Original Article

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Paediatrics Section

Association between Corrected QT Interval, QT Dispersion and Clinico-biochemical Severity of Diabetic Ketoacidosis in Children Aged 1-12 Years: A Prospective Cohort Study

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ABSTRACT

Introduction: Diabetic Ketoacidosis (DKA) is a serious complication in children with type 1 diabetes, characterised by hyperglycaemia, metabolic acidosis and ketosis. It often leads to cardiac arrhythmias due to electrolyte imbalances and prolonged corrected QT interval (QTc).

Aim: To investigate the frequency of QTc prolongation and QT dispersion (QTd) in paediatric DKA patients and their relationship with serum electrolytes and pH.

Materials and Methods: A prospective cohort study was conducted over a one-year period (August 2022 to August 2023) in the paediatric Intensive Care Unit (ICU) of the Institute of Child Health and Hospital for Children, Egmore, Chennai, Tamil Nadu, India. The study included 150 children aged 1 to 12 years diagnosed with DKA. Electrocardiograms (ECGs) were recorded both at presentation and after recovery from DKA, with QTc and QTd evaluated using a 12-lead ECG. The Mann-Whitney U test was employed to compare medians and Interquartile Ranges (IQR) between two groups, while Spearman's correlation coefficient was used to assess relationships between variables, with results illustrated through a scatter plot. A p-value <0.05 was considered statistically significant.

Results: Significant correlations were found between prolonged QTc intervals and severe acidosis, with affected patients exhibiting lower bicarbonate levels and higher anion gaps and blood ketone levels. After treatment, both QTc intervals and QTd significantly decreased, indicating improved cardiac repolarisation. Strong positive correlations were noted between QTc intervals and both the anion gap and blood ketone levels.

Conclusion: The study highlights the QTc prolongation and QTd in children with DKA, which are closely linked to metabolic derangements. Routine ECG monitoring is essential for the early detection of QT prolongation, aiding in the prevention of life-threatening arrhythmias and improving patient outcomes.

Keywords: Cardiac arrhythmias, Electrocardiogram, QTc prolongation

INTRODUCTION

DKA is an acute medical emergency that occurs in children with both type 1 and type 2 diabetes. DKA affects 20-40% of newly diagnosed type 1 diabetics [1]. It is a catabolic state characterised by a relative or absolute insulin deficiency, resulting in hyperglycaemia and the accumulation of ketoacids in the blood, which leads to metabolic acidosis. The characteristics of DKA include hyperglycaemia (blood glucose >200 mg/dL), venous pH <7.3 or serum bicarbonate <18 mmol/L, and ketonemia (blood ß-hydroxybutyrate ≥3 mmol/L) or moderate to large ketonuria. The Clinical Practice Guidelines of the International Society of Paediatric and Adolescent Diabetes (ISPAD) provide a comprehensive set of recommendations for the diagnosis and management of diabetes in children, adolescents and young adults [2].

The three main pathophysiological pathways of DKA include insulin deficiency, elevated levels of cortisol, glucagon, growth hormone and catecholamines, as well as increased peripheral insulin resistance, which cause hyperglycaemia, dehydration, ketosis and electrolyte imbalances. The absence of insulin is the trigger for acidosis [2]. Additionally, accelerated glycogenolysis, reduced glucose uptake, and absolute insulin deficiency all contribute to the development of hyperglycaemia in a child with DKA. Increased lipolysis and decreased lipogenesis lead to the conversion of excess free fatty acids into ketones such as acetoacetate, acetone and beta-hydroxybutyrate [3].

