ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 6, Issue – 2, April - 2023, Page No. : 53 - 65

Association between Biochemical parameters and in-hospital outcome in Patients with Diabetic Keto Acidosis (DKA): A Cross Sectional study

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How to citation this article: Dr Aswathy A, Dr Alavi K P, Dr Ali Akbar, "Association between Biochemical parameters and in-hospital outcome in Patients with Diabetic Keto Acidosis (DKA): A Cross Sectional study", IJMACR- April - 2023, Volume – 6, Issue - 2, P. No. 53 – 65.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Aims and Objectives: To find the association between biochemical factors and in hospital outcome in patients admitted with DKA in a tertiary care hospital.

Materials and Methods: A hospital based cross-sectional study was conducted among the age group of 15 years and above, who admitted and diagnosed with DKA according to 2019 ADA criteria in MES medical college during the period of 1st January 2021 to 31st December 2021. Sample size required was 70. A detailed clinical history, physical examination and baseline investigations were done and entered in the predesigned proforma. Demographic and biochemical profile of all patients were recorded at the time of presentation to see whether there is any correlation between these and in hospital outcome of DKA.Data were entered into MS excel and analysis was done using SSPS free software®.

Results: A total 70 patients diagnosed with DKA were included in the study analysis.Out of the 70 participants in our study, 60 patients (85.7%) were recovered from DKAwhereas 10 patients (14.3%) were died. In our study, majority (45.71%) of the participants was found in the age group >50 years. The mean age ± SD for death in DKA 47.3 ± 14.2years and mean age ± SD for recovery was 47.1 ± 18years. 52.85% had normal serum corrected sodium levels and 70% had normal serum potassium levels at the time of admission. 51.42% had hypochloremia, 21.42% had hypomagnesemia and 30% had hypocalcemia. Mortality was high in those with serum bicarbonate level <10meq/L and less in those with normal serum corrected calcium levels.Other biochemical parameters were found to be having statistically insignificant association with the outcome.

Conclusion: Mortality in our study was 14.3%. Mortality was higher in patients with serum bicarbonate levels <10meq/L (p=0.002).Patients with normal levels of serum corrected calcium had higher recovery rate (p<0.01).

Keywords: Diabetes mellitus, T1DM, T2DM, Diabetic ketoacidosis

Introduction

One of the most prevalent health issues in the globe is diabetes. A series of metabolic illnesses known as diabetes mellitus (DM) are defined by hyperglycaemia brought on by deficiencies in insulin secretion, action, or both.

Type 1 and Type 2 diabetes are the two general categories for diabetes mellitus. Due to the autoimmune death of pancreatic beta cells, type 1 diabetes (T1DM) results in a complete or nearly complete lack of insulin. Rather than having a complete lack of insulin, type 2 diabetes (T2DM) is caused by insulin resistance. Insulin resistance is important in this situation. The hazardous signs of diabetes mellitus known as diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) represent the two ends of the uncontrolled diabetic condition spectrum.

One of the most prevalent hyperglycemic emergencies in people with diabetes mellitus is DKA, which has a high morbidity and fatality rate. A complete or relative absence of insulin and concurrent increase of counter-regulatory hormones result in DKA. [1] Cortisol, glucagon, growth hormone, and catecholamine levels rise, which promotes glycogenolysis and gluconeogenesis and causes hyperglycemia. Lack of insulin encourages lipolysis and ketogenesis. Metabolic acidosis is caused by the production of keto acids such beta-hydroxybutyric acid and acetoacetic acid. DKA can therefore result from prolonged hyperglycemia, the buildup of significant amounts of ketone bodies, and the ensuing metabolic acidosis. DKA is characterized by the triad of hyperglycemia, acidosis and ketonuria.

It primarily affects persons with type 1 diabetes mellitus, which is uncontrolled and caused by the body's immune system attacking the beta cells in the islets of Langerhans. However it can also happen in adolescents with newly diagnosed type 2 diabetes mellitus and in adults with poorly controlled type 2 diabetes. DKA can be triggered by stressful situations such illnesses that require medical treatment or surgery and physiological pressures. DKA may also be triggered by inadequate insulin dosage, insulin, or the absence of anti-diabetic medications. Urinary tract infections and gastroenteritis are the two most common infections that can cause DKA.[1]

Individuals with DKA will exhibit polyuria, thirst, weight loss, generalised fatigue, nausea, vomiting, blurred vision, and abdominal pain as symptoms. Dehydration, hypotension, peripheral cyanosis, tachycardia, air hunger (Kussmaul's breathing), hypothermia, confusion, lethargy, and coma are possible symptoms of the patient.

