Scientific Research Publishing

Open Journal of Endocrine and Metabolic Diseases, 2025, 15(1), 8-21
https://www.scirp.org/journal/ojemd
ISSN Online: 2165-7432
ISSN Print: 2165-7424

Diagnostic and Evolutionary Aspects of Inaugural Diabetic Ketoacidosis in the Dakar Hospital Setting: A Descriptive and Analytical Cross-Sectional Study in the Endocrinology-Metabolism-Nutrition Department of CHN de Pikine

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How to cite this paper: Sall, S.A.B., Ndiaye, N., Diack, N.D., Leye, M.Y., Leye, P.A., Diallo, V.M.P.C. and Leye, A. (2025) Diagnostic and Evolutionary Aspects of Inaugural Diabetic Ketoacidosis in the Dakar Hospital Setting: A Descriptive and Analytical Cross-Sectional Study in the Endocrinology-Metabolism-Nutrition Department of CHN de Pikine. *Open Journal of Endocrine and Metabolic Diseases*, **15**, 8-21.
https://doi.org/10.4236/ojemd.2025.151002

Received: November 28, 2024
Accepted: January 20, 2025
Published: January 23, 2025

Abstract

Introduction: Diabetic ketoacidosis (DKA) is the most common metabolic complication of diabetes. Although historically reported in children, it is increasingly observed in adults. Short-term outcomes and prognosis vary widely across healthcare settings. Therefore, we aimed to determine the prevalence, diagnostic characteristics, and outcomes of newly diagnosed diabetic ketoacidosis in a hospital setting in Dakar. **Methodology:** We conducted a descriptive and analytical cross-sectional study from January 1, 2020, to January 31, 2021, in the Endocrinology-Metabolism Department of the Pikine National Hospital Center. All diabetic patients hospitalized for newly diagnosed diabetic ketoacidosis during this period were included. **Results:** A total of 54 patients were enrolled in the study. The prevalence of newly diagnosed DKA in the facility was 17.1%. The mean age was 38.54 years, with a male predominance and a sex ratio of 1.16. Thirty-five patients had a family history of diabetes. Upon admission, 14.9% of patients had altered consciousness, and 92.4% exhibited cardinal symptoms, with an average duration of 14.2 days. Kussmaul breathing and gastrointestinal symptoms were observed in 13% and 33% of cases, respectively. The mean capillary blood glucose level at admission was 3.43 g/L. Infection was identified as the precipitating factor in half of the cases, while no triggering factor was found in the remaining cases. The mean BMI

was 25.27 kg/m², and the average glycated hemoglobin (HbA1c) level was 12.2%. We recorded the diabetes classification for all patients. Nine had type 1 diabetes, twelve had type 1b diabetes, thirty-one had type 2 diabetes, and two had diabetes secondary to corticosteroid therapy. All patients received continuous intravenous insulin therapy via a syringe pump. The mean time to ketosis resolution was 34.6 hours. The average total dose of regular insulin administered was 442.3 IU. The mean duration of hospitalization was 7.6 days. One patient died, and fifty-one were discharged while continuing insulin therapy, with an average dose of 53 IU. **Conclusion:** The prevalence of newly diagnosed DKA remains high. With proper management, the prognosis is generally favorable. Diabetes classification is crucial, as it determines subsequent treatment strategies.

Keywords

Inaugural Diabetic Ketoacidosis, Ketosis-Prone Diabetes, Africa, Diabetes

1. Introduction

Diabetic ketoacidosis (DKA) is the most common acute metabolic complication of diabetes [1], marked by ketone buildup in the blood and often accompanied by acidosis [2]. It is a critical metabolic emergency with a mortality rate below 5% in recent decades, which varies depending on the patient's condition and the timeliness of therapeutic intervention [3] [4]. DKA can be the initial sign of diabetes in 15% - 30% of adults and up to 40% of children with type 1 diabetes [5] [6]. Although it is historically linked to type 1 diabetes, it is increasingly observed in adults with type 2 diabetes, especially in sub-Saharan Africa [7] [8]. This change has led to the inclusion of "ketosis-prone diabetes" (KPD) syndromes in the updated 2018 classification [6] [9]. Recognizing "atypical diabetes" is crucial, as its management and progression differ from classical diabetes [7]. While ketosis-prone diabetes appears in various populations, detailed reports from sub-Saharan Africa remain limited [7]. The short-term progression and prognosis of diabetic ketoacidosis vary across centers, affecting durations of ketosis resolution and hospital stay [10]. This study aims to identify the epidemiological profile, diagnostic features, and progression of initial diabetic ketoacidosis in a Dakar hospital.

