STUDY OF CLINICAL PROFILE IN DIABETIC KETOACIDOSIS AND ITS OUTCOME IN RELATION TO BICARBONATE LEVELS

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THANJAVUR MEDICAL COLLEGE AND HOSPITAL, THANJAVUR-613004

MAY 2023

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TITLE OF THE STUDY:

Study of clinical profile in diabetic ketoacidosis and its outcome in relation to bicarbonate levels

PRINCIPAL INVESTIGATOR:

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This to certify that the protocol submitted by the principal investigator of the above mentioned study has been reviewed as per standard ethical guidelines and the same has been **APPROVED** by the members of the Institutional ethical committee at its meeting held on **21.01.2021**.

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DECLARATION

I declare that this dissertation titled **"STUDY OF CLINICAL PROFILE IN DIABETIC KETOACIDOSIS AND ITS OUTCOME IN RELATION TO BICARBONATE LEVELS"** done at Thanjavur Medical College and hospital, Thanjavur represents a genuine work of mine. I, also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, neither in India nor abroad. This is submitted to the The Tamil Nadu Dr. MGR Medical University, Chennai in fulfillment of the rules and regulations for the award of **M.D. Degree (Branch -I) in General Medicine.**

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LIST OF ABBREVATIONS:

BP – BLOOD PRESSURE

DKA- DIABETIC KETOACIDOSIS

DM- DIABETES MELLLITUS

HbA1C- GLYCATED HEMOGLOBIN

NC- NON COMPLIANCE

OHA- ORAL HYPOGLYCAEMIC AGENTS

RBS – RANDOM BLOOD SUGAR

REC- RECOVERED

RR – RESPIRATORY RATE

SD- STANDARD DEVIATION

T1DM-TYPE 1 DIABETES MELLTUS

T2DM- TYPE 2 DIABETES MELLITUS

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1 ABSTRACT:

Introduction:

Diabetic Ketoacidosis (DKA) is a common complication of diabetes which comes to the emergency as the disease diabetes turns to be the common non communicable disease affecting the world. The understanding of the pathophysiology of the sickness, features, early diagnosis, and adequate treatment of DKA, and the triggering causes, can change how the disease affects the study population. This will help to reduce the disease's rates of morbidity and mortality.

Objectives:

- To evaluate age , sex distribution , common presentation and precipitating factor in diabetes ketoacidosis
- To study the incidence of DKA in poorly controlled diabetes by HbA1c level.
- To study the correlation between serum bicarbonate level and duration of hospital stay in DKA patients.
- To study the outcome of DKA during treatment

Methodology:

The study was a prospective study conducted among 46 subjects with an aim to assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects. The subjects with diabetic ketoacidosis were included in the study. Patient with hyperglycaemic hyperosmolar coma, chronic renal disease, with hyperemesis

gravidarum, with starvation ketosis and severe anaemia. After selecting the subjects the clinical profile including age, Sex, Total stay in Hospital, family history and treatment history was assessed. Clinical findings including level of consciousness, heart rate, respiratory rate, blood pressure, random blood sugar, renal function test, bicarbonate, plasma/urine acetone was assessed. The data was entered into Microsoft excel and analysed using SPSS 23.

Results:

In the study the mean Age (years) among the subjects was 47.87 (± 17.15) years ranging from 17 to 85 years. Among the subjects, 28 (60.87%) were Males and 18 (39.13%) were Females. In our study among the subjects, 33 (71.74%) had Type 2 and 13 (28.26%) had Type 1 Diabetes. In the study the mean Serum Bicarbonate (mEq/L) among the subjects was 13.84 (± 3.53) ranging from 7 to 21 mEq/L. The mean HbA1C (%) among the subjects was 8.98 (± 1.07) ranging from 6.7 to 11.4. Among the subjects, 10 (21.74%) had Vomiting, 9 (19.57%) had Fever And 5 (10.87%) had Altered Sensorium/Fever. Among the subjects, 32 (69.57%) had due to Non-Compliance, 6 (13.04%) had due to Infection and 5 (10.87%) had due to Inadequate dose. Among the subjects, 43 (93.48%) had Recovered and 3 (6.52%) had Death. Comparing the Treatment History with Outcome distribution, OHA + Insulin had significantly higher proportion of death with 28.57% followed by Insulin with 5.88% and least in OHA with 0%. The mean Random Blood Sugar (mg/dl) among subjects with Death was significantly higher 600 (± 0) vs. 450.35 (± 68.02). The mean Serum Bicarbonate (mEq/L) among subjects with Death outcome was significantly lower than recovered outcome subjects was 7.6 (± 0.53) vs.14.28 (± 3.22) .The mean HbA1C (%) among Death was 10.5 (± 1.31) which is higher by 1.63 and statistically significant compared to 8.87 (± 0.98) in Recovered. Serum Bicarbonate (mEq/L) has a significantly negative correlation

with Duration of Stay (days) with a correlation coefficient of -0.84. Duration of Stay (days) decreases by -0.65 times for each unit increase in Serum Bicarbonate (mEq/L).

Conclusion:

Diabetic Ketoacidosis with increased blood glucose value, decreased serum bicarbonate , and higher glycosylated haemoglobulin had death as an outcome. As the bicarbonate level decreases the duration of hospital stay increases.

Keywords:

Bicarbonate level, Clinical Profile, Diabetes Mellitus, Diabetic Ketoacidosis,

2 INTRODUCTION

Persistent hyperglycaemia and anomalies in the metabolism of meals including carbohydtrates, lipids, and proteins are characteristics of the metabolic disorder known as diabetes mellitus, which is brought on by deficiencies in insulin secretion, insulin action, or both. It is associated with widespread hormonal, metabolic, and microvascular abnormalities, as well as issues with the functionality of several organ systems. Type 2 diabetes mellitus is a group of illnesses characterised by variable degrees of insulin resistance, reduced insulin secretion, and increased glucose production. (1)The prevalence was reported to be 11.8% in adults over 50 years old in the Ministry of Health and Family Welfare's 2019 National Diabetes and Diabetic Retinopathy Survey report. (2,3)

One in 2000 people develops Diabetic Ketoacidosis (DKA), an acute consequence of diabetes that is a medical emergency. Although it can happen in type II diabetes, it often manifests in type I diabetes mellitus. DKA frequently shows signs of ketosis, ketoacidosis, pre-coma, and coma. In DKA patients, mean random blood glucose and pH significantly correlate with neurological state. However, the clinical appearance of the triggering illness frequently hides signs. (4,5)The blood pH, serum bicarbonate level, and serum osmolality all support the diagnosis. (6)

Lack of insulin, increased lipolysis and ketogenesis, the development of ketone bodies and free fatty acids, and insulin insufficiency are the major causes of DKA. Increased gluconeogenesis, lipolysis, ketogenesis, and reduced glycolysis are further traits of diabetic ketoacidosis. (7) Hyperglycaemia, metabolic acidosis, and a rise in ketone bodies in the blood and urine make up the diagnostic triad for DKA. (8,9)

Catabolic stress from acute illnesses or traumas like trauma, surgery, or infections may be a trigger in both Type 1 and type 2 populations. Non-compliance, newly diagnosed diabetes, and other acute illnesses are frequent causes of DKA. Pneumonia and urinary tract infections are the most typical forms of infections. DKA can also be triggered by other illnesses such alcoholism, trauma, pulmonary embolism, and myocardial infarction. DKA may be triggered by medications that alter carbohydrate metabolisms, including corticosteroids, thiazides, sympathomimetic drugs, and pentamidine. Antipsychotic medications, both traditional and atypical, have a small risk of causing hyperglycaemia and DKA.(10,11)

Insulin replacement can bring plasma glucagon levels back to normal in people with diabetes mellitus, which is characterised by insulin insufficiency and elevated levels. [12] Normally, as serum glucose levels rise, it penetrates pancreatic beta cells and triggers the synthesis of insulin. By blocking glycogenolysis and gluconeogenesis, insulin lowers hepatic glucose synthesis. Insulin increases the absorption of glucose by skeletal muscle and adipose tissue. The lowering of blood sugar is the outcome of both of these mechanisms. Increased gluconeogenesis, rapid glycogenolysis, and decreased glucose utilisation can result from insulin shortage and elevated counter-regulatory hormones in diabetic ketoacidosis. In the end, this will result in hyperglycaemia that gets worse. Lack of insulin and elevated levels of counterregulatory hormones also cause the release of free fatty acids from adipose tissue into the bloodstream (lipolysis). These fatty acids are then oxidised in the liver to form ketone bodies (beta-hydroxybutyrate and acetoacetate), which cause ketonemia and metabolic acidosis.(11,12)

Table 1: DKA Diagnosis and criteria*(13,14)*

DKA	Mild	Moderate	Severe
Plasma glucose (mg/dl)	>250 mg/dl	>250mg/dl	250mg/dl
Arterial pH	7.25-7.30	7.00-7.24	<7.00
Serum bicarbonate (mEq/L)	15-18	10- 15	<10
Urine ketone*	+	+	+
Serum ketone*	+	+	+
Effective Serum Osmolality**	Variable	Variable	Variable
Anion Gap***	>10	>12	>12
Mental Status	Alert	Alert/drowsy	Stupor/coma

Need for the study / Justification of the study:

The outcomes and findings of this study will be useful in understanding the illness pathophysiology, early diagnosis, and adequate treatment of DKA, avoiding the triggering causes, which in turn can influence how the disease affects the study population. This will aid in lowering the disease's morbidity and fatality rates.

3 AIM AND OBJECTIVES

3.1 **AIM:**

To assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects.

3.2 **OBJECTIVES:**

- To evaluate age , sex distribution , common presentation and precipitating factor in diabetes ketoacidosis
- To study the incidence of DKA in poorly controlled diabetes by HbA1c level.
- To study the correlation between serum bicarbonate level and duration of hospital stay in DKA patients.
- To study the outcome of DKA during treatment

4 REVIEW OF LITERATURE

Review of Literature of this study is discussed under the following heads:

a. Diabetes mellitus- pathophysiology

b. Complications of diabetes

c. DKA-pathology, clinical features, diagnosis, classification

d. Studies related to the topic

4.1 DIABETES MELLITUS- PATHOPHYSIOLOGY

Diabetes Mellitus(DM) is a chronic disease characterised by elevated blood glucose levels. This condition may be brought on by a reduction in insulin sensitivity (Type II) or Type I insulin synthesis in the cells. (15) The general mechanism is that hyperglycaemia develops as a result of inadequate glucose utilisation. (16)

Table 2: Classification of diabetes*(17)*

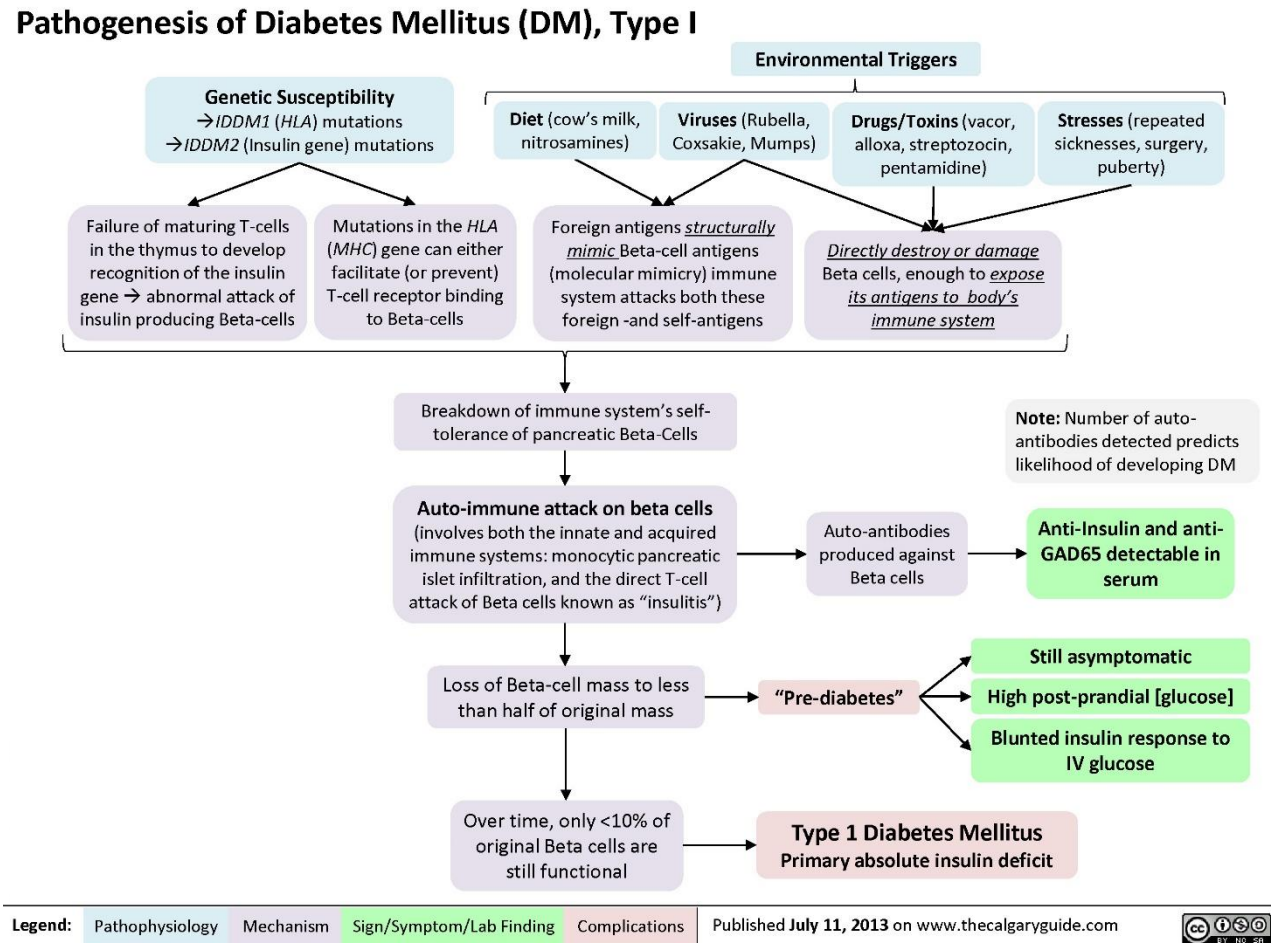
Type 1 diabetes
Type 2 diabetes
Hybrid forms of diabetes
Slowly evolving immune-mediated diabetes of adults
Ketosis prone type 2 diabetes
Other specific types (see Tables)
Monogenic diabetes
- Monogenic defects of β-cell function
- Monogenic defects in insulin action
Diseases of the exocrine pancreas
Endocrine disorders
Drug- or chemical-induced
Infections
Uncommon specific forms of immune-mediated diabetes
Other genetic syndromes sometimes associated with diabetes
Unclassified diabetes
This category should be used temporarily when there is not a clear diagnostic category especially close to the time of diagnosis of diabetes
Hyperglyacemia first detected during pregnancy
Diabetes mellitus in pregnancy
Gestational diabetes mellitus

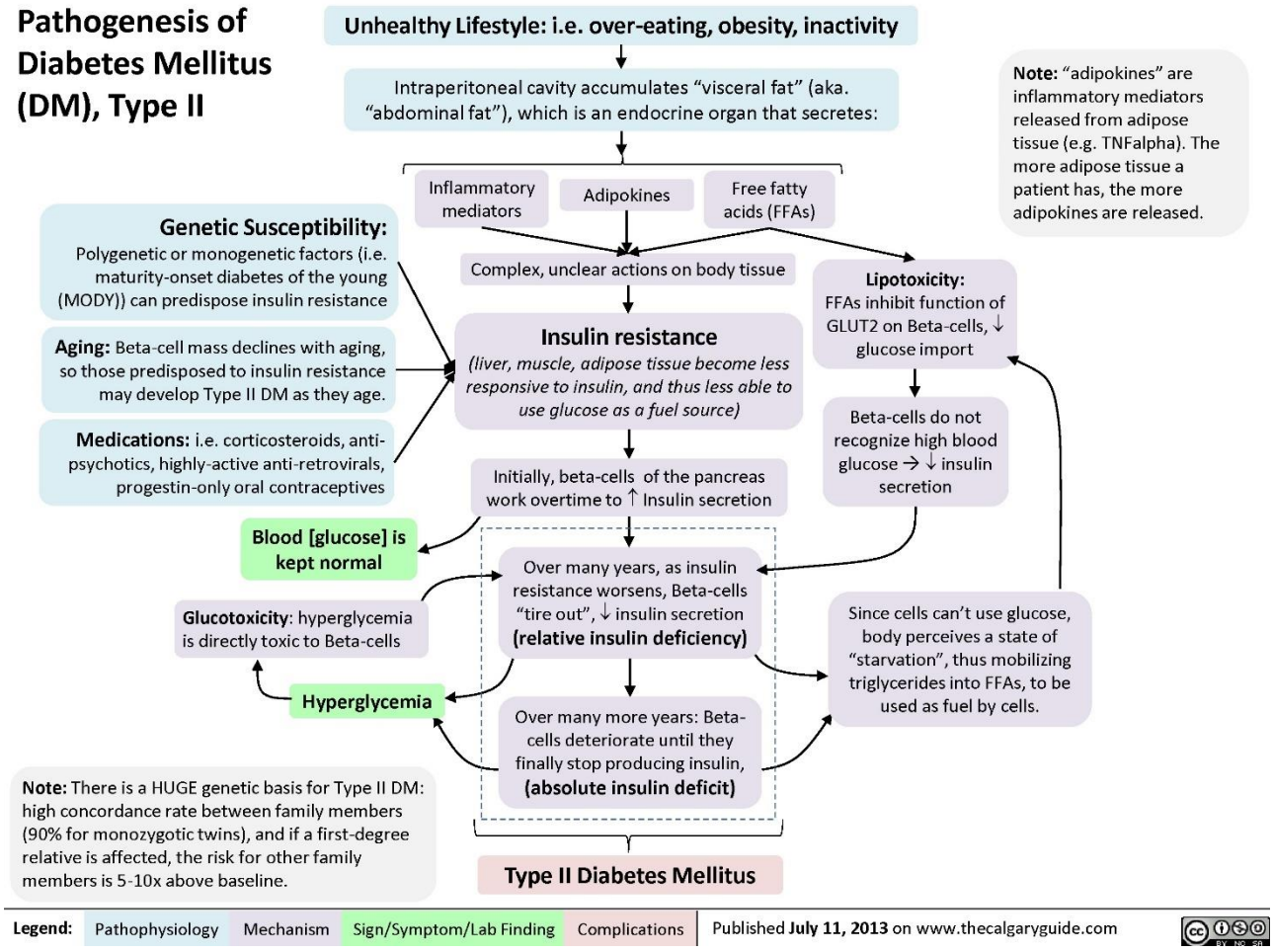
Pancreatic beta cells are destroyed by cellular-mediated, autoimmune processes in Type 1 DM. Strong genetic propensity exists for Type 1 DM. According to reports, the major histocompatibility complex (MHC), commonly referred to as human leukocyte antigens (HLA), is responsible for between 40 and 50 percent of the familial aggregation of Type 1 DM.

Beta-cell dysfunction is a component of type 2 DM, an insulin-resistance syndrome. Disruptions in various cellular pathways cause insulin resistance, which reduces the sensitivity of cells in peripheral tissues, particularly those in the muscle, liver, and adipose tissue, to insulin. Initial compensation involves a rise in insulin release, which keeps blood sugar levels within the usual range. As the illness worsens, beta cells alter, making insulin secretion unable to keep glucose homeostasis in check, leading to hyperglycaemia. The majority of type 2 DM patients are obese or have a greater body fat percentage, which is primarily distributed in the abdominal area. Through numerous inflammatory processes, including increased Free fatty acids release and dysregulated adipokines, this adipose tissue itself encourages insulin resistance.(18–21)

Pathophysiology

Figure 1: Pathophysiology of type1 and 2 DM



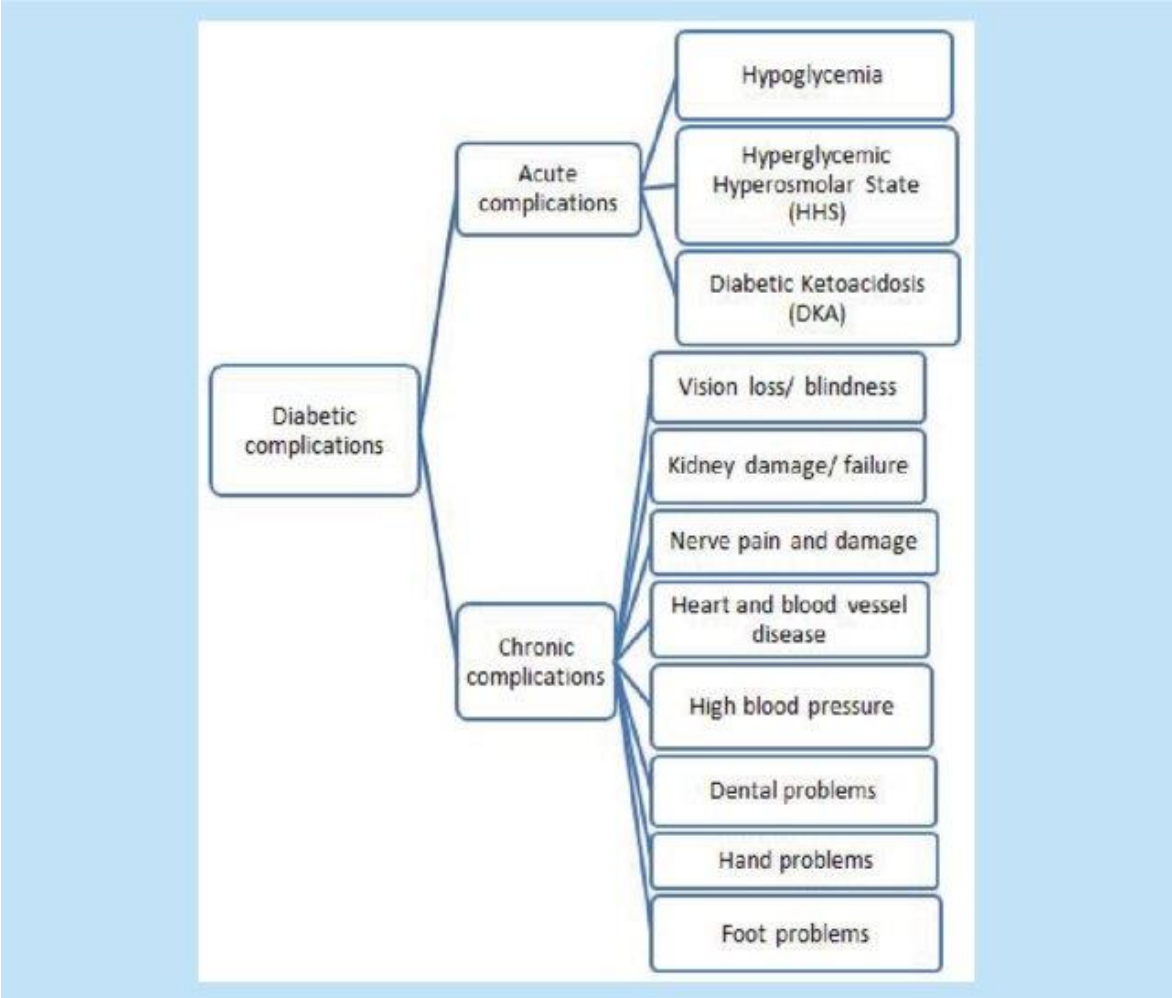


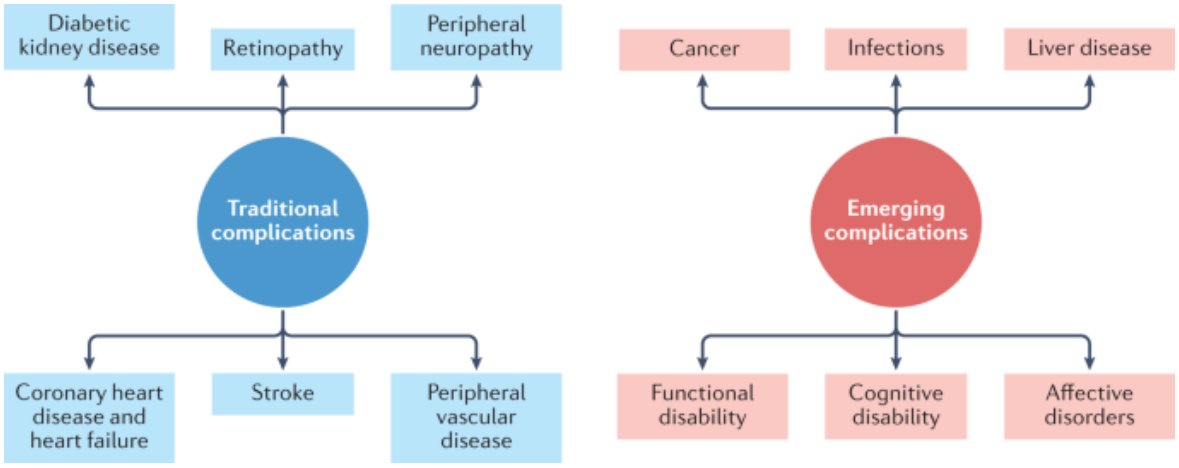
4.2 DIABETES MELLITUS-COMPLICATIONS

Numerous physiological organs are impacted by hyperglycaemia and the related protein, lipid, and carbohydrate metabolic dysfunctions, which prevent them from operating normally. The adverse effects of hyperglycaemia and its associated metabolic anomalies on the normal structure and function of the micro- and macro vasculature, which are at the foundation of organ structure and function throughout the body, are the primary cause of these disruptions, which develop gradually over time. Microvascular and macrovascular problems result from structural and functional disturbances in the organ system vasculature. These issues impact body organs, including the eyes, kidneys, heart, and nerves, causing organ damage, dysfunction, and eventually organ failure. Retinopathy is the result of eye-

related issues, and eventually progresses to blindness. Nephropathy and possible renal failure are caused by issues related to the kidneys. Coronary heart disease and hypertension are two heart-related problems. Neuropathy, which can be autonomic and/or peripheral, is caused by issues related to the nerves. Long-term peripheral neuropathy is frequently linked to foot infections, including ulcers that necessitate amputations and Charcot joint (osteoarthropathy), whereas cardiovascular, gastrointestinal, and genitourinary (including sexual) dysfunctions are characteristic manifestations of autonomic neuropathy. One of the main causes of diabetes-related morbidity and death is atherosclerotic cardiovascular disease, which is the term used to describe cerebrovascular disease, peripheral arterial disease, and coronary heart disease combined. (22–24)

