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Original Research Article

Clinical and biochemical profile among children admitted with diabetic ketoacidosis and their correlation in prognosis of children admitted in pediatric intensive care unit

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ABSTRACT

Background: The present study aimed to analyse clinical and biochemical profile among children admitted with diabetic ketoacidosis (DKA) and their correlation in prognosis of children admitted in PICU.
Methods: The prospective study was conducted in pediatric intensive care unit (PICU) of RNT medical college, Udaipur among 50 DKA children in the age group of (1 month-18 years) for a period of one year after approval of institutional ethics committee. In the study, HbA1c level was measured by automated analyzer method to find out the past 3-month duration of glycemic control. Pearson correlation coefficient was used to find the correlation between serum osmolality and GCS, serum osmolality and duration of hospital stay, correlation between HbA1c levels and GCS, HbA1c levels and duration of hospital stay.
Result: The most common presenting complaint was respiratory distress (72%) followed by vomiting (54%), polyuria (34%), polydipsia (24%), pain in abdomen (24%), fever (24%) and polyphagia (4%). There was negative correlation between HbA1c and depressed sensorium, i.e. patients with higher HbA1c levels had poor GCS and it was statistically significant. Patients with higher HbA1c levels had longer duration of hospital stay, although this observation was not statistically significant.
Conclusions: The present study concluded that patients presenting with DKA had severe derangement in acid-base parameters. The DKA patients who had higher serum osmolality and poor glycemic control had depressed sensorium at the time of hospital admission and a longer recovery time leading to a prolonged hospital stay, adding to the morbidity associated with the disease.

Keywords: DKA, Diabetes mellitus, Biochemical profile, PICU

INTRODUCTION

Diabetes mellitus is a metabolic disease with several etiologies that is characterized by persistent hyperglycemia and abnormalities in the metabolism of fat, protein, and carbohydrates resulting from defects in insulin secretion, insulin action, or both.[1]

The major forms of diabetes are differentiated by insulin deficiency vs insulin resistance: type 1 diabetes mellitus (T1DM) results from deficiency of insulin secretion because of pancreatic β-cell damage; type 2 diabetes mellitus (T2DM) is a consequence of insulin resistance occurring at the level of skeletal muscle, liver, and adipose tissue, with various degrees of β-cell impairment. T1DM is the most common endocrine-metabolic disorder

of childhood and adolescence.[1] The criteria for the diagnosis of diabetes mellitus include classic symptoms of diabetes or hyperglycemic crisis with plasma glucose concentration ≥11.1 mmol/l (200 mg/dl). Or fasting plasma glucose ≥7.0 mmol/l (≥126 mg/dl). Two-hour post-load glucose ≥11.1 mmol/l (≥200 mg/dl) during an oral glucose tolerance test (OGTT). Or HbA1c ≥6.5%.[1] Glycosylated haemoglobin (HbA1c) is formed by the non-enzymatic glycation of haemoglobin and typically reflects the average blood glucose level during the life span of the red blood cell which is about 2-3 months. For the purpose of evaluating long-term glycaemic control in diabetes mellitus, it is now the gold standard.[2]

The incidence and prevalence of T1DM is suspected to be high in India, but in the absence of a nation-wide registry, it is not possible to be sure of the numbers. The Diabetes Atlas 2017 estimates that there are 128,500 children and adolescents with diabetes in India.[3] 10th edition of the international diabetes federation diabetes Atlas estimates that 1,211,900 children and adolescents younger than 20 years have T1DM globally. T1DM incidence varies around the world with some regions having much higher incidences than others. In almost all of the nations examined, the incidence has been rising in recent decades; however, there is evidence that in certain high-income countries, this increase may be tapering off or has stopped. Although the causes of this increase are unknown, a number of environmental factors have been suggested and are being looked at. Globally, 108,300 children and adolescents under the age of 15 received a diagnosis in 2021; when the age range is extended to include those under age of 20, number jumps to 149,500.[4]