2. Patients and Methods

This cross-sectional study, conducted from January 1, 2020, to January 31, 2021, included all patients with diabetic ketoacidosis hospitalized in the Endocrinology-Metabolism-Nutrition department at the National Hospital Center of Pikine. The diagnosis of initial diabetic ketoacidosis was based on specific criteria:

- Capillary hyperglycemia over 2.5 g/L (measured with an Accu-Chek glucometer).
- Ketonuria exceeding two crosses (determined by Ketodiastix strips) and/or pH

< 7.

- Patients not previously diagnosed with diabetes.

Pregnant women were excluded.

For each patient, the following parameters were studied:

- **Epidemiological Data:** This includes age, gender, and lifestyle.
- **Clinical Data:** Patient data included medical and surgical history, family history of diabetes, physical condition, presence of Kussmaul breathing, consciousness disturbances, cardinal symptoms, time to consultation before admission, vital signs, body measurements, and other clinical findings.
- **Paraclinical Data:** First-line tests included anti-GAD and/or anti-IA2 antibodies, alongside HbA1c, blood count, inflammatory markers, lipid profile, and blood ionogram.
- **Therapeutic and Evolutionary Data:** Key metrics included ketosis correction time, total rapid insulin doses used, metformin prescription during or soon after hospitalization, mortality, complications (e.g., hypokalemia, venous thromboembolism, infections), hospital stay length, and six-month glycemic status, including HbA1c, average glucose levels, and hypoglycemia incidence.
- **Diabetes Typing:** Diabetes classification relied on anti-GAD and/or anti-IA2 antibody tests or clinical and evolutionary factors (e.g. patient's age, morphotype, symptom onset, family history, treatment response, and exogenous insulin needs). Diabetes classifications included:
  - **Type 1 Diabetes (T1D):** Age under 40, thin or normal BMI, no family history.
  - **Type 1b/Atypical/Ketosis-Prone Diabetes (KPD):** Remission within 14 weeks post-hospitalization, insulin cessation due to hypoglycemia, HbA1c ≤ 6.5% for three months with oral medication or diet, no complications at diagnosis.
  - **Type 2 Diabetes (T2D):** Age over 40, family history of diabetes, history of macrosomia in women, complications at diagnosis.
  - **Other Diabetes Types:** Based on ADA guidelines.

Data Collection and Analysis

Data were recorded on a pre-established form and analyzed using SPSS version 18.

- **Descriptive Analysis**: Calculated frequencies and proportions for qualitative variables and means with standard deviations for quantitative variables.
- **Analytical Analysis**: Cross-tabulations, Pearson Chi-square, or Fisher's exact test was used for frequency **comparisons**. Variance analysis tested means, with significance set at $p < 0.05$. Grouped parameter analysis compared findings with existing literature in the discussion.

The text processing was done using Word XP Professional.

3. Results

- **Descriptive Study**
- **Sociodemographic Data**

In this study, 54 patients were hospitalized for inaugural diabetic ketoacidosis (DKA). The prevalence of inaugural DKA among diabetic patients admitted during this period was 17.1%. The mean age of patients was 38.54 years (range: 16–60 years). There was a male predominance, with a sex ratio of 1.16. Smoking was noted in 6 patients, and 24 patients did not engage in physical activity. Hypertension was reported in seven patients, five of whom had poor control, and two were on treatment. Two patients were undergoing corticosteroid therapy. Psychiatric follow-up was reported in two patients: one receiving haloperidol and anticholinergic therapy, and the other receiving haloperidol and chlorpromazine. Two female patients reported a history of macrosomia. A family history of diabetes was found in 35 patients, with fathers being the most commonly affected (50%), followed by mothers (44.11%).

- **Clinical Data**

Among clinical signs of DKA, severe acidosis with altered consciousness was present in 14.9% of cases (13% with confusion and 1.9% in a coma). Kussmaul breathing was observed in 13% of patients upon admission. The cardinal symptoms of diabetes were present in 92.4% of cases. The average time from the onset of cardinal symptoms to consultation was 14.2 days. Gastrointestinal symptoms were present in 18 patients (31.5%), with 11 patients (20.4%) reporting vomiting associated with abdominal pain. The average capillary blood glucose level was 3.43 g/L. Glycosuria (++ level) was observed in 57.4% of cases, while ketonuria (++ level) was noted in 40.7%.

Additional clinical signs were observed in 24 patients. Infections, identified as the precipitating factor in 50% of cases, were mostly soft tissue-related, followed by urinary tract, foot infections, and pneumonia. No precipitating factors were found in the remaining cases, and no cardiovascular events were reported (Table 1).

50 patients did not report fever at admission, and 27 presented with tachycardia. The mean BMI was 25.27 kg/m^2 (range: 17.2 - 39.5 kg/m^2), and half of the patients had abdominal obesity. The mean waist circumference was 86.9 cm (range: 56 - 152 cm) (Table 2).

Table 1. Distribution of patients based on urine test results.

Urine Test	Cases (N = 54)	Percentage (%)
Glycosuria		
++	31	57.4
+++	17	31.5
++++	6	11.1
Ketonuria		
++	22	40.7
+++	20	37.0
++++	12	22.2

Table 2. Distribution of patients by infection site and identified pathogens.