When given the right care, DKA patients typically make a full recovery. However, if complications like cerebral edoema, thromboembolism, acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), electrolyte abnormalities, myocardial infarction, infections, and acute circulatory failure are not addressed, patients may also experience these conditions.

A key factor in lowering disease mortality is early detection of DKA. Patients who get standardised care, which includes the injection of insulin, the management

of triggering variables, and the correction of electrolytes, experience reduced DKA-related death rates[2]. According to recent epidemiological studies, hospitalisations for DKA have risen during the previous 20 years. The higher incidence of T2DM3 may contribute to some of this increased frequency of admissions.[3]

Diabetes Mellitus

Diabetes is a metabolic illness with multiple aetiologies defined by protracted hyperglycemia and abnormalities in the metabolism of proteins, fats, and carbohydrates. Defective insulin secretion, insulin action, or both are to blame. Diabetes' persistent hyperglycemia is linked to long-term harm, dysfunction, and failure of many organs, particularly the heart, blood vessels, kidneys, eyes, nerves, and kidneys.. Retinopathy, nephropathy, neuropathy, Charcot joints, and autonomic dysfunction such postural hypotension, sexual dysfunction, etc. are the long-term sequelae of diabetes. DKA and HHS are the short-term consequences of diabetes mellitus. Both DKA and HHS are potentially fatal side effects of untreated diabetes. Diabetes patients are more likely to develop cerebrovascular, peripheral arterial, and cardiovascular disorders with atherosclerosis[4].

Prediabetes

Prediabetes is sometimes referred to as impaired glucose tolerance (IGT) and impaired fasting glucose (IFG). It is the region between normal blood sugar levels and diabetic levels, called the "grey area". Patients are at risk of both acquiring diabetes and its consequences when in this range. At this point, a change in lifestyle may bring everything back to normal. Fasting Plasma Glucose (FPG) values of 100 to 125 mg/dl, Two Hour Postprandial Plasma Glucose values of 140 to 199 mg/dl, and HbA1c values of 5.7 to 6.4% are used to define prediabetes.

The two main kinds of diabetes mellitus previously recognised were "Insulin Dependent Diabetes Mellitus" (IDDM) and "Non-Insulin Dependent Diabetes Mellitus" (NIDDM). The correct classification of the kind of diabetes mellitus into the aforementioned two divisions grew muddled when newer treatment guidelines appeared. It became challenging to categorise NIIDM who were receiving insulin treatment accurately. This classification was further complicated by the identification of additional forms of diabetes with distinct pathophysiology that did not fall into either of these two categories. New understandings of the mechanisms of diabetes mellitus and these challenges served as a primary impetus for the creation of a new classification scheme5. Type 1 diabetes mellitus (T1DM), Type 2 diabetes mellitus (T2DM), and certain types of diabetes caused by other causes, such as gestational diabetes mellitus, are included in the new categorization (GDM).[6]

Type 1 Diabetes Mellitus

a) Immune mediated form

Type I diabetes was once known as IDDM or juvenile diabetes. It is characterised by the complete lack of insulin caused by the loss of beta cells as a result of an autoimmune process in which antibodies are created against islet cells. Type 1 diabetes mellitus in this form is referred to as "immune mediated form." The condition typically manifests itself acutely. Also, it has been discovered that these patients frequently have have autoimmune diseases such Hashimoto's thyroiditis, Addison's disease, vitiligo, or pernicious anaemia. [5,6,7]. Anti-insulin receptor antibodies and "Stiff Man" syndrome are uncommon types of immune-mediated diabetes.

