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Admissions for diabetic ketoacidosis in ethnic minority groups in a city hospital

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Abstract

Hospitalization for diabetic ketoacidosis (DKA) is increasing, perhaps due to the rising incidence of DKA in patients with type 2 diabetes mellitus (T2DM). Ethnic minority groups are at increased risk for T2DM. This study aimed at elucidating the characteristics of patients with ketosis-prone diabetes in a predominantly ethnic minority population. We performed a retrospective analysis of adults admitted with DKA at the Bronx Lebanon Hospital Center, Bronx, NY over 3 years. The patients were divided into cohorts based on type of diabetes and ethnicity. The cohorts were described and compared using statistical methods. We recorded 219 cases of DKA in 168 patients, 97% of whom were African American or Hispanic. Fifty-three (32%) patients had T2DM. New-onset diabetes, which was more common in T2DM ($P < .0001$), and African Americans ($P = .008$), occurred in 42 patients (25%). Readmission with DKA was more common in the Hispanic patients with type 1 diabetes mellitus (T1DM) ($P = .0001$). Type 2 diabetes mellitus was more prevalent in the African Americans ($P = .04$). Patients with T1DM had more severe acidosis than patients with T2DM (lower pH and bicarbonate and larger anion gap; $P = .03$, .02, and .005, respectively). Creatinine level was higher in patients with T2DM ($P = .04$) who were also less likely to have identifiable precipitating causes ($P = .02$). Hemoglobin A_{1c} level was higher in patients with new-onset diabetes ($P < .05$), but did not differ between those with T1DM and T2DM. Mortality, which was 2%, occurred only in the African Americans with T2DM. We conclude that DKA is an important mode of initial presentation of T2DM, with new-onset T2DM accounting for about 60% of all new cases of DKA. African American patients with T2DM, in comparison with the Hispanic patients, are more susceptible to developing DKA. Diabetic ketoacidosis could occur in T2DM without any identifiable precipitant. The rising incidence of DKA may be attributable to its increasing occurrence in T2DM; therefore, measures aimed at primary prevention of T2DM are worthwhile.

1. Introduction

The burden of diabetes in the United States continues to rise, especially among the ethnic minority groups (Blacks, Hispanics, and Native Americans), where its prevalence is 2 to 4 times higher compared with the majority population [1]. This high prevalence rate is largely due to type 2 diabetes mellitus (T2DM), which accounts for 90% to 95% of the cases. Hospital admission for diabetic ketoacidosis (DKA) is also on the increase [2], perhaps due to the rising incidence of DKA in patients with T2DM.

A previous study showed that 93% of patients diagnosed with DKA as initial manifestation of diabetes had T2DM after a follow-up period of at least two and a half years [3]. Although DKA is thought to be a far more characteristic feature of type 1 diabetes mellitus (T1DM), which is seen in T2DM under conditions of severe stress [4], significant proportions of patients from ethnic minority groups who presented with DKA have been found to have T2DM [3,5,6].

Furthermore, patients with T2DM presenting with DKA differ in some respects from the typical type 1 diabetic patients [3,5,7]. They are more likely to be obese and show absence of autoimmune markers [5,6,8]. However, although Banerji et al [6] found HLA association in African

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Americans with T2DM who presented with DKA, Umpierrez and coworkers [5] did not. Other studies have identified clinical and biochemical differences between patients with T1DM and T2DM [3,7]. These differences have raised the question of heterogeneity in ketosis-prone diabetes [9,10]. Studies that would foster understanding of the clinical and biochemical profiles of these patients should be helpful in their classification and long-term management. Given the high number of ethnic minority patients seen at the Bronx Lebanon Hospital Center (BLHC), Bronx, NY, we hypothesized that T2DM would be an important contributor to DKA cases seen at BLHC. This study therefore aimed at elucidating the characteristics of patients with ketosis-prone diabetes in a population at a higher risk for diabetes.

2. Research design and methods

We performed a retrospective analysis of hospital admissions for DKA in patients aged 18 years and older admitted to the BLHC between July 2001 and June 2004. Computer records were used to identify DKA admissions, using the *International Classification of Diseases, Ninth Revision* code for DKA (250.1). All available medical records of the identified patients were reviewed. Patients who met the diagnostic criteria below had their data on demographics, personal and family history of diabetes, social habits, precipitants of DKA, and biochemical profiles collected in an abstraction form. Records revealed 337 admissions, of which 100 cases were excluded for not meeting the diagnostic criteria. Medical records were not available for review in 18 admissions, which were also excluded from this study. Thus, we reviewed 219 cases of DKA over a 3-year period.

