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# COMPARISON OF CLINICAL PROFILE AND BIOCHEMICAL PROFILE IN DIABETIC KETOACIDOSIS PATIENTS

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## Abstract

**Introduction:** One of the most frequent medical emergencies in the world is diabetic ketoacidosis (DKA). The patient may exhibit a variety of symptoms, including ketosis, ketoacidosis, and pre-coma ketosis and coma[1] however frequently the clinical presentation bury these signs. In type 1 diabetes has a higher prevalence diabetes mellitus additionally, type 2 diabetes experiences it, particularly in instances such infections and additional co-morbid conditions. Numerous advancements have been made alterations in the original history of this condition have been made as result of improvements in the early detection and therapy of both ketoacidosis and the comorbidities bad health.

**Materials and Methods:** 50 patients admitted to the Government General Hospital in Anantapuramu are the subjects under this study. The following patients were the role of the investigation:- 1) Patients with diabetic ketoacidosis who were known to be type 1 or type 2 diabetes. 2) Patients who were initially admitted for different illnesses but who unintentionally had diabetic ketoacidosis. Patients have to fulfill all of the following criteria in order to be admitted to the protocol: Hyperglycemia >250 mg/dl, Acidosis with blood pH <7.3, Serum bicarbonate <15 mEq/L, Urine positive for ketones. Upon admission, a comprehensive clinical examination and a careful and detailed history were taken. All of the proforma bullet points were noted down. The following inquiries were conducted, and any additional information was recorded:

**Results:** Out of 50 the cases of dka, 42 were differentiated under type2 diabetes mellitus (84%) and 8 were under type1 diabetes mellitus (16%). Out of the 50 patients, 25 were about males (50%) and 25 were about females (50%). M: F ratio was around 1:1. In this study, the minimum age was around 14 years and the max. Age observed was around 69 years and mean is 42.9±12.9 years. Maximum count of cases 20 (40%) significant were at the age group 41- 50 years. Minimum number of cases 2 (4%) were at the age group 21- 30 years. Majority of cases are at the age group o f 31- 60, that patient (78%). Out of 50 patients 41 (82%) were dehydrated and 40(80%) were breathless at the time of t he admission.9 (18%) patients were drowsy and 4 (8%) were stuporous at the time of admission. 35 (74%) patients were fully conscious at admission.

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**Conclusion:** The most typical clinical signs and symptoms include nausea, vomiting, abdominal discomfort, dehydration, acidotic breathing, impaired sensory function, and fever. The clinical and the biochemical profiles of patients with the type 1 and the type 2 DM who develop ketoacidosis are not significantly different. Diabetes-related ketoacidosis has a mortality rate of 4%. Severity of the altered sensorium, severity of the comorbid disease, severity in dehydration, having severe acidosis, doses and length of insulin requiring for clearing UKB are some clinical and the biochemical indicators that may signal poor prognosis.

**Key Words:** diabetic ketoacidosis, vomiting, abdominaldiscomfort, dehydration.

## INTRODUCTION

One of the most frequent medical emergencies in the world is diabetic ketoacidosis (DKA). The patient may exhibit a variety of symptoms, including ketosis, ketoacidosis, and pre-coma ketosis and coma[1] however frequently the clinical presentation bury these signs.[1] In type 1 diabetes has a higher prevalence diabetes mellitus additionally, type 2 diabetes experiences it, particularly in instances such infections and additional co-morbid conditions. Numerous advancements have been made alterations in the original history of this condition have been made as result of improvements in the early detection and therapy of both ketoacidosis and the comorbidities bad health.[2]

Majority of the patients who come with DKA are already receiving therapy for their diabetes, and infection (sepsis) and forgetting to take insulin[3] were the common frequent triggering events. Nausea, vomitings, polyuria, and polydipsia are the common frequent presenting complaints, and the predominant clinical findings are dehydration, acidotic breathing, disorientation, or coma[4]. In such cases, mean random blood sugars, pH, and osmolality [1]correlate statistically strongly with neurological state.

Parameters are
A) The period of DKA previous to the admission,
B) Severity of the acidosis,
C) Severity of the peripheral vascular insufficiency,
D) Coexisting diseases are the key factors associated with death[5].

Therefore, this study was conducted to compare the clinical characteristics and the biochemical profile of DKA patients with the responses of those receiving the recommended treatment for the condition.

## OBJECTIVES OF THE STUDY

- To compare the clinical features and the biochemical profile in diabetic ketoacidosis.
- To assess the response in the patients with standard treatment of diabetic ketoacidosis.