Idiopathic form

They are also included in this classification and are referred to as having type 1 diabetes mellitus' "idiopathic form," which is rare and typically affects people of African or Asian descent. [5,6,7].

b) Latent Autoimmune Diabetes in Adults (LADA)

It is a kind of autoimmune diabetes that progresses gradually. Autoantibodies are made against islet cells, just like the immune-mediated type. However the death of beta cells by the autoimmune system happens gradually.[8].

Type 2 diabetes mellitus

Type 2 diabetes or adult-onset diabetes mellitus was once known as NIDDM. Insulin resistance in peripheral tissue and a beta cell deficiency in insulin secretion are its defining features. Impaired glucose tolerance is eventually brought on by insulin resistance and hyperinsulinemia.. [5,6,7].

Other specific types

Monogenic diabetes syndrome, diseases of the exocrine pancreas (such as pancreatitis or cystic fibrosis), people with dysfunction associated with other endocrinopathies (e.g., acromegaly), and people with pancreatic dysfunction caused by drugs, chemicals, or infections (such as with the use of glucocorticoids, in the treatment of HIV/AIDS, or after organ transplantation) are included in this category.[5]

A. Genetic defects of beta cell function

Individuals with diabetes who have genetic abnormalities in beta-cell activity were previously known as having young-onset maturity-onset diabetes (MODY). It is a kind of diabetes mellitus that is not insulin-dependent and is typically discovered in young adults. Patients who are non-obese and have diabetes that was diagnosed when they were young (less than 30 years old) and who have a significant family history of the disease should be suspect. Patients with MODY have intact pancreatic beta-cell function three to five years following diagnosis, in contrast to those with type 1 diabetes. MODY 3 is the most prevalent variant[9].

B. Genetic defects in insulin action[10,11] like Type A insulin resistance syndrome , Leprechaunism (Donohue syndrome), Rabson-Mendenhall syndrome ,Lipoatrophic diabetes

C. Diseases of the exocrine pancreas[12] like Pancreatitis, Trauma/pancreatectomy, Neoplasia, Cystic fibrosis, Hemochromatosis, Fibrocalculous pancreatopathy and Others

D. Endocrinopathies[13] like Acromegaly, Hyperprolactinaemia, Hypercortisolism , Hyperthyroidism, Hypothyroidism , Cushing's syndrome, Glucagonoma, Pheochromocytoma, Primary aldosteronism and others

E. Drug or chemical induced[14] like Pentamidine, Nicotinic acid , Glucocorticoids, Thyroid hormone etc

F. Infections like Congenital rubella syndrome, Cytomegalovirus, Mumps, Coxasackie

G. Other genetic syndromes sometimes associated with diabetes[15] are Down's syndrome, Klinefelter's syndrome , Turner's syndrome , Freiderich's ataxia , Huntington's chorea ,Laurence-Moon-Biedl syndrome

One of the biggest concerns in healthcare today is the growing prevalence of type 2 diabetes. Type 2 diabetes impacted 462 million people worldwide in 2017, accounting for 6.28% of the population (4.4% of those aged 15 to 49, 15% of people aged 50 to 69, and 22% of people over 70). This corresponds to a prevalence rate of 6059 cases per 100,000 people.[16]. Diabetes is a potentially epidemic health problem that is quickly spreading throughout low- and middle-income nations like India. According to projections, India would have

69.9 million cases of diabetes by 2025, the great majority of which will go undiagnosed[17]. Diabetes was the tenth most common cause of death globally in 2019, accounting for an estimated 1.5 million of those fatalities. [18]

In India, the incidence of diabetes increased from 7.1% in 2009 to 8.9% in 2019. IGT is thought to affect 25.2 million persons currently, and 35.7 million adults are thought to have it by 2045. In the world's diabetes epidemic, India is in second place to China with 77 million diabetics. 12.1 million of them, or 27.5 million by the year 2045, are predicted to be older than 65. In India, the number of persons with diabetes is estimated to be close to 57%, or 43.9 million, undiagnosed[19]. In 2017, estimates for the incidence and prevalence of type 1 diabetes worldwide were 2,34,710 and 90,04,610, respectively. With 17% of the world's population, high-income countries were responsible for 49% of incident cases and 52% of prevalent cases worldwide. Asia had the highest occurrence (32%) and prevalent (31%) rates of type 1 diabetes, while having 60% of the world's population[20].