The usual causes of complications and mortality in DKA include cerebral oedema (incidence: 0.5-0.9%; mortality: 20%), hyperkalemia, pneumonia and arrhythmias [4]. Cardiac arrhythmias and cardiac arrest in DKA are common complications caused by electrolyte imbalances, such as hyperkalemia or hypomagnesemia [3,5]. Another cause of arrhythmia, QT interval lengthening (the duration between the beginning of the Q wave and the end of the T wave), is frequently overlooked and is not necessarily caused by an electrolyte imbalance. The QT interval, which begins with ventricular systole and progresses from ventricular isovolumetric contraction to isovolumetric relaxation, measures the time required for ventricular depolarisation and repolarisation. A small number of children on a ketogenic diet have also been found to have an extended QTc [5,6]. The QTc estimates the QT interval at a standard heart rate of 60 bpm, facilitating the comparison of QT values over time at different heart rates, in addition to improving the detection rate of patients at risk of arrhythmias [7]. Ventricular arrhythmias, including bigeminy and trigeminy, have been documented, and numerous reports highlight cases of individuals with diabetes experiencing sudden death during sleep, potentially due to malignant arrhythmias [8].

The ketosis or acidosis present in a child with DKA directly affects cardiac repolarisation, leading to QTc prolongation, arrhythmias and subsequent cardiac arrest. An ECG performed during cardiac monitoring displays the QT interval, measured from the onset of the QRS complex to the end of the T wave, representing the duration of 'electrical' ventricular contraction. The QT interval is highly influenced

by heart rate [9]. The QTc interval, observed using digital ECGs commonly employed in emergency and ICU settings, serves as a simple and cost-effective predictor of all-cause cardiovascular mortality. While electrolyte imbalance is a primary cause of cardiac arrest in children with DKA, QTc prolongation has also been noted in children on ketogenic diets and in other ketosis-related conditions that are not associated with electrolyte disturbances [10].

This study aimed to evaluate the QTc and QTd in children with DKA. It also sought to reassess the prolonged QTc following the resolution of DKA and to correlate QTc and QTd with the clinical and biochemical severity of the condition.

MATERIALS AND METHODS

This prospective cohort study was conducted at Institute of Child Health and Hospital for Children, Egmore, Chennai, Tamil Nadu, India, over one year from August 2022 to August 2023, after obtaining approval from the Institutional Ethics Committee (IEC) at Madras Medical College (IEC Number: 11092021). Enrollment of children was conducted following the acquisition of informed consent from their parents or guardians.

Inclusion criteria: The study involved 150 children aged 1 to 12 years diagnosed with DKA according to the ISPAD 2022 criteria in the paediatric ICU at the Institute of Child Health and Hospital for Children in Chennai.

Exclusion criteria: Participants on medications associated with QT prolongation (such as azithromycin, chloroquine, sotalol, or tricyclic antidepressants), had congenital or rheumatic heart disease, left ventricular systolic dysfunction, acquired heart disease, unreliable identification of the T wave end on ECG, or if their parents did not provide consent for participation were excluded from the study.

Study Procedure

The clinical severity was evaluated using the Glasgow Coma Scale (GCS) score and vital signs, along with monitoring for any signs of complications related to DKA. The biochemical severity was determined by measuring blood haemoglobin levels, serum ketones, Arterial Blood Gas (ABG) parameters (pH, anion gap and bicarbonate), renal and liver function tests and serum electrolytes {sodium (Na), potassium (K), calcium (Ca), and magnesium (Mg)}.

Patients were treated according to the established DKA protocol, which involves initial evaluations of ABGs and serum electrolytes, specifically assessing blood ketones, Na, K, Ca, Mg and phosphorus. The anion gap was determined using the formula: Anion Gap=Na-(Cl+HCO_3) [11]. A 12-lead ECG was conducted upon admission and evaluated by a single paediatric cardiologist to maintain assessment consistency. QTc was determined using Bazett's formula [12], with the RR interval assessed in relation to the preceding QRS complex. A 12-lead ECG was recorded at a speed of 25 mm/s, along with a lead II rhythm strip at 50 mm/s. QT and RR intervals were measured.

The QT interval is measured from the beginning of the QRS complex to the end of the T wave. The end of the QT interval is determined as the point where a tangent drawn along the steepest downslope of the dominant repolarisation wave intersects the isoelectric line. Lead II was utilised for QTc measurements. A QTc of 0.44 seconds (440 milliseconds) or greater was considered prolonged.