Infection Site	Cases (N=27)	Percentage (%)
Soft tissue	7	26.0
Urinary tract	6	22.2
Lungs	5	18.5
Diabetic foot	4	14.8
Blood	2	7.4
Meninges	2	7.4
Gastroenteritis	1	3.7

- **Laboratory Data**

Among non-specific findings, anemia occurred in 10 patients (18.5%). Leukocytosis with neutrophilia was noted in 24 patients (44.4%) and linked to elevated CRP in 19 cases. Acute kidney injury, resolved by rehydration, was seen in 21 patients (38.9%). At admission, 25 patients (46.3%) had dysnatremia, 12 (22.3%) had dyskalemia, and 6 showed hyperosmolarity with DKA.

In microbiology, urinary cultures were positive in 6 cases, blood cultures in 1, malaria detected in 1, pus cultures in 2, and COVID-19 PCR positive in 1 patient.

For glycemic control, the average HbA1c was 12.2% (range: 7.8% - 16.9%). Average LDL was 1.21 g/L, HDL was 0.40 g/L, and triglycerides were 1.61 g/L. Mixed dyslipidemia appeared in 14 patients (25.9%). Troponin tests were negative in all patients with repolarization abnormalities. Radiographic and ECG anomalies were found in 4 patients; the ECG showed 3 with repolarization disorders and 1 with necrosis evidence.

- **Therapeutic Data**

Patients with severe acidosis and altered consciousness were managed in intensive care units using insulin therapy via an infusion pump. Regular insulin was administered, starting at a rate of 10. The average total insulin dose was 442.3 IU (range: 80 - 1800). Twenty-nine patients (53.7%) received total doses between 100 and 400 IU of insulin. The average time for ketosis compensation was 34.6 hours (range: 4 - 172), with twenty patients (37%) compensating between 10 and 40 hours.

Rehydration was performed in all patients. The volume and type of solution varied depending on hydration status and electrolytic parameters. Rehydration began with a sodium chloride solution at a dose of 1 liter in the first hour, then was reduced in the following hours. Once the blood glucose level fell below 250 mg/dl, the sodium chloride solution was replaced with a 5% glucose solution.

Antibiotics were given to 38 patients, with 16 (42.1%) receiving ceftriaxone and 15 (39.4%) amoxicillin-clavulanic acid. Metformin was prescribed to 23 patients (43.6%) during hospitalization. Prophylactic anticoagulation was started in 38 patients (70.3%), and statins were given to those with mixed dyslipidemia. The COVID-19 positive patient was moved to an epidemic treatment center. All

patients received therapeutic education.

- **Evolutionary Data**

The average length of hospital stay was 7.6 days (range: 3 - 29). Regarding complications, two patients developed hypokalemia after insulin therapy and received oral potassium supplementation. One patient experienced worsening consciousness disturbances, requiring transfer to the intensive care unit, and subsequently died. Among the patients with diabetic foot, three had poor outcomes. One diabetic foot, classified as 2D according to the Texas classification, progressed to necrosis of the 4th toe, leading to an amputation. Another diabetic foot, also classified as 2D, progressed to gangrene of the posterior and lateral aspects of the forefoot, necessitating debridement. The amputation site developed wound suppuration with necrosis of the stump, requiring a reoperation with leg amputation.

The average total insulin dose at discharge was 53 IU (range: 24 - 104). Regarding insulin therapy continuation at discharge, 51 patients received insulin therapy. Regular premixed insulin was prescribed for 41 patients, and premixed insulin analogue for 6 patients. Two patients were placed on a basal-bolus regimen with long-acting analogue insulin (glargine) and rapid-acting analogue insulin (aspart). Two patients were placed on a basal-bolus regimen with regular premixed insulin and regular rapid-acting insulin. Two patients refused insulin therapy and were given a combination of oral antidiabetic medications.

Regarding glycemic status at 6 months, 17 patients (31%) were lost to follow-up. Insulin therapy was discontinued in 22 patients who switched to oral antidiabetic medications.

- **Diabetes Typing**

Fifteen patients underwent antibody testing. Three tested positive, and twelve tested negative. According to WHO classification, the patients were categorized as follows (Figure 1):