Figure 2: Complications of diabetes mellitus*(25)*





4.3 DKA-PATHOLOGY, CLINICAL FEATURES, DIAGNOSIS, CLASSIFICATION

A dangerous and potentially fatal diabetes consequence is diabetic ketoacidosis (DKA). Most persons with type 1 diabetes experience DKA. DKA can occur in people who have type 2 diabetes as well.(26) DKA causes the body's metabolism of proteins, lipids, and carbohydrates to be drastically disrupted. The body enters a catabolic state with rapid degradation of glycogen reserves, triglyceride hydrolysis, and mobilisation of amino acids, which causes the liver to produce glucose and ketone bodies later, aggravating the metabolic decompensation.(27–29)

Precipitating factors

- Infection- pancreatitis, pneumonia
- Infarction-myocardial, stroke
- Non adherence to treatment
- Use of Drugs
- New onset diabetes
- Medical stress- trauma, surgery- through release of counter regulatory hormones
- Pregnancy

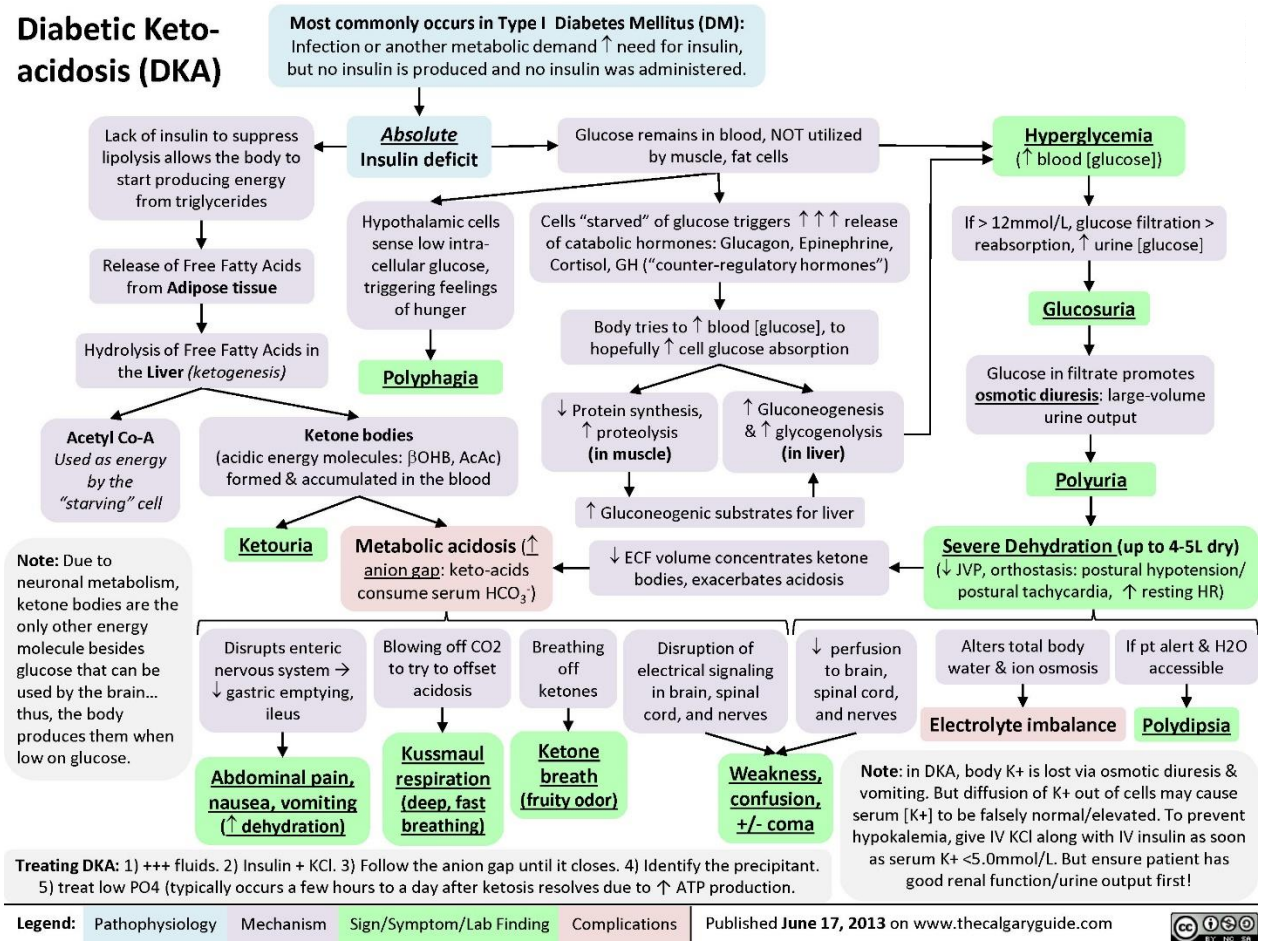
- Iatrogenic
- Idiopathic
- diseases of endocrine axis (acromegaly, Cushing`s syndrome, hyperthyroidism)
- diuretics, beta-blockers, corticosteroids, second-generation anti-psychotics, anti-convulsants, sodium-glucose cotransporter-2 (SGLT-2) inhibitors, and/or immune checkpoint inhibitors
- psychological problems, eating disorders, insulin pump malfunction

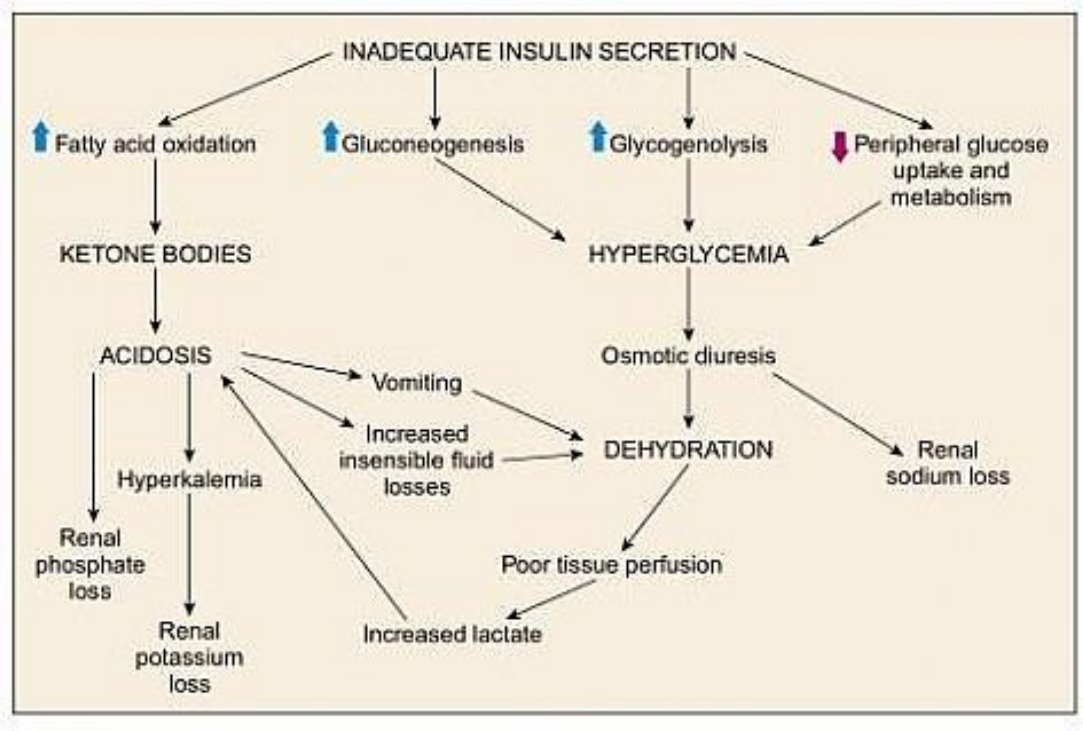
Pathophysiology

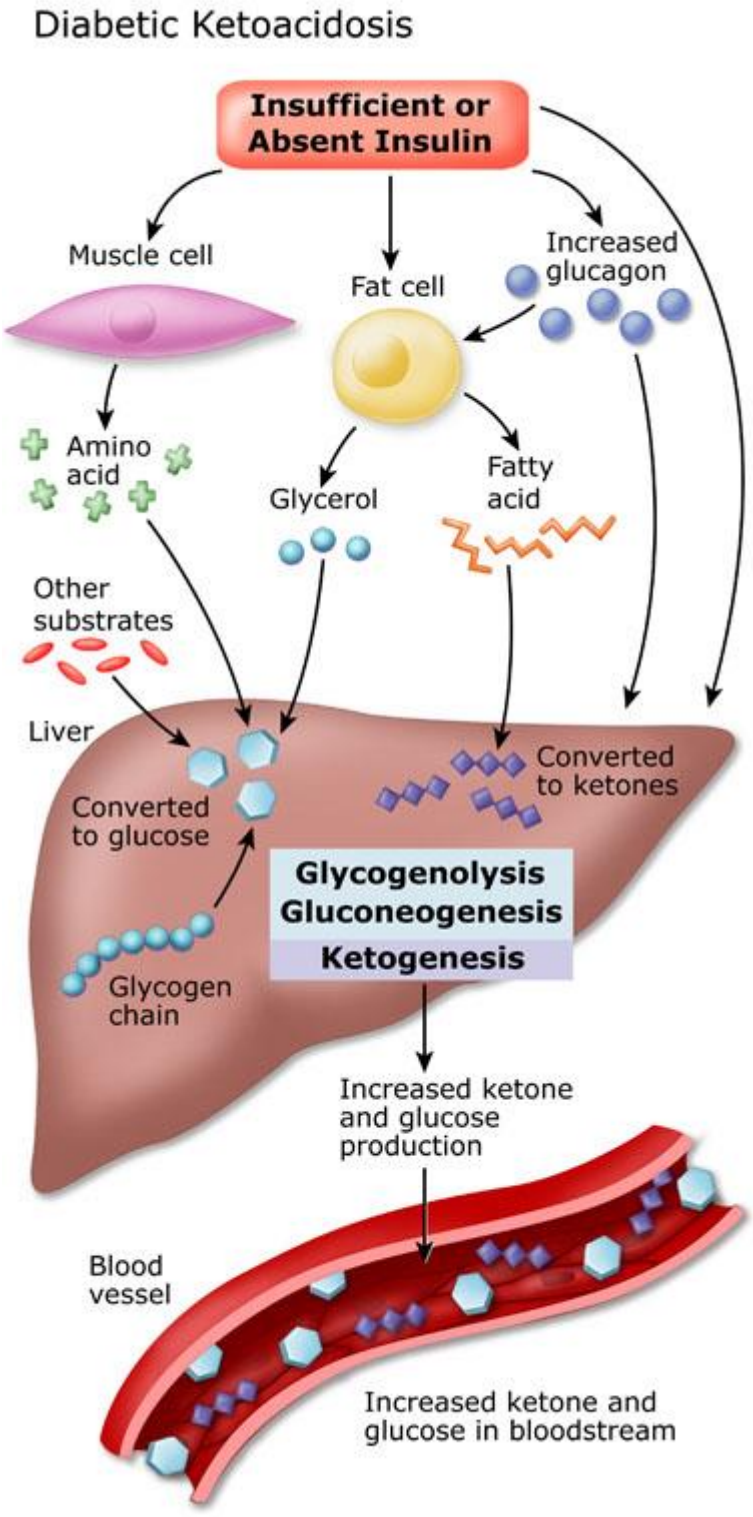
The pathogenesis of DKA is based on insulin insufficiency, elevated insulin counter-regulatory hormones (cortisol, glucagon, growth hormone, and catecholamines), and peripheral insulin resistance. These factors cause hyperglycemia, dehydration, ketosis, and electrolyte imbalance.

Accelerated glycogenolysis, reduced glucose uptake, and absolute insulin insufficiency all contribute to the development of hyperglycaemia in DKA patients. Noteworthy is the possibility of euglycemic DKA in diabetes patients who acquired DKA while being treated with SGLT-2 inhibitors. Abundant free fatty acids are transformed into the ketone bodies acetoacetate, acetone, and β-hydroxybutyrate (β -OHB) as a result of greater lipolysis and decreased lipogenesis. If oral fluid intake is insufficient, the osmotic diuresis brought on by hyperglycaemia results in dehydration, hyperosmolarity, electrolyte loss, and a reduction in glomerular filtration. Glycosuria decreases and hyperglycaemia/hyperosmolality worsens with a deterioration in renal function. Skeletal muscle utilises potassium much less efficiently with decreased insulin action and hyperosmolality, which causes intracellular potassium depletion. Additionally, osmotic diuresis results in the loss of potassium, which severely depletes the body of potassium. As a result, a wide range of serum potassium concentrations can be found in DKA patients.

Figure 3: Pathophysiology of DKA(30)






Diabetic Ketoacidosis
Insufficient or Absent Insulin
Muscle cell
Fat cell
Increased glucagon
Amino acid
Glycerol
Fatty acid
Other substrates
Liver
Converted to glucose
Converted to ketones
Glycogenolysis
Gluconeogenesis
Ketogenesis
Glycogen chain
Increased ketone and glucose production
Blood vessel
Increased ketone and glucose in bloodstream

Clinical features

Most clinical features are featured by hyperglycaemia and acidosis, including weight loss, among which the complications, cerebral oedema (CE) is the most deadly complication.(31) The complications of DKA are hypoglycaemia, cerebral oedema, thromboembolism, infections, shock, pulmonary oedema, electrolyte abnormalities.(32)

The following table represents the Clinical Signs, Physical Examination Findings, and Serum Biochemical Changes Commonly encountered in patients with Diabetic Ketoacidosis, (33)

Table 3. Clinical Features of Diabetic ketoacidosis:

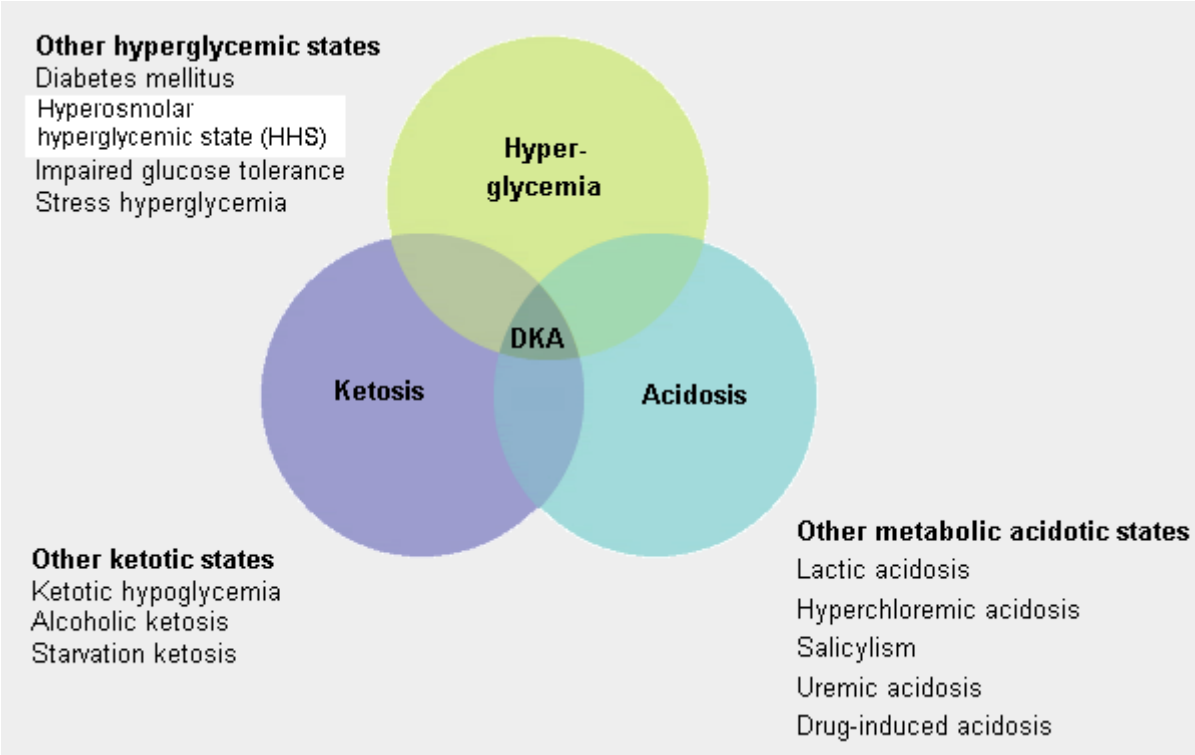
Clinical and Historical Signs	Clinical Pathology Findings
Anorexia	Azotemia
Dehydration	Elevated hepatic transaminases
Lethargy	Glucosuria
Previous polyuria or polydipsia	Hypercholesterolemia
Vomiting or nausea	Hyperglycemia
Physical Examination Findings	Hypertriglyceridemia
Dehydration	Hypochloremia
Ketone breath	Hypokalemia
Mental dullness	Hypomagnesemia
Thin body condition	Hyponatremia
Unkempt haircoat	Hypophosphatemia
Weakness	Ketonuria
	Metabolic acidosis

The diagnostic criteria for DKA includes

- mainly Blood glucose: > 250 mg per dL (13.9 mmol per L)
- pH <7.3
- Serum bicarbonate level < 15 mEq per L
- Urinary ketone levels ≥3+. (34)

The diagnostic algorithm of diabetic ketoacidosis includes ketones, acidosis and Hyperglycaemia as represented in the following figure, (34)

Figure 4. Diagnostic algorithm of diabetic ketoacidosis:



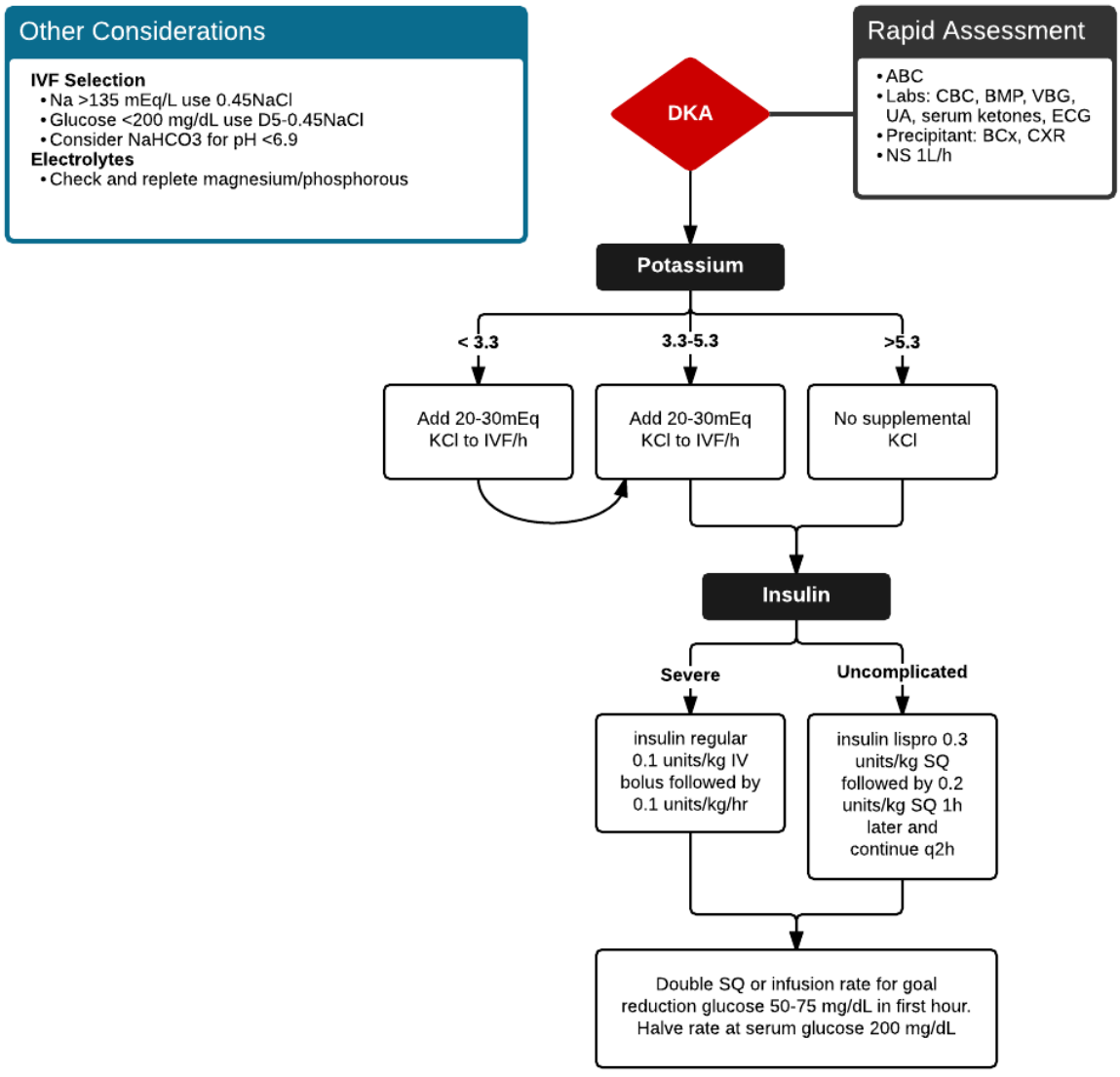
Classification of DKA

Table 4: DKA classification(35,36)