QTd refers to the variability of the QT interval among the various leads in a standard 12-lead ECG. This method is believed to provide an indirect evaluation of the heterogeneity of myocardial repolarisation, a critical element in the onset of arrhythmias [13]. ECGs were obtained at the time of DKA presentation in the emergency room and subsequently after recovery from DKA, following the correction of acidosis. The data were then analysed accordingly.

STATISTICAL ANALYSIS

Statistical analysis was performed using IBM Statistical Package for the Social Sciences (SPSS) version 29.0. Descriptive statistics were presented in tabular form, with means and standard deviations reported for quantitative variables, and frequencies and proportions for categorical variables. The Shapiro-Wilk test was used to assess the normality of the distribution for each variable. For quantitative variables that were not normally distributed, medians and IQR were compared using the Mann-Whitney U test. Spearman's correlation coefficient was used to evaluate relationships between variables, with findings visualised in a scatter plot. A p-value <0.05 was considered statistically significant.

RESULTS

The mean age of the children in the study was 6.55±3.33 years and the mean duration of illness was 2.1±0.57 days. The mean GCS score was 12.49±1.48. Out of the 150 children enrolled, 125 had QTc prolongation.

The laboratory results revealed significant metabolic acidosis, as evidenced by low mean pH and bicarbonate levels, along with an elevated anion gap, indicating a severe accumulation of ketoacids. Electrolyte analysis showed hyponatremia and normal potassium levels, suggesting potential dysregulation in fluid balance. Additionally, serum calcium and renal function parameters reflected stress on the kidneys, with elevated urea and creatinine levels. The mean QTc interval was prolonged, and the notable QTd suggested an increased risk of cardiac arrhythmias, highlighting the critical need for monitoring and managing cardiac function in children with DKA [Table/Fig-1].

Descriptives	Mean	SD	Reference range
Age (yrs)	6.55	3.33	
pH	7.07	0.15	7.35-7.45
Bicarbonate (mEq/dL)	7.15	5.13	22-26
Anion gap (mEq/L)	26.09	7.85	12-16
QTC at presentation (ms)	453.36	15.7	<440
QT dispersion at presentation (ms)	9.41	1.907	10-71
Duration of illness (days)	2.1	0.57	
GCS	12.49	1.48	13-15
Blood ketone (mEq/dL)	27.34	7.53	<6
S. Calcium (mmol/L)	9.44	1.41	7.4-10.5
S. Ionised calcium (mmol/L)	0.97	0.28	1.2-1.38
Haemoglobin (gm/dL)	10.98	2.25	>11
WBC (10^3 cells/mm^3)	15.07	2.85	4-11
S. Sodium (mEq/dL)	129.9	5.71	135-143
S. Potassium (mEq/dL)	4.21	0.98	3.9-4.9
Serum urea (md/dL)	48.1	11.81	11-40
Serum creatinine (mg/dL)	0.82	0.25	0.3-1
SGOT (IU)	57.39	25.74	3-35

[Table/Fig-1]: Patient characteristics and laboratory results.

The study showed that 25 children had normal QTc, while 125 children had prolonged QTc. Patients exhibiting prolonged QTc demonstrated a significantly reduced pH (median 7.04) compared to those with normal QTc (median 7.24), suggesting a more severe acidotic condition (p-value <0.001). Bicarbonate levels were significantly lower in the prolonged QTc group (median 4.53 mEq/dL) compared to the normal QTc group (median 16.01 mEq/dL, p-value <0.001), indicating a greater degree of acidosis. The anion gap was notably elevated in patients with prolonged QTc (median 29.38 mEq/dL) relative to those with normal QTc (median 22.01 mEq/dL, p-value <0.001), aligning with the occurrence

of increased anion gap metabolic acidosis. Blood ketone levels were elevated in the prolonged QTc group (median 30.36 mEq/dL) compared to the normal QTc group (median 19.61 mEq/dL, p-value <0.001), suggesting a more significant state of ketosis. Serum calcium levels were significantly reduced in the prolonged QTc group (median 9.23 mg/dL) compared to the normal QTc group (median 10.21 mg/dL, p-value=0.004). There were no statistically significant differences between the two groups in other parameters, including age, duration of illness, GCS score, Ionised calcium, haemoglobin, WBC count, sodium, potassium, urea, creatinine, and Serum Glutamic Oxaloacetic Transaminase (SGOT) [Table/Fig-2].

