPU), University of Maiduguri Teaching Hospital (UMTH), Maiduguri.

The Study subjects comprised children aged 1 month to 15 years who were admitted to the EPU of UMTH, Maiduguri, during the study period. Children whose caregivers refused to give consent for participation in the study were excluded.

Sample size was determined using the Cochran Formula[14,] considering the finite population of less than 10,000

$n_0 = \frac{Z^2pq}{e^2}$

Where:

n_0 = Minimum sample size

Z = Confidence limit set at 95% confidence interval = 1.96

P = Prevalence of hyperglycaemia /hypoglycaemia in a study conducted in Enugu[13] is 39% and 20.7%, respectively. Larger prevalence that is 39% was used = 0.39

q = Complementary probability = 1 – p, = 1 – 0.39= 0.61

e = Degree of precision, set at 0.05 (5%)

Thus,

$n_0 = \frac{(1.96)^2 \times 0.39 \times 0.6}{0.0025}$

n_0= 365

The population is finite, so the correction formula was used.

$$n = \frac{n_0}{1 + \frac{(n_0 - 1)}{N}}$$

n_0 = Is the Cochran's sample size recommendation, which is 365

N Population size, which is 3,979

N= New adjusted (corrected) sample size according to Cochran

Hence,

$n = \frac{365}{1 + 0.09}$

$n = \frac{365}{1 + 0.09}$

n = 335

Eligible patients were recruited consecutively as they presented to the EPU over the study period. For each of the patients, a pre-designed interviewer-administered questionnaire was used to collect information on socio-demographic characteristics. Other clinical data, including presenting symptoms, duration since last meal, anthropometric indices, clinical diagnosis and other clinical features were also recorded.

Random blood glucose (RBG) testing was performed for all the patients using a point-of-care test glucometer (ACCU-CHECK[R] with strips), according to the manufacturer's instructions. 2ml of venous blood was collected into a fluoride oxalate container by the researcher or assistant at the point of cannula insertion. Finger prick to obtain capillary blood was avoided to minimise false positive hypoglycaemia secondary to tissue fluid diluting the capillary blood while squeezing the finger to obtain blood. The performance of the glucometer was previously validated and correlates very well with standard laboratory blood glucose measurement with a correlation coefficient of 0.96 and 99% values in the Parke's zones A and B.[15] A drop of blood was applied at the sample area (after mixing it by rolling the specimen bottle) when the screen indicated readiness

to drop the blood and the reading was displayed on the screen in mmol/L and recorded. All patients that were found to be dysglycaemic had blood ketone determined from the same sample using the ketone meter and strips, On Call[R] GK dual blood ketone meter, another point of care test device. The ketone meter switches on when the strip is inserted into the strip chamber. Blood was applied to the end of the test strip, the blood application area, when the screen indicated readiness for blood application. The level of blood ketone was displayed in mmol/L on the display area of the meter. Blood ketone level >1mmol/l was considered high blood ketone.[16] hypoglycaemia and hyperglycaemia were defined as RBG of <2.2mmol/l[17] and >7.8mmol/l[18] respectively.

Approval for the study was obtained from the Health Research and Ethics Committee of UMTH, Maiduguri. The research procedure was duly explained to the child and /or the caregiver, and informed consent and ascent (were eligible) were obtained. Failure to give consent had no consequence whatsoever on the subsequent care of the child. All patients detected to have dysglycaemia were managed according to the recommended unit guidelines.

Data was entered into and analysed using SPSS version 25. Age was presented in median (IQR), while sex, nutritional status and clinical diagnoses were described in frequency and percentage. Blood ketone levels were categorised into two groups: normal and high blood ketone levels, and presented in a bar chart. The association between blood ketone levels and types of dysglycaemia, sociodemographic features, as well as clinical diagnoses, were tested using chi-square or, where indicated, Fisher's Exact test. Independent association between blood ketone levels and factors found significant at bivariate analysis was assessed using a logistic regression model, while simple binary logistic regression was used to assess the relationship between blood ketone levels and clinical outcome. A p-value of <0.05 was considered statistically significant.