## MATERIALS AND METHODS

50 patients admitted to the Government General Hospital in Anantapuramu are the subjectsunder this

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study.

The following patients were the role of the investigation:-

1. Patients with diabetic ketoacidosis who were known to be type 1 or type 2 diabetes.
2. Patients who were initially admitted for different illnesses but who unintentionally haddiabetic ketoacidosis.

Patients have to fulfill all of the following criteria in order to be admitted to the protocol:

1. Hyperglycemia >250 mg/dl
2. Acidosis with blood pH <7.3
3. Serum bicarbonate <15 mEq/L
4. Urine positive for ketones.

Upon admission, a comprehensive clinical examination and a careful and detailed history weretaken. All of the proforma's bullet points were noted down. The following inquiries were conducted, and any additional information was recorded:

**Hematological and biochemical investigations:**

a) Hb%, WBC count total and differential.
b) Blood sugar estimation was done by folin Wu method.
c) Serum electrolytes by flame photometry.

Normal range of serum sodium is 136 to 145 mEq/l Normal range of serum potassium is 3.6 to 5.5 mEq/l D) Blood urea estimation by Urea nesselerization methodNormal range is 25 to 40 mg/dl E) serum creatinine normal range is 0.5 to 1.9mg/dl.

**Radiological Investigation:**

Chest x ray PA view to see any lung pathology.

Electrocardiogram : to see evidence of ischemic cardiac disease and potassiumchanges in serial ECGs

Urine examination for routines and microscopyUrine for the culture and the sensitivity

**Urine Sugars:**

Benedict's Qualitative Test:

If the actual blue colour of reagent changes to green/yellow- orange/ brick red. Although this is a very simple and practical tool for detecting diabetes and assessing its degree of control, urine sugar estimates are frequently inaccurate because of varying renal threshold and it wont accurately represent the blood sugar levels. One of the important popular methods used now is urine sugar estimate since it is practical, simple to use at home, and able taught to the patients.

**Urine Albumin**

Results of the heat and the acetic acid test range from 1 to 4+. This is one of the simplest tests to determine involvement of kidney in diabetes patients is this one. Proteinuria severity is correlated with renal damage.

**Urine for Ketone Bodies:**

Rothera's Test is for acetone and acetoacetic acid:-

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Ammonium sulphate crystals are oversaturated in the volume of 10 ml urine. Then, around two ml of a potent ammonia solution and three drops of a powerfully recently produced sodium nitroprusside solution are added. Acetones and acetoacetic acid combine to generate a rich permanganate colour. Ketones are not present if Rothera's test yields a negative result. Ketone bodies seen in urine are indicative for diabetic ketoacidosis. Sequential urine sugars and acetone estimates in diabetic ketoacidosis provides insight into the progression of the conditionand the effectiveness of treatment.

**Blood pH:**

Normal arterial blood pH is 7.35 to 7.45.

Arterial pH between 7.25- 7.3 is mild DKA. pH between 7.24 – 7.1 is moderate DKAand pH <7 is severe DKA.

**Serum bicarbonate:**

It is measured in mEq/L. Normal value is 22-26 mEq/L

Mild DKA 15-18 mEq/L, Moderate DKA 10-15 mEq/L, Severe DKA <10mEq/L

## RESULTS

**Table 1: Distribution of the Type 1 and Type 2 diabetes mellitus in diabeticketoacidosis**

| Total num. of cases | Type 1 | Type 2 |
|---|---|---|
| 50 | 8(16%) | 42 (84%) |

Out of 50 the cases of dka, 42 were differentiated under type2 diabetes mellitus(84%) and 8 were under type1 diabetes mellitus (16%).

**Table 2: Distribution of Patients according to their Sex**

| Sex | No.of case | Percentage |
|---|---|---|
| Males | 25 | 50 |
| Females | 25 | 50 |

Out of the 50 patients, 25 were about males (50%) and 25 were about females (50%).M: Fratio was around 1:1.