2.1. Ethical considerations

The institutional review board of the BLHC approved this study. Confidentiality was maintained throughout the study.

2.2. Diagnostic criteria for DKA

Diabetic ketoacidosis is defined as the presence of the following.

1. Blood glucose of 250 mg/dL or higher.
2. Ketonemia determined by positive moderate to severe nitroprusside reaction in the serum.
3. Acidosis demonstrated by 1 or more of the following:
   a. Anion gap of more than 14.
   b. pH of 7.30 or lower.
   c. Serum bicarbonate level of less than 15 mEq/L.
4. Ketosis and acidosis should not be due to poisoning, alcohol intoxication, hyperemesis gravidarum, or preexisting intrinsic renal failure.

2.3. Diagnostic criteria for diabetes

Type 1 diabetes mellitus.

1. Previous ketosis or
2. Exclusive insulin therapy since diagnosis.

Type 2 diabetes mellitus.

1. Features of insulin resistance (obesity, acanthosis nigricans, hypertension, hyperlipidemia), or
2. History of control with oral hypoglycemic agents and
3. No previous ketosis.

Table 1
Clinical and biochemical profiles of patients

	T1DM (n = 112)		T2DM (n = 51)		*P* (type 1 vs type 2)
	African American	Hispanic	African American	Hispanic	
No. of patients	55	57	33	18	.04[a]
No. of admissions (% readmission)	70 (27.3)	90 (57.9)	NA	NA	.0001[b]
Age (y)	34.1 ± 12.8	31.1 ± 11.2	52.8 ± 14.3	48.5 ± 10.9	<.0001
Sex (M/F)	28/27	33/24	18/15	14/4	.32
Family history of DM (%)	18 (32.7)	13 (22.82)	13 (39.4)	9 (50.0)	.05
Duration of DM (y)	9.1 ± 7.2	11.2 ± 8.0	17.5 ± 12.6	4.3 ± 1.5	.83
New onset (%)	13 (23.6)	5 (8.8)	17 (51.5)	7 (38.9)	<.0001
BMI (kg/m^2)	24.1 ± 4.7	24.5 ± 5.8	32.1 ± 7.7	32.5 ± 8.4	<.0001
Patients with precipitant (%)	44 (80)	45 (79.8)	19 (57.6)	12 (70.6)	.02
Glucose (mg/dL)	641.4 ± 267.8	631.4 ± 192.3	608.1 ± 227.5	620.7 ± 287.6	.55
pH	7.18 ± 0.12	7.17 ± 0.12	7.20 ± 0.11	7.23 ± 0.07	.03
HCO_3 (mEq)	13.3 ± 5.7	14.7 ± 5.6	14.6 ± 6.3	15.2 ± 4.2	.02
Anion gap	25.0 ± 8.6	25.5 ± 7.5	21.9 ± 7.5	21.0 ± 4.8	.005
Plasma osmolality (mOsm/L)	309.2 ± 20.2	304.7 ± 15.8	306.4 ± 27.1	305.4 ± 24.4	.83
HbA_{1c}	13.5 ± 2.7	12.8 ± 1.9	13.0 ± 2.5	11.3 ± 1.8	.33
Potassium (mmol/L)	5.3 + 1.1	5.2 + 0.8	5.2 + 0.9	4.7 + 1.1	.07
Creatinine (mg/dL)	1.46 + 0.87	1.21 + 0.47	1.60 + 0.96	1.80 + 1.1	.04
Mortality	0	0	4	0	

NA indicates not applicable; BMI, body mass index.
[a] Compares the ethnic groups with T2DM.
[b] Compares the ethnic groups with T1DM.