	Mild DKA	Moderate DKA	Severe DKA
Plasma glucose (mg/dL)	>250	>250	>250
pH	7.25–7.3	7.0–7.24	<7.0
Serum bicarbonate (mEq/L)	15–18	10–15	<10
Ketones (urine or serum)	Positive	Positive	Positive
Anion gap	>10	>12	>12
Osmolality (mOsm/kg)	Variable	Variable	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma

Management

Figure 5: Management of DKA



4.4 STUDIES RELATED TO THE TOPIC

Eledrisi et al in 2022 did a study to evaluate the clinical traits and outcomes of DKA subjects who are hospitalized. 1330 individuals in total, with 1613 episodes of DKA, were analysed, of whom 37.3% had type 1 diabetes and 62.7 % had type 2 diabetes. While there was no difference in the severity of DKA, laboratory values at admission, or the time it took for DKA to resolve, patients with type 2 diabetes were older than those with type1 diabetes [48.0 (38.0-60.0), 26.0 (21.0-31.0) years]. In patients with type 2 diabetes compared to type 1 diabetes, admission to the critical care unit was greater (38.9 % vs. 26.6 %), with a longer hospital stay [5 (2.0-9.0) vs. 2 (2.0-4.0) days, and noticeably higher mortality (7.4 % vs. 1 %).(37)

Ooi et al in 2021 did a study to explore the clinical and biochemical variations in the racial and ethnic composition, clinical manifestations, and treatment of diabetic ketoacidosis (DKA) in individuals with type 1 and type 2 diabetes. Compared to those with type 1 diabetes, those with type 2 diabetes were older and more likely to be members of ethnic minorities. In both groups, concurrent sickness (39.8%) and poor compliance (26.8%) were the two most frequent triggering causes of DKA. Both groups had identical levels of DKA severity at presentation as measured by pH, glucose, and lactate. Total insulin requirements were the same for both types of diabetes (13.9 units for type 1 diabetes (9.1-21.9) and 13.9 units for type 2 diabetes (7.7-21.1)). However, those with type 2 diabetes required a considerably longer hospital stay (11.0 days (5.0-23.1) vs. 3.0 days (1.7-6.1); type 1 diabetes).(38)

Hare et al in 2021 did a study to describe the clinical, psychosocial, and demographic characteristics that are present in individuals with DKA, focusing on those that are linked to recurrent admissions. 16 of them (13%) had more than one DKA admission. There was a history of depression in 41 (32%). The most frequent causes of presentation were lack of insulin (54%), infection (31%), excessive alcohol use (26%) and new diabetes diagnosis (16%). People with recurring DKA were more likely to smoke (69% vs 27%, P = 0.003), be unemployed (31% vs 11%, P = 0.04), and use illicit drugs (44% vs 17%, P = 0.02) than those with single admissions.(39)

Nunes et al in 2021 did a study to describe the prevalence, clinical features, laboratory findings, and initiating factors of diabetic ketoacidosis in emergency room patients at a tertiary level hospital and to determine both short- and long-term mortality after two years. There were 8.7 cases of diabetic ketoacidosis for every 1,000 hospitalizations. The most frequent causes of diabetic ketoacidosis were infection and noncompliance with prescribed treatment. The death rate for the immediate, first six months, the first year, and the second year was 5.8%, 9.6%, 13.5%, and 19.2%, respectively. Age, type 2 diabetes, hypoalbuminemia, infection at presentation, and a higher sequential organ failure assessment score upon admission were all related with death occurring within two years.(40)

Yotsapon Thewjitcharoen et al in 2019 did a study among hospitalised DKA patients and treatment outcomes during a ten-year period. 81 diabetic patients experienced a total of 94 DKA episodes (61.5% females, mean age 47.4 (20.4) years, T1DM 41.5%, T2DM 50.0%, ketosis-prone diabetes 8.5%, baseline A1C 10.8 (3.0)%). Infection was frequently the cause of T2DM precipitation, whereas insulin omission was typically the cause of T1DM precipitation. During continuous treatment, 26.6% of patients experienced hypokalaemia, and in this group of patients, supplementation was not recommended as directed.

Hypoglycaemia suffered over 13% of patients within the first 24 hours. DKA resolution took an average of 8.5 hours. In our study, the mortality rate was 4.3% and was caused by four T2DM patients dying from DKA, which was the precipitating reason.(41)

Sonwani, Arya and Saxena in 2018 did a study to evaluate the clinical characteristics and prognosis of DKA in Type 2 diabetes mellitus patients. The majority of our DKA patients were male (72%), aged 51 to 55 (21%), and obese (46%).70% of them were on oral hypoglycaemic medications. The mean values for the following variables like mean duration of diabetes, Body Mass Index, Haemoglobulin, Random blood Sugar, HbA1c and pH was 7.28(3.81) years, 29.00(3.58) kg/m^2, 9.8(1.42) gm%, 351.72(22.32) mg/dl, 9.3(1.23)%, and 7.14(0.10), respectively. Out of 100 patients, 86% were discharged, 7% died, and 7% dropped out of the trial. In DKA patients, hypertension (60%) obesity (47%), nausea and vomiting (86%), stomach pain (58%), poor compliance (53%) and infection, particularly pneumonia (24%), were the most prevalent co-morbidities, clinical symptoms, and precipitating factors. Most of the patients who died had diabetes for more than ten years (23.1%), had poor compliance (10.2%), had Cardiovascular Disease (40%), and had Acute Coronary Syndrome (40%).(42)

Prajapati in 2017 did a study to evaluate the clinical and laboratory characteristics of adults in the Dhulikhel hospital who have diabetic ketoacidosis. The study comprised 48 patients who met the requirements for diabetic ketoacidosis. The prevalence of type 2 diabetes mellitus among patients was 73%. 23 percent of the patients had just been diagnosed with diabetes mellitus. Patients with type 1 diabetes mellitus were more likely to appear with polyuria and polydipsia (p=0.002), whereas type 2 diabetes mellitus patients were more

likely to experience fever (p=0.03). Most individuals showed normal levels of serum sodium and potassium. Over one third of patients had high serum creatinine levels, while 42% of patients had high serum urea levels. Omission of insulin was the most frequent cause of diabetic ketoacidosis in people with type 1 diabetes mellitus, whereas in people with type 2 diabetes, it was infection.(43)

Kamata et al in 2017 did a study to analyse the clinical characteristics and in-depth care of related type 1 and type 2 diabetes patients who were hospitalised with DKA. For patients with type 1 diabetes and those with type 2 diabetes, ingestion of sugar-containing beverages was the most frequent triggering cause of DKA. Patients with type 2 diabetes had higher mean plasma glucose levels than those with type 1 diabetes (48.4(21.6) vs. 37.1(16.4)mmol/l) as well as higher serum levels of haemoglobin, blood urea nitrogen, and creatinine, all of which returned to normal once DKA was resolved. Patients with type 2 diabetes needed significantly more daily total insulin (35.9(37.0)U vs. 20.2(23.3)U), more replacement fluid (4.17(2.69)L vs. 2.29(1.57)L), and more potassium supplementation (23.9(36.5)mEq vs. 11.2(17.9)mEq) than patients with type 1 diabetes to resolve DKA.(44)

Xu et al in 2016 did a study to analyse the clinical characteristics of those who suffer from diabetic ketoacidosis at China's tertiary hospitals. The study included 643 diabetic ketoacidosis patients, including 308 Type 1 diabetes patients (47.9%), 294 Type 2 diabetes patients (45.7%), and 41 patients (6.4%) with atypical diabetes. Patients with known diabetes accounted for 388 (60.3%) of the 388 diabetic ketoacidosis incidents. Infection (40.1%), unidentified causes (36.9%), and non-compliance with anti-diabetes therapy (16.8%) were the most frequent triggering factors. At presentation, the Type 1 diabetes group had a higher prevalence of dehydration and gastrointestinal complaints. Only 74.4%

and 55.9% of individuals with new-onset diabetes had their -cell function and autoantibodies analysed for categorization, respectively. Only 67% of diabetic ketoacidosis patients received the proper fluid therapy, and only 56% of patients with severe acidosis received bicarbonate therapy. The patient spent 10.0 (7.0-14.0) days in the hospital. And the death rate was 1.7% and was significantly greater among Type 2 diabetes than in Type 1 diabetes (3.2% vs. 0.4%, P <0.01).(45)

Seth, Kaur and Maneet Kaur in 2015 did a study to investigate the clinical characteristics, contributing factors, and clinical outcome in patients who present with diabetic ketoacidosis in the emergency department of a tertiary care hospital. Out of 60 patients, 12 had Type 1 diabetes and 48 had Type 2 diabetes. 8.65 years on average had diabetes. Only 14 (23.3%) patients with diabetes mellitus were receiving regular treatment, compared to 32 (53.33%) patients receiving irregular treatment and eight (13.33%) patients receiving nil treatment at all. Six of the 12 Type 1 diabetic patients who presented with diabetic ketoacidosis complications had just received their diabetes diagnosis. The most frequent symptoms among these patients were nausea and vomiting (63.33%). The most frequent contributing factor to diabetic ketoacidosis was an infection (73.33%). 10% mortality was observed.(46)

Maskey et al in 2015 did a study to analyse the demographic profile, clinical attributes of subjects admitted with diabetic ketoacidosis Only 16 patients (seven types 1 and nine type 2DM) had DKA. The type 2 participants had substantially higher PH levels (7.11 s 7.28) and were older (56.8 s 25.7 years) than the 16 subjects with type 1 DM. The mean BMI for both type 1 and type 2 DM was 20.5(2.44). The most frequent precipitating factor was infection, which was followed by poor drug compliance (37.5%) and the first presentation (6.25%).(47)

Azevedo et al in 2014 did a study to explain the clinical results and level of care for adult intensive care unit patients with moderate- to severe diabetic ketoacidosis (DKA). Additionally, we wanted to compare their clinical course to that of matched non-DKA intensive care unit controls and find characteristics that predicted mortality and hospital readmission within a year. 39% of DKA patients required mechanical breathing, 17% required vasopressors, and 12% required renal replacement therapy. Mortality and readmission rates at one year were 9% and 36%, respectively. By using logistic regression, it was determined that the usage of insulin and treatment noncompliance had independent associations with mortality and/or readmission that occurred within a year. Patients with DKA had reduced mortality rates and were more likely to be sent home when compared to similarly matched non-DKA patients.(48)

Rao, Pradhan, Mallikarjuna and Reddy in 2012 did a study to assess the clinical features of DKA. 22 (81%) of the 27 individuals had type 2 diabetes, while just 5 (19%) had type 1 diabetes. There were 17 men and 10 women. The patients' ages ranged from 18 to 70, with a mean age of 45.3-year. In 60% of the admissions, precipitating circumstances were present. 50% of patients exhibited non-compliance or pharmacological therapy cessation. Average hospital stays in type 1 DM were somewhat longer than those in type 2 DM patients.(49)

5 RESEARCH QUESTION OR HYPOTHESIS

5.1 **RESEARCH QUESTION:**

What is the clinical profile, precipitating factors, outcome and bicarbonate level among DKA subjects?

5.2 **NULL HYPOTHESIS:**

There is no significant association in bicarbonate level and severity factors among DKA subjects.

5.3 **ALTERNATE HYPOTHESIS:**

There is a significant association in bicarbonate level and severity factors among DKA subjects.

6 METHODOLOGY

6.1 **STUDY SUBJECTS:**

DKA subjects admitted in the inpatient wards ,Department of General medicine,Thanjavur medical college.

6.2 **STUDY DESIGN:**

Prospective study

6.3 STUDY PERIOD:

Data collection – 1 year (

6.4 **STUDY SETTING:**

Department of General medicine,Thanjavur medical college.

6.5 **SAMPLING PROCEDURE:**

Subjects recruited after considering inclusion and exclusion criteria through convenient sampling

6.6 INCLUSION CRITERIA:

- ✓ Patients with diabetic ketoacidosis

6.7 EXCLUSION CRITERIA:

- Patient with hyperglycaemic hyperosmolar coma.
- Patient having chronic renal disease.
- Patient with hyperemesis gravidarum.
- Patient with starvation ketosis.
- Patient with severe anaemia.

6.8 SAMPLE SIZE:

According to Sathik Basha et al study(50), considering the prevalence of Low bicarbonate levels in DKA (p) as 14%, with a precision (d) of 10%, at 95% confidence interval ($Z_{1-\alpha}$ = 1.96), the sample size is calculated as

$N = Z^2_{1-\alpha} * p * (1 - p) / d^2$

$N = 1.96^2 * 0.14*(1 - 0.14) / 0.1^2 = 46$

Thus the total sample size required for the study is 46

6.9 **ETHICAL CONSIDERATION:**

Institutional Ethical Committee approval, from Tirunelveli Medical College, Tirunelveli, was obtained before the start of the study. Informed written consent was obtained.

Source of Funding: None declared

Conflict of Interest: None declared

6.10 **STUDY PROCEDURE:**

The subjects were assessed for these parameters

- CLINICAL PROFILE includes Age, Sex, socioeconomic status, Total stay in Hospital, personal history and family history, treatment history.
- CLINICAL FINDINGS includes level of consciousness, pupil size, heart rate, respiratory rate, blood pressure
- RBS, RFT, bicarbonate, plasma/urine acetone.
- Electrolyte Derangements

6.11 BUDGET:

Self. (No added investigation or intervention)

6.12 STATISTICAL METHODS:

I. Descriptive Statistics:

1. Numerical variables like Age , Random Blood Sugar (mg/dl), Serum Bicarbonate (mEq/L), HbA1C (%), Hemoglobin (mg/dl), Renal Function tests, Serum Electrolytes, Heart Rate , Respiratory rate and duration of stay etc are represented in mean, SD, median, and mode. Histograms are used wherever necessary.
2. Categorical variables like gender, Symptoms, Conscious level, Cause of DKA and Outcome etc., are represented in frequencies and percentages. Pie-charts and bar diagrams are used as appropriate.
3. Data was entered in MS excel sheet and analysed using SPSS software version 23.

II. Inferential Statistics:

1. When a Numerical variable like serum bicarbonate level is compared with the Numerical variables like duration of stay, Pearson's correlation test is used.
2. When a Categorical Variable like outcome – death and recovered is compared with a categorical variable types of diabetes mellitus and treatment history , the variables are represented in both by tables and bar diagrams. For test of significance, chi-square test or Fischer's exact test is used. When a Categorical Variable like outcome – death and recovered is compared with a continuous variable like Random Blood Sugar (mg/dl), Serum Bicarbonate (mEq/L), HbA1C (%), Hemoglobin (mg/dl), Renal Function tests, Serum Electrolytes, Heart Rate , Respiratory rate and duration of stay, independent t test was used for test of significance.
3. P-values less than 0.05 were considered statistically significant.

7 RESULTS

Results of the study, on Diabetic ketoacidosis clinical profile is discussed under the following headings:

I. Age (years)

II. Age group

III. Gender

IV. Diabetes Mellitus type

V. Family History

VI. Treatment History

VII. Random Blood Sugar (mg/dl)

VIII. Plasma acetone

IX. Serum Bicarbonate (mEq/L)

X. HbA1C (%)

XI. Hemoglobin (mg/dl)

XII. Renal Function tests

XIII. Serum Electrolytes

XIV. Symptoms

XV. Consciousness level

XVI. Heart Rate & Respiratory rate

XVII. Blood Pressure

XVIII. Duration of Stay (days)

XIX. Cause of DKA

XX. Outcome

XXI. Comparison of Diabetes Mellitus type with the Outcome

XXII. Comparison of Treatment History with the Outcome

XXIII. Random Blood Sugar (mg/dl) with Outcome

XXIV. Serum Bicarbonate (mEq/L) with Outcome

XXV. HbA1C (%) with Outcome

XXVI. Correlation between Serum Bicarbonate (mEq/L) and Duration of Stay (days)

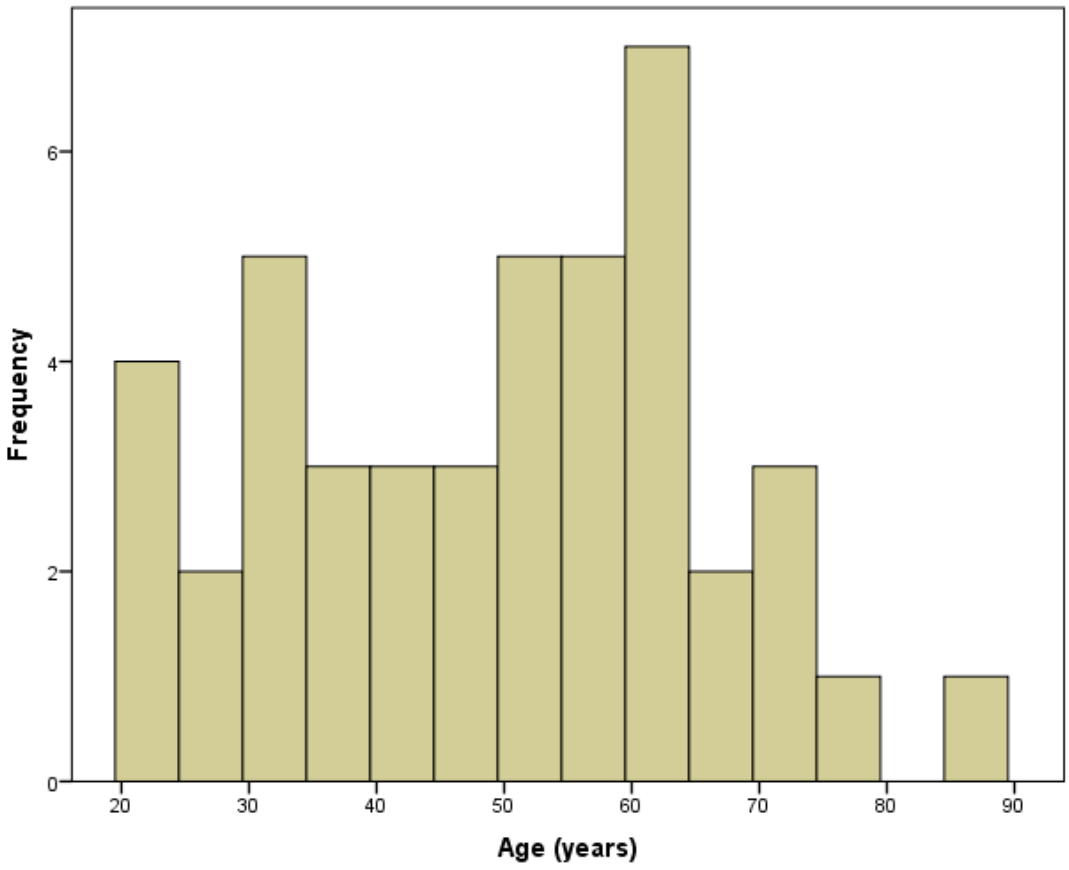
I. Age (years)

The mean Age (years) among the subjects was 47.87 (± 17.15) years ranging from 17 to 85 years.