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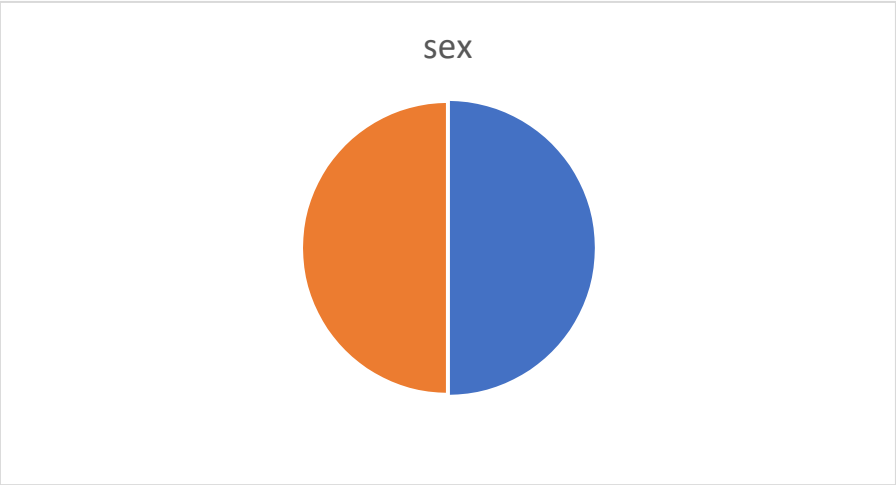
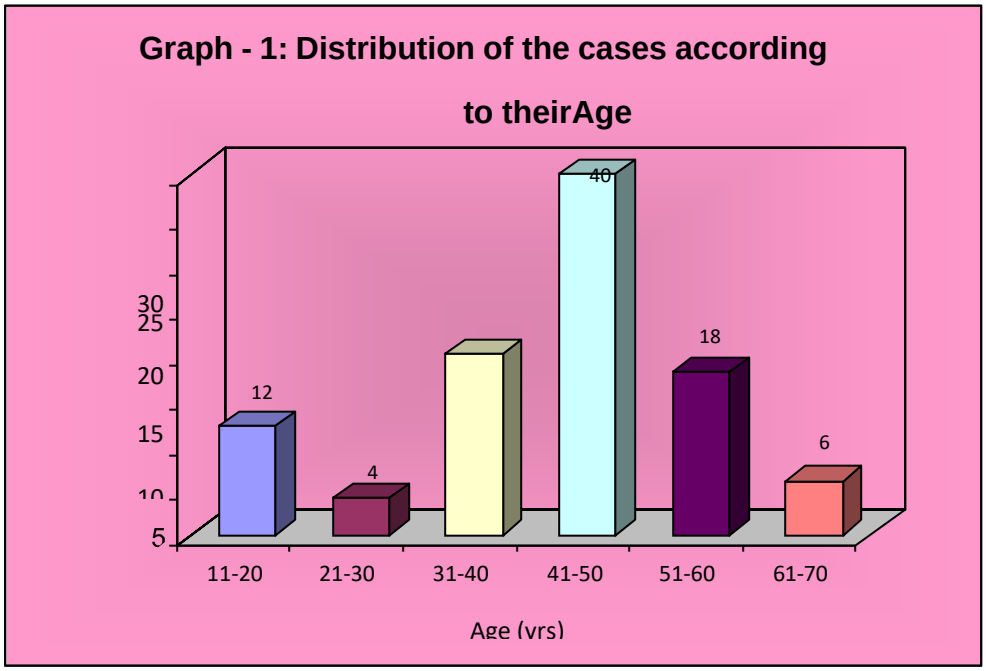



**Table 3: Distribution of the Cases according to their Age**

| Age (years) | No.of case | ntage |
|---|---|---|
| 11-20 | 6 | 12 |
| 21-30 | 2 | 4 |
| 31-40 | 10 | 20 |
| 41-50 | 20 | 40 |
| 51-60 | 9 | 18 |
| 61-70 | 3 | 6 |
| Total | 50 | 100 |