Table 2
Precipitants of DKA

Precipitant	Frequency (%)
Noncompliance	96 (43.8)
Infection	57 (26.0)
Pancreatitis	12 (5.5)
Acute myocardial infarction	5 (2.3)
Pregnancy	4 (1.8)
Acute asthmatic attack	3 (1.3)
Gastrointestinal bleeding	3 (1.3)
Deep vein thrombosis	1 (0.5)
Trauma with fracture	1 (0.5)
Angina	1 (0.5)
Pericarditis	1 (0.5)
Change in insulin regimen	1 (0.5)
Total	219 (100)

2.4. Treatment

All patients were managed initially in the emergency department based on American Diabetes Association guidelines [11] and were transferred to the intensive care unit as soon as possible. Intravenous infusion of normal saline was given for fluid repletion; this was replaced with dextrose infusion when blood glucose was less than 250 mg/dL. Each patient was started on intravenous regular insulin infusion at the rate of 4 to 6 U/h, after a bolus dose of 10 U intravenously. Blood glucose was monitored hourly and insulin drip was adjusted accordingly to reduce blood glucose by 50 to 70 mg/dL every hour. Serum electrolytes and venous pH were monitored every 2 hours. A flow chart of the blood glucose, serum electrolytes, venous pH, and fluids given was maintained. Electrolyte abnormalities—hypokalemia, hypophosphatemia—were corrected as indicated. Nine patients with severe acidosis (pH <7.00) were given sodium bicarbonate infusion. Insulin drip was continued until acidosis and ketosis resolved. As soon as patients could eat, long-acting insulin was started 2 hours before regular insulin drip was discontinued. The patients were managed by the intensive care unit team, which consisted of the attending critical care physician and residents in internal medicine. An attending endocrinologist reviewed the patients as soon as possible.

Noncompliance in this study was defined as omission of insulin therapy or oral hypoglycemic agents.

2.5. Data management and analysis

The data obtained were coded and entered into a PC for analysis using Epi Info 6 statistical package (Centers for Disease Control, Atlanta, GA) and SAS (SAS Institute, Cary, NC) as software. The patients were divided into 2 major groups (T1DM and T2DM) and were further subclassified depending on ethnicity. Descriptive statistics such as means and SD were used to characterize the groups. Discrete variables (eg, sex, family history of diabetes) between the groups were compared using χ^2/Fisher exact test. Continuous variables (eg, age, body mass index [BMI], pH, glucose) were compared using analysis of variance.

3. Results

We identified 219 cases of DKA, which occurred in 168 patients, giving average recurrence rate of 1.30 per patient or 30%. Hispanic patients with T1DM had significantly more readmissions than the African American patients—58% and 27% respectively ($P = .0001$) (see Table 1).

3.1. Demographics

There were 72 (43%) females and 96 (57%) males, giving a male-to-female ratio of 1.33:1. The mean age was 38.6 ± 14.8 years for the entire population, 39.2 ± 17.8 years for females, and 38.2 ± 12.2 years for the males. Nearly all (97%) of the patients were African Americans or

Table 3
Comparison of the clinical and biochemical parameters of the patients in this study with those of a similar study

	This study		Balasubramanyam et al [3]	
	T1DM	T2DM	T1DM	T2DM
No. of patients	112	51	75	55
No. of admissions (mean)	160 (1.4)	55 (1.0)	99 (1.14)	57 (1.04)
Age (y)	32.6 ± 11.7	47.5 ± 13.2	35.5 ± 10.1	47.3 ± 13.7
Sex (M/F)	61/51	32/19	42/32	34/23
New onset	18	24	3	26
BMI (kg/m^2)	24.3 ± 5.3	32.2 ± 7.8	24.2 ± 7.8	30.5 ± 7.5
Race/ethnicity				
African American	55	33	28	35
Hispanic	57	18	15	20
Patients with precipitant (%)	89/112 (79.5)	31/55 (60.8)	30/96 (31)	14/31 (45.2)
Glucose (mg/dL)	636.3 ± 231.7	612.5 ± 247.5	628.2 ± 316.8	730.8 ± 428.4
pH	7.17 ± 0.12	7.21 ± 0.09	7.14 ± 0.12	7.17 ± 0.12
HCO_3 (mEq)	13.5 ± 5.7	16.4 ± 5.6	13.0 ± 13	12.0 ± 4.0
Anion gap	26.7 ± 8.0	23.5 ± 6.6	27.8 ± 8.0	25.0 ± 7.0
Plasma osmolality (mOsm/L)	306.9 ± 18.2	306.4 ± 25.8	314 ± 23	318 ± 33
Potassium (mmol/L)	5.3 + 0.9	5.0 + 0.9	5.2 + 1.2	4.9 + 1.2
Creatinine (mg/dL)	1.46 + 0.70	1.95 + 1.01	1.80 + 1.3	1.80 + 1.09