According to the 6th edition of the international diabetes federation diabetes atlas, T1DM is on increase in India with a trend of 3-5% increase/year. India has three new instances of T1DM for every 100,000 children aged 0 to 14. There is variation in the prevalence of diabetes in India. Three sets of data indicate that Karnataka has 17.93 cases per 100,000 children, Chennai has 3.2 cases per 100,000 children, and Karnal has 10.2 cases per 100,000 children.[5]

DKA is an acute complication of T1 DM and is a sign of more advance disease. Clinical manifestations include nausea, vomiting, pain in abdomen, breathlessness, lethargy, altered mental status and in extreme cases, coma. Classical symptoms of polyuria, polydipsia and polyphagia may or may not be present.[6]

A shortage of circulating insulin and elevated levels of catecholamines, cortisol, growth hormone, and glucagon are the causes of DKA. When the concentrations of counter-regulatory hormones noticeably rise in response to stress, infection, or inadequate insulin, it is known as relative insulin insufficiency. When high levels of counter-regulatory hormones are combined with absolute or relative insulin shortage, the body enters an accelerated catabolic state, causing the liver and kidney to produce more glucose (via glycogenolysis and gluconeogenesis), and simultaneously impaired peripheral glucose utilization, which combine to result in hyperglycemia and hyperosmolarity; increased lipolysis and ketogenesis leading to ketonemia and metabolic acidosis. Hyperglycemia together with hyperketonemia cause osmotic diuresis, dehydration, and obligatory loss of electrolytes.[6] This leads to increase in serum osmolality. This increased serum osmolality has been associated with depressed mental function in children especially presenting in severe DKA.[7] The acidity is exacerbated by lactic acidosis resulting from hypoperfusion or infection. Thus, DKA triggers a dangerous chain of events that can be fatal, including hyperglycemia, hyperketonemia, osmotic diuresis, severe vomiting, dehydration, and the subsequent needless loss of electrolytes, increased production of stress hormones, and worsening insulin resistance. It would result in catastrophic hypoperfusion, dehydration, and eventually metabolic acidosis if it wasn't stopped by exogenous insulin, fluid, and electrolyte therapy.[6]

Most patients with DKA recover when diagnosed and treated promptly and if left untreated, patient may develop complications such as cerebral edema, thromboembolism, acute respiratory distress syndrome (ARDS), disseminated intra-vascular coagulation (DIC), electrolyte abnormalities (hypoglycemia, hypokalemia, non-anion gap hyperchloremic acidosis), rhabdomyolysis and acute circulatory failure.[8,9]

The cornerstone in managing a DKA patient is fluid therapy. Evidence regarding fluid type, volume, and rate of therapy is still evolving.[10] Early identification of ketoacidosis and aggressive management with insulin, intra venous fluid and electrolytes replacement may change the outcome of the disease.

Systematic reviews show that the risk factors for developing DKA are younger age (<2 years), low body mass index, ethnic minority background, misdiagnosis at first visit, delay in starting treatment, improper insulin administration at home and poor compliance to treatment in already diagnosed cases and history of recent infection. In addition, having immediate relatives with T1DM, parents with a higher education level or living in a region with a higher incidence of T1DM are factors that protect from presenting a DKA episode.[11]

What is already known?

The clinical and demographic profile of DKA has been researched in India with varying data emerging out of the studies. There have been a very few studies focusing on the metabolic derangements in children and their association with morbidity and mortality, and complications in DKA.

There is paucity of data on the biochemical profile and its corelation in prognosis of children admitted with DKA.

What this study will add?

Present study focuses on the clinical and biochemical profile and its correlation in prognosis in children admitted with DKA in PICU of Bal Chikitsalya, a tertiary care hospital in Southern Rajasthan.