Mean age ± SD 42.9 ± 12.9, Range 14-69



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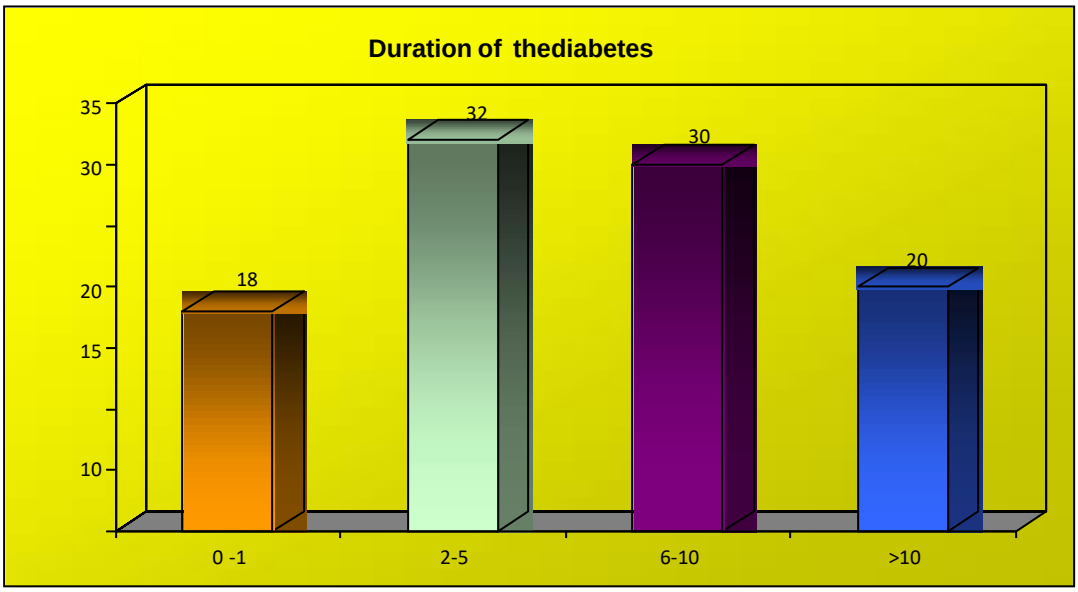
In this study, the minimum age was around 14 years and the max. age observed was around 69 years and mean i s 42.9±12.9 years. Maximum count of cases 20 (40%) significant were at the age group 41- 50 years. Minimum number of cases 2 (4%) were at the age group 21- 30 years. Majority of cases are at the age group o f 31-60, that patient (78%).

**Table 13: Distribution of patients according to duration of t h e diabetes inknown cases of dka**

| Duration of the diabetes (years) | No.of case | Percentage |
|---|---|---|
| 0-1 | 9 | 18 |
| 2-5 | 16 | 32 |
| 6-10 | 15 | 30 |
| >10 | 10 | 20 |
| Total | 50 | 100 |

Mean duration ± SD 6.7 ± 4.8, Range 0.6 – 17 years




In this study, minimum duration of the diabetes in DKA patients is 6 months and maximum duration is 17 years. Max. Number of patients i.e., 16 (32%) were diabetic for 2- 5 years. Patients with diabetes more than 10 years were 10(20%).

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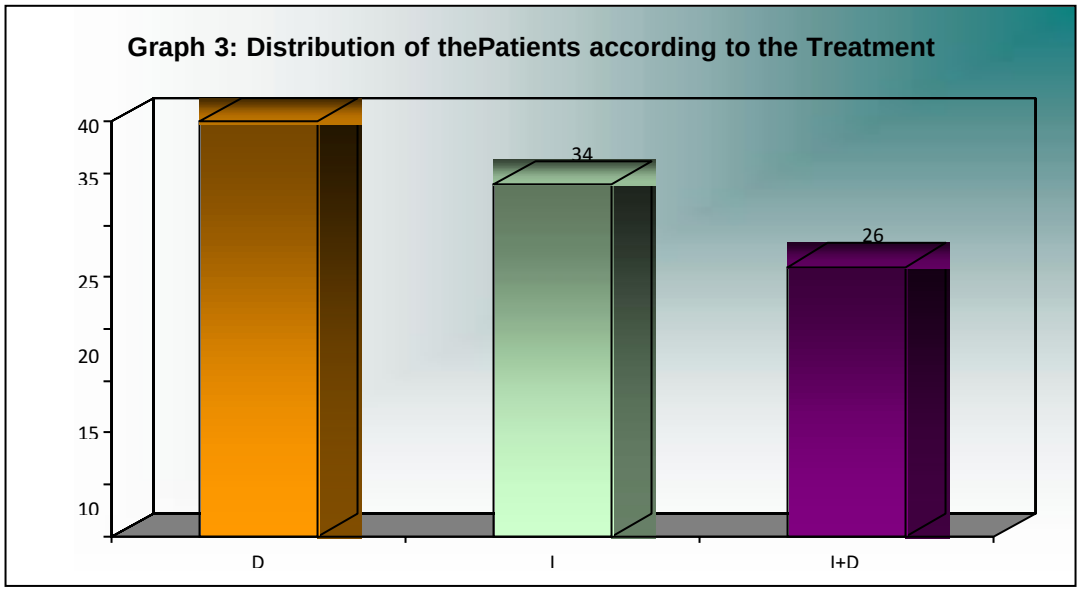
**Table 14: Distribution of the patients according to the Treatment History**

| Treatment | No.of case | Percentage |
|---|---|---|
| Insulin | 20 | 40 |
| insulin | 17 | 34 |
| Insulin + drugs | 13 | 26 |




We observed that the max. Number of the patients were using OHDs alone 20 (40%), 17 (34%) were on the insulin therapy alone 13 (26%) patients were using both insulin and OHD's.