Table 4
Causes of noncompliance

Factors	Noncompliant	Compliant	P
Cocaine			
Users	33	23	.01
Nonusers	63	100	
Cannabis			
Users	20	13	.04
Nonusers	72	110	
Smoking			
Active smokers	50	43	.01
Nonsmokers	46	80	
Alcohol			
Users	41	48	.58
Nonusers	55	75	
Insurance			
Insured	68	79	.30
Noninsured	28	44	

Hispanics, and their clinical and biochemical profiles are shown in Table 1. Five (3%) of the patients were whites or Asians. They were not entered in the analysis in Table 1 because of the small number. Patients with T1DM were younger than those with T2DM; the mean ages were 32.7 ± 11.7 and 51.1 ± 12.9 years, respectively ($P < .0001$).

3.2. Family history of diabetes

Patients with T2DM were more likely to have family history of diabetes than those with T1DM—43% and 27%, respectively ($P = .005$).

3.3. Duration of diabetes

The mean duration of diabetes among the previously diagnosed cases was 9.9 ± 7.8 years. Thirty-seven percent of the DKA episodes occurred within the first 5 years of being diagnosed with diabetes, and 66.3% occurred within 10 years of diagnosis. There were no statistically significant differences between the types of diabetes and the ethnic groups as shown in Table 1.

3.4. Obesity

Patients with T2DM were more obese than the patients with T1DM. Body mass index for the 2 groups were 24.3 ± 5.3 for type 1 diabetic and 32.2 ± 7.8 for type 2 diabetic patients ($P < .0001$).

3.5. Precipitating factors for DKA

The precipitating causes are outlined in Table 2.

No precipitant was found in 34 (15%) cases. Common infections identified were abscesses, sepsis, urinary tract infections, and upper respiratory tract infections. Patients with T2DM were less likely to have identifiable precipitating cause—62% against 79% in type 1 diabetic patients ($P = .02$). No ethnic differences were observed in this regard.

3.6. Types of diabetes

Fifty-three (31.5%) patients had T2DM. New-onset diabetes occurred in 42 (25%) patients, 57.1% of whom were type 2 diabetic patients. Of the 51 patients with T2DM, 24 (47%) had new-onset disease compared with 16% among type 1 diabetic patients ($P < .0001$). The African American patients had significantly more episodes of new-onset diabetes than the Hispanic patients—34% vs 16% ($P = .008$); 70% of the new-onset disease in T2DM occurred in African Americans. The prevalence of T1DM was equal in both ethnic groups (55:57), but T2DM was more frequent among the African American patients ($P = .04$) (see Table 1).

3.7. Biochemical profile

Patients with T1DM had more severe acidosis as shown by lower serum pH and bicarbonate levels and larger anion gap ($P = .03$, .02, and .005, respectively), but patients with T2DM had higher creatinine levels than type 1 diabetic patients ($P = .04$). Hemoglobin A_{1c} (HbA_{1c}) was significantly higher in those with new-onset diabetes (13.5 ± 2.1) compared with those previously known to have diabetes (12.4 ± 2.4) ($P < .05$).

The HbA_{1c} levels were comparable between the types of diabetes and the ethnic groups. Nineteen percent of the patients had calculated plasma osmolality of more than 320 mOsm/L. There were no statistically significant differences in plasma osmolality between the types of diabetes.

Table 3 compares the clinical and biochemical characteristics of the patients in this study with those of another study (reference [3]).

3.8. Noncompliance and DKA

Of the 219 episodes of DKA, 96 (44%) occurred in patients who were noncompliant. There was no difference

Table 5
Characteristics of the deceased patients

Patient's ID	A	B	C	D
Age (y)	42	85	62	75
Sex	F	F	M	F
Race	AA	AA	AA	AA
BMI (kg/m^2)	19.9	26.6	53.1	28.1
Type of DM	2	2	2	2
Precipitant/cause of death	AIDS/lymphoma, septic shock	Acute myocardial infarction	Pneumonia/septic shock	Septic shock
pH	7.27	7.29	7.24	7.10
Anion gap	30	20	18	31
Glucose (mg/dL)	298	635	634	999
Plasma osmolality (mOsm)	310	311	298	360

between the ethnic groups in terms of compliance ($\chi^2 = 7.9$, $P = .16$), but patients with T1DM were more noncompliant than those with T2DM ($\chi^2 = 18.8$, $P < .0001$). Factors associated with noncompliance included cocaine use, cannabis, and cigarette smoking. There was no association between accessibility to health care (measured by medical insurance rates) and noncompliance ($P = .30$) (see Table 4). There were no ethnic differences in medical insurance rates ($\chi^2 = 0.72$, $P = .40$).