Aim and objectives

Aims and objectives of the study are divided into five subparts. First, to study the clinical profile among children admitted with DKA. Second, to study the biochemical profile of children with DKA at the time of hospital admission. Third, to study the correlation between serum bicarbonate level and severity of disease and outcome in DKA patients. Fourth, to study the correlation of HbA1c level and severity of DKA and outcome in DKA patients. Fifth, to study the correlation of serum osmolality and sensorium of the patient and outcome in DKA patients.

METHODS

The present prospective study was conducted in PICU of RNT medical college, Udaipur among 50 DKA children in the age group of (1 month -18 years) admitted in PICU for a period of one year (i.e., July 2021 to December 2022) after approval of institutional ethics committee. Patients were enrolled in the study after taking proper informed consent of parents.

Inclusion criteria

All children (1 month-18 years) admitted with DKA and consenting to participate in the study were included-A diagnosis of DKA arterial pH <7.3, or serum. bicarbonate <15 mmol/l serum, glucose >200 mg/dl (11 mmol/l) together with the ketonemia, glucosuria and the ketonuria.[28]

Exclusion criteria

Non consenting individuals, no urine ketone bodies, normal ABG analysis, type 2 DM individuals and pre-existing comorbidity were excluded.

Study method

After an informed consent, all patients were thoroughly assessed at presentation, investigated and treated according to the protocol.

Their detailed clinical history, demographic profile and socio-economic status (Kuppuswamy scale 2020) were recorded. Address and contact number of patients were also be taken for further communication.[12]

General physical examination as well as complete systemic examination was done.

A diagnosis of DKA was made based on the clinical symptoms, random blood glucose, arterial/ venous blood gas, urine ketone bodies.

Biochemical investigations

The initial biochemical investigations such as random blood sugar, arterial/ venous blood gas, serum electrolytes (Sodium, potassium, and bicarbonate), blood urea, and serum creatinine was measured to confirm the diagnosis and to assess the severity of disease. Urine ketone bodies were identified by strip test. The test used in the urine test strips is based on the reaction of sodium nitroprusside (nitroferricyanide). In this reaction the acetoacetic acid in an alkali medium reacts with the sodium nitroprusside producing a magenta colour complex.

According to the age of presentation, the study group were divided into 4 groups: 1 month-2 years, >2-5 years, 6-10 years and >11 years.

In all the study patients, HbA1c level was measured by automated analyzer method to find out the past 3-month duration of glycemic control. In this study DKA patients were divided in to four groups based on the HbA1c level.

Group I-HbA1c level between 6.5-7.5%, group II-HbA1c level between 7.6-8.5%, group III-HbA1c level between 8.6-9.5% and group IV-HbA1c level more than 9.5%.

These four groups were compared to find out the role of long standing poor glycemic control for development of DKA. In these four groups the mean duration of hospital stay will be calculated separately to find the effect of HbA1c level in outcome of the DKA patients.

Serum osmolality

The serum osmolality is calculated from the following formula: 2×Na + blood sugar /18 + blood urea/5.6.

The normal range of serum osmolality is 285-295 mOsm/kg. Studies on serum osmolality and mental alteration have established a positive linear relationship between osmolality and mental alteration. In this study, patients of DKA were divided into three groups according to serum osmolality: Group A: Serum osmolality less than 295 mOsm/ kg, group B: Serum osmolality between 295-310 mOsm/kg and group C: Serum osmolality >310 mOsm/kg.

In each group, correlation between serum osmolality and duration of recovery to full consciousness and outcome were noted.

Serum bicarbonate level: Normal serum bicarbonate level is 21-28 mEq/l. On ABG, patients with acidosis will be divided into 3 groups: Group I=serum bicarbonate level

between 16-20 mEq/l, group II=serum bicarbonate level between 11-15 mEq/l and group III=serum bicarbonate level ≤10 mEq/l.

In these three groups, the duration of recovery of consciousness, duration of hospital stay was noted.

Data analysis

Data was compiled and cleaned in the Ms-excel and analysed using SPSS version 25.0. Summary statistics proportion, mean, median and standard deviation were calculated.