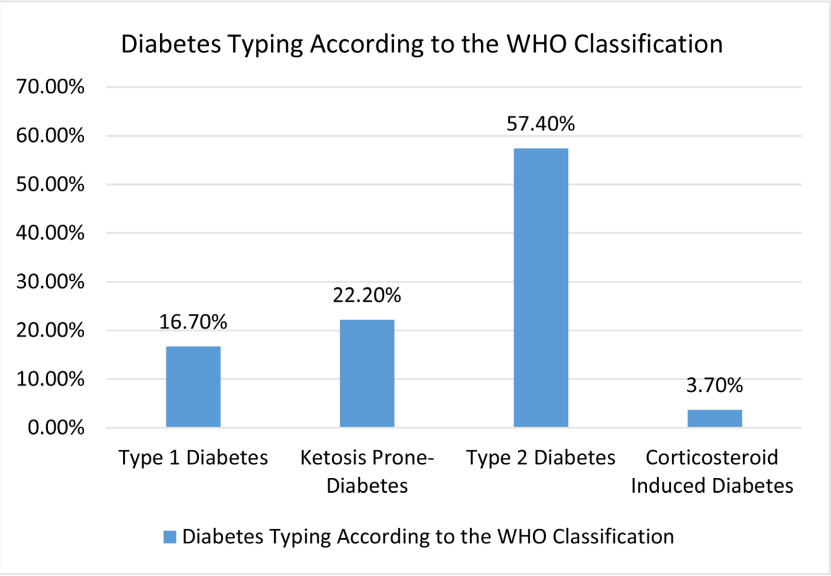

Figure 1. Diabetes typing according to the WHO classification.

Type 1 diabetes was diagnosed in 16.7% of patients.

Ketosis Prone-diabetes was diagnosed in 22.2% of patients.

Type 2 diabetes was diagnosed in 57.4% of patients.

Corticosteroid induced diabetes was diagnosed in 3.7% of patients.

➢ **Analytical Study**

- **Relationship between Consciousness Disorders and Patient Characteristics**

Table 3. Cross-tabulation between consciousness disorders and patient characteristics.

Patient Characteristics	No Consciousness Disorder	Obnubilation	Coma	P-value
Average Ketoneuria (cross)	2.76 ± 0.77	3.00 ± 0.82	4.00	0.234
Average Glucosuria (cross)	2.37 ± 0.80	2.50 ± 1.12	3	0.719
HbA1c (%)	12.12 ± 1.81	12.89 ± 1.97	15.10	0.187

The average ketonuria was higher in coma patients (4.00) than in obnibulated (3.00 ± 0.82) and conscious patients (2.76 ± 0.77). The difference was not statistically significant (P = 0.234).

The average glucosuria and ketonuria were higher in coma patients (3) than in obnibulated (2.50 ± 1.12) and conscious patients (2.37 ± 0.80). The difference was not statistically significant (P = 0.719).

The average HbA1c was higher in coma patients upon admission (15.10) than in obnibulated (12.89 ± 1.97) and conscious patients (12.12 ± 1.81), with a non-significant difference (P = 0.187) (Table 3).

- **Relationship Between Kussmaul's Dyspnea and Patient Characteristics**

Table 4. Cross-tabulation between kussmaul's dyspnea and patient characteristics.

Patient Characteristics	Presence of Kussmaul's Dyspnea	Absence of Kussmaul's Dyspnea	P-value
Average Blood Glucose (g/L)	3.50 ± 1.19	3.41 ± 1.02	0.845
Average Ketoneuria (cross)	2.86 ± 0.90	2.81 ± 0.77	0.879
Average glycosuria (cross)	2.26 ± 1.11	2.40 ± 0.80	0.89
HbA1c (%)	13.83 ± 1.93	12.05 ± 1.75	0.016

The average blood glucose levels were similar in patients with Kussmaul's dyspnea at admission (3.50 ± 1.19) and those without (3.41 ± 1.02), with no significant difference (P = 0.845).

The average ketonuria levels were also similar in patients with Kussmaul's dyspnea at admission (2.86 ± 0.90) and those without (2.81 ± 0.77), with no significant difference (P = 0.879).

However, the average HbA1c was significantly higher (P = 0.016) in patients with Kussmaul's dyspnea at admission (13.83 ± 1.93) compared to those without (12.05 ± 1.75) (Table 4).

- **Comparison of Characteristics of the Three Main Types of Diabetes Found (Type 1 Diabetes - Type 1b Diabetes - Type 2 Diabetes)**

Table 5. Characteristics of the Three Main Types of Diabetes Found (Type 1 Diabetes - Type 1b Diabetes - Type 2 Diabetes).

Patient Characteristics	Type 1 Diabetes (9 patients)	Type 1b Diabetes (12 patients)	Type 2 Diabetes (31 patients)	P-value
Age (years)	24.67 ± 6.46	35 ± 6.15	43.71 ± 9.20	0.000
Average Blood Glucose (g/L)	3.64 ± 0.93	3.36 ± 0.99	2.81 ± 0.79	0.764
Average Body Mass Index (kg/m^2)	21.73 ± 3.44	26.65 ± 6.63	25.96 ± 4.82	0.069
Normal BMI (24 patients)	8 (33.3%)	4 (16.7%)	12 (50%)	0.017
Infections	5/9 (55.5%)	2/12 (16.7%)	19/31 (61.3%)	0.03
Unspecified Causes	4/9 (44.4%)	10/12 (83.3%)	12/31 (38.7%)	0.03
Total Insulin Dose at Discharge (IU)	50.44 ± 11.78	63.83 ± 14.73	49.61 ± 23.82	0.124 (NS)
Insulin Discontinuation (patients)	0	12/12 (100%)	10/31 (32.2%)	0.0001