Variables	Normal QTc (n=25)		Prolonged QTc (n=125)		U	p-value
	Median	IQR	Median	IQR		
Age (years)	6.94	6.5	6.78	6.13	1446	0.557
pH	7.24	0.05	7.04	0.49	2947	<0.001
Bicarbonate (mEq/dL)	16.01	2.39	4.53	4.69	2997	<0.001
Anion gap (mEq/dL)	22.01	9.7	29.38	13.58	863	<0.001
Duration of illness (days)	2.01	1.07	2.15	1.01	1372.5	0.338
GCS	13	2.7	12.3	2.6	1806	0.219
Blood ketone (mEq/dL)	19.61	8.9	30.36	10	279	<0.001
S. Calcium (mg/dL)	10.21	1.53	9.23	4.98	2126	0.004
S. Ionised calcium (mmol/L)	0.94	0.52	0.95	0.47	1446.5	0.559
Haemoglobin (gm/dL)	11.48	4.94	10.86	3.26	1580	0.938
WBC (10^3 cells/mm^3)	14.28	2.72	15.39	4.84	1403	0.421
S. Sodium (mEq/dL)	128.52	17.87	129.3	9.97	1491	0.718
S. Potassium (mEq/dL)	4.53	1.45	4.14	1.46	1800	0.231
Serum urea (md/dL)	39.1	21.98	47.77	21.12	1362.5	0.313
Serum creatinine (mg/dL)	0.76	0.42	0.81	0.43	1605.5	0.828
SGOT (IU)	58.06	39.87	59.37	51.21	1586	0.906

[Table/Fig-2]: Comparing children with normal QTc and prolonged QTc (Mann-Whitney U Test).

The median QTc interval significantly decreased from 455.4 ms at presentation to 430.95 ms after treatment, with the IQR widening from 16.3 to 22.4 ms. The test statistic (W=778.5) and a p-value of <0.001 indicated that this reduction was statistically significant, suggesting that treatment effectively reduced QTc intervals.

Similarly, QTd also significantly decreased from a median of 9.4 ms at presentation to 7.65 ms after treatment, with the IQR slightly narrowing from 2.73 to 2.63 ms. The test statistic (W=1607) and a p-value of <0.001 confirmed that this reduction in QTd was statistically significant. These findings suggest that treatment led to a significant improvement in both QTc and QTd [Table/Fig-3].

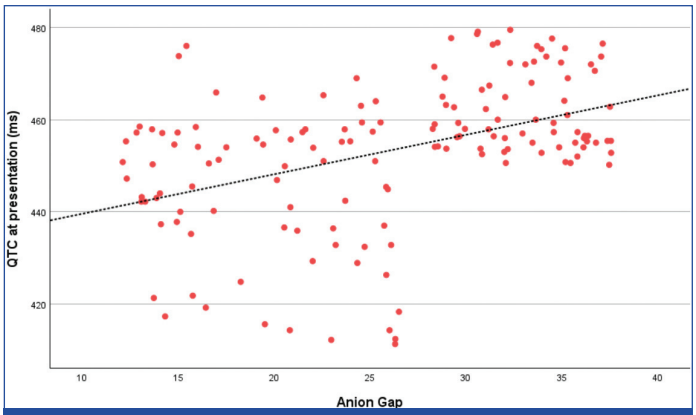
Interval	At presentation		After treatment		W	p-value
	Median	IQR	Median	IQR		
QTc (ms)	455.4	16.3	430.95	22.4	778.5	<0.001
QTd (ms)	9.4	2.73	7.65	2.63	1607	<0.001

[Table/Fig-3]: Pre and post intervention QTc and QTd (Wilcoxon single rank test).