Appropriate statistical tests (Pearson correlation coefficient) were used to find the correlation between serum osmolality and GCS, serum osmolality and duration of hospital stay. Similar statistical test was used to find the correlation between HbA1c levels and GCS, HbA1c levels and duration of hospital stay, coefficient of correlation was calculated and $p<0.05$ was considered statistically significant.

Treatment protocol: All the patients were treated according to the standard DKA protocol.[1]

Time: IST hour: -20 ml/kg IV bolus 0.9% NaCl or Ringer lactate. Insulin drip at 0.05 to 0.10 units/kg/hr.

Comments: Quick volume expansion; may be repeated. NPO. Monitor I/O, neurologic status. Use flow sheet. Have mannitol at bedside; 1 g/kg IV push for cerebral edema.

Second hour until DKA resolution: 0.45% NaCl: plus potassium 20 mEq/l in 5% glucose if blood sugar<250 mg/dl, continue insulin drip

Comments: IV rate=85ml/kg+ maintenance-bolus/ 23 hours

If K <3 mEq/l, give 0.5-1.0 mEq/kg as oral K solution or increase IV K to 80 mEq/l.

Note that the initial IV bolus is considered part of the total fluid allowed in the 1st 24 hr and is subtracted before calculating the IV rate. Maintenance (24 hr) = 100 ml/kg (for the first 10 kg) + 50 ml/kg (for the second 10 kg) + 25 ml/kg (for all remaining kg). DKA, DKA; I/O, input and output (urine, emesis); K, potassium; KAc, potassium acetate; KPhos, potassium phosphate; LR, lactated Ringer solution; NaCl, sodium chloride; NPO, nothing by mouth.

RESULTS

Out of 50 subjects, maximum was from age group of >11 years (42%) followed by 6-10 years (34%). Minimum subjects were from the age group of 1 months-2 years (8%) followed by >2-5 years (16%). There were 56% males and 44% females in the study as shown in Table 1.

Table 1: Age distribution and gender distribution among the study subjects.

Age group (in years)	N	%	Gender	N	%
1 months	4	8	Male	28	56
>2-5	8	16	Female	22	44
6-10	17	34	Total	50	100
>11	21	42			
Total	50	100			

Table 2: Chief complaints distribution among the study subjects.

Chief complaints	N	%
Respiratory distress	36	72
Vomiting	27	54
Polyuria	17	34
Polydipsia	12	24
Pain in abdomen	12	24
Fever	12	24
Polyphagia	2	4

Table 2 shows the chief complaints distribution among the study subjects. Respiratory distress (72%), vomiting (54%), polyuria (34%), polydipsia (24%), pain in abdomen (24%), fever (24%) and polyphagia (4%) was reported in 50 subjects. Hence, most common complaint was respiratory distress followed by vomiting.

Table 3: Newly diagnosed TIDM cases presenting in DKA.

Newly diagnosed	N	%
No	27	54
Yes	23	46
Total	50	100

The 46% of the subjects in this study were newly diagnosed TIDM cases who presented in DKA Table 3.

Table 4: Descriptive analysis of ABG parameters and serum osmolality.

Variables	Min	Max	Mean	SD
pH	6.6	7.29	6.97	0.15
pCO_2	7.0	20.0	12.78	3.23
Serum bicarbonate	2	13	5.97	3.23
Serum osmolality	288.0	315.0	299.13	11.87

Mean pH, pCO_2, serum osmolality and bicarbonate level were found to be 6.97±0.15, 12.78±3.23, 299.13±11.87 and 5.97±3.23 respectively Table 4.

Severity of DKA according to bicarbonate levels, mild DKA in 2% cases, moderate DKA in 12% cases and severe DKA in 86% cases, Table 5.

Table 5: Severity of DKA among study subjects according to bicarbonate levels.