The average age of diagnosis for Type 1 diabetic patients (24.67 ± 6.46) was younger compared to patients with ketosis-prone diabetes (35 ± 6.15) and Type 2 diabetic patients (43.71 ± 9.20). The body mass index (BMI) was significantly lower (P = 0.069) in Type 1 diabetic patients (21.73 ± 3.44) than in patients with ketosis-prone diabetes (26.65 ± 6.63) and Type 2 diabetic patients (25.96 ± 4.82). Infections were found in 61.3% of Type 2 diabetic patients (P = 0.03), while unspecified causes of diabetic ketoacidosis were found in 83.3% of patients with ketosis-prone diabetes, with significant differences (P = 0.03). Insulin therapy was discontinued in all patients with ketosis-prone diabetes and in 32.2% of Type 2 diabetic patients, with a significant difference (P = 0.0001) (Table 5).

4. Discussion

We conducted a descriptive, cross-sectional, analytical study over 12 months. An advantage of our study was the infrequency of missing data. It included 54 patients and revealed a hospital prevalence of 17.1%. The short-term progression and prognosis of diabetic ketoacidosis (DKA) vary widely between centers, with delays in ketosis compensation and hospital stay durations being highly variable.

However, our study has some limitations:

- Financial constraints and lack of cooperation from some patients after discharge.
- Absence of antibody testing for all patients.

Epidemiological Aspects

The prevalence of inaugural DKA ranged from 15% to 30% in adults and up to approximately 30% to 40% in children with type 1 diabetes (T1D) [1] [5] [11]. In Africa, it ranged from 30% to 40%, according to different series [12] [13]. In our study, the prevalence was 17.1%.

It is well known that diabetic ketoacidosis (DKA) most commonly occurs in children and adolescents with type 1 diabetes, but it is increasingly observed in adults as well [8]. The majority of patients in our study were over 40 years old. The average age was 38.54 years, consistent with some studies: slightly higher than Mee Kyoung K's study in Korea (34.24 years) [14], and close to Thewjitcharoen Y's study in Thailand, where the average age was 37.7 years [15]. A male predominance was observed in several African and Asian studies, though the reason for this sex difference remains unclear [15]. In our study, males predominated with a sex ratio of 1.16.

According to the literature, smoking increases the risk of developing type 2 diabetes by approximately 44% [16] and induces chronic inflammation [16]. Nicotine also has a direct toxic effect on the pancreas and insulin receptors, contributing to insulin resistance [16]. In our study, 11.1% of patients were smokers. Recent studies have demonstrated that chlorpromazine (CPZ) induces hyperglycemia and glucose intolerance in humans [17]. One of our patients was treated by chlorpromazine.

Diagnostic Aspects

Consciousness disturbances were reported in 14.9% of our patients, with 1.9% in a coma. Most patients had glycosuria and ketonuria at two crosses and were admitted to a pre-coma ketoacidotic state. This aligns with the literature, where only 10% of patients are comatose upon admission [13]. The average capillary blood glucose was 3.43 g/l, lower than the 6.40 g/l reported by Thewjitcharoen Y in Tunisia [15]. We attribute this lower average to the majority of patients being referred from other facilities where intravenous insulin therapy had already begun. Average glycosuria and ketonuria were higher in patients with acidotic syndrome, though the difference was not significant. The average HbA1c was higher in patients who presented with coma or Kussmaul's breathing, also with no significant difference. This is consistent with the literature, where glycosuria and ketonuria are elevated in the stage of acidotic coma [15]. The average HbA1c in our study was 12.2%, similar to that reported by Taieb A in Tunisia (12.17%) [2] but higher than that reported by El Jadi (10.7%) [18].

In our study, infection was identified as a decompensating factor in 50% of cases. This aligns with other series, including Taieb A. (37.1%) and Chihaoui M. in Tunisia (41.3%) [2] [19]. We found a predominance of urinary tract infections, soft tissue infections, pneumonia, and infected diabetic foot ulcers [20]. These results were consistent with those of Riden D. [20]. The predominance of infectious etiology in our regions may be correlated with several factors: ignorance of diabetes, poverty, the endemicity of certain diseases such as malaria, and delays in

consultation. For 50% of the patients, no decompensating factor could be identified. Thewjitcharoen Y. found no triggering factors in 52% of their patients [15].