**Table 15: Distribution of patients according to the family history ofdiabetes**

| Family history of DM | Type 1(n=8) | Type 2(n=42) | Total (n=50) |
|---|---|---|---|
| Present | 1 (12.5%) | 34 (81%) | 35 (70%) |
| Absent | 7 (87.5%) | 8 (19%) | 15 (30%) |

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Out of 50 patients, 35 (70%) had about family history of diabetes and 15(30%)did not gave any family history of diabetes. Amongst known cases under type 1 DM (8), 1 (12.5%) gave the family history of diabetes and 7 did not. Amongst known case of type 2 DM (42), 34 (81%) gave the family history of diabetes.

| The Precipitating factors | Type 1(n=8) | Type 2(n=42) | Total (n=50) |
|---|---|---|---|
| I) Irregular treatment | 5 (62.5%) | 3 (7%) | 8 (16%) |
| II) Infections: | 3 (37.5%) | 25 (60%) | 28 (56%) |
| - Urinary tract infection | - | 4 | 4 (8%) |
| - Acute gastroenteritis | 1 | 2 | 3 (6%) |
| - Diabetic foot | - | 2 | 2 (4%) |
| - Respiratory tract infection | 2 | 4 | 6 (12%) |
| - Perianal abscess | - | 2 | 2 (4%) |
| - Enteric fever | - | 3 | 3 (6%) |
| - CNSinfection | - | 2 | 2 (4%) |
| - Septic shock | - | 2 | 2 (4%) |
| - Acute cholecystitis | - | 2 | 2 (4%) |
| - Chronic pancreatitis | - | 2 | 2 (4%) |
| II) Others: | | 14 (33%) | 14 (28%) |
| - Cerebrovascular accident | - | 4 | 4 (8%) |
| - Head injury | - | 2 | 2 (4) |
| - Surgery | - | 5 | 5 (10%) |
| - IHD | - | 3 | 3 (6%) |

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In this study, the commonest precipitating factor was found as infection in 28 patients (56%).8 (16%) patients had either stopped treatment or were on irregular treatment. In 5 (10%) patients surgery was found as one of the precipitating factor and CVA and ischemic heart disease in 4 (8%) and 3 (6%) patients respectively. Amongst the infections, majorly i.e. 6(12%) patients had therespiratory tract infection and 4 (8%) had the urinary tract infections.

**Table 17: Dosage of Insulin required to clear UKB**

| Group | Dosage of insulin req clear UKB (in units) | Type 1(n=8) | Type 2(n=42) | Total (n=50) |
|---|---|---|---|---|
| I | 0 – 25 | 1 (12.5%) | 3 (7%) | 4 (8%) |
| II | 26 – 50 | 6 (75%) | 15 (36%) | 21 (42%) |
| III | 51 – 75 | -- | 7 (17%) | 7 (14%) |
| IV | 76 – 100 | -- | 4 (10%) | 4 (8%) |
| V | >100 | 1 (12.5%) | 13 (30%) | 14 (28%) |

UKB = Urine Ketone Bodies

In our study groups max i.e. 21 (42%) patients requiring insulin dosage between 26- 50units for the clearance of UKB.7 (14%) patients required insulin dose between 51- 75 units. 4 patients required <25 units for the clearance of UKB, and 14 patients required >100 units. In this study, 7 patients (87.5%) under type I DM required < 50 units of insulin dose to clear UKB.

**Table 18: Duration of the Insulin Therapy Required to Clear UKB**

| Group | Duration of the insulin therapy requiredfor clearance of UKB (in hours) | Type 1(n=8) | Type 2(n=42) | Total (n=50) |
|---|---|---|---|---|
| I | 0 – 12 | 1(12.5%) | 4 (10%) | 5 (10%) |
| II | 13 – 24 | 6 (75%) | 17 (40%) | 23 (46%) |
| III | 25 – 36 | -- | 7 (17%) | 7 (14%) |
| IV | 37 – 48 | -- | 9 (21%) | 9 (18%) |

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| | | | | |
|---|---|---|---|---|
| V | 49 – 72 | 1 (12.5%) | 3 (7%) | 4 (8%) |
| VI | >72 | -- | 2 (5%) | 2 (4%) |

Majority of patients in our study group required 13-24 hrs for the clearance of UKB from the start of insulin therapy.5 (10%) patient required <12 hr and 2 (4%) >72 hrs. Both the patients who expired required more than 72 hrs.