Results

Three hundred and forty children were recruited for the study. There were 206 (60.6%) males with a male-to-female ratio of 1.5:1. The age ranged between 1 month to fifteen years (180 months) with a median (IQR) age of 30.5 (12 - 80) months. Two hundred and twenty-six (66.5%) of the total subjects were < 5years. One hundred and seventy-five (51.5%) had normal weight for age, and the remaining 165 (48.5%) were at least moderately underweight. On the other hand, 255 (75.0%) had normal height/length for age, and the remaining 85 (25.0%) were at least moderately stunted. Severe acute malnutrition 53 (15.6%), pneumonia 51 (15.0%) and severe malaria 42 (12.4%) were the most frequent diagnoses. Other common diagnoses encountered include surgical cases 30 (8.80%), diarrhoeal disease 26 (7.6%), sickle cell crises 21 (6.2%), febrile convulsion 19 (5.6%), enteric fever 18 (5.3%), acute pharyngotonsillitis 17 (5.0%), urinary tract infection 15 (4.4%), severe sepsis 9 (2.6%), acute bacterial meningitis 9 (2.6%) and others 30 (8.8%).

One hundred and thirty-five (39.7%) of the 340 subjects had dysglycaemia, 54 (15.9%) had hypoglycaemia, and 81 (23.8%) had hyperglycaemia.

Of the 135 children with dysglycaemia, 46 (85.2%) of those with hypoglycaemia and 12 (14.8%) of those with hyperglycaemia had high blood ketone levels, figure 1. Hypoglycaemia was significantly associated with high blood ketone levels. P-value is <0.001, Table I.

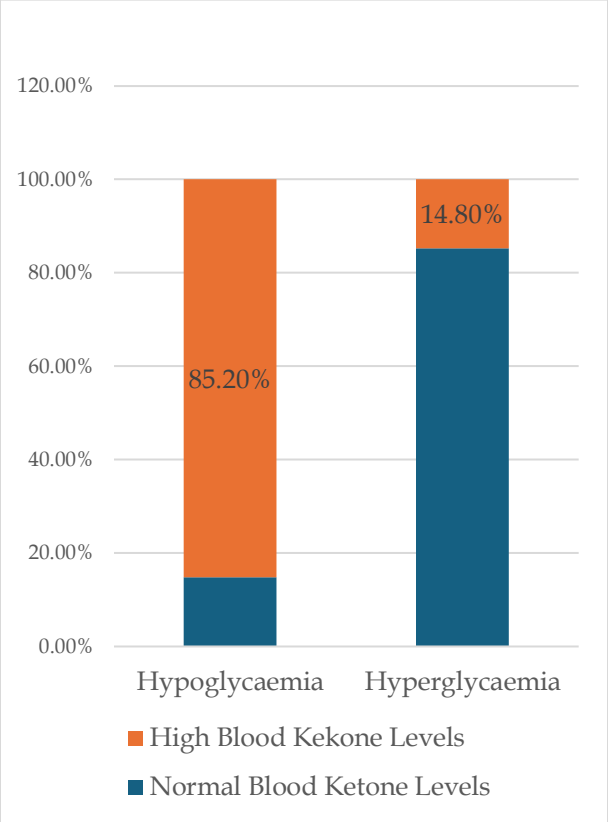

Figure 1: Blood ketone category distribution of dysglycaemic children

Table 1: Blood ketone pattern of dysglycaemic children

Dysglycaemia	KETONE		Total n (%)	X^2	p-value
	NORMAL n (%)	HIGH n (%)			
Hyperglycaemia	69 (89.6)	12 (20.7)	81	61.10	**<0.001***
Hypoglycaemia	8 (10.4)	46 (79.3)	54		
Total	77 (100)	58 (100)	135		

Table 2 shows the relationship between blood ketone categories with age, nutritional status and duration since last meal. Age and Duration since last meal were significantly associated with blood ketone levels, p=0.020 and 0.001 for age and duration since last meal, respectively

.

Table 2: Relationship between blood ketone level with age, nutritional status and duration since last meal.