Table 5. Age (years)

Age (years)	
Mean	47.87
Median	50.5
Std. Deviation	17.15
Range	68
Minimum	17
Maximum	85

Figure 6. Age (years)



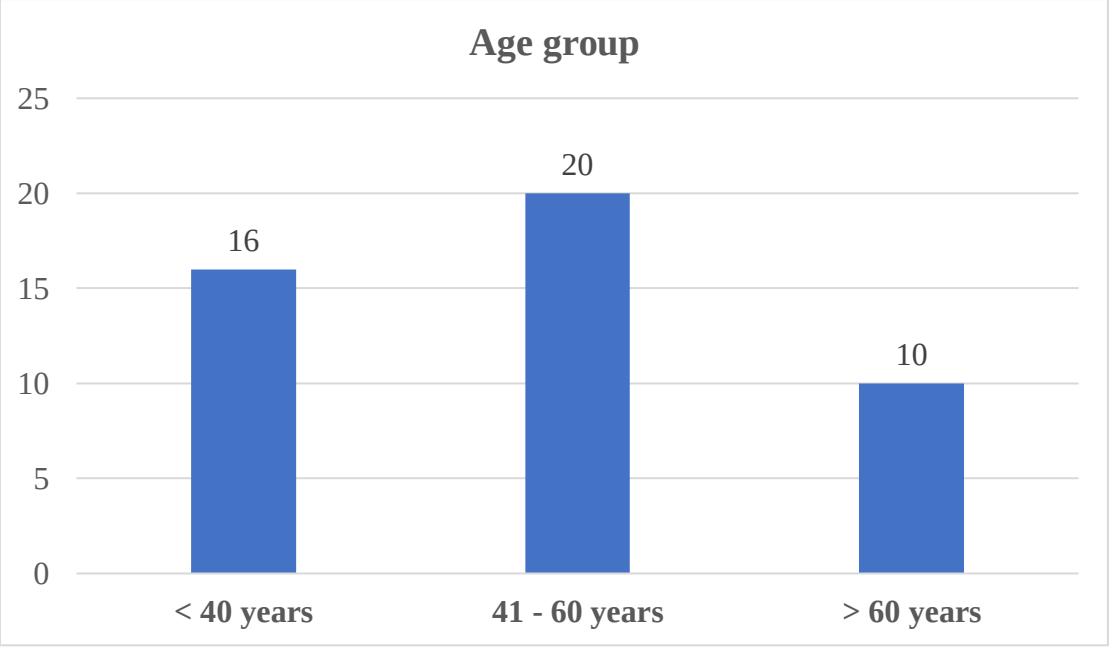
II. Age group

Among the subjects, 20 (43.48%) were in 41 - 60 years, 16 (34.78%) were in < 40 years and 10 (21.74%) were in > 60 years

Table 6. Age group

Age group	Frequency	Percent
< 40 years	16	34.78
41 - 60 years	20	43.48
> 60 years	10	21.74
Total	46	100.00

Figure 7. Age group



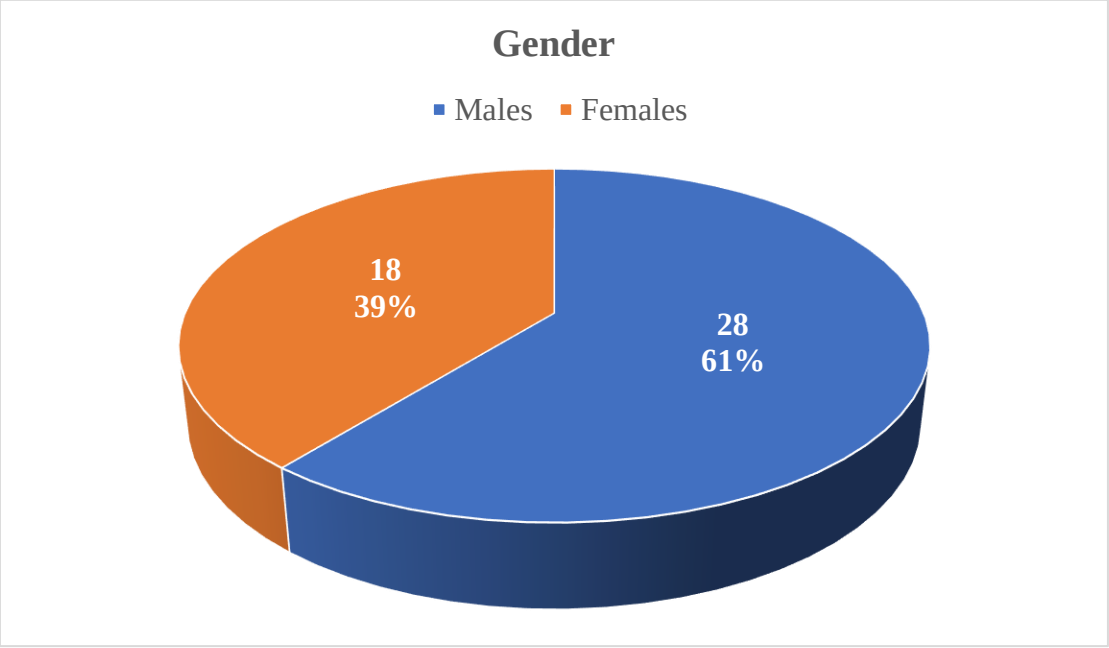
III. Gender

Among the subjects, 28 (60.87%) were Males and 18 (39.13%) were Females

Table 7. Gender

Gender	Frequency	Percent
Males	28	60.87
Females	18	39.13
Total	46	100.00

Figure 8. Gender



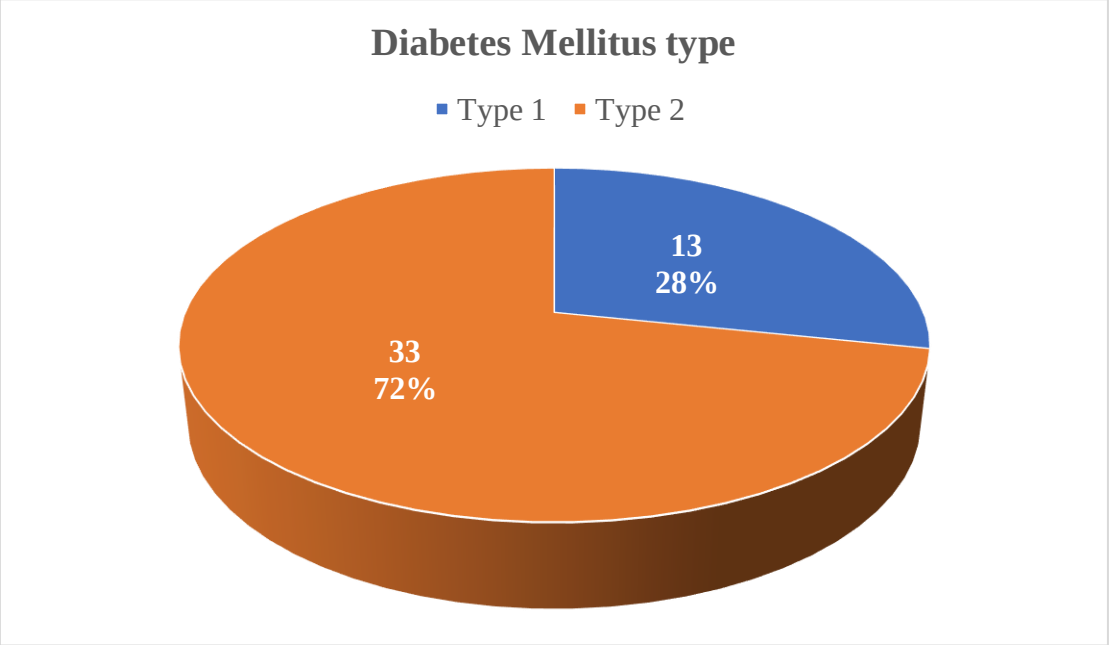
IV. Diabetes Mellitus type

Among the subjects, 33 (71.74%) had Type 2 and 13 (28.26%) had Type 1 Diabetes.

Table 8. Diabetes Mellitus type

Diabetes Mellitus type	Frequency	Percent
Type 1	13	28.26
Type 2	33	71.74
Total	46	100.00

Figure 9. Diabetes Mellitus type



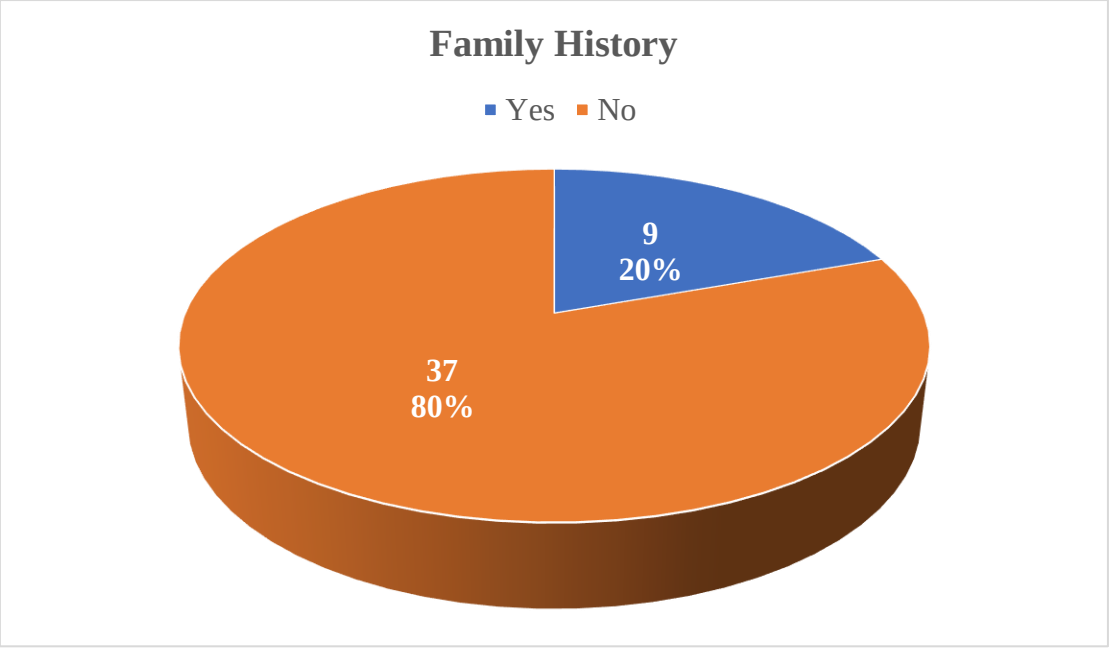
V. Family History

Among the subjects, 9 (19.57%) had Family History

Table 9. Family History

Family History	Frequency	Percent
Yes	9	19.57
No	37	80.43
Total	46	100.00

Figure 10. Family History



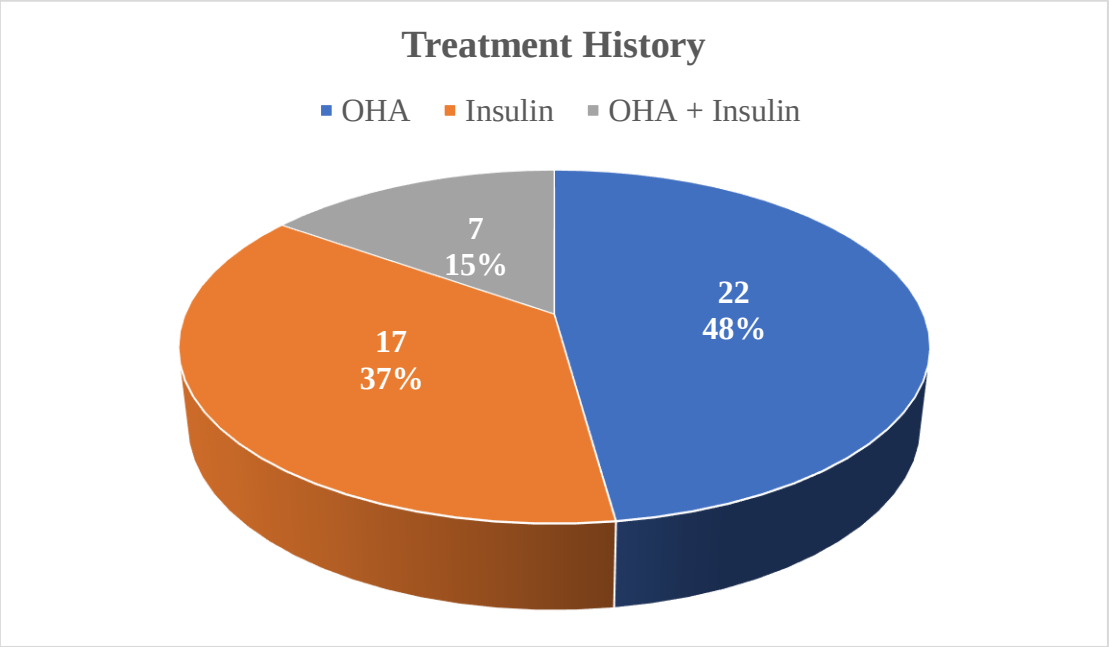
VI. Treatment History

Among the subjects, 22 (47.83%) had OHA, 17 (36.96%) had Insulin and 7 (15.22%) had OHA + Insulin

Table 10. Treatment History

Treatment History	Frequency	Percent
OHA	22	47.83
Insulin	17	36.96
OHA + Insulin	7	15.22
Total	46	100.00

Figure 11. Treatment History



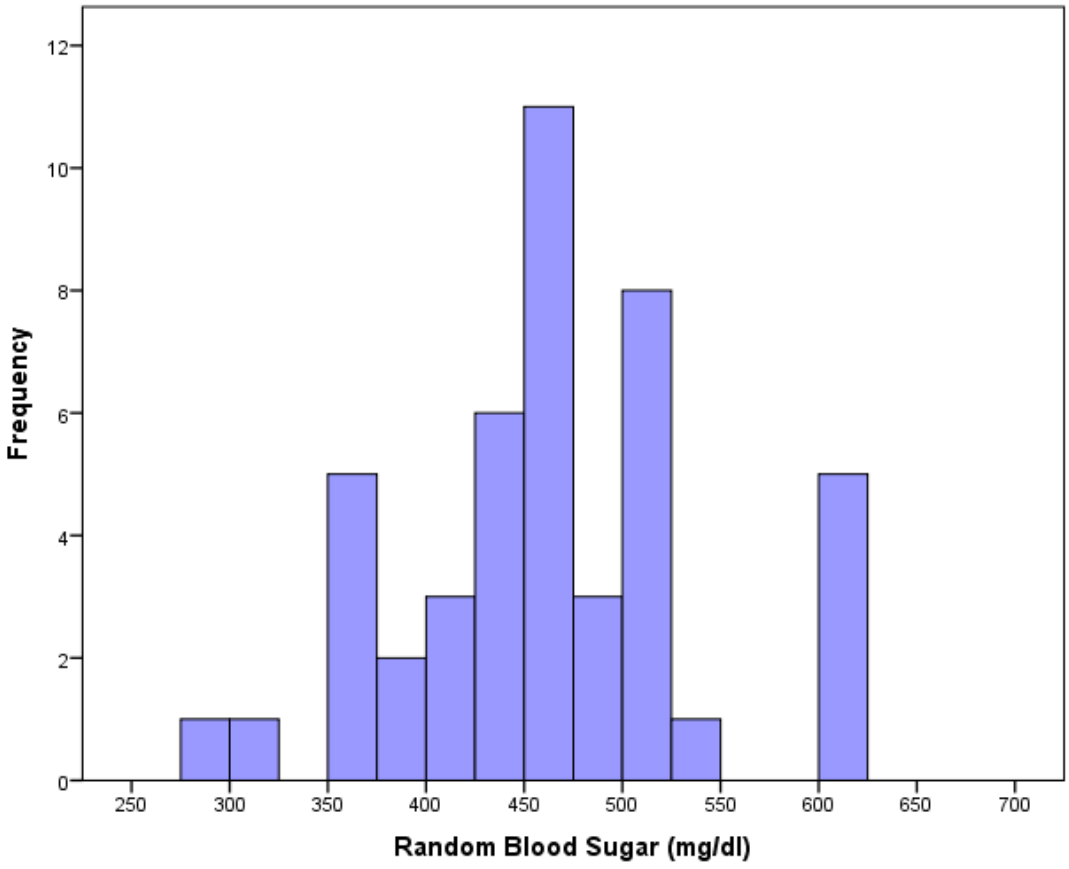
VII. Random Blood Sugar (mg/dl)

The mean Random Blood Sugar (mg/dl) among the subjects was 460.11 (± 75.59) ranging from 281 to 600

Table 11. Random Blood Sugar (mg/dl)

Random Blood Sugar (mg/dl)	
Mean	460.11
Median	465
Std. Deviation	75.59
Range	319
Minimum	281
Maximum	600

Figure 12. Random Blood Sugar (mg/dl)



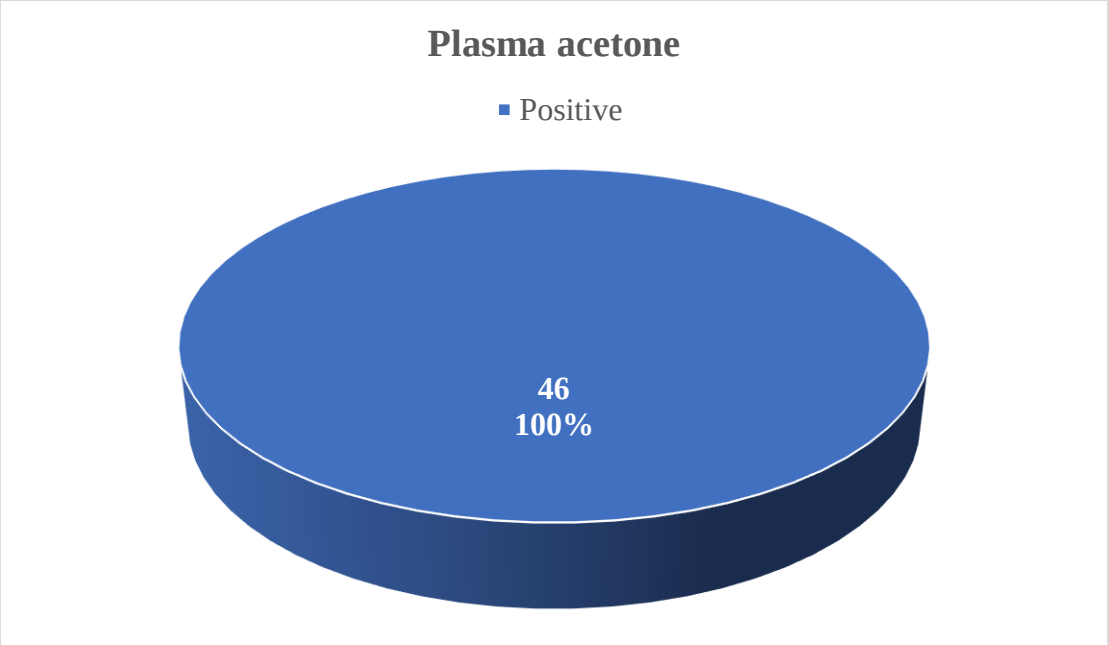
VIII. Plasma acetone

All the subjects, 46 (100%) had Plasma acetone

Table 12. Plasma acetone

Plasma acetone	Frequency	Percent
Positive	46	100.00

Figure 13. Plasma acetone



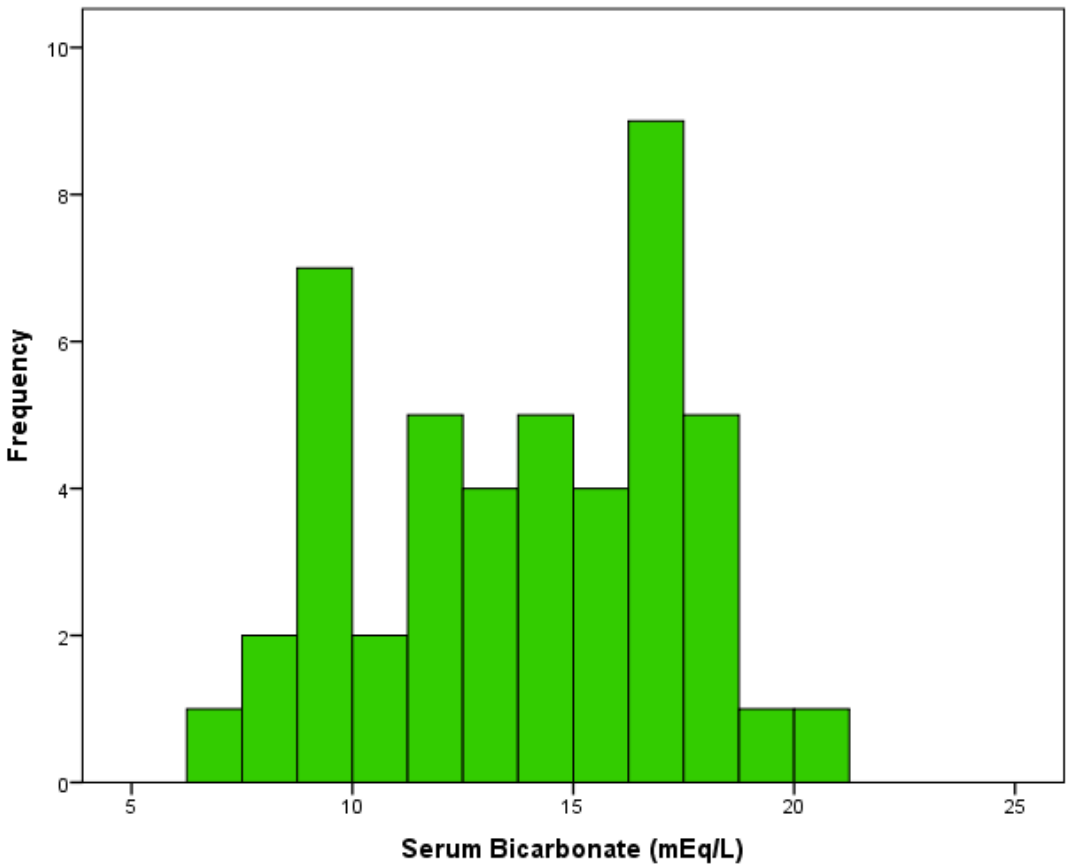
IX. Serum Bicarbonate (mEq/L)

The mean Serum Bicarbonate (mEq/L) among the subjects was 13.84 (± 3.53) ranging from 7 to 21 mEq/L.

Table 13. Serum Bicarbonate (mEq/L)

Serum Bicarbonate (mEq/L)	
Mean	13.84
Median	14.05
Std. Deviation	3.53
Range	14
Minimum	7
Maximum	21

Figure 14. Serum Bicarbonate (mEq/L)



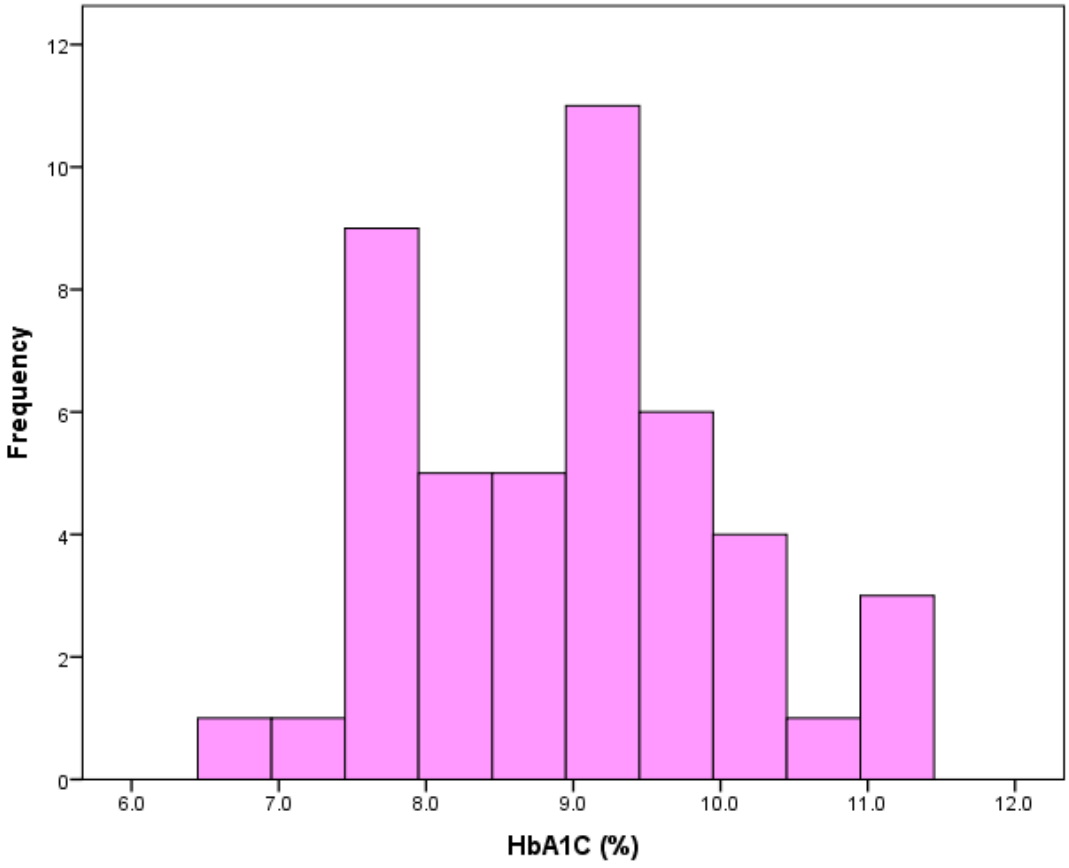
X. HbA1C (%)

The mean HbA1C (%) among the subjects was 8.98 (± 1.07) ranging from 6.7 to 11.4

Table 14. HbA1C (%)

HbA1C (%)	
Mean	8.98
Median	9
Std. Deviation	1.07
Range	4.7
Minimum	6.7
Maximum	11.4

Figure 15. HbA1C (%)