Nine patients (16.6%) were classified as type 1 diabetics (T1D). This proportion was lower than those reported by H. X. Liu in China and Riden D., which were 30.1% and 33.3%, respectively [20] [21]. DKA is a classic mode of discovery for T1D. The average age of T1D in our series was 24.67 years; T1D generally occurs in younger individuals. The average blood glucose at the time of diabetes discovery was higher in T1D (3.64 ± 0.93 g/l) compared to those with KPD (3.36 ± 0.99 g/l) and those with T2D (2.81 ± 0.73 g/l). This is consistent with the literature, where patients with KPD present with an initial discovery similar to that of T1D [22]. Another form of T1D occurring in older individuals is LADA. One of our patients was diagnosed with T1D at the age of 35.

Twelve patients were classified as type 1b diabetics, also known as ketosis-prone diabetes (KPD). This subtype of diabetes is situated between T1D and T2D [23]. The lack of consensus regarding its classification poses an obstacle to conducting studies on KPD. The WHO and ADA classify it as "idiopathic type 1 diabetes" or "type 1B" [23]. A study conducted over four months revealed a high frequency of KPD (28.3%) in Cameroon, suggesting it may be more widespread than previously described. The average age of onset of KPD in our study (35 ± 6.15) is similar to other studies, typically between 35 and 45 years [24]. It is higher than that of T1D (24.67 ± 6.46) and lower than that of T2D (43.71 ± 9.20), as reported in the literature. A family history of diabetes was found in 75% of patients with KPD. This frequency was higher than that reported by Brunel G. and Chihaoui M., who reported 37.5% and 57.3%, respectively [19] [24]. The difference in frequency between the studies is difficult to explain, as patients come from different socio-economic backgrounds and countries [24]. In our study, men were more frequently affected by KPD, with a sex ratio of 2, consistent with other series (58% to 76%) [24]. The average BMI at KPD discovery was 26.65 ± 6.63 kg/m^2, similar to French studies (25 - 26 kg/m^2) but differing from American studies (29 - 37 kg/m^2). An undifferentiated cause was most common in KPD patients (83.33%), showing a significant difference ($p = 0.03$), aligning with the literature. In Brazzaville, no decompensating factor was found in 65.4% of KPD patients [22].

Thirty-one patients were classified as type 2 diabetics. T2D typically occurs in patients with a family history of diabetes and may be associated with dyslipidemia. In our study, 61.8% of patients had a family history of diabetes, and 54.8% had dyslipidemia. The higher rate of ketosis in T2D compared to T1D can be explained by the study's focus on hospitalized patients in an adult endocrinology department. Some teams argue that the diagnosis of T2D during inaugural DKA may constitute a distinct entity from classic type 2 diabetes. This has led to the development of the modified French ADA classification, which has been adopted in some studies, such as Chihaoui's [19]. In this classification, diabetics discovered in an acidotic state with positive autoantibodies are classified as "type 1," while those without autoantibodies are classified as "ketosis-prone type 2 diabetes"

(equivalent to KPD). However, T2D is a latent disease that may be asymptomatic for years, especially in contexts where early screening is less common than in Western countries. Moreover, T2D can present with ketosis in the presence of a triggering factor, even though ketosis is not a classic initial presentation of the disease. It will, therefore present as an acute initial case with severe hyperglycemia and ketosis, indicating acute insulin deficiency, similar to T1D. Furthermore, infection was found more frequently in T2D (61.3%) with a significant difference ($p = 0.03$). T2D can present with ketosis in the presence of a precipitating factor. Hence, we chose to classify our patients according to the WHO classification. Other studies have also preferred to adopt the ABeta classification, as in the study by Taieb [2]. Thus, the search for pancreatic autoantibodies and the assessment of beta cell secretion capacity through C-peptide measurement may be helpful. Unfortunately, these biological parameters are not routinely available in our country, so we could not adopt this classification.

Two patients developed diabetes due to corticosteroid therapy. Among patients hospitalized in internal medicine, approximately 11% receive high doses of corticosteroids (>40 mg prednisone/day for at least 48 hours), with 64% experiencing at least one episode of hyperglycemia and nearly half developing diabetes [18]. One patient had lupus and the other had vasculitis. They were on 40 mg prednisone for 5 months and 60 mg prednisone for 4 months, respectively, without dose reduction as they were lost to follow-up.

The HbA1c level at diagnosis was not discriminative in our study and was nearly equal across the three patient groups. This is consistent with the results of Brunel [24].

Therapeutic and Evolutionary Aspects

Intravenous insulin therapy and rehydration were implemented for all our patients. The average length of hospitalization was 7.6 days. Riden D. [20] reported an average hospitalization duration of 8.5 days. The main complication observed was hypokalemia, affecting 13% of our patients. DKA is associated with a potassium deficit, which can initially be masked by hyperkalemia due to acidosis, proteolysis, and insulin deficiency [25]. The correction of these disorders through hydration and insulin therapy can reveal this deficit. Ach Taieb and his team reported fewer cases of hypokalemia (2.8% compared to 13% in our study) [2]. One patient experienced worsening consciousness disturbances, resulting in death. The mortality rate reported in the literature is less than 1%.