Variable		Ketone category			X^2	P-Value
		High 58 (100%)	Normal 77(100%)	Total 135		
Age Category	1month -<5yrs	43 (74.1)	47 (61.0)	90	7.492	**0.020****
	5 -<10yrs	12 (20.7)	13 (16.9)	25		
	10 -15yrs	3 (5.2)	17 (22.1)	20		
WAZ	Normal	27(46.6)	42 (54.5)	69	2.473	0.296
	Moderate underweight	6 (10.3)	12 (15.6)	18		
	Severe underweight	25(43.1)	23 (29.9)	48		
HAZ	Normal	46(79.3)	64 (83.1)	110	1.815	0.403
	Moderate stunting	2 (3.5)	5 (6.5)	7		
	Severe stunting	10 (17.2)	8 (10.4)	18		
Duration since last meal	<8hours	8 (13.8)	23 (29.9)	31	27.790	**0.001***
	8-<16hours	9 (15.5)	26 (33.8)	35		
	16-24hours	24 (41.4)	27 (35.0)	51		
	>24hours	17 (29.3)	1(1.3)	18		

*Pearson's chi-square/** Fishers exact-significant P-value). WAZ- weight-for-age Z-score, HAZ- height/length-for-age Z-score.

Of all the diagnoses, only severe sepsis had a significant association with the blood ketone category.

Table 3: Relationship between blood ketone category and various diagnoses

DIAGNOSIS	BLOOD KETONE CATEGORY			X^2	P-VALUE
	HIGH 58 (100%)	NORMAL 77 (100%)	TOTAL 135		
Severe Malaria	13(22.4)	15(19.5)	28	0.173	0.677
Pneumonia	10(17.2)	10(13.0)	20	0.094	0.759
Diarrhoeal disease	5(8.6)	3(3.9)	8	1.325	0.216
Enteric fever	0(0.0)	5(6.5)	5	3.911	0.057
Meningitis	2(3.4)	5(6.5)	7	0.624	0.353
Febrile Convulsion	2(3.4)	10(13.0)	12	3.717	0.070
Malnutrition	11(19.0)	10(13.0)	21	0.900	0.484
Surgical	5(8.6)	6(7.8)	11	0.030	1.000
Severe Sepsis	8(13.8)	1(1.3)	9	8.300	**0.005****
SCA	0(0.0)	2(2.6)	2	1.529	0.323
UTI	0(0.0)	2(2.6)	2	1.529	0.323
Pharyngotonsilitis	2(3.4)	4(5.2)	6	0.238	0.482
^Others	0(0.0)	4(5.2)	4	3.105	0.102

** significant P-Value, SCA sickle cell anaemia, UTI= urinary tract infection, n (%) = frequency (percentage of total for each variable), ^Others: tuberculosis, acute poisoning, tetanus, afebrile seizure, measles, asthma, malignancies, nephrotic syndrome, acute glomerulonephritis, chronic kidney disease, HIV infection, congenital heart disease.

Table 4 shows multiple regression models for factors significantly associated with high blood ketone levels. Age < 5 years, duration since last meal of ≥ 24 hours, severe sepsis and hypoglycaemia were found to be independently associated with high blood ketone level. Children aged < 5 years were at least 34 times more likely to have high blood ketone levels when compared to those aged 10 years and above. Similarly, children who last ate ≥ 24 hours or had severe sepsis or hypoglycaemia were 142, 151 and 50 times more likely to have high blood ketone levels when compared to those who last ate < 8hours, without severe sepsis and with hyperglycaemia, respectively.

Table 4: Multiple logistic regression model for factors associated with high blood ketone category.

Variables		Total enrolled	High ketone	OR (95% CI)	P-Value
Age	-<5years	90	43 (47.8)	34.455 (2.147-550.458)	**0.012***
	5-<10years	25	12 (48.0)	7.332 (0.433-124.100)	0.167
	10-15years	20	3 (15.0)	1	
Duration since last meal	<8hours	31	8 (25.8)	1	
	8- <16hours	35	9 (25.8)	1.470 (0.252-8.663)	0.663
	16- <24 hour	51	24 (47.1)	2.624 (0.466-13.981)	0.249
	>24hours	18	17 (94.4)	142.92(3.094-6589.604)	**0.011***
Severe Sepsis	Yes	9	8 (88.9)	151.26(9.184-2495.760)	**<0.001***
	No	126	50 (39.7)	1	
Dysglycaemia	Hypoglycaemia	54	46 (85.2)	50.475 (12.968-196.457)	**<0.001***
	hyperglycaemia	81	12 (14.8)	1	

OR-odds ratio, CI-confidence interval, *significant P-value

Approximately 70% of deaths were among those with high blood ketone, while approximately 60%, 73 (59.8%) of discharges were among those with normal ketone category. High blood ketone levels had a significant association with death, and the odds of dying among those with high blood ketone levels were about 3 times higher when compared to those with normal blood ketone levels, Table 5.