Over 30 million Americans have diabetes, with nearly 95% of those having T2DM. Prediabetes affects an additional 86 million people, greatly increasing their risk of acquiring T2DM. Youth are becoming more susceptible to it. The chance of acquiring T2DM is highly associated with advancing age. People over 65 have diabetes at a rate of more than one in four, and more than half have prediabetes. In the US, men (6.9%) are more likely than women (5.9%) to have type 2 diabetes. There is a lot of variation in T2DM prevalence around the world. Adults with diabetes are more prevalent in East Asia, South Asia, and Australia than any other region (153 million). The largest prevalence rate is in North America and the Caribbean, where one in eight people are affected[21].

DKA

In our clinical practise, emergency admissions resulting from acute metabolic crises such DKA, HHS, and hypoglycemia in patients with diabetes continue to rank among the most frequent and difficult situations. To help in the diagnosis of these disorders, a history, clinical examination, signs and symptoms, and biochemical tests are necessary. A potentially catastrophic metabolic side effect of untreated diabetes mellitus is called DKA. Hyperglycemia, ketonaemia, and large anion gap metabolic acidosis make up the traditional triad of DKA. As DKA and HHS are quite similar in many ways, it is crucial to distinguish between the two acute situations. Despite providing the foundation for both illnesses, hyperglycemia. Between the two, there are differences in the degree of hyperglycemia, which are more severe in HHS. DKA22 is characterised by ketoacidosis. DKA is defined by the 2019 ADA recommendations as Blood Glucose > 250 mg/dl, Ketonemia > 3.0 mmol/L or substantial ketonuria (greater than 2+ on standard urine dip stick), Metabolic acidosis - PH 7.30, and serum bicarbonate 15 mmol/L.

DKA is primarily associated with T1DM and may be the first symptom in up to 25% of cases. Due to the prevalence of insulin resistance in T2DM, ketoacidosis can happen even when there is no insulin deficiency. The pathogenesis of type 2 diabetes and an increase in the levels of counter-regulatory hormones such cortisol, glucagon, adrenaline, and growth hormone are two of the many mechanisms that mediate insulin resistance. DKA may arise from any physiological stress in a patient with type 1 diabetes because of high levels of hormones that regulate metabolism. Most recently,

SGLT2 inhibitor-treated T1DM and T2DM patients have been reported to have euglycemic diabetic ketoacidosis (EDKA). Hence, EDKA should be ruled out in a sick person with diabetes who is not hyperglycemic and is on SGLT2 inhibitors23,24. When gluconeogenesis is hindered due to alcoholism or liver disease, euglycaemic DKA is also seen in pregnant individuals. Hence, the severity of DKA is not determined by plasma glucose levels [25,26] .

Hyperglycemia

By boosting the supply of gluconeogenic precursors to the liver, activating the enzymes in the gluconeogenic pathway, and raising glucagon secretion, insulin shortage and/or resistance increase and expedite hepatic gluconeogenesis. Fatty acid oxidation produces the metabolic energy needed to power gluconeogenesis.

Ketone production

The production of non-esterified fatty acids (NEFA) and glycerol from the breakdown of triglycerides increases in DKA due to decreased effective insulin action and higher concentrations of counterregulatory hormones, particularly epinephrine, which activates hormone-sensitive lipase in adipose tissue. The substrate for gluconeogenesis is glycerol. The primary substrate for ketogenesis in the liver is assumed to be NEFA in this instance. Hepatocytes absorb the fatty acids once they have been delivered and bound to albumin. NEFA is subjected to oxidation, which causes the liver to produce ketone bodies. Glucagon is the main biological stimulator of this process. They are also utilised to make diacylglycerol, which can increase very low-density proteins and hyperlipidemia (VLDL). The rate-limiting enzyme in ketogenesis, carnitine acyltransferase, is stimulated by hyperglucagonemia, which raises hepatic carnitine concentrations and lowers hepatic malonyl CoA levels (CAT1). Due to low insulin concentrations, elevated glucocorticoids, and decreased peripheral glucose consumption, the elimination of ketone bodies is further compromised in DKA[27,28,29]. A small amount of insulin is present in HHS and will prevent ketosis, but the amount is insufficient to effectively regulate hyperglycemia. Even in a hyperosmolar state, there are less free fatty acids available for ketogenesis[30]. While South India has a sufficient amount of information on the causes of DKA and its clinical symptoms, information on the variables influencing in-hospital outcomes is lacking. So, the goal of this study is to identify biochemical variables that influence patients who have been admitted with DKA. The underlying biochemical markers and hospital outcome for DKA patients are thought to be unrelated.