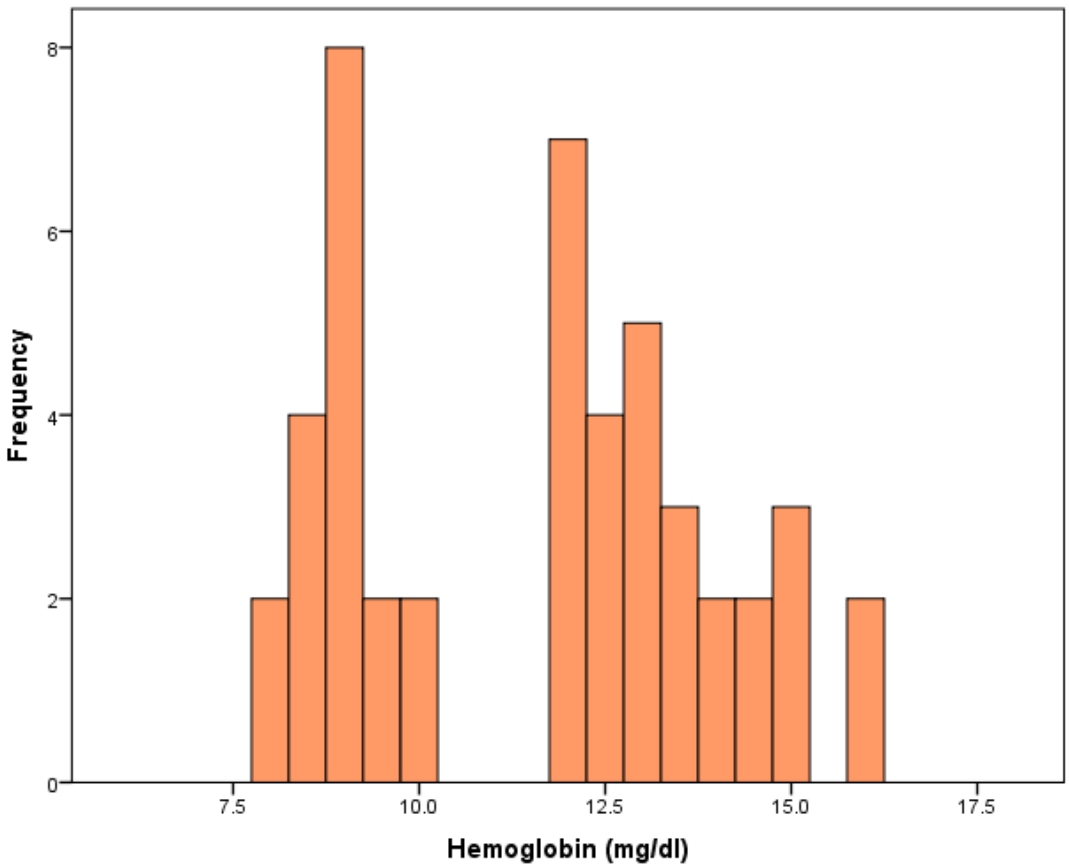
XI. Hemoglobin (mg/dl)

The mean Hemoglobin (mg/dl) among the subjects was 11.64 (± 2.4) ranging from 8 to 16.2

Table 15. Hemoglobin (mg/dl)

Hemoglobin (mg/dl)	
Mean	11.64
Median	12.05
Std. Deviation	2.40
Range	8.2
Minimum	8
Maximum	16.2

Figure 16. Hemoglobin (mg/dl)



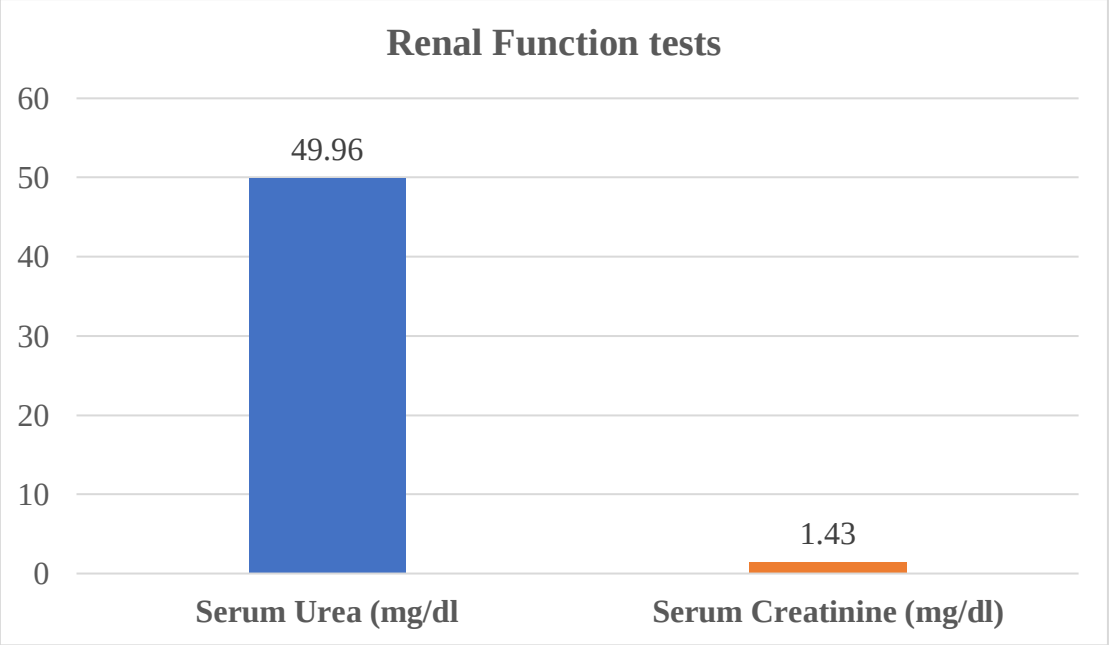
XII. Renal Function tests

The mean Serum Urea (mg/dl) among the subjects was 49.96 (± 12.61) ranging from 30 to 84 mg/dl. The mean Serum Creatinine (mg/dl) among the subjects was 1.43 (± 0.42) ranging from 0.7 to 2.2 mg/dl.

Table 16. Renal Function tests

	N	Mean	Std. Deviation	Minimum	Maximum
Serum Urea (mg/dl	46	49.96	12.61	30.0	84.0
Serum Creatinine (mg/dl)	46	1.43	0.42	0.7	2.2

Figure 17. Renal Function tests



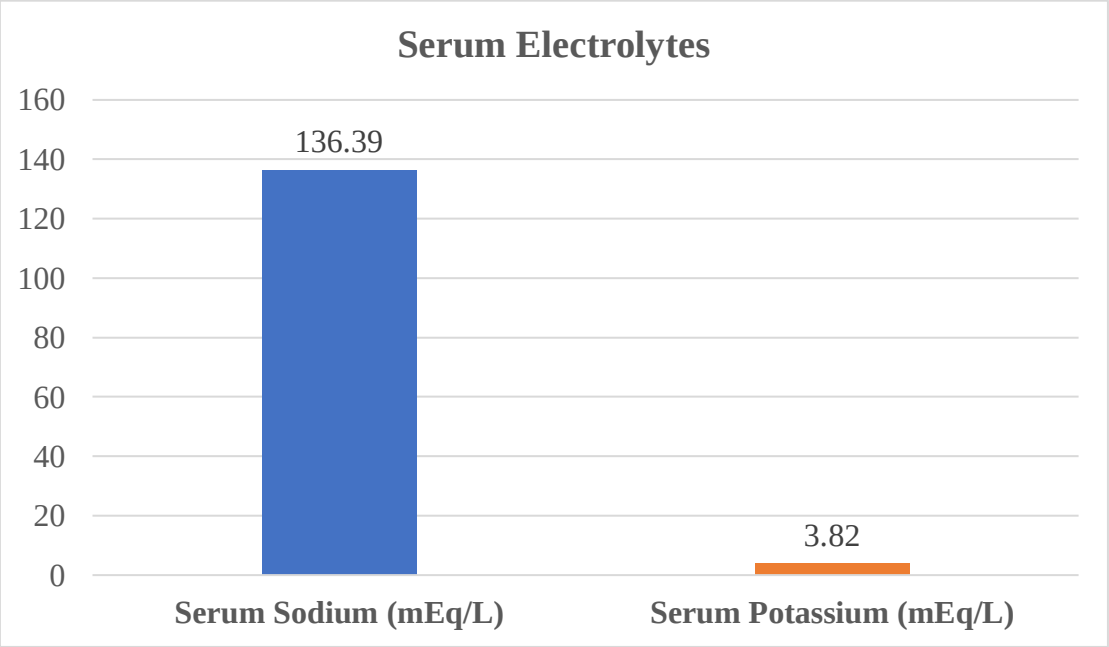
XIII. Serum Electrolytes

The mean Serum Sodium (mEq/L) among the subjects was 136.39 (± 6.61) ranging from 121 to 150 mEq/L. The mean Serum Potassium (mEq/L) among the subjects was 3.82 (± 0.73) ranging from 2.7 to 5.4 mEq/L.

Table 17. Serum Electrolytes

	N	Mean	Std. Deviation	Minimum	Maximum
Serum Sodium (mEq/L)	46	136.39	6.61	121.0	150.0
Serum Potassium (mEq/L)	46	3.82	0.73	2.7	5.4

Figure 18. Serum Electrolytes



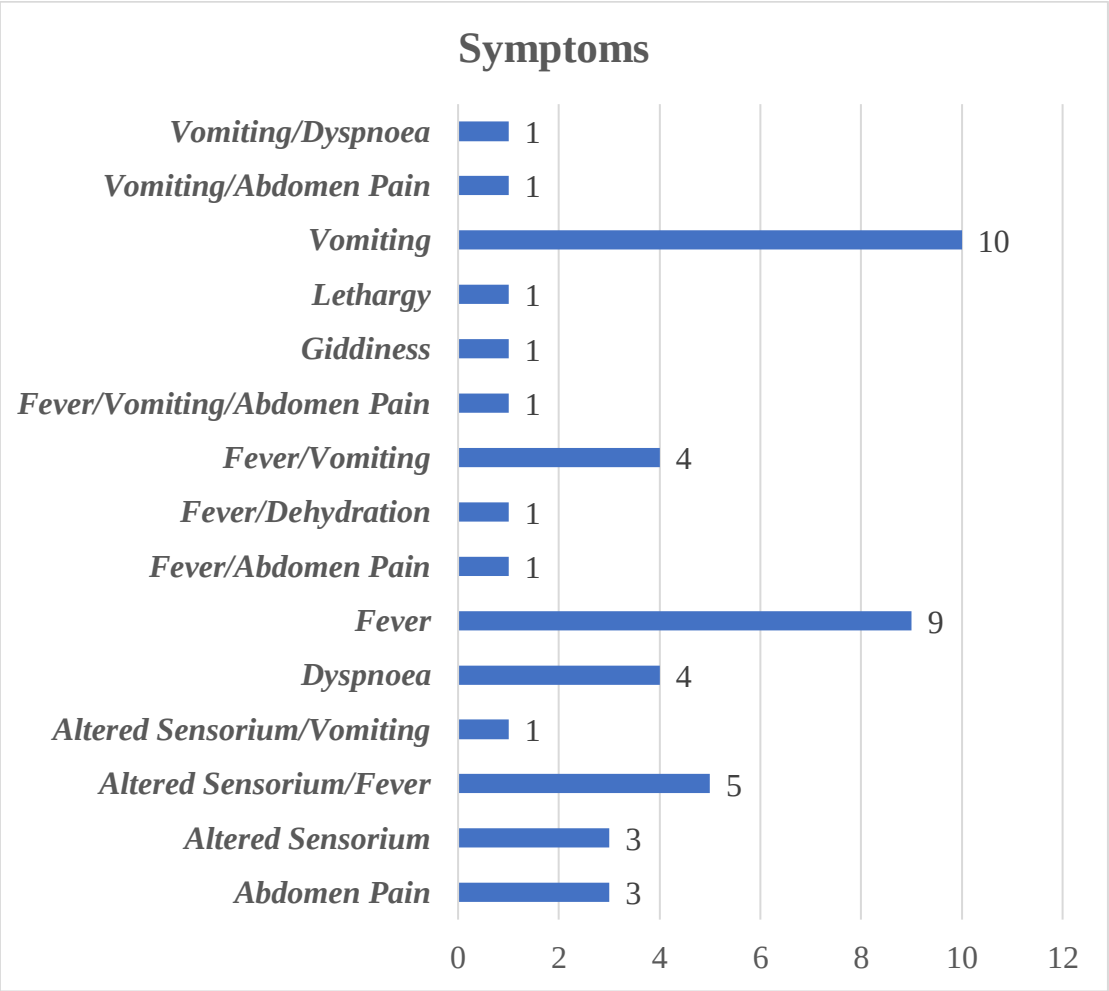
XIV. Symptoms

Among the subjects, 10 (21.74%) had Vomiting, 9 (19.57%) had Fever And 5 (10.87%) had Altered Sensorium/Fever

Table 18. Symptoms

Symptoms	Frequency	Percent
Abdomen Pain	3	6.52
Altered Sensorium	3	6.52
Altered Sensorium/Fever	5	10.87
Altered Sensorium/Vomiting	1	2.17
Dyspnoea	4	8.70
Fever	9	19.57
Fever/Abdomen Pain	1	2.17
Fever/Dehydration	1	2.17
Fever/Vomiting	4	8.70
Fever/Vomiting/Abdomen Pain	1	2.17
Giddiness	1	2.17
Lethargy	1	2.17
Vomiting	10	21.74
Vomiting/Abdomen Pain	1	2.17
Vomiting/Dyspnoea	1	2.17
Total	46	100.00

Figure 19. Symptoms



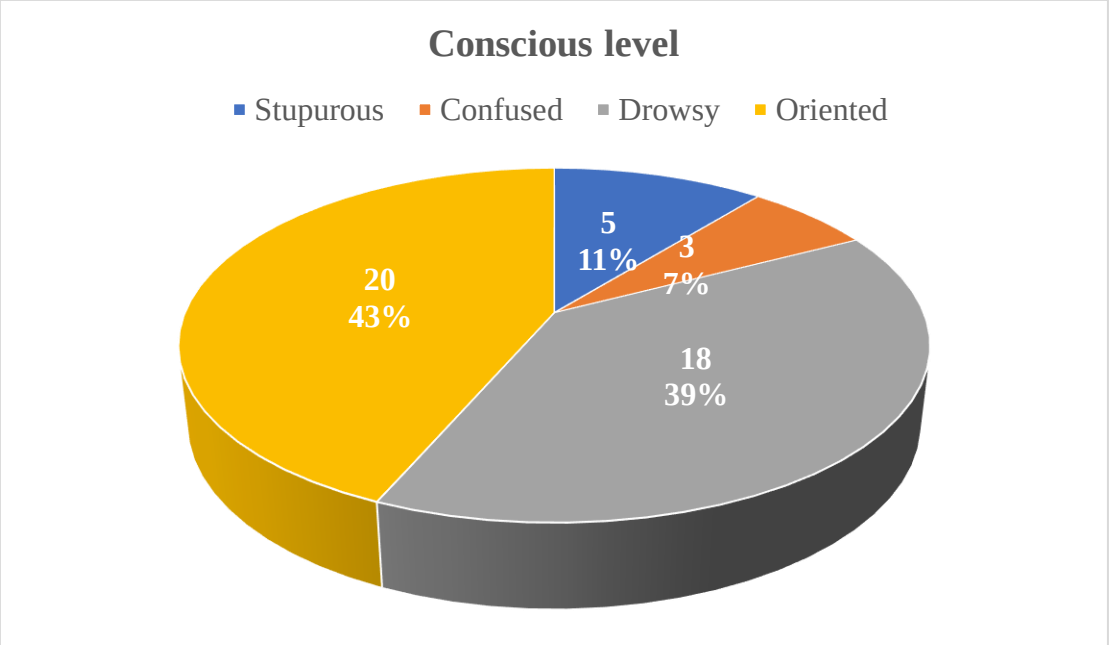
XV. Consciousness level

Among the subjects, 20 (43.48%) were Oriented, 18 (39.13%) were Drowsy and 5 (10.87%) were Stuporous.

Table 19. Conscious level

Consciousness level	Frequency	Percent
Stuporous	5	10.87
Confused	3	6.52
Drowsy	18	39.13
Oriented	20	43.48
Total	46	100.00

Figure 20. Conscious level



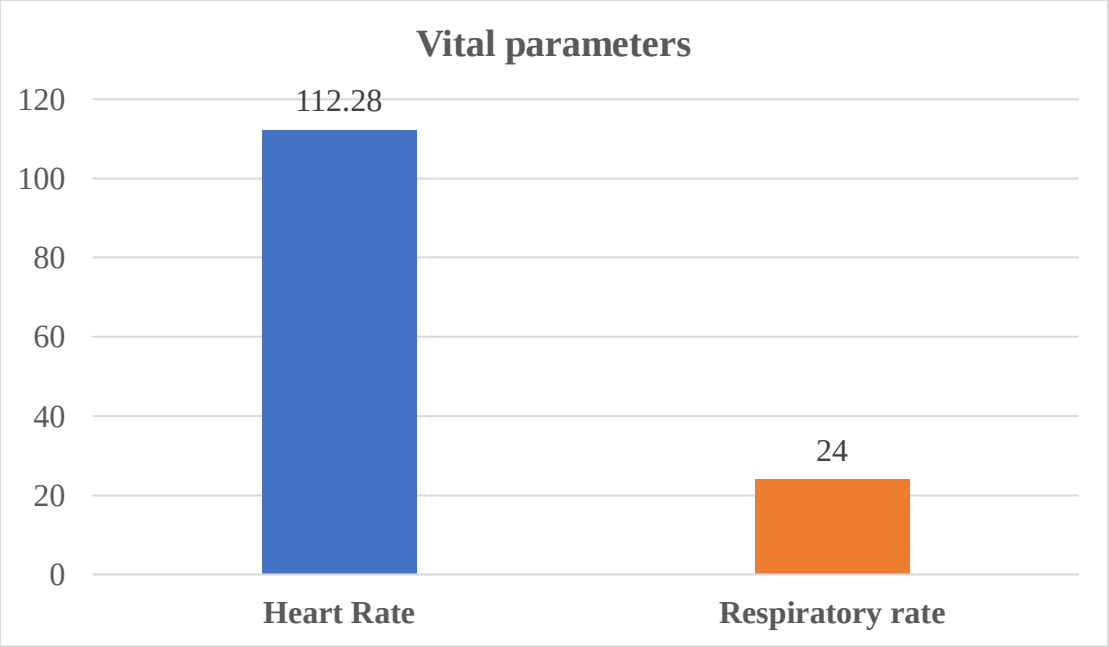
XVI. Heart Rate & Respiratory rate

The mean Heart Rate among the subjects was 112.28 (± 12.35) ranging from 74 to 133. The mean Respiratory rate among the subjects was 24 (± 3.2) ranging from 18 to 32.

Table 20. Heart Rate & Respiratory rate

	N	Mean	Std. Deviation	Minimum	Maximum
Heart Rate	46	112.28	12.35	74.0	133.0
Respiratory rate	46	24.00	3.20	18.0	32.0

Figure 21. Heart Rate & Respiratory rate



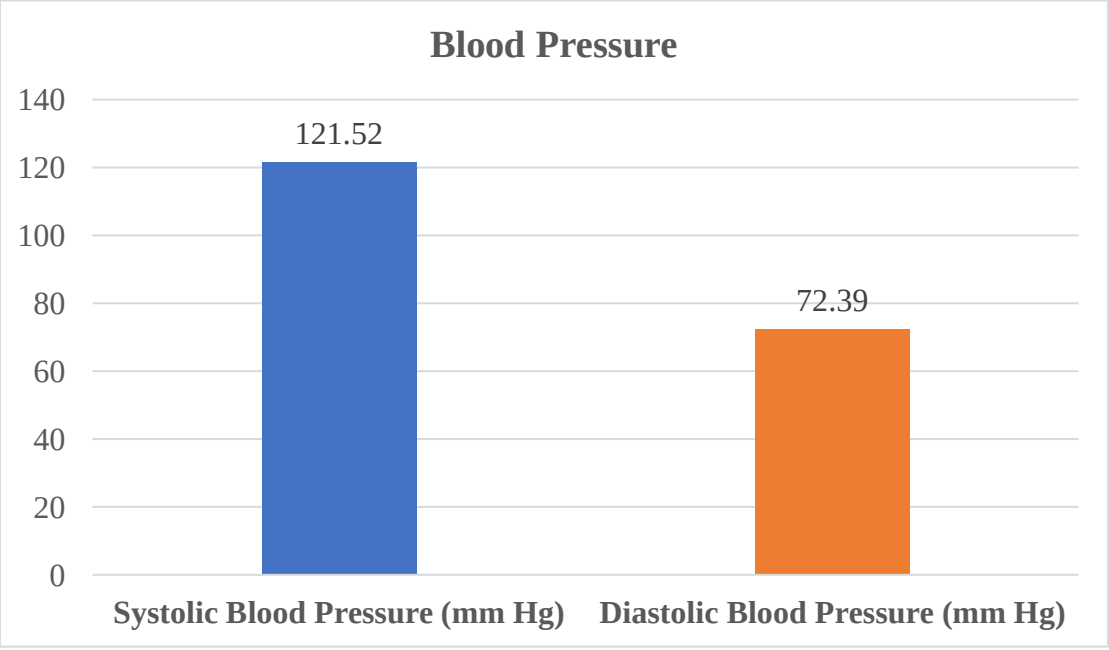
XVII. Blood Pressure

The mean Systolic Blood Pressure among the subjects was 121.52 (± 15.91) ranging from 100 to 150 mm Hg. The mean Diastolic Blood Pressure among the subjects was 72.39 (± 7.05) ranging from 60 to 90 mm Hg.

Table 21. Blood Pressure

	N	Mean	Std. Deviation	Minimum	Maximum
Systolic Blood Pressure (mm Hg)	46	121.52	15.91	100.0	150.0
Diastolic Blood Pressure (mm Hg)	46	72.39	7.05	60.0	90.0

Figure 22. Blood Pressure



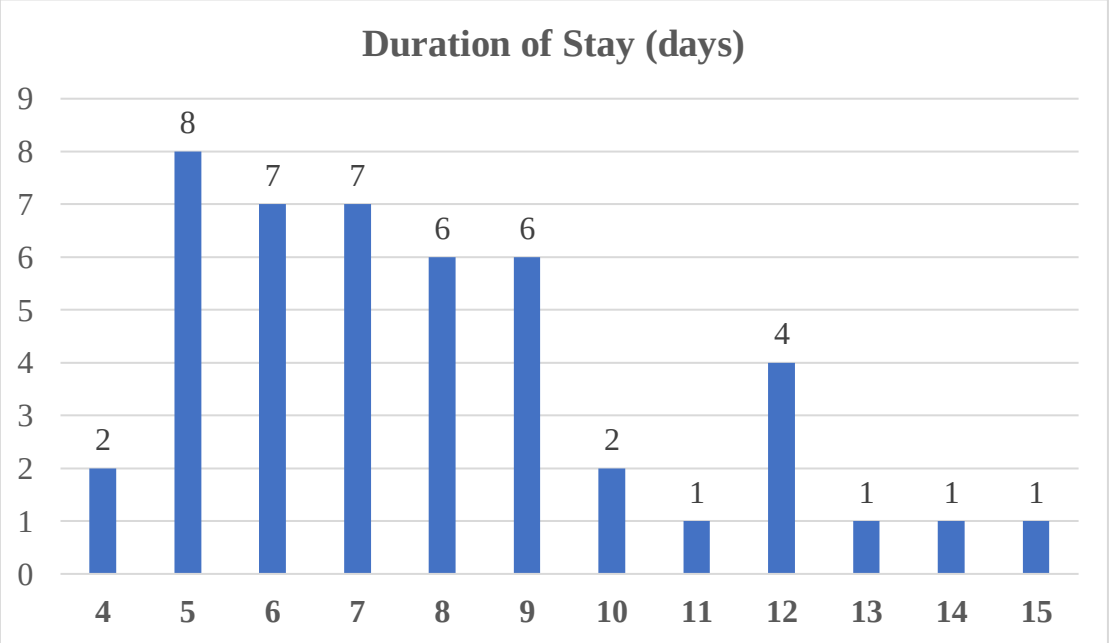
XVIII. Duration of Stay (days)

The mean Duration of Stay (days) among the subjects was 7.87 (± 2.73) ranging from 4 to 15 days.