Table 5: Relationship between blood ketone category and clinical outcome

	Outcome					
Blood Ketone category	Death n (%)	Discharged n (%)	Total	P-Value	OR	(95% CI)
High Ketone	9(69.2)	49 (40.2)	58	**0.044***	3.352	0.978– 11.493
Normal Ketone	4(30.8)	73 (59.8)	77			
Total	13(100)	122 (100)	135			

OR- Odds ratio, n (%) = frequency (percentage). OR- Odds ratio, CI- confidence interval. *Significant P-Value

Discussion

This study determined blood ketone levels among children with dysglycaemia and the association between blood ketone levels and clinical outcomes. Dysglycaemia was found in 135 (39.7%) of the studied children. Forty-six (85.2%) of those with hypoglycaemia and 12 (14.8%) of those with hyperglycaemia had high blood ketone levels. Children with high blood ketone levels were 3 times more likely to die compared to those with normal blood ketone levels.

The prevalence of dysglycaemia of 39.7% is within the range previously reported of 10.9%- 57.4%.[3,10,12] This prevalence is composed of 23.8% for hyperglycaemia and 15.9% for hypoglycaemia. The prevalence of hypoglycaemia in this study of 15.9% was higher than the 0.3% reported by Oluwayemi *et al*[19] from Nigeria in 2018 and 3.1% by Sambany *et al*[12] from Madagascar in 2013. While the current study recruited acutely ill children admitted into the children's emergency, Oluwayemi *et al. (*[19] studied children in the outpatient Clinic, and dysglycaemia is known to complicate severe illness. Similarly, while the current study was in a malaria endemic area, the Madagascar study was in non-malaria areas, and hypoglycaemia is a defining feature of severe malaria. Conversely, the prevalence of 15.9% was lower than 20.7% and 22.1% in Nigeria

reported by Uleanya *et al.* [13] in 2017 and Abdullahi *et al.* [20] in 2019. This lower prevalence may be due to the difference in the definition of hypoglycaemia. While this study defined hypoglycaemia as RBG <2.2mmol/l, Abdullahi *et al*[20] and Uleanya *et al*[13] defined it as RBG <2.8mmol/l and <3.6 mmol/l, respectively, thus increasing the range of patients recruited as hypoglycaemic in these studies. On the other hand, the prevalence of hyperglycaemia of 23.8% from this study was lower than 27.2% and 53% reported by Soliman *et al*[21] from Egypt in 2021 and Kirti *et al*[10] from India in 2017. The lower prevalence may be due to the differences in the subjects used. While the current study used less severely ill patients who do not require ICU care, the other two studies were carried out in the more stressful environments of PICU among critically ill and peri-operative patients with traumatic brain injury, respectively. The prevalence was, however, higher than 2.7% by Osier *et al*[3] from Kenya and 10.7% by Sambany *et al*[12] from Madagascar. The higher prevalence could be due to the differences in the definition of hyperglycaemia.

In this study, high blood ketone level was found to be a common occurrence among non-diabetic dysglycaemic children, with 43% of them affected. Whereas hyperglycaemia, consistent with previous studies,[10,12,21,] was the more prevalent type of dysglycaemia, high blood ketone level was significantly more common among the hypoglycaemic group, 85.2%. Kim *et al*[22] from South Korea reported a comparable high prevalence, 80.5%, of ketosis among children with hypoglycaemia. Monde *et al*[23] from Cote d'Ivoire reported a slightly lower prevalence of 70%, which is not surprising due to the difference in subjects studied. While the current study and that by Kim *et al*[22] studied acutely ill children in the emergency unit, Monde *et al*[23] studied school children who weren't ill. These findings are in consonance with the expected physiologic response to hypoglycaemia in an acutely ill child free of hyperinsulinism, disorder of fatty acid and urea cycle metabolism.[6] Hypoglycaemia through the elaboration of the counter-regulatory hormones activates the release and breakdown of free fatty acid into Acetyl-CoA for gluconeogenesis and ketone bodies for utilisation in the brain.[17] The reduced yield of glucose from glycogenolysis and gluconeogenesis during periods of increased metabolic demand and poor intake associated with illness predisposes these patients to hypoglycaemia and ketosis. [9,24]