At the time of DKA discovery, patients with KPD typically require insulin therapy [26]. Subsequently, hypoglycemic episodes are observed in most patients. After the acute episode, the clinical course resembles classic T2D and may not require insulin. Most of these patients remain in near-normoglycemic remission without insulin for several months or years. The remission phase is defined by insulin withdrawal with an HbA1c level below 6.5% for more than 3 months. In the literature, this remission typically begins between 9 and 14 weeks after the discovery of diabetes and can last for several years [26]. In our study, patients experienced hypoglycemic episodes that

required insulin discontinuation within the first three months, with HbA1c remaining below 6.5% for more than 3 months.

5. Conclusion

In conclusion, inaugural diabetic ketoacidosis is common among overweight adults with Type 2 diabetes and often occurs in Ketosis-Prone Diabetes (Type 1b). Proper typing is crucial for guiding treatment and follow-up. Distinguishing Type 1 from Ketosis-Prone and Type 2 diabetes requires antibody testing, which is unavailable to all patients. However, rapid remission within 14 weeks post-hospitalization helps diagnose ketosis-prone diabetes retrospectively. Under specialized care, these patients generally show favorable outcomes. Since undiagnosed diabetes increases the risk of DKA and diabetes prevalence is high in our area, we recommend more mass diabetes screenings.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Elzouki, A. and Eledrisi, M. (2020) Management of Diabetic Ketoacidosis in Adults: A Narrative Review. *Saudi Journal of Medicine and Medical Sciences*, **8**, 165-173. https://doi.org/10.4103/sjmms.sjmms_478_19

[2] Taieb, A., Cheikh, A.B., Hasni, Y., Maaroufi, A., Kacem, M., Chaieb, M., *et al.* (2018) Etude sur le diabète aigu cétosique inaugural dans un hôpital du Centre-Est Tunisien. *Pan African Medical Journal*, **31**, Article 134. https://doi.org/10.11604/pamj.2018.31.134.12207

[3] Jouini, S., Aloui, A., Slimani, O., Hebaieb, F., Kaddour, R.B., Manai, H., *et al.* (2019) Profils épidémiologiques des acidocétoses diabètiques aux urgences. *Pan African Medical Journal*, **33**, Article 322. https://doi.org/10.11604/pamj.2019.33.322.17161

[4] Orban, J. and Ichai, C. (2008) Complications métaboliques aiguës du diabète. *Réanimation*, **17**, 761-767. https://doi.org/10.1016/j.reaurg.2008.09.006

[5] Klingensmith, G.J., Tamborlane, W.V., Wood, J., Haller, M.J., Silverstein, J., Cengiz, E., *et al.* (2013) Diabetic Ketoacidosis at Diabetes Onset: Still an All Too Common Threat in Youth. *The Journal of Pediatrics*, **162**, 330-334.e1. https://doi.org/10.1016/j.jpeds.2012.06.058

[6] Gaba, R., Mehta, P. and Balasubramanyam, A. (2019) Evaluation and Management of Ketosis-Prone Diabetes. *Expert Review of Endocrinology & Metabolism*, **14**, 43-48. https://doi.org/10.1080/17446651.2019.1561270

[7] Lontchi-Yimagou, E., Nguewa, J.L., Assah, F., Noubiap, J.J., Boudou, P., Djahmeni, E., *et al.* (2016) Ketosis-prone Atypical Diabetes in Cameroonian People with Hyperglycaemic Crisis: Frequency, Clinical and Metabolic Phenotypes. *Diabetic Medicine*, **34**, 426-431. https://doi.org/10.1111/dme.13264

[8] Balasubramanyam, A. (2019) Syndromes of Ketosis-Prone Diabetes. *Transactions of the American Clinical and Climatological Association*, **130**, 145-155.

[9] American Diabetes Association (2017) 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes—2018. *Diabetes Care*, **41**, S13-S27. https://doi.org/10.2337/dc18-s002

[10] Große, J., Hornstein, H., Manuwald, U., Kugler, J., Glauche, I. and Rothe, U. (2018) Incidence of Diabetic Ketoacidosis of New-Onset Type 1 Diabetes in Children and Adolescents in Different Countries Correlates with Human Development Index (HDI): An Updated Systematic Review, Meta-Analysis, and Meta-Regression. *Hormone and Metabolic Research*, **50**, 209-222. https://doi.org/10.1055/s-0044-102090

[11] Dabelea, D., Rewers, A., Stafford, J.M., Standiford, D.A., Lawrence, J.M., Saydah, S., *et al.* (2014) Trends in the Prevalence of Ketoacidosis at Diabetes Diagnosis: The SEARCH for Diabetes in Youth Study. *Pediatrics*, **133**, e938-e945. https://doi.org/10.1542/peds.2013-2795

[12] Nemi, K.D., Djalogue, L., Djagadou, K.A., Tchamdja, T., Tsevi, Y.M. and Balaka, A. (2019) Les modes de révélation du diabète sucré au CHU Sylvanus Olympio de Lomé. *Pan African Medical Journal*, **34**, Article 99. https://doi.org/10.11604/pamj.2019.34.99.20012

[13] Leye, Y.M., Leye, A., Ndiaye, N., Diack, N., Ngo Bikai, V., Toure, P.S., *et al.* (2016) Aspects épidémiologiques et diagnostiques de la cétoacidose diabétique en milieu hospitalier à Dakar. Analyse de 102 cas au CHU de Pikine. *RAFMI*, **3**, 8-11.