Severity	N	%
Mild (16-20 mEq/l)	1	2
Moderate (11-15 mEq/l)	6	12
Severe (<10 mEq/l)	43	86

Table 6: Duration of hospital stay among the study subjects.

Hospital stays (in days)	N	%
1-3	4	8
4-6	14	28
>6	32	64
Total	50	100
Mean	7.93	
SD	3.17	
Median	8	

Mean duration of hospital stay among the study subjects was 7.93±3.17 days as shown in Table 6.

Table 7: Complications and outcome observed among the study subjects.

Complications	N	%	Mortality	N	%
Cerebral oedema	4	8	Yes	7	14
Sepsis	2	4	No	43	86
ARDS	2	4	Total	50	100
DIC	1	2			

Complications viz. cerebral oedema, sepsis, ARDS and DIC was shown in 8%, 4%, 4% and 2% of the subjects respectively. Hence, cerebral oedema was most common complication followed by sepsis as well as ARDS and mortality among the study subjects was 14%, Table 7.

Table 8: Correlation between serum osmolality and GCS as well as duration of hospital stay.

Variables	R value	P value
GCS	-0.49	<0.01*
Duration of hospital stay	0.32	0.008*

*Statistically significant

According to Pearson correlation analysis, there was significant negative correlation between osmolality and GCS (r=-0.49, p<0.01) while significant positive correlation between osmolality and duration of hospital stays (r=0.32, p=0.008) as shown in Table 8.

Table 9: Correlation between HbA1c and GCS as well as duration of hospital stay.

Variables	R value	P value
GCS	-0.29	0.04*
Duration of hospital stay	0.11	0.47

*Statistically significant.

According to Pearson correlation analysis, there was significant negative correlation between HbA1c and GCS (r=-0.29, p=0.04) while positive correlation between HbA1c and duration of hospital stay (r=0.11, p=0.47), though statistically insignificant as shown in Table 9.

Table 10: Complications observed among the study subjects and its relation to severity of DKA.

Complications	N	%	Severity of DKA
Cerebral oedema	4	8	Severe
Sepsis	2	4	Severe
ARDS	2	4	Severe
DIC	1	2	Severe

All the complications viz. cerebral oedema (8%), sepsis (4%), ARDS (4%) and DIC (2%) were observed in subjects with severe DKA. Hence, complications were significantly associated with severe DKA Table 10.

DISCUSSION

The eventual result of the metabolic anomalies brought on by a significant insulin deficit or ineffectiveness of insulin is DKA. The latter occurs during stress as counter-regulatory hormones block insulin action. T1DM mostly presents as DKA which is a common pediatric endocrine emergency and is a state of absolute or relative insulin deficiency. About 25-40% of the newly diagnosed T1DM children present with DKA whereas the risk of developing DKA in established T1DM children is 1-8% per patient per year.[13]

The mortality rate associated with DKA among children in industrialized countries has declined to 0.15%-0.31%.[14,15] However, in less developed countries, the risk of death from DKA remains high at 3.4% to 13.4% and children may die before receiving adequate treatment or during treatment.[16,17] To reduce complications and mortality, DKA must be identified quickly and treated appropriately.

The present prospective study was conducted in PICU of M. B. govt. hospital, Udaipur among 50 DKA children in the age group of (1 month-18 years) admitted in PICU for a period of one year after approval of institutional ethics committee. The aim of the study was to analyse clinical and biochemical profile among children admitted with DKA and their correlation in prognosis of children admitted in PICU.

In this study, we evaluated 50 patients admitted in PICU over a period of 1 year, the majority of the patients were >11 years of age (42%) which was similar to a study conducted by Bhardwaj et al where the mean age of presentation was>11 years in >58% of the subjects.[18] Similar findings were also observed in study done by Barot et al where the mean age of presentation was 10.76±3.88 years.[19] However, a study done in North India by Prasad et al showed 60% of the subjects of age group

<10 years.[20] Similarly, in studies done by Ganesh et al and Kanwal et al mean age of presentation was 8.2 years and 7.4 years respectively.[21,22] The difference in age of presentation can be due to difference in demographic profile of that place.