Materials & Methods

Ethical clearance

Obtained from institutional ethical committee of MES Medical College, Kerala with IEC number – IEC/MES/13/2020

This cross sectional study was conducted in the department of Internal medicine at MES Medical College, Perinthalmanna. All patients diagnosed with DKA in the inpatient facility of MES Medical College during the period of 1/01/2021 to 31/12/2021, satisfying the inclusion criteria were considered as the study population through convenient sampling. As per 2019 ADA guidelines[6] DKA is defined as

•Blood Glucose > 250mg /dl

•Ketonemia > 3.0 mmol /L or significant ketonuria (more than 2+ on standard urine dip stick)

•Metabolic acidosis - PH < 7.30 & serum bicarbonate <15mmol /L

Patients will be further classified into three groups based on the severity of DKA

Electrolyte abnormalities

Serum sodium concentrations below 135meq/L are considered hyponatremia[31].

Serum salt levels greater than 145meq/L are considered hypernatremia[31].

Serum potassium concentrations below 3.5meq/L are considered hypokalemic[31].

Serum potassium concentrations over 5.5 meq/L are considered hyperkalemia[31.]

A blood calcium level of less than 8.2 mg/dl is considered hypocalcemia[32].

Lower than 1.6 mg/dl serum magnesium levels are considered hypomagnesemia[33].

Serum phosphate values of less than 2.5 mg/dl are considered hypophosphatemia[33].

Serum chloride values more than 105 mmol/L are considered hyperchloriemia[34].

Hypobicarbonatemia[6]

Mild: serum bicarbonate level 15-18 meq/L

Moderate: serum bicarbonate level10-15meq/L

Severe: serum bicarbonate level <10meq/L

Anemia[72] is defined as hemoglobin <13 g/dl

All patients diagnosed with DKA according to 2019 ADA criteria were included in the study and those Patients aged < 15 years, Pregnant women and Patients not giving consent to participate in the study were excluded. Sample size was scientifically estimatged according to a previous study[11], infection was the most common precipitating factor of DKA (22.5%) and this value is used to calculate sample size.

Sample size = $4pq / d^2p = 22.5$

$= 4 \times 22.5 \times 77.5 / 10^2q = 100-22.5 = 77.5$

$= 70d = 10$

Methodology

Between January 1 and December 31, 2021, all patients who met the inclusion criteria were included in the research. All participants gave their written consent after being fully informed. A thorough physical examination, clinical history, and baseline tests were completed and put into the predesigned proforma. All patients' demographic, clinical, and biochemical profiles were taken at the time of presentation in order to determine whether there was a relationship between them and the outcome of DKA in hospitals. GCS was used to document the patient's mental state at the time of admission. On the day of admission, information from regular tests was also gathered, including blood glucose level, ABG, urine ketones, serum electrolytes, RFT, LFT, HbA1c level, and CBC. ABG analysis was done in our laboratory using ABL 800, biochemical parameters using VITOS 5.1 FS machine, CBC using MINDRAY 6200 machine, and urine ketone body using FUS 1000 machine by strip method. Serum osmolality was calculated by using the formula 2× S. sodium + (plasma glucose ÷ 18) + (BUN ÷ 2.8) and serum anion gap by serum sodium - (serum chloride + bicarbonate). Based on plasma glucose (mg/dl), arterial pH, ketonemia/ketonuria, anion gap, and sensorium - patients with DKA were graded as mild, moderate and severe. All the individuals received the usual treatment for DKA, which included intravenous fluids, insulin, and electrolyte correction. The precipitating component was found and fixed. After providing routine care, patients were followed during their hospital stay to determine whether they would recover or die.