The study also showed an independent significant association between high blood ketone levels and age less than five years, duration since last meal and severe sepsis. Several other studies have documented the significant relationship between age less than five years and ketotic hypoglycaemia. Paul *et al*[25] from the USA reported a median age of 2.9 years, while Al-Amin *et al*.[9] from Bangladesh reported an age range of 0.5 – 6 years with remission at 8-9 years. Although the underlying reason for this relationship is far from settled, it is believed that younger children are prone to ketotic hypoglycaemia due to limitations in their glucose homeostasis compared to older children and adults. They have reduced hepatic glucose output, coupled with their relatively higher rates of glucose consumption due to their larger brain-to-body mass ratio.[8] They are deficient in alanine, a major gluconeogenic precursor derived from the muscles, due to their small muscle mass and hence the subsequent remission with an increase in muscle mass by 8-9 years.[9]

The mechanism linking severe sepsis to high blood ketone levels, though inconclusive, may involve several mechanisms depending on the infecting organism and severity of the infection. Inhibition of gluconeogenesis,[26] endotoxin-induced insulin secretion,[26-28] direct toxin-induced depletion of hepatic glycogen store,[28] increase metabolic demand from fever, 28 and anaerobic respiration in septic shock are all mechanisms through which sepsis predisposes to hypoglycaemia and then the subsequent ketosis. On the other hand, disordered glucose metabolism from counter-regulatory hormones, insulin resistance from increased elaboration of cytokines, enhanced Warburg effect and excessive degradation of dipeptidyl peptidase IV are mechanisms leading to hyperglycaemia in severe sepsis.[29] Failure of peripheral glucose utilisation in turn leads to lipolysis, release and breakdown of free fatty acids into acetyl-CoA and ketone bodies.

The relationship between high blood ketone levels and long fasting is a known and well-demonstrated phenomenon, which is attributable to glycogen store depletion and the switch to the beta-oxidative pathway for ATP generation.[7,8,17] In this study duration since the last meal ≥ 24 hours showed an independent association with high blood ketone level. While Onyearugha *et al*[30] and Onyiriuka *et al*[31,] both from Nigeria, found duration since last meal of >12 hours to be significantly associated with hypoglycaemia, these studies did not assess blood ketone level and its association with duration since last meal. However, children prone to idiopathic ketotic hypoglycaemia are likely to develop

hypoglycaemia and ketosis within 14 to 24 hours of fast against 36 hours in healthy normal children of similar age not prone to idiopathic ketotic hypoglycaemia.[9]

High blood ketone was found to have a significant relationship with mortality; the unadjusted odds ratio showed that those with high blood ketone levels were more than 3 times likely to die than their counterpart with normal blood ketone levels. This finding may be important in prognostication among children with dysglycaemia. The mechanism by which high blood ketone levels lead to increased mortality may not be immediately clear, but children with idiopathic ketotic hypoglycaemia are thought to represent the low end of the spectrum of capacity to tolerate fasting.[9] In addition, dysglycaemia with ketosis is a marker of severity of illness, particularly in stress-induced hyperglycaemia.[4,5] The literature search did not yield any previous studies on blood ketone levels among non-diabetic children with acute dysglycaemia and its association with clinical outcome.

This study has demonstrated that high blood ketone levels in children with dysglycaemia may, after all, be a poor prognostic factor. Since the rise in blood ketone and its appearance in the urine, ketonuria, precedes hypoglycaemia in idiopathic ketotic hypoglycaemia and prolonged fast by several hours, routine ketone determination in the blood or urine in at-risk children may help avert hypoglycaemia by initiating appropriate prophylaxis. This principle may not apply to children living with diabetes mellitus. The study is, however, limited by the inability of the authors to specifically exclude hormonal and other metabolic causes of hypoglycaemia in all the children with ketotic hypoglycaemia.