In this study, males (56%) were affected more as compared to females (44%), this is in similarity with the studies done by Bhardwaj et al, Ganesh et al, and Prasad et al.[18,20,21] However, a difference was observed in studies conducted by Barot et al, Kanwal et al, Krishnan et al where a female preponderance was observed.[18,22,23] This might be due to difference in study area and design.

DKA is often difficult to diagnose because of its non-specific symptoms and signs like pain abdomen, vomiting and fast breathing and may be misdiagnosed as pneumonia, gastroenteritis or acute abdomen. It is even more challenging to diagnose DKA in newly diagnosed DM due to lack of awareness regarding signs and symptoms of T1DM among general population and primary care physician and this contributes to T1DM complicating as DKA.

In our study, the most common presenting complaint was respiratory distress (72%) followed by vomiting (54%), where as in studies conducted by Kanwal et al, Barot et al, Krishnan et al, Vasudev et al and Pozoa et al vomiting was the prime complaint in the patients.[19,23-25] However, according to Bhardwaj et al abdominal pain (62%) was the most common symptom noted followed by polyuria (58.6%), fast breathing (58.6%) and vomiting (55.1%).[18] In a study done by Ganesh et al polyuria (65%) was the most common presenting complaint followed by fever (33%), vomiting (23%) and pain in abdomen (23%).[21] These varying presentations of the disease highlight the importance of measuring blood sugar in every patient presenting to PICU with similar complaints and eliciting a detailed clinical history to correlate with the diagnosis.

In our study, already known case of TIDM were higher (54%) then the newly diagnosed ones (46%). These findings were similar to the study done by Barot et al where 59.25% cases were already known cases of TIDM.[19] A similar observation was also made by Bhardwaj et al, Kanwal et al and Krishnan et al the already known cases of TIDM in their studies were 51.8%, 43.6% and 35.3% respectively.[18,22,23] However, in studies done by Vasudev et al, Mohammed et al newly diagnosed TIDM cases formed the majority with 62% and 58.1% cases respectively. This difference can be due to the fact that many T1DM cases remain undiagnosed.[24,26]

In our study, 86% of the cases presented in severe acidosis with HCO_3 levels of <10 mEq/l, these findings were similar to the studies done by Bhardwaj et al, Ganesh et al and Krishnan et al where the cases with severe acidosis were 48%, 57.1% and 62% respectively.[18,21,23] These observations suggest that in developing countries like ours, resources are limited and timely referral to a health care facility from periphery is often delayed and the child lands up in more advanced disease state. A very similar observation was made by Musoma et al in their study done in Kenya which is also a low middle income country, the number of severe acidosis cases in their study were 41.1%.[27]

HbA1c represents a chronic hyperglycemic state and is indicator of glycemic control in already known cases of T1DM. Recently, it has been introduced as one of the criteria to diagnose T1DM. In our study, newly diagnosed cases who presented in severe acidosis also had higher HbA1c levels. This indicates, although, DKA is an acute complication but the subjects had chronic hyperglycemia which went undiagnosed because of lack of clinical symptoms and if present, being unrecognized and lack of screening of the susceptible population.

In our study, we correlated serum osmolality with depressed sensorium using GCS and, there was significant negative correlation ($r=-0.49$, $p<0.01$) between serum osmolality and depressed sensorium. This is in similarity with a retrospective study conducted in Tennessee by Nyenwe et al where they observed severe acidosis and higher osmolality, together, were responsible for severe depressed GCS at presentation.[7]