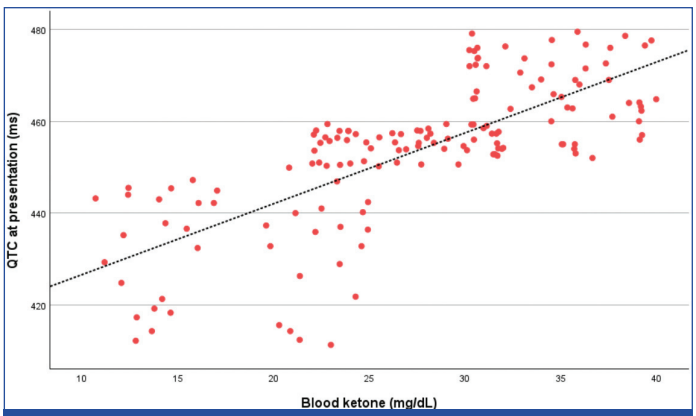
The Spearman correlation coefficient for the anion gap and QTc interval was 0.428 (p-value <0.001), indicating a moderate positive correlation between these variables. This suggests that as the anion gap increases, the QTc interval tends to increase as well. The Spearman correlation coefficient between blood ketone levels and the QTc interval was 0.755 (p-value <0.001), indicating a strong positive correlation. This suggests that higher blood ketone levels are strongly associated with longer QTc intervals. In contrast, serum bicarbonate showed a negative correlation with the QTc interval, with a Spearman coefficient of -0.697 (p-value <0.001) [Table/Fig-4-7].

Variables	QTc (ms)	p-value
Bicarbonate	-0.697	<0.001
Anion gap	0.428	<0.001
Blood ketone	0.755	<0.001
Sodium	0.090	0.272
Potassium	-0.250	0.758
Calium	-0.118	0.149
Magnesium	0.054	0.515
S. urea	0.105	0.200
S. creatinine	-0.040	0.624
SGOT	0.028	0.733

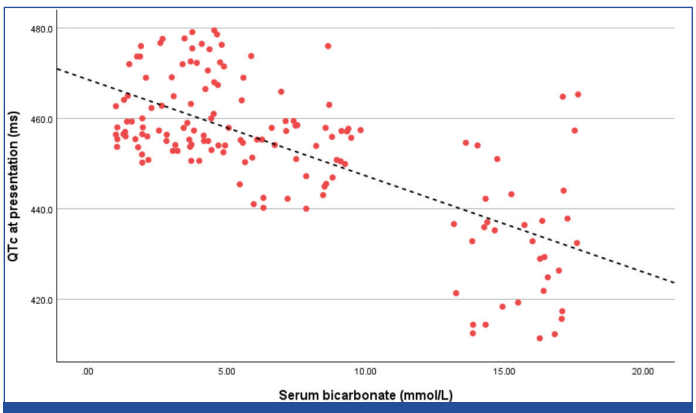
[Table/Fig-4]: Correlation between biochemical parameters with QTc at presentation (Spearman correlation).



[Table/Fig-5]: Correlation between anion gap with QTc at presentation.



[Table/Fig-6]: Correlation between blood ketone with QTc at presentation.



[Table/Fig-7]: Correlation between serum bicarbonate with QTc at presentation.

The correlations with serum sodium, potassium, calcium, magnesium, urea, creatinine, and liver enzymes were tested and found not to be statistically significant [Table/Fig-4].

The Spearman correlation coefficient for the anion gap and QTd interval was 0.205 (p-value=0.012), indicating a positive correlation between these variables. This suggests that as the anion gap increases, the QTd interval tends to increase as well. In contrast, serum bicarbonate showed a negative correlation with the QTd interval, with a Spearman coefficient of -0.259 (p-value <0.001) [Table/Fig-8].

Variables	QTd (ms)	p-value
Bicarbonate	-0.259	<0.001
Anion gap	0.205	0.012
Blood ketone	0.087	0.289
Sodium	0.078	0.346
Potassium	0.209	0.010
Calcium	0.090	0.272
Magnesium	0.158	0.054
S. Urea	-0.032	0.700
S. Creatinine	0.022	0.790
SGOT	-0.043	0.598

[Table/Fig-8]: Correlation between biochemical parameters with QTd at presentation (Spearman correlation).