Table 22. Duration of Stay (days)

Duration of Stay (days)	
Mean	7.87
Median	7
Std. Deviation	2.73
Range	11
Minimum	4
Maximum	15

Figure 23. Duration of Stay (days)



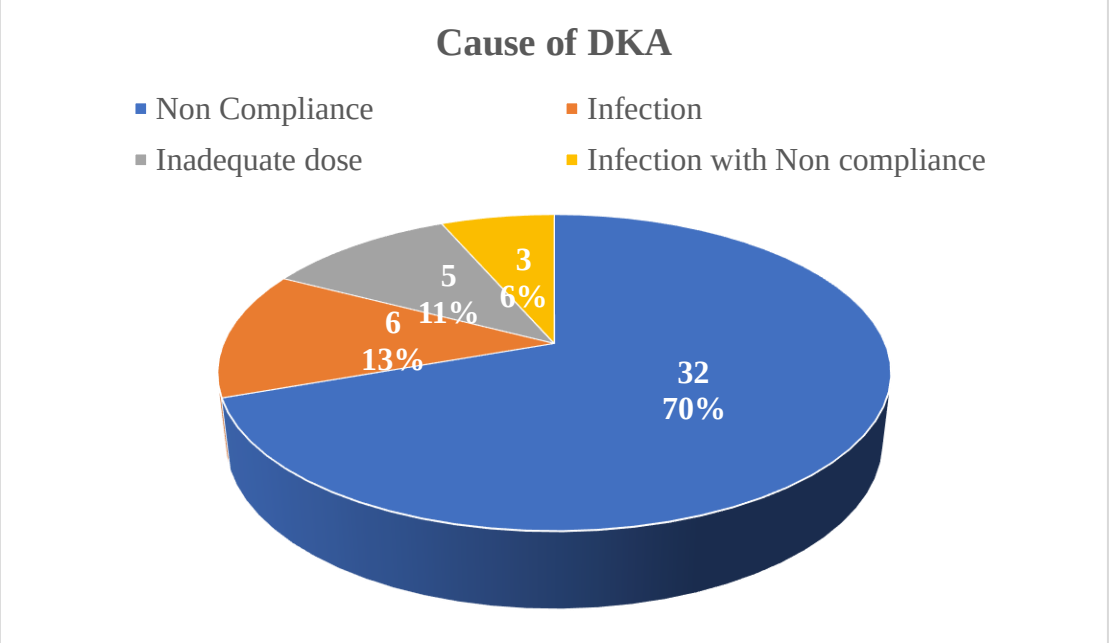
XIX. Cause of DKA

Among the subjects, 32 (69.57%) had due to Non Compliance, 6 (13.04%) had due to Infection and 5 (10.87%) had due to Inadequate dose.

Table 23. Cause of DKA

Cause of DKA	Frequency	Percent
Non-Compliance	32	69.57
Infection	6	13.04
Inadequate dose	5	10.87
Infection with Noncompliance	3	6.52
Total	46	100.00

Figure 24. Cause of DKA



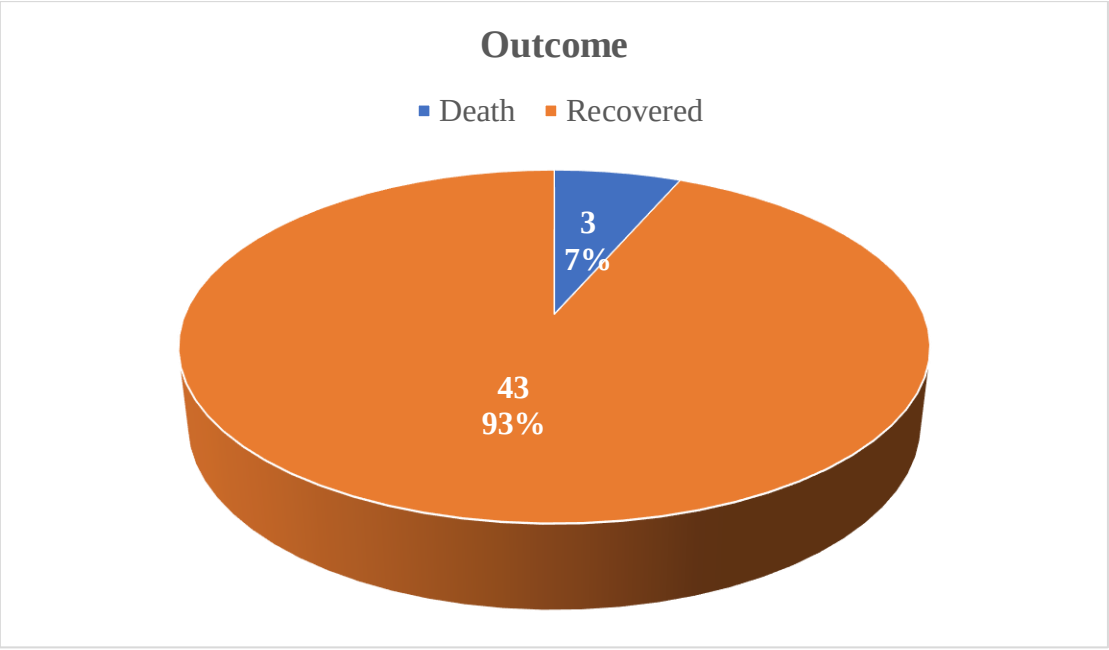
XX. Outcome

Among the subjects, 43 (93.48%) had Recovered and 3 (6.52%) had Death

Table 24. Outcome

Outcome	Frequency	Percent
Death	3	6.52
Recovered	43	93.48
Total	46	100.00

Figure 25. Outcome



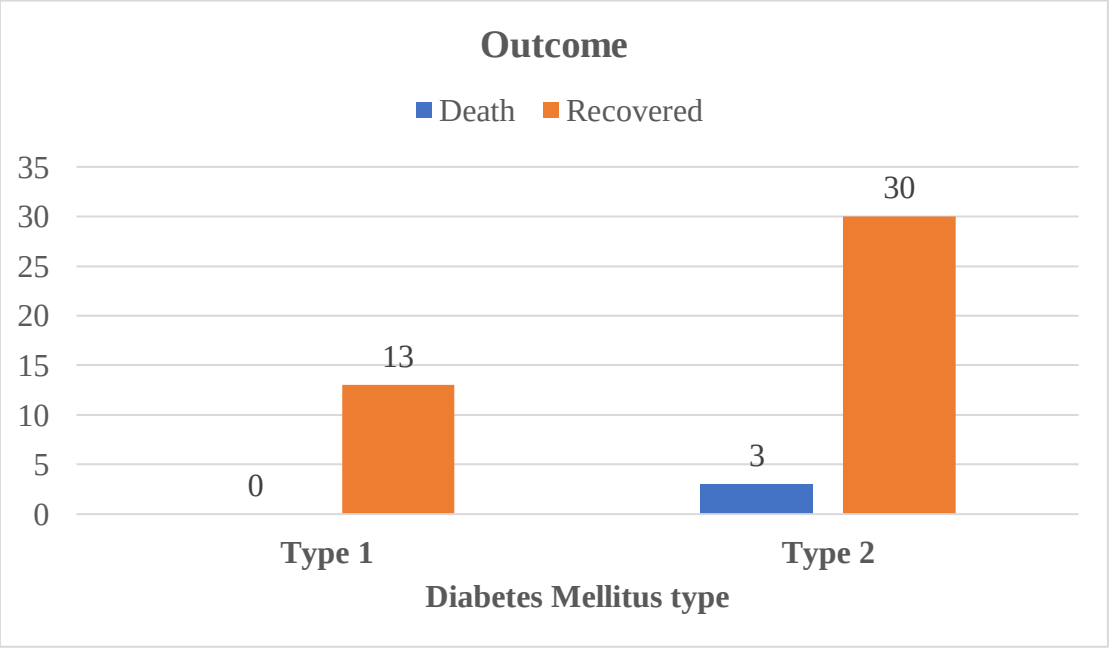
XXI. Comparison of Diabetes Mellitus type with the Outcome

Comparing the Diabetes Mellitus type with Outcome distribution, 0% of the subjects with Type 1 Diabetes Mellitus had death which is lower compared to subjects with Type 2 Diabetes Mellitus of whom 9.09% had death and the difference was not statistically significant ($p > 0.05$).

Table 25. Comparison of Diabetes Mellitus type with the Outcome

Diabetes Mellitus type	Outcome		Total	Fisher exact p value
	Death	Recovered		
Type 1	0 (0%)	13 (100%)	13 (100%)	0.36
Type 2	3 (9.09%)	30 (90.9%)	33 (100%)	
Total	3 (6.52%)	43 (93.47%)	46 (100%)	

Figure 26. Comparison of Diabetes Mellitus type with the Outcome



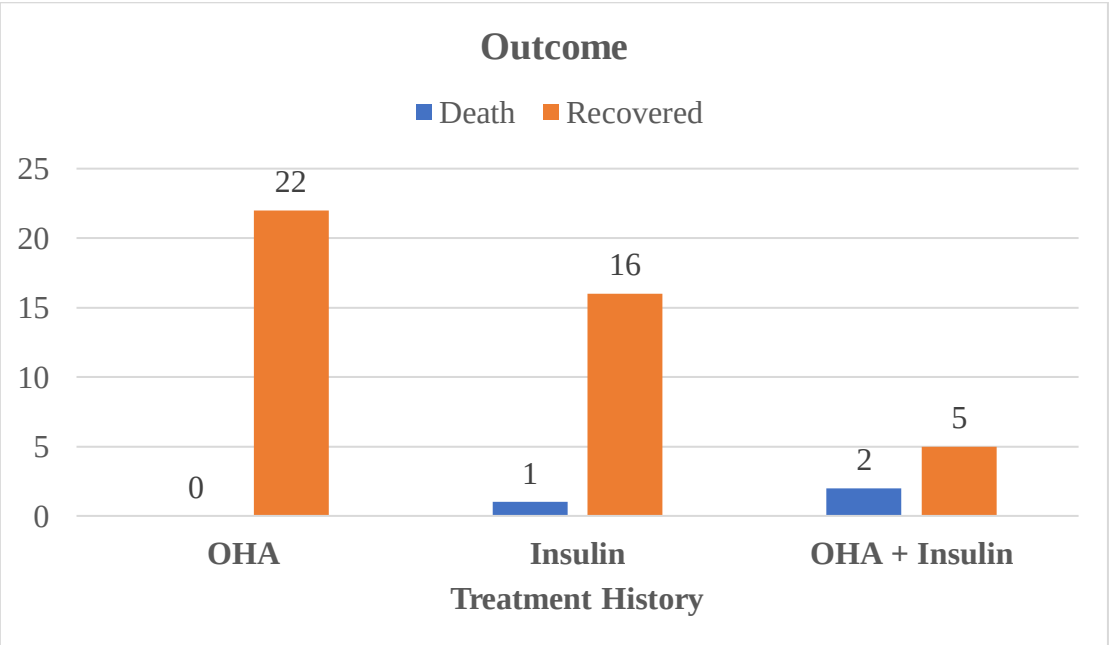
XXII. Comparison of Treatment History with the Outcome

Comparing the Treatment History with Outcome distribution, OHA + Insulin had higher proportion of death with 28.57% followed by Insulin with 5.88% and least in OHA with 0%. The difference in Outcome distribution between different Treatment History was statistically significant ($p < 0.05$)

Table 26. Comparison of Treatment History with the Outcome

Treatment History	Outcome		Total	Fisher exact p value
	Death	Recovered		
OHA	0 (0%)	22 (100%)	22 (100%)	0.024
Insulin	1 (5.88%)	16 (94.11%)	17 (100%)	
OHA + Insulin	2 (28.57%)	5 (71.42%)	7 (100%)	
Total	3 (6.52%)	43 (93.47%)	46 (100%)	

Figure 27. Comparison of Treatment History with the Outcome



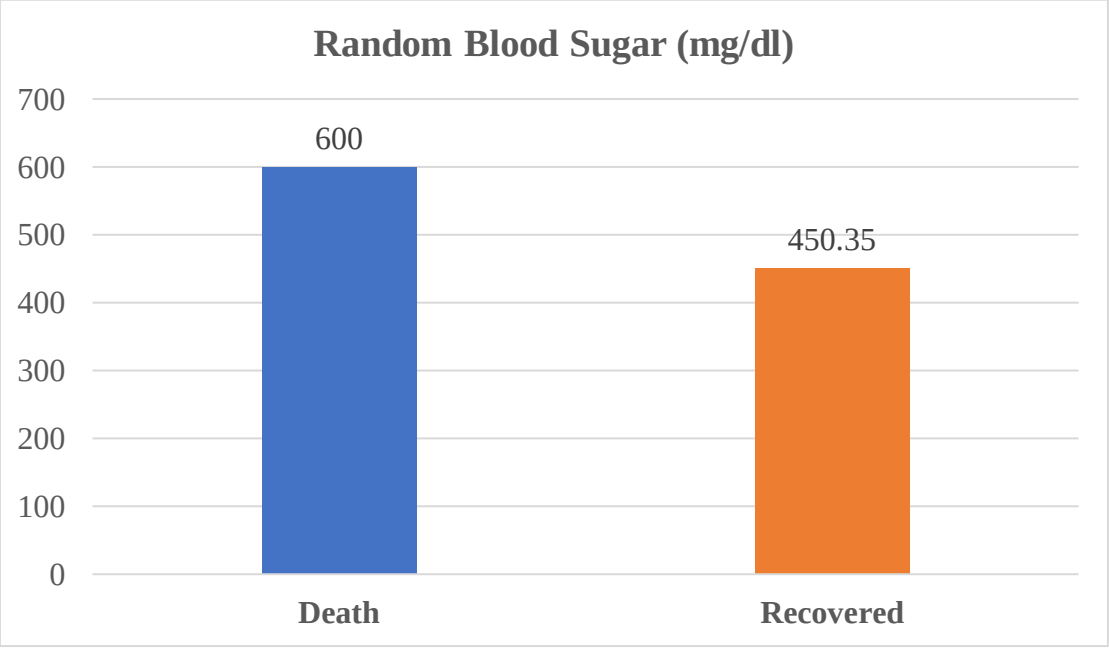
XXIII. Random Blood Sugar (mg/dl) with Outcome

The mean Random Blood Sugar (mg/dl) among Death was 600 (± 0) which is higher by 149.65 and statistically significant compared to 450.35 (± 68.02) in Recovered.

Table 27. Random Blood Sugar (mg/dl) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Random Blood Sugar (mg/dl)	**Death**	3	600.00	0.00	149.651	0.001
	Recovered	43	450.35	68.02		

Figure 28. Random Blood Sugar (mg/dl) with Outcome



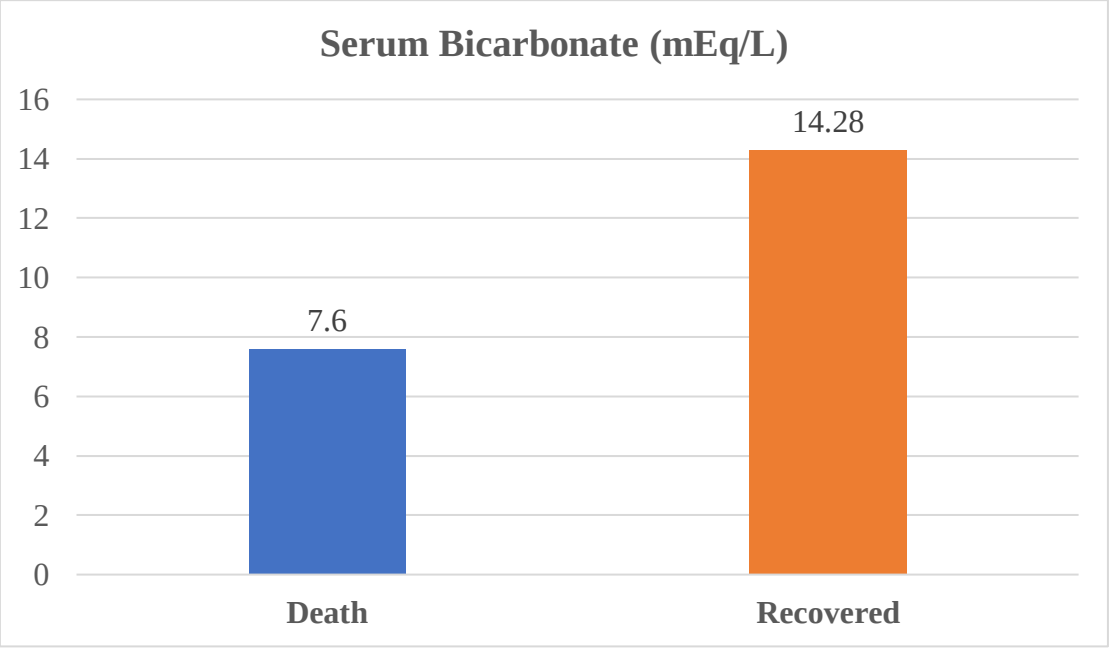
XXIV. Serum Bicarbonate (mEq/L) with Outcome

The mean Serum Bicarbonate (mEq/L) among Death was 7.6 (± 0.53) which is lower by 6.68 and statistically significant compared to 14.28 (± 3.22) in Recovered

Table 28. Serum Bicarbonate (mEq/L) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Serum Bicarbonate (mEq/L)	**Death**	3	7.60	0.53	6.679	0.001
	Recovered	43	14.28	3.22		

Figure 29. Serum Bicarbonate (mEq/L) with Outcome



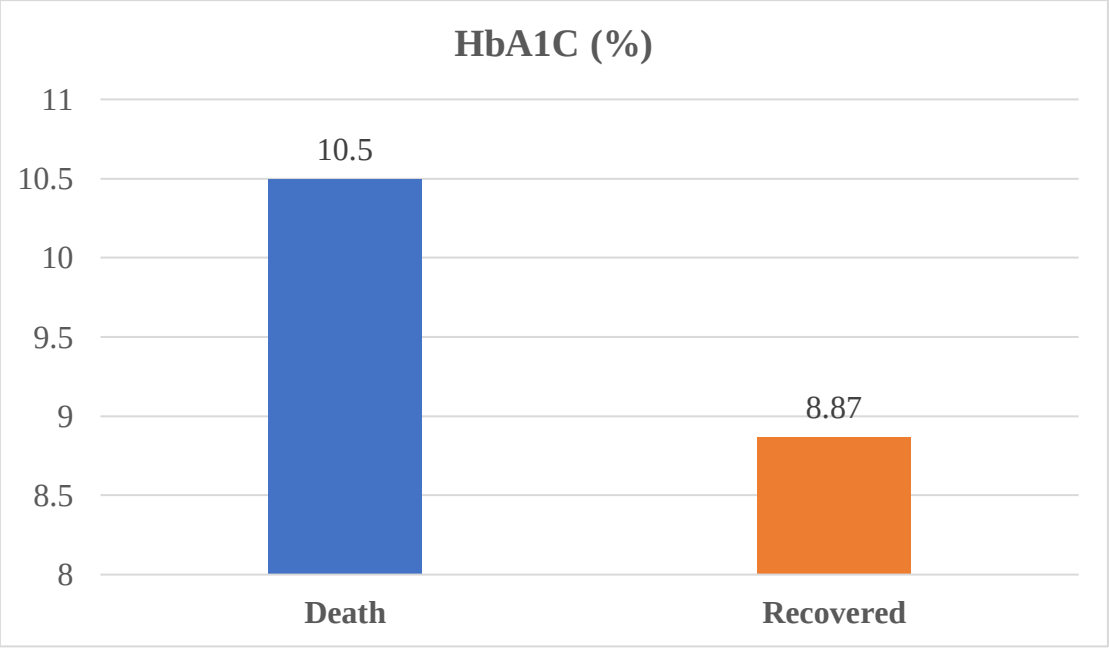
XXV. HbA1C (%) with Outcome

The mean HbA1C (%) among Death was 10.5 (± 1.31) which is higher by 1.63 and statistically significant compared to 8.87 (± 0.98) in Recovered

Table 29. HbA1C (%) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
HbA1C (%)	**Death**	3	10.50	1.31	1.628	0.009
	Recovered	43	8.87	0.98		

Figure 30. HbA1C (%) with Outcome



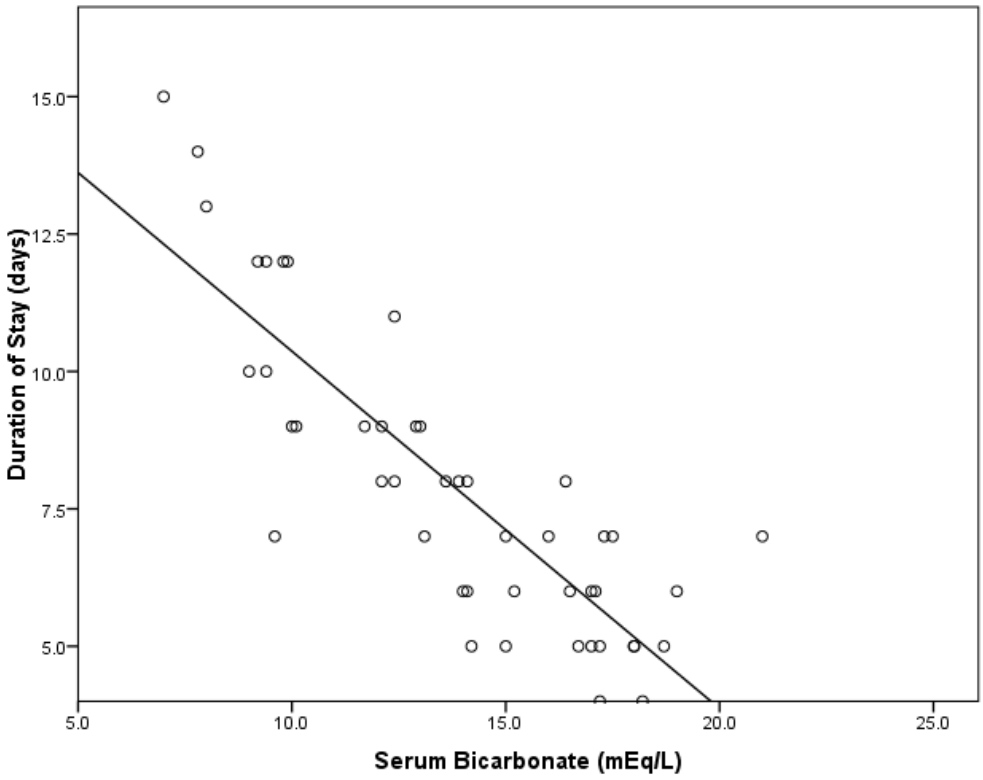
XXVI. Correlation between Serum Bicarbonate (mEq/L) and Duration of Stay (days)

Serum Bicarbonate (mEq/L) has a negative correlation with Duration of Stay (days) with a correlation coefficient of -0.84. Duration of Stay (days) decreases by -0.65 times for each unit increase in Serum Bicarbonate (mEq/L). The correlation between Duration of Stay (days) and Serum Bicarbonate (mEq/L) was statistically significant.