Data analysis

Data collected according to the pre-structured proforma, were entered in Microsoft excel and further analysis was done using SPSS (Statistical Package for Social Science) software version 20.0. Frequencies of DKA severity was found by using descriptive statistics. Categorical and quantitative variables were expressed as frequency (percentage) and mean ± SD respectively. Chi-square test and Fisher's exact test were used to find association between categorical variables. Mann-Whitney U Test was used to compare ordinal parameters between groups. For all statistical interpretations, $p<0.05$ was considered the threshold for statistical significance.

Results

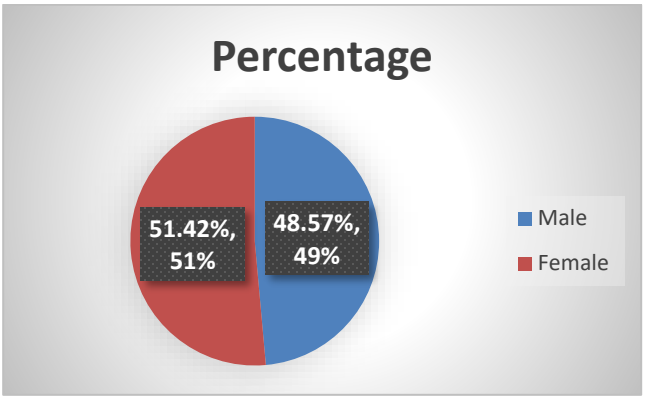
This is a hospital based cross sectional study conducted in MES medical college done during the period of January 2021 to December 2021. 70 DKA patients were selected for the study, among which 85.7% recovered from DKA whereas 14.3% died.

Table 1: Age distribution based on outcome.

Age	Death		Discharged	
(years)	Count	Percent	Count	Percent
<30	1	6.7	14	93.3
30 - 49	6	26.1	17	73.9
≥50	3	9.4	29	90.6
Mean ± SD	47.3 ± 14.2		47.1 ± 18.7	

The mean age of death was 47.3± 14.2 years whereas mean age of recovered patients was 47.1 ± 18.7 years.

Figure 1: DKA according to gender



In the study population, 48.57% were males and 51.42% were females.

Table 2: Electrolyte abnormalities

Electrolytes	Number	Percentage (%)
Serum corrected sodium		
Hyponatremia	7	10
Normal level	37	52.85
Hypernatremia	26	37.14
Serum potassium		
Hypokalemia	15	21.42
Normal level	49	70
hyperkalemia	6	8.57
Serum chloride		
Hypochloremia	36	51.42
Normal level	22	31.42
Hyperchloremia	12	17.14
Serum magnesium levels		
Hypomagnesemia	15	21.42
Normal level	55	78.57
Serum calcium levels		
Hypocalcemia	21	30
Normal level	48	68.57
Hypercalcemia	1	1.42

52.85% had normal serum corrected sodium levels and 70% had normal serum potassium levels at the time of admission. 51.42% had hypochloremia, 21.42% had hypomagnesemia and 30% had hypocalcemia.

Table 3: Comparison of biochemical parameters with the outcome

Biochemical Parameters	Death Number (%)	Discharged Number (%)	χ^2	P value
1)Bicarbonate				
<10	9(28.1)	23(71.9)		
≥ 10	1(2.6)	37(97.4)	9.22	**0.002**
2) Serum calcium				
Hypocalcemia	8(38.1)	13(61.9)		
Normal level	2(4.2)	46(95.8)	13.9	**<0.01**

Test Applied : Cho square test P value <0.05 statistically significant

Test Applied: Cho square test P value <0.05 statistically significant.

Mortality was high in those with serum bicarbonate level <10meq/L and less in those with normal serum corrected calcium levels. Other biochemical parameters were found to be having statistically insignificant association with the outcome.

Discussion

The most prevalent acute metabolic consequence of diabetes is DKA. Due to an insulin deficit and a corresponding rise in the counter-regulatory hormones, it is characterised by a triad of hyperglycemia, ketonemia/ketonuria, and metabolic acidosis. The analysis of the study included 70 participants with DKA in total. The outcome of DKA was compared with the demographic, clinical, and biochemical factors present at the time of admission. Death and DKA recovery were the study's two outcomes. 85.7% of the 70 trial subjects recovered from DKA, whereas 14.3% passed away. Sonwani et al. and the outcome of DKA were associated.[3]

Demographic parameters

Age

45.71% of the participants in our study were over the age of 50. The mean age with SD for death was 47.3 years and for recovery it was 47.1 years and with SD. This was related to the demographics of the Sonwani et al.[35] study, where 21% of the participants were aged 51 to 55. The mean age in another study by Sotiropoulos A et al.[36] was 64.9 14.6 years, but the mean age in a retrospective analysis by Tadesse Melaku Abegaz et al.[37] was 33.30 14.96 years.