Conclusion

High blood ketone level is a common occurrence among children with dysglycaemia. Children under the age of five years, prolonged fast and severe sepsis are independent factors predicting high blood ketone levels, which may indicate poor prognosis among children in this cohort. We recommend routine determination of blood ketone level among children with dysglycaemia or at-risk groups at the Emergency Paediatric Unit.

References

1. Metwalley KA, Farghaly HS. Idiopathic ketotic hypoglycemia in children: an update. Ann Pediatr Endocrinol Metab. 2024;29(3):152–155. doi: 10.6065/apem.2346156.078
2. Argyropoulos T, Korakas E, Gikas A, Kountouri A, Kostaridou-Nikolopoulou S, Raptis A, et al. Stress Hyperglycemia in Children and Adolescents as a Prognostic Indicator for the Development of Type 1 Diabetes Mellitus. Front Pediatr. 2021; 9:670976. doi: 10.3389/fped 2021.670976.
3. Osier FHA, Berley JA, Sanderson F, Ross A, Mohammad S, Newton C. Abnormal blood glucose concentrations on admission to a rural Kenyan district hospital: prevalence and outcome. Arch Dis Child. 2003 Jul;88(7):621-5. Doi: 10.1136 /adc.88.7.621.
4. Vedantam D, Poman DS, Motwani L, Asif N, Patel A, Anne KK. Stress-Induced Hyperglycemia: Consequences and Management. Cureus. 2022;14(7): e26714. doi: 10.7759/cureus 26714.
5. Ganesan G, Vidyasagar S, Bolanthakodi N, Sukumar CA. Clinical profile of stress hyperglycemia in medical intensive care units. Clin Epidemiol Global Health. 2025; 32:101941.
6. Freeman VS. Carbohydrates. In: Bishop ML, Fodym EP, Schoeff LF, editors. Clinical chemistry: techniques, principles and correlation. 6th ed. Burlington: Jones & Bartlett Learning; 2010. p. 309–28.
7. Sacks DB, Carbohydrates. In: Burtis CA, Ashwood ER, Bruns DE, Tietz NW, editors. Tietz's textbook of clinical chemistry and molecular diagnosis. 5th ed. Edinburgh: Elsevier/Saunders; 2012.p. 709–30.
8. Thornton TS, Finegold DN, Stanley CA, SM. Hypoglycaemia in infants and children. 2nd ed. Sperling MA, editor. Philadelphia: Saunders; 2002. 135–159.
9. Al Amin, MD, Abdul M. Ketotic Hypoglycemia in Children: A Review. Bangladesh J Child Health. 2019;43(2):113-116.
10. Kirti MN, Banani P. Blood Glucose Variability and Outcomes in Critically Ill Children. Ind J Crit Med. 2017;68(149):134–68.
11. Emmanuel A, Kwame A, Peter Y, Jean-Pierre C. Abnormal Blood Glucose as a Prognostic Factor for Adverse Clinical Outcome in Children Admitted to the Paediatric Emergency Unit at Komfo Anokye Teaching Hospital, Kumasi, Ghana. Int J Paediatr. 2014; 2014:1–6.
12. Sambany E, Pussard E, Rajaonarivo C, Raobijaona H, Barennes H. Childhood Dysglycemia: Prevalence and Outcome in a Referral Hospital. PLoS One. 2013; 8(5): e65193. Doi: 10.1371/journal.pone.0065193.
13. Uleanya ND, Aniwada EC, Nwokoye IC. Relationship between Glycemic Levels and Treatment Outcome among Critically Ill Children admitted into Emergency Room in Enugu. BMC Paediatr. 2017; 17:1–7.