Table 30. Correlation between Serum Bicarbonate (mEq/L) and Duration of Stay (days)

Predictor for Duration of Stay (days)	Pearson Correlation coefficient "r"	B (95% C.I.)	p value
Serum Bicarbonate (mEq/L)	-0.842	-0.65 (-0.78 to -0.52)	0.001

Figure 31. Correlation between Serum Bicarbonate (mEq/L) and Duration of Stay (days)



8 DISCUSSION

The main aim of the study is to assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects.

In the study the mean Age (years) among the subjects was 47.87 (± 17.15) years ranging from 17 to 85 years. Among the subjects, 28 (60.87%) were Males and 18 (39.13%) were Females.

Eledrisi et al in 2022 showed that 59% were males and 41% were females with a mean age of 46.46 years.(37) Ooi et al in 2021 showed that the median age was 38.2 years and the male to female ratio was 1:1.04..(38) Nunes et al in 2021 showed that the median age of DKA subjects was 28 years with a interquartile range of 20-44 and males were 44% in the study.(40)Yotsapon Thewjitcharoen et al in 2019 showed that females were 61.5% in the study and mean age of the population was 47.4 (20.4) years.(41) Sonwani, Arya and Saxena in 2018 showed that mean (SD) age of DKA subjects was 56.10(10.40) years and 72% were males.(42) Kamata et al in 2017 showed that 47.26% were males and the mean age was about 48 years**.**(44) Xu et al in 2016 showed that 52.25% were males and the mean age was about 48.4 years..(45) Seth, Kaur and Maneet Kaur in 2015 showed that 34 (56.66%) were males and 26 (43.33%) were females with a male: female ratio was 1.3:1 and mean age was 51.46 years.(46)Maskey et al in 2015 showed that 1:1.25 was male: female ratio and 48.2 years was the mean age.(47) Rao et al in..(51) Rao, Pradhan, Mallikarjuna and Reddy in 2012 showed that The female to male ratio is 1: 1.7 with a mean age of 43.1 years (49) Bedaso et al showed that 26.7 % subjects were between 25-34 years with 64.1% were males.(52) The variation in study results may be due to the difference in study setting, admission into hospitals and disparity in the study selection criteria.

In our study among the subjects, 33 (71.74%) had Type 2 and 13 (28.26%) had Type 1 Diabetes

Table 31: Comparison of results with other studies.

	Type 1	Type 2
In our study	28.26%	71.4%
Bedaso et al (52)	58.46%	41.54%
Edo et al(53)	11.9%	88.1%
Seth et al(46)	20%	80%
Maskey et al(47)	43.75%	56.25%
Xu et al(45)	47.64%	36%
Thewjitcharoen et al(41)	41.5%	50%
Nunes et al(40)	65.4%	19.%

In the study the mean Serum Bicarbonate (mEq/L) among the subjects was 13.84 (± 3.53) ranging from 7 to 21 mEq/L. The mean HbA1C (%) among the subjects was 8.98 (± 1.07) ranging from 6.7 to 11.4. Among the subjects, 10 (21.74%) had Vomiting, 9 (19.57%) had Fever And 5 (10.87%) had Altered Sensorium/Fever. Among the subjects, 32 (69.57%) had due to Non-Compliance, 6 (13.04%) had due to Infection and 5 (10.87%) had due to Inadequate dose. Among the subjects, 43 (93.48%) had Recovered and 3 (6.52%) had Death. Comparing the Treatment History with Outcome distribution, OHA + Insulin had significantly higher proportion of death with 28.57% followed by Insulin with 5.88% and least in OHA with 0%. The mean Random Blood Sugar (mg/dl) among subjects with Death was significantly higher 600 (± 0) vs. 450.35 (± 68.02). The mean Serum Bicarbonate

(mEq/L) among subjects with Death outcome was significantly lower than recovered outcome subjects was 7.6 (± 0.53) vs.14.28 (± 3.22) .The mean HbA1C (%) among Death was 10.5 (± 1.31) which is higher by 1.63 and statistically significant compared to 8.87 (± 0.98) in Recovered. Serum Bicarbonate (mEq/L) has a significantly negative correlation with Duration of Stay (days) with a correlation coefficient of -0.84. Duration of Stay (days) decreases by -0.65 times for each unit increase in Serum Bicarbonate (mEq/L).

Eledrisi et al in 2022 showed that patients with type 2 diabetes were older than those with type1 diabetes [48.0 (38.0-60.0), 26.0 (21.0-31.0) years]. In patients with type 2 diabetes compared to type 1 diabetes, admission to the critical care unit was greater (38.9 % vs. 26.6 %), with a longer hospital stay [5 (2.0-9.0) vs. 2 (2.0-4.0) days, and noticeably higher mortality (7.4 % vs. 1 %).(37) Ooi et al in 2021 showed that compared to those with type 1 diabetes, those with type 2 diabetes were older and more likely to be members of ethnic minorities. In both groups, concurrent sickness (39.8%) and poor compliance (26.8%) were the two most frequent triggering causes of DKA. Total insulin requirements were the same for both types of diabetes (13.9 units for type 1 diabetes (9.1-21.9) and 13.9 units for type 2 diabetes (7.7-21.1)). However, those with type 2 diabetes required a considerably longer hospital stay (11.0 days (5.0-23.1) vs. 3.0 days (1.7-6.1); type 1 diabetes).(38)

Hare et al in 2021 showed that the most frequent causes of presentation were lack of insulin (54%), infection (31%), excessive alcohol use (26%) and new diabetes diagnosis (16%). People with recurring DKA were more likely to smoke (69% vs 27%, P = 0.003), be unemployed (31% vs 11%, P = 0.04), and use illicit drugs (44% vs 17%, P = 0.02) than those with single admissions.(39) Nunes et al in 2021 showed that the most frequent causes of diabetic ketoacidosis were infection and noncompliance with prescribed treatment. The

death rate for the immediate, first six months, the first year, and the second year was 5.8%, 9.6%, 13.5%, and 19.2%, respectively. Age, type 2 diabetes, hypoalbuminemia, infection at presentation, and a higher sequential organ failure assessment score upon admission were all related with death occurring within two years.(40)

Yotsapon Thewjitcharoen et al in 2019 showed that Infection was frequently the cause of T2DM precipitation, whereas insulin omission was typically the cause of T1DM precipitation. During continuous treatment, 26.6% of patients experienced hypokalaemia, and in this group of patients, supplementation was not recommended as directed. In our study, the mortality rate was 4.3% and was caused by four T2DM patients dying from DKA, which was the precipitating reason.(41)Sonwani, Arya and Saxena in 2018 showed that out of 100 DKA patients, 86% were discharged, 7% died, and 7% dropped out of the trial. In DKA patients, hypertension (60%) obesity (47%), nausea and vomiting (86%), stomach pain (58%), poor compliance (53%) and infection, particularly pneumonia (24%), were the most prevalent co-morbidities, clinical symptoms, and precipitating factors. Most of the patients who died had diabetes for more than ten years (23.1%), had poor compliance (10.2%), had Cardiovascular Disease (40%), and had Acute Coronary Syndrome (40%).(42)

Prajapati in 2017 showed that patients with type 1 diabetes mellitus were more likely to appear with polyuria and polydipsia (p=0.002), whereas type 2 diabetes mellitus patients were more likely to experience fever (p=0.03). Most individuals showed normal levels of serum sodium and potassium. Over one third of patients had high serum creatinine levels, while 42% of patients had high serum urea levels. Omission of insulin was the most frequent

cause of diabetic ketoacidosis in people with type 1 diabetes mellitus, whereas in people with type 2 diabetes, it was infection.(43)

Kamata et al in 2017 showed that patients with type 2 diabetes had higher mean plasma glucose levels than those with type 1 diabetes (48.4(21.6) vs. 37.1(16.4)mmol/l) as well as higher serum levels of haemoglobin, blood urea nitrogen, and creatinine, all of which returned to normal once DKA was resolved. Patients with type 2 diabetes needed significantly more daily total insulin (35.9(37.0)U vs. 20.2(23.3)U), more replacement fluid (4.17(2.69)L vs. 2.29(1.57)L), and more potassium supplementation (23.9(36.5)mEq vs. 11.2(17.9)mEq) than patients with type 1 diabetes to resolve DKA.(44) Xu et al in 2016 showed that patients with known diabetes accounted for 388 (60.3%) of the 388 diabetic ketoacidosis incidents. Infection (40.1%), unidentified causes (36.9%), and non-compliance with anti-diabetes therapy (16.8%) were the most frequent triggering factors. And the death rate was 1.7% and was significantly greater among Type 2 diabetes than in Type 1 diabetes (3.2% vs. 0.4%, P <0.01).(45)

Seth, Kaur and Maneet Kaur in 2015 showed that Six of the 12 Type 1 diabetic patients who presented with diabetic ketoacidosis complications had just received their diabetes diagnosis. The most frequent symptoms among these patients were nausea and vomiting (63.33%). The most frequent contributing factor to diabetic ketoacidosis was an infection (73.33%). 10% mortality was observed.(46)Maskey et al in 2015 showed that the type 2 participants had substantially higher PH levels (7.11 s 7.28) and were older (56.8 s 25.7 years) than the 16 subjects with type 1 DM. The mean BMI for both type 1 and type 2 DM was 20.5(2.44). The most frequent precipitating factor was infection, which was followed by poor drug compliance (37.5%) and the first presentation (6.25%).(47)

Rao, Pradhan, Mallikarjuna and Reddy in 2012 showed that 60% of the admissions, precipitating circumstances were present. 50% of patients exhibited non-compliance or pharmacological therapy cessation. Average hospital stays in type 1 DM were somewhat longer than those in type 2 DM patients.(49)

Most of the findings in our study is similar to other studies. The disparities between the studies are based on the geographical distribution and selection criteria among the subjects.

9 STRENGTHS

- The inclusion of both type 1 and type 2 diabetes mellitus
- Prospective evaluation to assess the outcome of the study

10 LIMITATIONS

The main aim of the study is to assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects.

The limitations are

- Single centre study
- Classification of diabetes mellitus was not verified
- Non compliance was not assessed properly by scales
- Other factors to contribute like socioeconomic status was not analysed
- The presence of any associate comorbidity was not analysed

11 RECOMMENDATIONS

The main aim of the study is to assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects.

The recommendations are

- Further studies to assess the missing cases in the area
- Recurrent admissions should be assessed and intervention strategies to address should be formulated
- Proper profile- including time of development of DKA, any delay in admission to the centre and the various modalities intervened should be assessed.
- Comparison of paediatric and adult DKA

12 SUMMARY OF RESULTS

- The mean Age (years) among the subjects was 47.87 (± 17.15) years ranging from 17 to 85 years.
- Among the subjects, 20 (43.48%) were in 41 - 60 years, 16 (34.78%) were in < 40 years and 10 (21.74%) were in > 60 years
- Among the subjects, 28 (60.87%) were Males and 18 (39.13%) were Females
- Among the subjects, 33 (71.74%) had Type 2 and 13 (28.26%) had Type 1 Diabetes.
- Among the subjects, 9 (19.57%) had Family History
- Among the subjects, 22 (47.83%) had OHA, 17 (36.96%) had Insulin and 7 (15.22%) had OHA + Insulin
- The mean Random Blood Sugar (mg/dl) among the subjects was 460.11 (± 75.59) ranging from 281 to 600
- All the subjects, 46 (100%) had Plasma acetone
- The mean Serum Bicarbonate (mEq/L) among the subjects was 13.84 (± 3.53) ranging from 7 to 21 mEq/L.
- The mean HbA1C (%) among the subjects was 8.98 (± 1.07) ranging from 6.7 to 11.4
- The mean Hemoglobin (mg/dl) among the subjects was 11.64 (± 2.4) ranging from 8 to 16.2
- The mean Serum Urea (mg/dl) among the subjects was 49.96 (± 12.61) ranging from 30 to 84 mg/dl. The mean Serum Creatinine (mg/dl) among the subjects was 1.43 (± 0.42) ranging from 0.7 to 2.2 mg/dl.

- The mean Serum Sodium (mEq/L) among the subjects was 136.39 (± 6.61) ranging from 121 to 150 mEq/L. The mean Serum Potassium (mEq/L) among the subjects was 3.82 (± 0.73) ranging from 2.7 to 5.4 mEq/L.
- Among the subjects, 10 (21.74%) had Vomiting, 9 (19.57%) had Fever And 5 (10.87%) had Altered Sensorium/Fever
- Among the subjects, 20 (43.48%) were Oriented, 18 (39.13%) were Drowsy and 5 (10.87%) were Stuporous
- The mean Heart Rate among the subjects was 112.28 (± 12.35) ranging from 74 to 133. The mean Respiratory rate among the subjects was 24 (± 3.2) ranging from 18 to 32.
- The mean Systolic Blood Pressure among the subjects was 121.52 (± 15.91) ranging from 100 to 150 mm Hg. The mean Diastolic Blood Pressure among the subjects was 72.39 (± 7.05) ranging from 60 to 90 mm Hg.
- The mean Duration of Stay (days) among the subjects was 7.87 (± 2.73) ranging from 4 to 15 days
- Among the subjects, 32 (69.57%) had due to Non Compliance, 6 (13.04%) had due to Infection and 5 (10.87%) had due to Inadequate dose.
- Among the subjects, 43 (93.48%) had Recovered and 3 (6.52%) had Death
- Comparing the Diabetes Mellitus type with Outcome distribution, 0% of the subjects with Type 1 Diabetes Mellitus had death which is lower compared to subjects with Type 2 Diabetes Mellitus of whom 9.09% had death and the difference was not statistically significant ($p > 0.05$).
- Comparing the Treatment History with Outcome distribution, OHA + Insulin had higher proportion of death with 28.57% followed by Insulin with 5.88% and least in

OHA with 0%. The difference in Outcome distribution between different Treatment History was statistically significant ($p < 0.05$)

- The mean Random Blood Sugar (mg/dl) among Death was 600 (± 0) which is higher by 149.65 and statistically significant compared to 450.35 (± 68.02) in Recovered.
- The mean Serum Bicarbonate (mEq/L) among Death was 7.6 (± 0.53) which is lower by 6.68 and statistically significant compared to 14.28 (± 3.22) in Recovered
- The mean HbA1C (%) among Death was 10.5 (± 1.31) which is higher by 1.63 and statistically significant compared to 8.87 (± 0.98) in Recovered
- Serum Bicarbonate (mEq/L) has a negative correlation with Duration of Stay (days) with a correlation coefficient of -0.84. Duration of Stay (days) decreases by -0.65 times for each unit increase in Serum Bicarbonate (mEq/L). The correlation between Duration of Stay (days) and Serum Bicarbonate (mEq/L) was statistically significant.

13 CONCLUSION

The study was a prospective study conducted among 46 subjects with an aim to assess the clinical profile, precipitating factors, outcome and correlation of bicarbonate level among DKA subjects. The subjects with diabetic ketoacidosis were included in the study. After selecting the subjects the clinical profile including age, Sex, Total stay in Hospital, family history and treatment history was assessed. Clinical findings including level of consciousness, heart rate, respiratory rate, blood pressure, random blood sugar, renal function test, bicarbonate, plasma/urine acetone was assessed.

Among the subjects, 43 (93.48%) had Recovered and 3 (6.52%) had Death. Comparing the Treatment History with Outcome distribution, OHA + Insulin had significantly higher proportion of death with 28.57% followed by Insulin with 5.88% and least in OHA with 0%. The mean Random Blood Sugar (mg/dl) among subjects with Death was significantly higher 600 (± 0) vs. 450.35 (± 68.02). The mean Serum Bicarbonate (mEq/L) among subjects with Death outcome was significantly lower than recovered outcome subjects was 7.6 (± 0.53) vs.14.28 (± 3.22) .The mean HbA1C (%) among Death was 10.5 (± 1.31) which is higher by 1.63 and statistically significant compared to 8.87 (± 0.98) in Recovered. Serum Bicarbonate (mEq/L) has a significantly negative correlation with Duration of Stay (days) with a correlation coefficient of -0.84. Duration of Stay (days) decreases by -0.65 times for each unit increase in Serum Bicarbonate (mEq/L).

14 REFERENCES

1. AC P. Harrison's Principles of Internal Medicine, 20e | AccessMedicine | McGraw-Hill Medical. McGraw-Hill. 2018. 2968–9 p.

2. Nagarathna R, Bali P, Anand A, Srivastava V, Patil S, Sharma G, et al. Prevalence of Diabetes and Its Determinants in the Young Adults Indian Population-Call for Yoga Intervention. Front Endocrinol (Lausanne) [Internet]. 2020;11:846. Available from: https://www.frontiersin.org/article/10.3389/fendo.2020.507064

3. Vijayakumar G, Manghat S, Vijayakumar R, Simon L, Scaria LM, Vijayakumar A, et al. No Title. BMC Public Health [Internet]. 2019 Jan 31 [cited 2020 Oct 23];19(1):140. Available from: https://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-019-6445-6

4. Dalfrà MG, Burlina S. Ketoacidosis. In: Frontiers in Diabetes. S. Karger AG; 2019. p. 123–31.

5. Laffel L. Ketone bodies: a review of physiology, pathophysiology and application of monitoring to diabetes. Diabetes Metab Res Rev. 1999 Nov;15(6):412–26.

6. Choi BCK, Shi F. Risk factors for diabetes mellitus by age and sex: Results of the National Population Health Survey. Diabetologia. 2001;44(10):1221–31.

7. Kitabchi AE, Wall BM. Management of Diabetic Ketoacidosis. Am Fam Physician. 1999 Aug;60(2):455–64.

8. Rawla P, Vellipuram AR, Bandaru SS, Pradeep Raj J. Euglycemic diabetic ketoacidosis: a diagnostic and therapeutic dilemma. Endocrinol Diabetes Metab

Case Reports. 2017 Sep;2017.

9. Timilsina S, Timilsina S, Mandal A, Paudel R, Gayam V. Triad of Diabetic Ketoacidosis, Hypertriglyceridemia, and Acute Pancreatitis: Severity of Acute Pancreatitis May Correlate with the Level of Hypertriglyceridemia. Cureus. 2019 Jun;11(6).

10. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009 Jul;32(7):1335–43.

11. Lizzo JM, Goyal A, Gupta V. Adult Diabetic Ketoacidosis. StatPearls [Internet]. 2022 Jul 12 [cited 2022 Dec 3]; Available from: https://www.ncbi.nlm.nih.gov/books/NBK560723/

12. Barnes AJ, Bloom SR, Goerge K, Alberti GM, Smythe P, Alford FP, et al. Ketoacidosis in pancreatectomized man. N Engl J Med. 1977 Jun;296(22):1250–3.

13. Feingold KR, Anawalt B, Boyce A, Chrousos G, de Herder WW, Dhatariya K, et al., editors. Endotext. South Dartmouth (MA); 2000.

14. Gosmanov AR, Kitabchi AE. Diabetic Ketoacidosis. Endotext [Internet]. 2018 Apr 28 [cited 2022 Dec 3]; Available from: https://www.ncbi.nlm.nih.gov/books/NBK279146/

15. Stogdale L. Definition of diabetes mellitus. Vol. 76, The Cornell veterinarian. 1986. p. 156–74.

16. Palicka V. Pathophysiology of Diabetes Mellitus. EJIFCC. 2002 Dec;13(5):140–4.

17. World Health Organization Geneva S. CLASSIFICATION OF DIABETES

MELLITUS. 2019.

18. Introduction: Standards of Medical Care in Diabetes-2018. Diabetes Care. 2018 Jan;41(Suppl 1):S1–2.

19. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2014 Jan;37 Suppl 1:S81-90.

20. Sapra A, Bhandari P. Diabetes Mellitus. StatPearls [Internet]. 2022 Jun 26 [cited 2022 Dec 3]; Available from: https://www.ncbi.nlm.nih.gov/books/NBK551501/

21. Regina CC, Mu'ti A, Fitriany E. Diabetes Mellitus Type 2. Verdure Heal Sci J [Internet]. 2022 Jun 19 [cited 2022 Dec 3];3(1):8–17. Available from: https://www.ncbi.nlm.nih.gov/books/NBK513253/

22. Rawshani A, Rawshani A, Franzén S, Eliasson B, Svensson A-M, Miftaraj M, et al. Mortality and Cardiovascular Disease in Type 1 and Type 2 Diabetes. N Engl J Med. 2017 Apr;376(15):1407–18.

23. Association AD. 9. Cardiovascular Disease and Risk Management: Standards of Medical Care in Diabetes-2018. Diabetes Care. 2018 Jan;41(Suppl 1):S86–104.

24. Association AD. 10. Microvascular Complications and Foot Care: Standards of Medical Care in Diabetes-2018. Diabetes Care. 2018 Jan;41(Suppl 1):S105–18.

25. Ma J. Pharmaceutical Diabetes : pharmacological history and future management strategies. 2018;(April).

26. Diabetic Ketoacidosis | Diabetes | CDC [Internet]. [cited 2022 Dec 3]. Available from: https://www.cdc.gov/diabetes/basics/diabetic-ketoacidosis.html

27. Suwarto S, Sutrisna B, Waspadji S, Pohan HT. Predictors of five days mortality in diabetic ketoacidosis patients: a prospective cohort study. Acta Med Indones. 2014 Jan;46(1):18–23.

28. Barski L, Nevzorov R, Rabaev E, Jotkowitz A, Harman-Boehm I, Zektser M, et al. Diabetic ketoacidosis: clinical characteristics, precipitating factors and outcomes of care. Isr Med Assoc J. 2012 May;14(5):299–303.

29. Umpierrez G, Korytkowski M. Diabetic emergencies - ketoacidosis, hyperglycaemic hyperosmolar state and hypoglycaemia. Nat Rev Endocrinol. 2016 Apr;12(4):222–32.

30. Diabetic Ketoacidosis (DKA): Symptoms, Causes, Criteria, DKA Pathophysiology and Management - Jotscroll [Internet]. [cited 2022 Dec 4]. Available from: https://www.jotscroll.com/forums/11/posts/245/diabetic-ketoacidosis-dka-criteria-dka-pathophysiology-management-sign.html

31. Koul PB. Diabetic ketoacidosis: A current appraisal of pathophysiology and management. Vol. 48, Clinical Pediatrics. SAGE PublicationsSage CA: Los Angeles, CA; 2009. p. 135–44.

32. Van den Berghe G, Kitabchi AE, Fisher JN. Hyperglycemic Crises: Diabetic Ketoacidosis (DKA) and Hyperglycemic Hyperosmolar State (HHS). In: Acute Endocrinology. Humana Press; 2008. p. 119–47.

33. Kerl ME. Diabetic Ketoacidosis: Pathophysiology and Clinical and Laboratory Presentation. Compend Contin Educ Pract Vet. 2001;23(3):220–8.

34. Kitabchi AE, Wall BM. Diabetic ketoacidosis. Med Clin North Am. 1995;79(1):9–

37.