**Table 19: Clinical and biochemical profile of the patients who expired**

| | **Patient 1** | **Patient 2** |
|---|---|---|
| Age | 65 | 52 |
| Sex | M | F |
| DOD (Years) | 15 | 17 |
| Type | T2 | T2 |
| Dehydration | Severe | Severe |
| Mental status | Coma | Coma |
| UKB | 4+ | 4+ |
| RBS | 585 | 548 |
| HCO3 | 6 | 5 |
| pH | 6.95 | 6.98 |
| Insulin dose required | 160 | 148 |
| Duration of the insulin | 78 hours | 72 hours |

DOD = Duration of the diabetes

In our study we observed that both the patients who were died, were comatosed at the time of the presentation, had the septicemic shock, type 2 diabetic, severely dehydrated, and insulin requiring to clear UKB was >140 units and duration of insulin therapy to clear UKB was >78 hrs in one and >72 hrs in the other. RBS was very high 566.5±26.2 at the time of presentation. Both the patients were in the severe acidosis 7.0±0.0 (6.95- 6.98) and HCO3 was 5.5±0.7 (5-6).

**Table 20: Clinical profile in the diabetic ketoacidosis before and after treatment**

| | **Admission (n=50)** | | **Post treatment(n=48)** | |
|---|---|---|---|---|
| | **No.of cases** | **%** | **No.of cases** | **%** |

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| | | | | |
|---|---|---|---|---|
| Fever | 19 | 38 | 4 | 8.3 |
| Dehydration | 41 | 82 | 4 | 8.3 |
| Abdominal pain | 25 | 50 | - | - |
| Vomiting | 37 | 74 | 4 | 8.3 |
| Mental status | | | | |
| Coma | 2 | 4 | - | - |
| Conscious | 35 | 70 | 48 | 100 |
| Drowsy | 9 | 18 | - | - |
| Stupor | 4 | 8 | - | - |
| Acidotic breathing | 40 | 80 | - | - |

Out of 50 patients 41 (82%) were dehydrated and 40(80%) were breathless at the time of the admission.9 (18%) patients were drowsy and 4 (8%) were stuporousat the time of admission. 35 (74%) patients were fully conscious at admission.

Conscious level of all the patients who survived i.e., 48 patients improved with treatment. After treatment fever, dehydration and vomiting were present in 4 eachpatients (8.3%).

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**Table 21: Urine ketone bodies before and after treatment**

| UKB admission | Admission | | Post treatment | |
|---|---|---|---|---|
| | No.of cases | % | No.of cases | % |
| 2+ | 12 | 24 | - | - |
| 3+ | 22 | 44 | 2* | - |
| 4+ | 16 | 32 | - | - |
| Nil | - | - | 48 | - |
| Total | 50 | 100 | 50 | 100 |

***** Expired

In this study, urine ketone bodies of majority of patients at admissionwere 3+,i.e., 22 patient (44%).Urine ketone bodies of expired patient were positive during the time of death.

**Table 22: Comparison of the biochemical profile before and after treatment**

| Measurement | Admission | | After treatment | | Mean red. | P-value* |
|---|---|---|---|---|---|---|
| | Range | Mean±SD | Range | Mean±SD | | |
| RBS | 218-585 | 355.3±69.1 | 126-258 | 163.8±28.8 | 191.5 □(54%) | <.001 HS |
| pH | 6.95-7.31 | 7.2±0.1 | 7.27-7.38 | 7.4±0.02 | 0.2 □(3%) | <.001 HS |
| HCO3 | 5-20 | 14.9±3.4 | 20-25 | 22.9±1.2 | 8.0 □(35%) | <.001 HS |

HS: Highly significant,* Paired t-test

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At admission, Mean±SD of RBS, pH and bicarbonate values were 355.3±69.1,7.2±0.1 and 14.9±3.4 respectively. After treatment, there were significant changes in RBS, pH and HCO3 values i.e., Mean ± SD were 163.8±28.8, 7.4±0.02 and 22.9±1.2 respectively.

**Table 23: Comparison of the Biochemical Profile of Patients who Survived and Expired**

| | **RBS** | **pH** | **HCO3** | **Requirement of insulin(units)** | **Duration of insulin (Hrs)** |
|---|---|---|---|---|---|
| Survived | 346.5±54.6 | 7.2±0.1 | 15.3±2.9 | 63.8±36.7 | 27.7±13.5 |
| Expired | 566.5±26.2 | 7.0±0.0 | 5.5±0.7 | 154.0±8.5 | 75.0±4.2 |
| P- value | <.05, S | <.05, S | <.01, S | <.01, S | <.01, S |

Student's t-test, S-Significant

We observed in our study that, there was a significant difference in many of the biochemical parameters in between the patients who survived and expired; There is also other significant difference in requirement of insulin and the duration of insulin between the patients who were survived and expired.