14. Cochrane WG. Sampling technique. 3rd ed. New York: John Walley and Sons. 1977. Chapter 4, The estimation of sample size; pp. 72 -114.
15. Choukem SP, Sih C, Nebongo D, Tientcheu P, Kengne AP. Accuracy and precision of four main glucometers used in a Sub-Saharan African Country: a cross-sectional study. Pan Afr Med J. 2019; 32:118. doi: 10.11604/pamj.2019.32.118.15553.
16. Hopkins R, Warburton V. UK Metabolic Biochemistry Network Recommendation for the Investigation of Hypoglycaemia in Infants and Children [Internet]. Liverpool: National Metabolic Biochemistry Network; March 2025 [cited 2025 Aug 20]. Available from: https://metbio.net/wp-content/uploads/MetBioNet-Guideline-Hypoglycaemia-2025-Final.pdf
17. Sperling MA. Hypoglycemia. In: Behrman RE, Klignman RJH, editors. Nelson Textbook of Paediatrics. 18th ed. Philadelphia: Elsevier; 2004.p. 504–518.
18. IDF/ISPAD. Global guideline for Diabetes in Childhood and Adolescence. Int Diabetes Fed. 2014.
19. Oluwayemi IO, Ogundare EO, Ajite AB, Raimi TH. Profile of Random Blood Glucose of Children Seen at the Children's Outpatient Department of Ekiti State University Teaching Hospital, Ado-Ekiti, Nigeria. Glob J Endocrinol Metab. 2018;1(4):1–5.
20. Musa A, Ilah BG, Sakajiki AM, Adeniji AO, Yusuf I. Prevalence and outcome of hypoglycemia in children attending the emergency pediatric unit of a specialist hospital in Nigeria. Sahel Med J. 2019; 22(2):77–81.
21. Soliman AA, Abdelaal SHH, Mohamad IL. Prevalence and Course of Stress Hyperglycemia in Critically Ill Children Admitted to Pediatric Intensive Care Unit of Assiut University Children's Hospital. J Curr Med Res and Pract. 2025;10(3):145-152
22. Kim JY, Shin JS, Kim HS, Lee SJ. Prevalence and Clinical Features of Ketotic Hypoglycemia in Childhood. Ann Pediatr Endocrinol Metab. 2004; 9(2):192-8.
23. Monde AA, Djessou SP, Camara CM, Tiahou GG, Koffi G, Djohan F, et al. Prevalence of ketotic hypoglycaemia among schoolchildren in the village of Ahoué in Côte-d'Ivoire. Bull Soc Pathol Exot. 2010;103(2):96-9. doi: 10.1007/s13149-010-0040-5.
24. Hoffman RP. Pediatric Hypoglycemia [Internet]. Newark: Medscape; [updated 2024 Apr 03; cited 2025 Aug 24]. Available from: https://emedicine.medscape.com/article/921936-overview?form=fpf
25. Kaplowitz P, Sekizkardes H. Clinical and laboratory characteristics and follow-up of 62 cases of ketotic hypoglycemia: a retrospective study. Int J Pediatr Endocrinol. 2019; 2019:3. doi: 10.1186/s13633-019-0066-9.
26. Sihan L, Xinchen Q, Jian L, Weida L, Chao Z, Zhen-Ning Z, et al. Suppression of CRTC2-mediated hepatic gluconeogenesis by TRAF6 contributes to hypoglycemia in septic shock. Cell Discov. 2016; 2:16046. Doi: 10.1038/celldisc 2016.46.
27. Moreira APB, Alfenas ACG. The influence of endotoxemia on the molecular mechanisms of insulin resistance. Nutr Hosp. 2012;27(2):382-90. doi:10.1590/S0212-16112012000200007.
28. Miller SI, Wallace RJ Jr, Musher DM, Septimus EJ, Kohi S, Baughn RE. Hypoglycemia as a Manifestation of Sepsis. Am J Med. 1980; 68:649–54.
29. Wang T, Gong B, Shi K, Hu G, Sun L, Xu Y. Hyperglycemia in sepsis. Clinica Chimica Acta 2026; 579:120702. Doi: 10.1016/j.cca.2025.120702
30. Onyearugha CN, George IO. Incidence of Hypoglycaemia in a Paediatric Emergency Ward in Nigeria. BJMMR. 2015; 10(7):1–8.
31. Onyiruka AN, Peters OO, Awaeba PO. Hypoglycaemia at point of hospital admission of children below five years of age with falciparum malaria: prevalence and risk. Niger J Paed. 2013; 40(3):238–42