35. Eledrisi M, Elzouki A-N. Management of diabetic ketoacidosis in adults: A narrative review. Saudi J Med Med Sci. 2020;8(3):165.

36. Ionescu-Tîrgovişte C, Imincu I. Classification of diabetic ketoacidosis. Med Interne. 1975;13(3):227–34.

37. Eledrisi MS, Alkabbani H, Aboawon M, Ali A, Alabdulrazzak I, Elhaj M, et al. Clinical characteristics and outcomes of care in patients hospitalized with diabetic ketoacidosis. Diabetes Res Clin Pract [Internet]. 2022;192:110041. Available from: https://www.sciencedirect.com/science/article/pii/S0168822722008555

38. Ooi E, Nash K, Rengarajan L, Melson E, Thomas L, Johnson A, et al. Clinical and biochemical profile of 786 sequential episodes of diabetic ketoacidosis in adults with type 1 and type 2 diabetes mellitus. BMJ open diabetes Res care. 2021 Dec;9(2).

39. Hare MJL, Deitch JM, Kang MJY, Bach LA. Clinical, psychological and demographic factors in a contemporary adult cohort with diabetic ketoacidosis and type 1 diabetes. Intern Med J [Internet]. 2021 Aug 1;51(8):1292–7. Available from: https://doi.org/10.1111/imj.14877

40. Nunes RTL, Mota CFMGP, Lins PRG, Reis FS, Resende TC de F, Barberino L de A, et al. Incidence, characteristics and long-term outcomes of patients with diabetic ketoacidosis: a prospective prognosis cohort study in an emergency department. Sao Paulo Med J. 2021;139(1):10–7.

41. Thewjitcharoen Y, Plianpan P, Chotjirat A, Nakasatien S, Chotwanvirat P,

Wanothayaroj E, et al. Clinical characteristics and outcomes of care in adult patients with diabetic ketoacidosis: A retrospective study from a tertiary diabetes center in Thailand. J Clin Transl Endocrinol [Internet]. 2019;16:100188. Available from: https://www.sciencedirect.com/science/article/pii/S2214623719300249

42. Sonwani S, Arya A, Saxena RS. A Prospective Study of Risk Factors , Clinical Profile and Outcome in Patients of Diabetic Ketoacidosis (DKA) in Type II Diabetes Patients Section : Medicine. 2018;5(4):21–4.

43. Prajapati BK. Clinical Profile of Diabetic Ketoacidosis in Adults in Dhulikhel Hospital. Kathmandu Univ Med J (KUMJ). 2017;15(57):25–8.

44. Kamata Y, Takano K, Kishihara E, Watanabe M, Ichikawa R, Shichiri M. Distinct clinical characteristics and therapeutic modalities for diabetic ketoacidosis in type 1 and type 2 diabetes mellitus. J Diabetes Complications. 2017 Feb;31(2):468–72.

45. Xu Y, Bai J, Wang G, Zhong S, Su X, Huang Z, et al. Clinical profile of diabetic ketoacidosis in tertiary hospitals in China: a multicentre, clinic-based study. Diabet Med. 2016 Feb;33(2):261–8.

46. Seth P, Kaur H, Kaur M. Clinical Profile of Diabetic Ketoacidosis: A Prospective Study in a Tertiary Care Hospital. J Clin Diagn Res. 2015 Jun;9(6):OC01-4.

47. Maskey R, Shakya DR, Nikesh B, Krishna KA, Lavaju P, Kattel V, et al. Clinical profile of diabetic ketoacidosis in tertiary care hospital of Eastern Nepal. Indian J Endocrinol Metab. 2015;19(5):673–5.

48. Azevedo LCP, Choi H, Simmonds K, Davidow J, Bagshaw SM. Incidence and long-term outcomes of critically ill adult patients with moderate-to-severe diabetic

ketoacidosis: retrospective matched cohort study. J Crit Care. 2014 Dec;29(6):971–7.

49. Rao VD, Pradhan B, Mallikarjuna Y, Reddy R. Clinical profile of diabetic ketoacidosis in adults. Heal Renaiss. 2012;10(2):80–6.

50. Sathik Basha K. A Study on Clinical and Biochemical Profile in Diabetic Ketoacidosis. 2013.

51. Rao VD, Pradhan B, Mallikarjuna Y, Reddy R, V1 DR, Pradhan2 B, et al. Clinical profile of diabetic ketoacidosis in adults. Heal Renaiss. 2012;10(2):80–6.

52. Bedaso A, Oltaye Z, Geja E, Ayalew M. Diabetic ketoacidosis among adult patients with diabetes mellitus admitted to emergency unit of Hawassa university comprehensive specialized hospital. BMC Res Notes [Internet]. 2019;12(1):137. Available from: https://doi.org/10.1186/s13104-019-4186-3

53. Edo AE. Clinical profile and outcomes of adult patients with hyperglycemic emergencies managed at a tertiary care hospital in Nigeria. Niger Med J. 2012 Jul;53(3):121–5.

15 ANNEXURES

15.1.1 QUESTIONNAIRE

Name:

Age/sex:

IP no:

Socioeconomic status:

Presenting complaints

History of fever/vomiting/abdomen pain/altered sensorium/seizures/breathing difficulty

Past history:

H/o DM,TB,BA, CLD,CAD,CKD,SHTN,CVA,THYROID OR SEIZURE DISORDER

Personal history

Alcoholic/ non alcoholic ,Smoker/non smoker

Family history

h/o known diabetes mellitus in family members

Treatment history

Diabetes mellitus treatment – OHA/Insulin

Compliance-yes /no

Dose of OHA/INSULN

Clinical Examination -General Examination

Consciousness,orientation, pallor,Jaundice, clubbing, lymphadenopathy, pedal edema

Vital BP

PR

RR

Spo2

Pupil size

Systemic examination- CVS-

RS-

ABDOMEN-

CNS

Bed side investigation:

TC- HB- MCV- PLT HCT-

RBS- mg/dl, serum urea- mg/dl serum creatinine- mg/dl plasma acetone-

Serum sodum- mEq/L SERUM POTTASSIUM- mEq/L HbA1C- %

ABG- PH-

Pco2- Hco3-

Total duration of stay in hospital-

Outcome-

15.1.2 **CONSENT FORM**

PARTICIPANT CONSENT FORM

Participants name :

Address :

Title of the sudy:

"STUDY OF CLINICAL PROFILE IN DIABETIC KETOACIDOSIS AND ITS OUTCOME IN RELATION TO BICARBONATE LEVELS "

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes.i have been given an information sheet giving details of the study. I fully consent to participate in the above study

Signature of the participant : Date :

Signature of the witness : Date :

Signature of the investigator : Date :

Consent Forms

"STUDY OF CLINICAL PROFILE IN DIABETIC KETOACIDOSIS AND ITS OUTCOME IN RELATION TO BICARBONATE LEVELS "

Investigator: **Dr.SAKTHIKUMAR.S**

Please initial each box & sign at bottom

I confirm that I have read and understood the 'Patient

1. Information Sheet' for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

I understand that my participation is voluntary and that I am

2. free to withdraw at any time, without giving any reason and without my medical care or legal rights being affected ☐

I understand that relevant sections of any of my medical notes and data collected during the study may be looked at ☐

3. by responsible individuals from TMC Hospital. I give permission for these individuals to have access to my records. ☐

4. I consent to my data being retained if I withdraw from the study ☐

5. I agree to take part in the above study. ☐

நோயாளியின் ஒப்புதல் படிவம்

நோயாளியின் பெயர் :

முகவரி :

1 .மேற்கூறிய ஆய்வினை பற்றி எழுத்து மூலமாகவும் , என் சொந்த மொழியிலும் முழுவிவரம் அறிந்துகொண்டேன்.

2.இந்த ஆய்வில் நான் பங்கேற்பதற்கான தேவையை பற்றியும், எந்த சமயத்திலும் ஆய்வில் இருந்து வெளியேற முடியும் என்றும் தெரிந்து கொண்டேன்.

3.ஆய்வின்போது பெறப்படும் எனது தகவல்கள் மற்றும் மருத்துவக்குறிப்புகள் அனைத்தையும் எந்தவித தடையும் இன்றி ஆராய்ச்சிக்கு பயன்படுத்தி கொள்ளலாம்.

4. மேற் கூறிய ஆய்விற்கு எனது முழு சம்மந்தம் தெரிவிக்கிறேன்.

நோயாளியின் கையொப்பம்: தேதி :

சாட்சியின் கையொப்பம் : தேதி:

ஆராய்ச்சியாளரின் கையொப்பம் : தேதி:

15.1.3 PATIENT INFORMATION SHEET

I, Dr. SAKTHIKUMAR.S(PH No; 8667581220) am conducting a

"STUDY OF CLINICAL PROFILE IN DIABETIC KETOACIDOSIS AND ITS OUTCOME IN RELATION TO BICARBONATE LEVELS "

You are being invited to take part in a research study. Before you decide whether to take part, it is important for you to understand why the research is being done. Feel free to discuss the study with others if you wish. Please take time to decide whether or not you wish to take part.

The aim of this study is to collect information on patients such as yourself presenting with diabetic ketoacidosis and to study clinical profile, and outcome in relation to bicarbonate levels.The study will analyse patients with diabetic ketoacidosis admitted to THANJAVUR MEDICAL COLLEGE Hospital over a period of time. The type of treatment patients receive will not be altered by taking part in this study. If you agree to take part you will be observed from the time you agree to take part until you leave the hospital. No additional tests will be undertaken as part of the study, but you will be asked to give permission for your medical records to be examined in detail, in order to collect information about your health status and any treatments that you have throughout your hospital stay.Your treatment options will not be altered in any way by taking part in this study. Your doctor will decide on the best treatment for you. Participation in the study does not restrict your ability to change from one treatment option to another.There will be no additional visits as part of the study.There are no anticipated

disadvantages or risks involved in this study as it is an observational study of the patients with diabetic ketoacidosis'.All information that is collected about you during the course of this study will be kept strictly confidential.This study was reviewed and approved by the Institutional Review Board and Ethics'

Contact details:

Please contact **Dr.SAKTHIKUMAR.S** at Department of Medicine ,THANJAVUR MEDICAL COLLEGE. PHONE NO:8667581220

15.1.4 DATA SHEET

S.NO	NAME	AGE	SEX	TYPE OF DM	FAMILY HISTORY	TREATMENT	RBS(MG/DL)	PLASMA ACETONE	BICARBONATE LEVEL(MEQ/L)	HBA1C	HB IN G/DL	UREA (mg/dl)	CREATININE(mg/dl)	sodium m(meq/l)	POTASSIUM(meq/l)	SYMPTOMS	CONSCIOUS LEVEL	HR	RR	BP in mmHg	TOTAL DURATION OF STAY in days	CAUSE	OUTCOME
1	GANAPATHY	57	M	2	N	OHA	430	POSITIVE	16.5	7.6	12.4	43	1.5	137	3.4	VOMITING	ORIENTED	110	22	130 BY 70	6	NC	REC
2	RUKUMANI	35	F	2	N		501	POSITIVE	9.4	9.9	9.9	30	1.1	131	4.2	FEVER/ADBOMEN PAIN	CONFUSED	124	31	140 BY 70	10	INFECTION	REC
3	JAYAPAL	36	M	2	YES	INSULIN	446	POSITIVE	16	8.9	13.2	31	0.9	134	4.4	DYSPNEA	DROWSY	114	26	130 BY 70	7	NC	REC
4	SUBRAMANI	43	M	2	N	INSULIN	491	POSITIVE	13	7.7	12.7	38	1.3	121	4.1	VOMITING	DROWSY	130	25	110 BY 70	9	NC	REC
5	MURUGAN	31	M	1	N	INSULIN	512	POSITIVE	16.4	7.4	14.8	56	2	134	5.3	LETHARGY	ORIENTED	108	26	130 BY 70	8	NC	REC
6	AKASH	20	M	1	N	INSULIN	376	POSITIVE	17	9.1	15.1	34	0.7	132	3.6	VOMITING	CONFUSED	121	27	140 BY 90	5	INADEQUATE DOSE	REC
7	CHELLAMMAL	60	F	2	YES		ABOVE 600	POSITIVE	15	8.1	8.8	56	1.6	140	5.3	FEVER/CONFUSION	CONFUSED	110	24	110 BY 70	7	NC	REC
8	SRIMATHI	23	F	1	N	INSULIN	430	POSITIVE	14.2	7.7	9.7	42	1.2	138	5.2	ABDOMEN PAIN	ORIENTED	102	20	150 BY 80	5	NC	REC
9	SELVAMBIGAI	49	F	2	N	OHA +INSULIN	470	POSITIVE	14.1	9	9.2	40	1.4	132	4.7	FEVER	DROWSY	116	24	140 BY 70	8	INFECTION	REC
11	KUMAR	28	M	1	N	INSULIN	368	POSITIVE	21	9.3	16.1	444	1.5	144	3.8	VOMITING	ORIENTED	106	23	100 BY 70	7	NC	REC
12	NALINI	17	F	1	N	INSULIN	470	POSITIVE	19	9	10.2	40	1.2	150	3.6	FEVER	ORIENTED	90	20	110 BY 70	6	NC	REC
13	SUNDARAM	57	M	2	YES	OHA+INSULIN	512	POSITIVE	18.7	11	11.9	62	1.7	148	3.4	GIDDINESS	ORIENTED	102	18	100 BY 70	5	NC	REC
14	RASHEER	20	M	1	N	INSULIN	476	POSITIVE	17.2	8.4	14.1	52	1.2	139	4.4	FEVER	ORIENTED	114	22	110 BY 70	7	NC	REC

2	S.NO	NAME	AGE	SEX	TYPE OF DM	FAMILY HISTORY	TREATMENT HISTORY	RBS(MG/DL)	PLASMA ACETONE	BICORBONCATE LEVEL(MEQ/L)	HBA1	HB IN G/DL	UREA (mg/dl)	CREATININE(mg/dl)	sodium(meq/l)	POTTASSIUM(meq/l)	SYMPTOMS	CONSCIOUS LEVEL	HR	RR	BP in mmHg	TOTAL DURATION OF STAY in days	CAUSE	OUTCOME
16	14	BASHEER BASHA	29	M	1	N	INSULIN	476	POSITIVE	17.3	8.4	14.1	52	1.3	139	4.4	FEVER	ORIENTED	114	22	110 BY 70	7	NC	REC
17	15	CHINNAMMAL	50	F	2	YES	INSULIN	446	POSITIVE	18	6.7	8.5	54	1.6	136	4.3	ABDOMEN PAIN	CONSCIOUS	94	22	130 BY 70	5	NC	REC
18	16	AROKIYARAJ	34	M	1	N	INSULIN	470	POSITIVE	9.2	9.2	13.6	51	0.8	128	3.3	FEVER/ALTERED SENSORIUM	STUPUROUS	132	28	100 BY 70	12	INFECTION	REC
19	17	KARTHICK	18	M	1	N	INSULIN	410	POSITIVE	17.1	9.5	14.7	40	1.3	144	3.9	FEVER/ DEHYDRATION	ORIENTED	102	22	110 BY 70	6	INFECTION	REC
20	18	KUPPUSAMY	61	M	2	N		520	POSITIVE	12.4	7.8	11.8	43	1.1	140	4.6	FEVER/VOMITING	DROWSY	122	26	130 BY 70	8	NC	REC
21	19	MAHESHWARI	33	F	1	N	INSULIN	402	POSITIVE	15	8.4	8.9	33	0.8	146	4	VOMITING	ORIENTED	107	24	100 BY 70	5	INADEQUATE DOSE	REC
22	20	RENGASAMY	56	M	2	N		427	POSITIVE	14	9.3	12.3	40	1.1	137	3.3	ALTERED SENSORIUM	DROWSY	110	22	140 BY 80	6	INADEQUATE DOSE	REC
23	21	VIGNESH	22	M	1	N	INSULIN	ABOVE 600	POSITIVE	17	7.8	14.3	36	0.8	131	3.6	ALTERED SENSORIUM	STUPUROUS	124	28	130 BY 80	6	NC	REC
24	22	UMA	46	F	2	N		451	POSITIVE	18	9.1	9.2	32	0.7	131	5.1	FEVER	ORIENTED	101	24	120 BY 60	5	NC	REC
25	23	GANDHI	63	F	2	YES		433	POSITIVE	9.6	10.3	8.9	54	1.9	128	5.2	ALTERED SENSORIUM/FEVER	DROWSY	120	26	130 BY 70	7	NC	REC

2	S.NO	NAME	AGE	SEX	TYPE OF DM	FAMILY HISTORY	TREATMENT HISTORY	RBS(MG/DL)	PLASMA ACETONE	BICORBONCATE LEVEL(MEQ/L)	HBA1	HB IN G/DL	UREA (mg/dl)	CREATININE(mg/dl)	sodium(meq/l)	POTTASSIUM(meq/l)	SYMPTOMS	CONSCIOUS LEVEL	HR	RR	BP in mmHg	TOTAL DURATION OF STAY in days	CAUSE	OUTCOME
26	24	SELVAM	21	M	1	N	INSULIN	471	POSITIVE	13.6	8	16.2	38	1	134	4.4	ALTERED SENSORIUM/ VOMITING	STUPUROUS	133	30	100 BY 60	8	NC	REC
27	25	RAMESH	34	M	1	N	INSULIN	450	POSITIVE	9	9.1	15.2	48	1	142	3.5	FEVER/VOMITING/ ABDOMEN PAIN	DROWSY	128	27	110 BY 70	10	INFECTION/ INADEQUATE DOSE	REC
28	26	SELVARAJ	57	M	2	N	OHA+INSULIN	512	POSITIVE	17.5	10.2	14	62	1.7	135	3.7	ALTERED SENSORIUM	DROWSY	116	24	100 BY 70	7	NC	REC
29	27	NALAN KANNI	54	F	2	N	OHA+INSULIN	ABOVE 600	POSITIVE	8	11.4	9.2	39	1.1	149	5.4	FEVER/VOMITING	DROWSY	74	21	110 BY 70	13	INFECTION	DEATH
30	28	DEVI	41	F	2	YES		351	POSITIVE	15.2	9.1	9	52	1.9	131	3.1	FEVER	CONSCIOUS ORIENTED	112	22	140 BY 90	6	INFECTION	REC
31	29	UDHAYAKUMAAR	43	M	2	N		281	POSITIVE	16.7	8.4	13	57	1.9	137	3.6	VOMITING	ORIENTED	91	19	130 BY 80	5	NC	REC
32	30	MANIMARAN	57	M	2	N		302	POSITIVE	17.2	8.7	13.1	64	1.8	141	3.5	FEVER	ORIENTED	90	20	140 BY 80	4	INFECTION/ NC	REC
33	31	BALAMURUGAN	32	M	1	N	INSULIN	460	POSITIVE	12.1	9.6	12.9	38	0.9	142	3	FEVER/VOMITING	DROWSY	114	25	100 BY 60	8	NC	REC
34	32	SAKTHIVEL	39	M	2	N		370	POSITIVE	13.9	8.7	13.4	43	1.3	129	3.2	ADBOMEN PAIN	ORIENTED	120	24	100 BY70	8	NC	REC

2	S.NO	NAME	AGE	SEX	TYPE OF DM	FAMILY HISTORY	TREATMENT HISTORY	RBS(MG/DL)	PLASMA ACETONE	BICORBONCATE LEVEL(MEQ/L)	HBA1	HB IN G/DL	UREA (mg/dl)	CREATININE(mg/dl)	sodium(meq/l)	POTTASSIUM(meq/l)	SYMPTOMS	CONSCIOUS LEVEL	HR	RR	BP in mmHg	TOTAL DURATION OF STAY in days	CAUSE	OUTCOME
35	33	BHUVANA	65	F	2	N	OHA+INSULIN	ABOVE 600	POSITIVE	7	9	9.3	69	2.1	131	3.3	FEVER/ALTERED SENSORIUM	STUPUROUS	130	26	140 BY 60	15	INFECTION/ NC	DEATH
36	34	KRISHNAMMAL	75	F	2	N		459	POSITIVE	9.4	10.7	8.1	79	2	135	3	FEVER	DROWSY	127	30	110 BY 70	12	NC	REC
37	35	MARIAMMAL	60	F	2	N		371	POSITIVE	17.2	7.8	8.4	60	0.9	130	3.7	VOMITING	ORIENTED	110	21	120 BY 80	5	INADEQUATE DOSE	REC
38	36	KUMARASAMY	51	M	2	N		450	POSITIVE	12.9	7.9	11.9	47	1.6	145	3.9	VOMITING	DROWSY	102	24	150 BY 80	9	NC	REC
39	37	JAYAPAL	60	M	2	N	OHA+INSULIN	470	POSITIVE	10.1	9.6	12.5	55	1.4	138	3.7	FEVER	ORIENTED	114	22	130 BY 80	9	NC	REC
40	38	MANOHAR	49	M	2	YES		530	POSITIVE	9.9	10.2	13.4	47	1.3	148	3.9	VOMITING/ DYSPNEA	DROWSY	115	25	130 BY 80	12	NC	REC
41	39	MUTHAMMAL	74	F	2	N		506	POSITIVE	12.1	9.8	8	66	1.6	131	3.3	FEVER/VOMITING	DROWSY	120	22	140 BY 70	9	NC	REC
42	40	CHINNASAMY	85	M	2	N	INSULIN	ABOVE 600	POSITIVE	7.8	11.1	12.1	84	2.2	130	2.7	ALTERED SENSORIUM/FEVER	STUPUROUS	121	26	140 BY 70	14	NC	DEATH
43	41	PAPPA	61	F	2	N		350	POSITIVE	11.7	7.9	8.7	54	1.4	138	3.4	VOMITING/ ABDOMEN PAIN	ORIENTED	110	22	130 BY 80	9	NC	REC

2	S.NO	NAME	AGE	SEX	TYPE OF DM	FAMILY HISTORY	TREATMENT HISTORY	RBS(MG/DL)	PLASMA ACETONE	BICORBONCATE LEVEL(MEQ/L)	HBA1	HB IN G/DL	UREA (mg/dl)	CREATININE(mg/dl)	sodium(meq/l)	POTTASSIUM(meq/l)	SYMPTOMS	CONSCIOUS LEVEL	HR	RR	BP in mmHg	TOTAL DURATION OF STAY in days	CAUSE	OUTCOME
44	42	CHELLAPANDIAN	71	M	2	N		520	POSITIVE	9.8	9.7	12	65	2.1	142	2.9	DYSPNEA / DYSPNEA	DROWSY	108	25	130 BY 80	12	NC	REC
45	43	KAMATCHI	60	F	2	YES		402	POSITIVE	12.4	8.8	8.6	49	1.8	129	2.8	DYSPNEA	DROWSY	116	24	120 BY 70	11	NC	REC
46	44	UTHTHAMI	54	F	2	YES	OHA+INSULIN	399	POSITIVE	18.2	7.7	9.2	47	1.7	130	3.3	VOMITING	ORIENTED	102	19	120 BY 80	4	INADEQUATE DOSE	REC
47	45	GNANAVEL	69	M	2	N		512	POSITIVE	14.1	8.9	12.1	55	1.9	137	3.1	FEVER	ORIENTED	112	21	100 BY 70	6	NC	REC
48	46	VEERAIYAN	70	M	2	N		470	POSITIVE	13.1	9.1	12	61	1.8	129	3.2	VOMITING	DROWSY	117	23	100 BY 60	7	NC	REC
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