## DISCUSSION

**AGE**:

In this study, participants' ages ranged from 14 years old to 69 years old. 42.9±12.9 yrs years old was the average age. In one study, participants ranged in age from 6 to 80 years, with a mean age of 36. The average age of patients admitted for the management of diabetic ketoacidosis was about 40 to 50 years old, according to several additional research.

According to the Balasubramanyam A et al, from Department of general medicine, Baylor college of Medicine , Houston had done a cross sectional study, 53% were type 1 diabetes , 39% were type 2 diabetes and 8% of the patients were couldnot be identified as either type 1 or type 2 DM.[6]

**Sex:**

Male: female ratio in our study is1:1. According to one study, women tend to have diabetic ketoacidosis more frequently than males do. A female majority has been noted in certain other investigations. According to Daad Hassan Akbar conducted a study in king Abdul Aziz university hospital, Saudi Arabia, in 102 Diabetic keto acidosis patient. In the study, 75 percentage of patients were type 1 and 25 percentage of patients were type 2 diabetes. Male : female sex ratio was 2.1 : 1

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In this study, poor compliance and sub optimal insulin dosage was the major precipitatin factors. the major presenting problems in this study was nausea and vomiting. According to Berhane Seyonum, conducted a study in university school ofMedicine, Detroit, Males to Females ratio was 2:1.

**Diabetes duration:**

Our patients had diabetes for anywhere from 6 months to 17 years. Nine cases of dka(18%) occurred during the first year of the illness. The age ranges from 2- 5 yrs. had the highest percentage of cases (16), making up 32%. 15 cases had dka during 6 to 10 yrs of duration with percentage of 30.10 cases had greater than 10 yrs of duration with percentage of 20. According to one study, the length of diabetes ranged as follows: The prevalence of DKA was from 2.2% up to duration of 1 year to 2.8% up to 5 years, 2.9% up to 10 years, and 4.3% after 10 years.[7]

**Treatment history**

In this study, 20 number of cases are on drugs with percentage of 40.17 number of cases are on insulin dosage with percentage of 34. 13 numbers of cases were on both insulin usage and oral hypoglycemic drugs with percentage of 26. Sometimes patients may skip the dosage of insulin or skipped to take drugs.

This will be one of the major drawback for this study. According to another study, patients were mostly on insulin dosage with percentage around 38.

**Family history**:

In this study out of 50 cases, 35 cases had given the history of diabetes in their families with the percentage of 70. 15 cases doesn't had any history of diabetes in their families with the percentage of 30.

In the patients having type1 diabetes, among those 8 cases only one had family history with percentage of 12.5. In the 42 patients having type 2 diabetes had family history of diabetes with the percentage around 81.according to another study, 80 percentage of cases gave history in their families among type 2 daibetes.[8]

**Precipitating Factors:**

In our analysis, infection is the most prevalent precipitating factor (56%).The most frequently occurring infection are ,respiratory tract infections, after those UTIs are most frequently occurred.

In this study, irregular treatment mostly seen in type 1 diabetes that are around 5 cases in type 1 diabetes mellitus with percentage around 62.5. In type 2 diabetes mellitus, that are around 3 cases with the percentage of 7.

In type 1 diabetes, among the infections, respiratory tract infections are commonly one of the factor for dka in 2 cases. In type 2 diabetes, mostly commonly attributed to UTI, respiratory tract infections, with the percentage of around 8 in both these type of infections. After that enteric

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fever had seen in 3 cases with percentage around 6.

Later with the patients suffering from acute GE, diabetic foot disease, perianal abscess, acute cholecystitis, chronic pancreatitis with the percentage of 4.

In other factors like surgery is one of the factor in around 5 cases with percentage of 10. Cerebro vascular accidents seen in 4 cases with percentage of 8. IHD seen in 3 cases with percentage of 6. Head injury seen as one of the factor in 2 cases with percentage of 4.

According to other research, infection is the most frequently occuring factor contributing to development of diabetic ketoacidosis, along with CVA, omission, and inconsistent therapy. According to one study, 41% of cases had infection as primary triggering cause.

Infection (30.5%), non-compliance with the treatment (63.7%), and the newly diagnosed (5.8%) were the causes, according to another study.

According to Akhter khan et al conducted a study, in Aga khan university, infection is the most crucial cause for aggrevating diabetes in about 45.2 %. He observed that those who are having high blood sugars, low blood pH, increased serum osmolality having alteration in their mental status.