DISCUSSION

Arrhythmias and cardiac arrest have been reported as complications of DKA, often attributed to electrolyte abnormalities such as hypokalemia, hypomagnesemia and hypocalcemia. In this study, out of 150 children, QTc prolongation was observed in 125 children during DKA. Since electrolyte imbalances and hypoglycaemia were not present in the studied cases, the observed prolongations in QTc and QTd suggest that ketoacidosis itself was a significant contributing factor to delayed cardiac repolarisation. This finding aligns with the research conducted by Perez MM et al., which reported a mean QTc interval of 470 ms in paediatric patients with DKA, indicating a notable increase compared to normal ranges. Their study emphasised that the metabolic disturbances associated with ketoacidosis, particularly acidosis and elevated ketone levels, can directly impact cardiac electrical activity, leading to a heightened risk of arrhythmias [13]. QTc prolongation is a critical condition that predisposes patients to life-threatening arrhythmias, such as torsades de pointes. A study has also linked other ketotic states, such as ketogenic diets, to QTc prolongation and sudden cardiac deaths, reinforcing the potential role of ketosis in these cardiac disturbances [5].

Notably, a few patients in this study with normal QTc intervals exhibited prolonged QTd, indicating that QTd may serve as a superior marker of cardiac risk in DKA. This finding aligns with the research by Youssef OI and Farid SM, who identified prolonged QTd as a significant predictor of cardiac mortality in diabetic patients [14]. They reported that a QTd greater than 60 ms correlated with an increased risk of adverse cardiac events, highlighting its relevance in assessing cardiac health. Unlike QTc, which measures overall repolarisation time, QTd reflects the heterogeneity of myocardial repolarization, thereby capturing arrhythmogenic potential. This underscores the importance of monitoring QTd in DKA patients to better identify those at heightened risk for cardiac complications. Notably, the anion gap was significantly higher at presentation in children with prolonged QTc and QTd intervals. A strong positive correlation was observed between QTc/QTd values and the initial anion gap, suggesting that ketosis plays a role in prolonging QTc and QTd intervals. This finding was consistent with prior research, including studies by Best TH et al., which reported QTc prolongation in children on ketogenic diets [5]. This study utilised the anion gap as an indicator of ketosis, but the lack of direct serum ketone measurements was a limitation. Prolonged QTc and a higher incidence of cardiac autonomic dysfunction abnormalities, often linked to diabetic neuropathy, have been reported in children with long-standing diabetes, even in the absence of DKA [15]. However, in this study, QTc prolongation was not attributed to pre-existing diabetic neuropathy, as QTc normalised after DKA resolution in most patients, including those with both new-onset and established diabetes. By the time of discharge, both QTc and QTd values had returned to the normal range. These findings emphasise that prolonged QTc is a common phenomenon during DKA and is strongly associated with ketosis, underscoring the importance of vigilant ECG and cardiac monitoring in children with DKA to reduce the risk of severe arrhythmias.

Limitation(s)

The small sample size of 150 patients and the study being conducted at a single centre may limit the generalisability of the results. Additionally, excluding patients on QT-prolonging medications and those with pre-existing conditions may have skewed the findings.

Further multicentric studies with a larger sample size and longer follow-up can be conducted in the future.

CONCLUSION(S)

This study emphasises the significant occurrence of QTc and QTd prolongation in paediatric patients with DKA, revealing a strong correlation between prolonged QT intervals and metabolic disturbances, particularly acidosis and elevated blood ketone concentrations. These findings underscore the necessity for careful cardiac monitoring of paediatric DKA cases due to the heightened risk of life-threatening arrhythmias during this metabolic crisis. Effective treatment of DKA normalises QTc and QTd, potentially reducing mortality risk. The study highlights the importance of routine ECG assessments, as early detection of QT prolongation is crucial for preventing serious cardiac complications.

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