Sex

The gender split of the 70 participants in our survey was 51.42% women and 48.57% men. Men recovered at a rate of 85.3% and women at a rate of 86.1%. Five of the total ten deaths among the participants (five) were split evenly between the sexes. No correlation between gender and the result of DKA was found in our investigation. Retrospective studies by Tadesse Melaku Abegaz et al.[37] and Raya Almazrouei et al.[38] both contained 54.5% and 74% female participants, respectively.

Serum bicarbonate levels

97.4% of the study's healed patients had bicarbonate levels more than 10 meq/L. When serum bicarbonate was below 10meq/L, 90% of patients passed away. Data demonstrates that those with extremely low serum bicarbonate levels had a higher mortality rate (P=0.002).

Severity of DKA according to pH

In our study, 12.86% of participants had severe DKA, 44.28% had mild DKA, and 42.86% had moderate DKA. 71.73 percent of patients had severe DKA, 22.1% had moderate DKA, and 6.07% had light DKA, according to Jiménez-Castillo et al. In contrast to the study stated above, there was a lower incidence of severe DKA in our study. Because most of our patients sought medical care as soon as possible.

Biochemical parameters

Leucocyte count and outcome were not found to be significantly correlated in our study. Leucocytosis is typically present in DKA, especially when infection or hemoconcentration are present[1,30]. In our analysis, infection was the most frequent precipitating reason (32%), while only 25.71% of patients had high leucocyte counts.

Serum bicarbonate, serum creatinine, serum albumin, and several electrolytes, including corrected sodium, potassium, magnesium, phosphate, chloride, and corrected calcium, were the biochemical parameters analysed in our study.

Out of 70 participants in our study, 70% had normal potassium levels, 21.42% had hypokalemia and 8.55% had hyperkalemia at the time of admission. 85.7% of those who had normal potassium levels recovered. we couldn't find any association between serum potassium levels with the outcome of DKA (p=0.981). According to Gavrielatos G et al[39], 10.7% had hypokalemia.

Of the 70 participants in our study, 37 had corrected sodium in the normal range, 7 had hyponatremia and 26 had hypernatremia. 94.6% of those who had normal corrected sodium recovered. 6 participants with hypernatremia died. In our study, 78.57% of the participants had normal serum magnesium levels, 21.42% had hypomagnesemia. In our study, 51.42% had hypochloridemia. 95.5% of those with normal serum chloride levels at the time admission recovered. Out of the total 10 deaths, 80% occurred in hypochloridemia group. Among the total study population, 68.57% had normal serum corrected calcium, 30% had hypocalcemia and 1.42% had hypercalcemia at the time of admission. 95.8% of those with normal serum corrected calcium recovered. 38.1% of those with hypocalcemia at the time of admission died. Our study did not show any major association between the various electrolyte disturbances and outcome, except for serum bicarbonate (p=0.002) and serum corrected calcium (p<0.01In our investigation, those with very low serum bicarbonate levels (10meq/L) died more frequently than those with normal adjusted calcium levels. In our investigation, there was no correlation between blood albumin and serum creatinine and the result. There is a risk of selection bias in this study due to the small study group and the fact that it was conducted in a single centre. In addition, the study individuals were not monitored for chronic complications of DM. Newly discovered cases might not be accurate since no prior blood test for the diagnosis of DM had been performed on them. Sample size with various DM types is a limiting factor because it significantly affects the course of the disease and its complications. Thus, we encourage further study using a large sample size.

Conclusion

Mortality in our study was 14.3%.Mortality was higher in patients with serum bicarbonate levels <10meq/L (p=0.002).Patients with normal levels of serum corrected calcium had higher recovery rate (p<0.01).

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