**Dosage of insulin required for clearing ketone bodies in urine**:

In this study, max. Around 21 patients required dosage of insulin between 26 to 50 units for clearing ketone bodies in the urine. 7 patients required dosage of insulin between 51 to 75 units for clearing ketone bodies in the urine.

4 patients require lesser than 25 units for clearing ketone bodies in the urine. 4 patients required 76 to 100 units of insulin dosage for clearing urine ketone bodies in the urine In our study, we compared between the clinical and biochemical profile of various who presented with diabetic keto acidosis. The patient who were died, were at a stage of coma during the admission time, were in the septic shock stage, with severe dehydration.[9]

The requirement of insulin dosage in those patient was about >140 units for clearing ketones was about >78 hours in one case, where as >72 hours in the other case. In both cases randomblood sugar test was very high during admission time. Both the cases were in acidotic stage.

**Clinical Profile:**

Vomiting and the abdominal pain were first and second in frequency in our survey (74% and50%, respectively). Dehydration (82% of cases) and the acidotic breathing (80% of cases) were the most frequently occuring symptoms.

50% of patients reported experiencing abdominal pain. In the 30% of individuals, altered in sensorium was identified. According to one study, the clinical symptoms included abdominal pain (51%), polyuria in (75.2%), polydipsia in (74.4%), polyphagia in (33%), nausea in (83.4%), and

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vomiting (78.5%). In another study, the most common clinical symptoms were polyuria, polydipsia, nausea, and vomiting.

**Biochemical Profile:**

In our investigation, pH levels ranged from 6.95 to 7.31 with a mean of 7.2 ± 0.1, and bicarbonate values ranged from 5 to 20, with a mean of 14.9 ± 3.4 RBS values ranges from 218 to 585 mg/dl. The metabolic profiles of type 1 and the type 2 DM patients did not significantly differ.

Most of these parameters in the individuals who passed away fit the criteria for severe dka and all of them had severe acidosis. Similar results from another study demonstrated that neither type 1 nor type 2 diabetes mellitus significantly differed in their propensity to develop diabetic ketoacidosis.

According to our study, various cases were present with fever, dehydration state, pain abdomen, vomitings, mental status in various states like conscious, Drowsy, stupor, coma, some cases presented with acidotic breathing When we compared between the biochemical profile before and after the treatment.

When we gare insulin dosage the blood sugars were reduced from the range of 218 – 585 to the range of 126-258 after giving treatment to the patients suffering from diabetic keto acidosis. After giving insulin dosage, the blood pH increased from the range 6.95 - 7.31 to the range of 7.27-7.38 after giving the treatment to the patients in diabetic keto acidosis and also after Insulin dosage, the serum bicarbonate valve increased from the range of 5-20 to the range of 20-25.

According to our study, there is a significant difference in the biochemical parameters in between the cases who surried and dead. The requirement of insulin dosage is about 63.8 ± 36.7 with period of insulin usage in hours 27.7 ± 13.5. The requirement of insulin dosage in death patient is about 154.0 ± 8.5 with period of insulinusage in hours is 75.0 ± 4.7.

After the treatment most of the cases got urine ketones negative. Those patients who died were had urine ketones positive at the time of death.[10]

**Predictors of bad prognosis or mortality:**

In this study, we discovered that the severity of the acidosis, the severity of the altered sensorium, severity of the dehydration, amount and the duration of insulin dosage therapy needed to clearing UKB and RBS during the time of presentation, and the comorbidity conditions were the factors that were significantly different or altered in the patients who died compared to those who survived. Both patients who passed away were experiencing septicemic shock.

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## CONCLUSION

The ratio of men to women is 1:1. 12% of people having DKA had diabetes before the age of 20. Diabetes mellitus type 2 typically first manifests in people between the ages of 40 and 60. A sizable portion of people with type 2 diabetes experience complications from or develop diabetic ketoacidosis. Infection is the most frequently cause for diabetic ketoacidosis, followed by neglect or inconsistent therapy. The most typical clinical signs and symptoms include nausea, vomiting, abdominal discomfort, dehydration, acidotic breathing, impaired sensory function, and fever. The clinical and the biochemical profiles of patients with the type 1 and the type 2 DM who develop ketoacidosis are not significantly different. Diabetes-related ketoacidosis has a mortality rate of 4%. Severity of the altered sensorium, severity of the comorbid disease, severity in dehydration, having severe acidosis, doses and length of insulin requiring for clearing UKB are some clinical and the biochemical indicators that may signal poor prognosis.

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