[14] Kim, M.K., Lee, S.H., Kim, J.H., Lee, J.I., Kim, J.H., Jang, E.H., *et al.* (2009) Clinical Characteristics of Korean Patients with New-Onset Diabetes Presenting with Diabetic Ketoacidosis. *Diabetes Research and Clinical Practice*, **85**, e8-e11. https://doi.org/10.1016/j.diabres.2009.04.017

[15] Thewjitcharoen, Y. and Sunthornyothin, S. (2010) Clinical Characteristics of Diabetic Ketoacidosis in Newly Diagnosed Adult Patients. *Diabetes Research and Clinical Practice*, **90**, e43-e45. https://doi.org/10.1016/j.diabres.2010.08.014

[16] Willi, C., Bodenmann, P., Ghali, W.A., Faris, P.D. and Cornuz, J. (2007) Active Smoking and the Risk of Type 2 Diabetes. *JAMA*, **298**, 2654-2664. https://doi.org/10.1001/jama.298.22.2654

[17] Erle, G., Basso, M., Federspil, G., Sicolo, N. and Scandellari, C. (1977) Effect of Chlorpromazine on Blood Glucose and Plasma Insulin in Man. *European Journal of Clinical Pharmacology*, **11**, 15-18. https://doi.org/10.1007/bf00561782

[18] El Jadi, H., Guerboub, A.A., Meftah, A., Moumen, A., Errahali, Y., Chakdoufi, S., *et al.* (2015) Statut dégénératif chez 105 patients admis pour cétose inaugurale. *Annales d'Endocrinologie*, **76**, 528. https://doi.org/10.1016/j.ando.2015.07.766

[19] Chihaoui, M. (2012) Characteristics of Ketosis-Onset Diabetes in Tunisian Population. *Journal of Diabetes & Metabolism*, **3**, Article ID: 1000175. https://doi.org/10.4172/2155-6156.1000175

[20] Riden, D., Khessairi, N., Chaker, F., Oueslati, I., Yazidi, M. and Chihaoui, M. (2020) Cétose diabétique inaugurale: Facteurs de décompensation. *Annales d' Endocrinologie*, **81**, 450-451. https://doi.org/10.1016/j.ando.2020.07.884

[21] Liu, H.X., Li, G.M., Zhou, Y.W., Luo, S.H., Zheng, X.Y., Yang, D.Z., *et al.* (2019) Clinical Characteristics and Classification Diagnosis of Newly Diagnosed Diabetes Onset with Ketosis or Ketoacidosis in Adult Patients]. *Chinese Medical Journal*, **99**, 1369-1374.

[22] Umpierrez, G.E., Smiley, D. and Kitabchi, A.E. (2006) Narrative Review: Ketosis-Prone Type 2 Diabetes Mellitus. *Annals of Internal Medicine*, **144**, 350-357. https://doi.org/10.7326/0003-4819-144-5-200603070-00011

[23] Umpierrez, G.E. and Kitabchi, A.E. (2003) Diabetic Ketoacidosis: Risk Factors and Management Strategies. *Treatments in Endocrinology*, **2**, 95-108. https://doi.org/10.2165/00024677-200302020-00003

[24] Brunel, G. (2015) Le diabète atypique à tendance cétosique de l'adulte originaire d'Afrique subsaharienne: Étude de 16 patients recensés en Seine-Maritime. Thèse Medicine Unirouen: Université de Rouen UFR santé, 86 p.

[25] Kitabchi, A.E. and Nyenwe, E.A. (2006) Hyperglycemic Crises in Diabetes Mellitus: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State. *Endocrinology and Metabolism Clinics of North America*, **35**, 725-751. https://doi.org/10.1016/j.ecl.2006.09.006

[26] Mauvais-Jarvis, F., Sobngwi, E., Porcher, R., Riveline, J., Kevorkian, J., Vaisse, C., *et al.* (2004) Ketosis-Prone Type 2 Diabetes in Patients of Sub-Saharan African Origin: Clinical Pathophysiology and Natural History of β-Cell Dysfunction and Insulin Resistance. *Diabetes*, **53**, 645-653. https://doi.org/10.2337/diabetes.53.3.645