3.9. Outcome

Of the 168 patients, 4 (2.4%) died. All these patients had recovered biochemically from DKA, but died of complications of the primary disease, which precipitated the DKA episode. The characteristics of the deceased patients are shown in Table 5. Of the 4 deaths, 3 were due to infection. Although there was no ethnic difference in infection rate ($\chi^2 = 0.03$, $P = .87$), the African Americans had more severe infections than the Hispanics (5 cases of sepsis in the African Americans vs 1 in the Hispanics).

4. Discussion

The results of this study showed that patients with T1DM were much younger and had more severe metabolic derangement as shown by acidosis than those with T2DM. Several other studies recorded younger type 1 diabetic patients [3,5,7]. We may therefore infer that the younger a patient is, the higher the chance he or she may have T1DM. However, T2DM occurs in pediatric patients as well, especially among the ethnic minority groups [11-16]. Late-onset T1DM or latent autoimmune diabetes in adults is also well known [17,18]. It accounts for 2% to 12% of all diabetic cases [19]. Up to 10% of Swedish patients aged 40 to 75 years with newly diagnosed diabetes were found to have immunologic markers for T1DM [20]. The finding of more severe metabolic abnormalities agrees with the reports of other workers [5,7], although another study [3] did not find any significant differences in the biochemical parameters between T1DM and T2DM. Patients with T1DM were also more likely to have identifiable precipitating factors for DKA. It seems plausible that the stress posed by the precipitant in the internal milieu of insulin deficiency produced more severe metabolic derangements in type 1 diabetic patients compared with type 2 diabetic patients. It has been shown that African American [5,21] and Hispanic [22] patients with T2DM have relatively well-preserved beta-cell secretory function as shown by C-peptide levels. The higher insulin levels in these patients mitigate against lipolysis and ketogenesis in T2DM, thus producing less acidosis. It should therefore take higher levels of stress and counter-regulatory hormones to produce DKA in type 2 diabetic patients [4] because the underlying pathogenetic mechanism in DKA is imbalance between insulin and its counter-regulatory hormones [23]. However, this study and indeed others [3,7] have shown that type 2 diabetic patients were less likely to have identifiable precipitating cause. Insulin resistance, which occurs in hyperglycemic crises [23], could worsen the state of relative insulin deficiency in T2DM and contribute to the events in DKA in the absence of a precipitating cause. Insulin resistance may explain the longer time needed for resolution of DKA in patients with T2DM, although they had less severe acidosis [7]. Chronic hyperglycemia has been shown to impair insulin action and glucose uptake in peripheral tissues [24]. Secondly, it has been shown that a state of transient insulinopenia exists in patients with ketosis-prone T2DM during an episode of DKA, which resolves after ketosis or hyperglycemia [21]. This sudden decompensation in beta-cell secretory function may explain the emergence of DKA without any apparent cause. The reason for this transient beta-cell failure is not known with certainty, but hypothesized mechanisms include glucotoxicity, lipotoxicity, and genetic preposition. It is also possible that psychological stress, which has not been measured in previous studies, may be a contributor in the patients in whom no precipitants were found. Studies that would compare the levels of insulin or C-peptide and counterregulatory hormones in type 1 and type 2 diabetic patients with DKA should be helpful in clarifying this paradox.

Patients with T2DM were more obese and more likely to have family history of diabetes and higher creatinine levels than type 1 diabetic patients. The higher creatinine levels in T2DM may be related to early onset of vascular complications. It is well known that more than 25% of patients with T2DM have complications at the time of diagnosis [25,26]; whereas nephropathy takes at least 10 years to develop in type 1 diabetic patients, a type 2 diabetic patient may have nephropathy at diagnosis [27,28].