In our study, 9 (18%) cases had complications during the course of illness and hospital stay. Cerebral oedema (8%) was the most common complication in our study followed by sepsis (4%), ARDS (4%) and DIC (2%). Similar findings were observed in a study done by Bhardwaj et al where 20.9% of the cases developed cerebral edema.[18] Kanwal et al in their study, reported a similar finding with cerebral oedema cases (14.5%) forming the majority.[22] Tiwari et al reported a similar finding, in their study, 26% cases developed cerebral oedema.[28] The higher rates of cerebral oedema being observed are due to delayed recognition of the preexisting neurological signs before commencement of the treatment, also, it is often precipitated by the overzealous fluid administration thereby disturbing the cerebral hemodynamics.[29] The complication rate varies due to delay in seeking medical attention, poor referral chain in low middle income countries, delay in diagnosis, and further management at the referral centre.

In our study, the mean duration of hospital stay was 7.93±3.17 days. The median duration of hospital in our study was 8 days which is in similarity to study done by Musoma et al where the overall median duration of hospital stay for children was 8 days.[27] In a study done by Barot et al mean duration of hospital stay was 9.19 days, which is slightly longer than observed in our study.[19] The mean duration of hospital stay varies due to variable clinical condition and complication seen in the patients and time of referral of the patient to intensive care unit.

In our study, mortality among the study subject was reported to be 14%. The mortality rate among DKA in developed countries is from 0.15-0.31% and in developing countries it is around 13%.[14-17] Therefore, the mortality rate observed in our study is comparable to the mortality rate mentioned in the literature. Similarly, Kannwal et al reported 7 (12.7%) deaths in their study Poovazhagi et al in their study described that 11.8% of the patient's died, which is comparatively lower to that reported from other developing countries.[2,15,22] Musoma et al in their study revealed mortality rate of 6.9% which is lesser as compared to the present study.[27] Syed et al and Bhardwaj et al in their studies both reported 3.4% deaths which is lower as compared to the present study.[16,18] The difference observed is because of the difference in number of study subjects, easy access to health care facility in developed countries, timely referral to tertiary care centre. In our study, all seven non survivors had severe DKA and cerebral oedema was the most common complication leading to mortality. This is in similarity to a cohort of children admitted at a PICU in north India and another study in Chennai, India, where, cerebral edema was the most common cause of mortality.[2] Similarly, Kannwal et al noticed cerebral edema with or without renal failure and sepsis were the common cause of deaths in Indian children.[22] According to Bhardwaj et al mortality occurred due to cerebral edema with septic shock.[18] Barot et al in their study similarly revealed that likelihood of death was significantly higher among the patients who had cerebral edema.[19] The varied mortality rate depends on severity of DKA and associated complications observed in the patients.

CONCLUSION

In our study, the clinical presentation of DKA was varied with non-specific complaints which reflects on why the disease often gets misdiagnosed and there is a delay in seeking medical attention. Our study concluded that patients presenting with DKA had severe derangement in acid-base parameters like metabolic acidosis and reduced serum bicarbonate level. The DKA patients who had higher serum osmolality and poor glycemic control had depressed sensorium at the time of hospital admission and a longer recovery time leading to a prolonged hospital stay, adding to the morbidity associated with the disease. In our study, the DKA patients who did not survive had severe metabolic acidosis, higher serum osmolality, and poor glycemic control along with complications like cerebral edema, sepsis, ARDS, and DIC. Through this study, we determined the correlation between the altered biochemical profile and prognosis in DKA subjects, thereby emphasizing the need for early diagnosis and management of disease along with meticulous monitoring of biochemical parameters for early recognition of complications and their management. The limitations of study are small sample size, single centre study, lack of control group.

Recommendations

Public education about T1DM symptoms and screening of the susceptible population when they seek medical care for other concerns can help in early diagnosis and management. Assessment of RBS as 6th vital sign in all admitted patients with other disease etiologies can further strengthen the screening process. A 3-month assessment of HbA1c levels should be advised and reinforcement on the correct method of insulin administration, site of administration, dosage and storage should be done among the parents and affected children to ensure good glycemic control and prevention of long-term morbidity associated with the disease.

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