In this study, we did not use autoantibodies such as anti–glutamic acid decarboxylase antibody (GAD 65) and C-peptide levels, which have been used in some clinical trials to classify diabetes; this constitutes a limitation of our study. We recognize that it would have been ideal to classify diabetes based on immune markers and C-peptide levels, but these measurements were not available. Therefore, we adopted clinical criteria to classify our patients as has been done in other studies [3,29]. In a more recent study, Otiniano et al [30] showed that the metabolic syndrome (which is included in our criteria) in a patient with DKA was a marker for T2DM, better preserved beta-cell function, and glycemic control, regardless of ethnicity. Classifying individuals with recurrent DKA as type 1 diabetic patients, as we did, could have placed some patients with T2DM among type 1 diabetic patients. However, the chances that this would have occurred is less than 1 in 25 because less than 4% of type 2 diabetic patients were found to have recurrent DKA in a study that followed patients over 2 and a half to 4 years after initial episode of DKA [3]. Although autoantibodies are accepted for classifying diabetes, they are not without a potential for confounding the process. Up to 10% to 17% of patients with T2DM have autoantibodies, whereas only 65% to 80% of type 1 diabetic patients have autoantibodies [5,31,32]. Therefore, even if

these markers were used, there is a chance that some patients could have been misclassified. Although the prevalence of T2DM in our study (32%) is similar to that of 2 other studies (26%-39%) [3,29], we wish to acknowledge that using exclusive insulin treatment to classify patients as having T1DM, as we did, could have underestimated the number of type 2 diabetic patients, especially those with new-onset diabetes. Some of these patients could have remained on insulin therapy because they were never tried on oral agents.

If the findings of this study and the others are validated in a properly controlled prospective investigation, it may be possible to derive a predictive model that could help categorize patients at the bedside. Such a study is ongoing and some data have already been published. The predictors that have been derived include preserved beta-cell mass [22,30,33], compliance with lifestyle modification [34], new-onset diabetes [35], presence of metabolic syndrome [30], and Hispanic ethnicity [29,30].

Diabetic ketoacidosis was the initial presentation of diabetes in 25% of our patients in whom 60% were found to have T2DM. African Americans comprised about 70% of this group. Umpierrez et al [21] reported that 25% to 50% of African American and Hispanic patients with new diagnosis of DKA had ketosis-prone T2DM. The inference we draw from this observation is that 3 of every 5 patients presenting with DKA as initial manifestation of diabetes may have T2DM. The chances are even higher if they are African American, obese, or older than 40 years. Because DKA is a potentially fatal and expensive disease, measures aimed at primary prevention of T2DM would be worthwhile.

We identified some differences between the African American and Hispanic patients. Whereas readmission was significantly higher in the Hispanic population, new-onset diabetes was more prevalent among the African Americans. Type 2 diabetes mellitus was more frequent in the African Americans compared with the Hispanic patients, thus demonstrating a clinical trend that DKA is an important presentation of T2DM particularly in the African American patient. Other workers had noted increased incidence of T2DM in African Americans who presented with DKA [6-8,21,30,33]. However, another study found a higher proportion of T2DM in Hispanic patients compared with the African Americans [3]. All the 4 patients who died in the period reviewed in this study were African Americans with T2DM. This may be due to the preponderance of more severe infection (sepsis) in African Americans. However, it is noteworthy that the higher mortality rate recorded in this study is consistent with the trend demonstrated by the National Diabetes Surveillance System, where the mortality from DKA in blacks is about 3-fold that of whites [2]. The reason for more frequent readmission in Hispanic patients with TIDM remains unclear. We explored access to health care, using medical insurance rates as surrogate, precipitants of DKA, and compliance with treatment, but found no differences between the ethnic groups. Although the reason for this finding remains unapparent, it can be applied clinically for better patient care. The message here is that a Hispanic patient with T1DM should be followed up closely to prevent recurrent DKA.

In conclusion, DKA is an important initial manifestation of T2DM in ethnic minority groups, especially in the African Americans. Because these patients have a higher risk for developing T2DM, the incidence of DKA is likely to increase as the current epidemic of T2DM continues. The clinical and biochemical characteristics of patients with DKA vary depending on ethnicity and type of diabetes; these differences can be exploited positively in the management of patients with DKA.

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