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Original Article

Fetal Mortality in Pregnancy Complicated by Diabetic Ketoacidosis Prevalence and Outcome at Emergency Department

Dr. Zalanda Shoaib[1], Dr. Muhammad Abas Khan[2], Tahira Zainab[3], Salaiha Gilani[4], Waqas Ahmad[5], Hitash Kumar[6], Dr. Tayyab Asmat Khan[7]

[1] Medical Officer, Emergency Department, Lady Reading Hospital, Peshawar, Pakistan
[2] Assistant Professor, Emergency Department, Lady Reading Hospital, Peshawar, Pakistan
[3] WMO, Gynae/OBS Department, Tehsil Headquarter Hospital, Pind Dadan Khan, Jhelum, Pakistan
[4] TMO, Bahawal Victoria Hospital, Bahawalpur, Punjab, Pakistan
[5] Medical Officer, King Abdullah Teaching Hospital, Mansehra, Pakistan
[6] Resident of Emergency Medicine, Jinnah Postgraduate Medical Center, Karachi, Pakistan
[7] TMO, Radiology Department; Graduate of Gajju Khan Medical College, Swabi, Pakistan; House Job, Bacha Khan Medical Complex, Swabi, Pakistan

*** Corresponding author: Dr. Muhammad Abas Khan, drabbaskhan83@gmail.com**

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ABSTRACT

Background: Diabetic ketoacidosis during pregnancy is an uncommon but serious metabolic emergency associated with substantial fetal risk despite improved maternal survival. Pregnancy-related insulin resistance, reduced buffering capacity, dehydration, ketonemia, and metabolic acidosis may rapidly compromise uteroplacental perfusion and fetal oxygenation. Objective: To determine fetal mortality and maternal outcomes among pregnant women presenting with diabetic ketoacidosis to the Emergency Department of Lady Reading Hospital, Peshawar. Methods: This prospective observational study was conducted from 1st July 2025 to 30th December 2025 after ethical approval. Pregnant women with diabetic ketoacidosis in any trimester were enrolled using non-probability consecutive sampling. Demographic, obstetric, clinical, laboratory, treatment, and outcome data were analyzed using SPSS version 27.0. The primary outcome was fetal mortality during the same hospital admission. Results: A total of 62 patients were included. Mean age was 28.9 ± 5.4 years, and mean gestational age was 29.6 ± 6.8 weeks. Fetal mortality occurred in 13 patients (21.0%), while maternal mortality occurred in 2 patients (3.2%). Intensive care unit admission was required in 21 patients (33.9%), and mechanical ventilation in 8 patients (12.9%). Severe metabolic acidosis and altered mental status independently predicted fetal mortality, with adjusted odds ratios of 3.84 and 3.21, respectively. Conclusion: Diabetic ketoacidosis during pregnancy is associated with considerable fetal mortality. Severe acidosis and altered mental status should prompt urgent multidisciplinary escalation. Keywords: Diabetic Ketoacidosis; Pregnancy; Fetal Mortality; Emergency Department; Maternal Outcomes; Diabetes Mellitus.

INTRODUCTION

Diabetes mellitus is a rapidly increasing global non-communicable disease and remains an important contributor to adverse maternal and perinatal outcomes. In 2021, more than 537 million adults were living with diabetes worldwide, and this burden is projected to rise substantially over the coming decades (1). Hyperglycemia in pregnancy affects approximately 16–17% of live births globally, with a disproportionate burden in low- and middle-income countries, where delayed diagnosis, inconsistent antenatal surveillance, limited specialist access, and suboptimal glycemic control may increase preventable maternal and fetal complications (2). Among the acute complications of diabetes, diabetic

ketoacidosis is uncommon in pregnancy but represents one of the most serious metabolic emergencies because it can progress rapidly and may threaten fetal survival even when maternal survival is achieved.

Pregnancy creates a physiologic state in which diabetic ketoacidosis may develop earlier, progress faster, and present with less marked hyperglycemia than in non-pregnant adults. Placental hormones, including human placental lactogen, progesterone, cortisol, prolactin, and growth hormone, progressively increase insulin resistance as gestation advances, while pregnancy-related respiratory alkalosis lowers serum bicarbonate concentration and reduces maternal buffering capacity (3). Increased lipolysis and accelerated ketone production further predispose pregnant women to ketosis and metabolic acidosis. As a result, diabetic ketoacidosis in pregnancy may occur at lower blood glucose levels, including euglycemic presentations, which can delay recognition in emergency settings if clinicians rely mainly on marked hyperglycemia for diagnosis (3,5).

Although maternal mortality from diabetic ketoacidosis has decreased in tertiary-care settings, fetal compromise remains frequent and clinically important. Published reports indicate that diabetic ketoacidosis occurs in approximately 0.5–3% of diabetic pregnancies, yet fetal mortality after an episode may range from 10% to 35% (3,4). Fetal injury during maternal diabetic ketoacidosis is multifactorial. Maternal dehydration reduces uteroplacental perfusion, metabolic acidosis impairs oxygen delivery, electrolyte disturbances may compromise fetal cardiac function, and persistent maternal ketonemia can contribute to fetal distress, intrauterine fetal demise, or medically indicated preterm delivery if maternal metabolic derangements are not corrected promptly (3,4). These mechanisms make early emergency recognition, aggressive resuscitation, insulin therapy, electrolyte correction, and coordinated obstetric monitoring essential to fetal survival.

The regional relevance of this problem is increasing because Asia carries a major share of the global diabetes burden, and South Asian populations have rising rates of type 2 diabetes among women of reproductive age due to urbanization, obesity, sedentary behavior, and delayed childbearing (1,2). Pakistan is among the countries with a particularly high diabetes burden, with recent estimates indicating that a substantial proportion of adults are affected by diabetes (1). This growing burden is likely to increase the number of pregnancies complicated by pre-existing diabetes, gestational diabetes, and acute metabolic emergencies. At the same time, Pakistan continues to face preventable adverse perinatal outcomes, including stillbirth, which may be worsened by delayed hospital presentation, limited antenatal follow-up, infection, poor insulin adherence, and restricted access to multidisciplinary diabetes-obstetric care (6).

Despite the clinical importance of diabetic ketoacidosis in pregnancy, prospective evidence from Pakistan remains limited, particularly from emergency departments where critically ill pregnant women often present first. Most available studies on diabetic ketoacidosis in pregnancy have been conducted in Europe, North America, or South Africa, and local evidence is frequently retrospective, limited by small sample sizes, or focused broadly on diabetes in pregnancy rather than emergency presentation with ketoacidosis (3,4). This creates an important knowledge gap regarding the proportion of fetal mortality, maternal outcomes, and clinically identifiable emergency-department predictors of fetal loss among pregnant women presenting with diabetic ketoacidosis in Pakistan. Local data are necessary for risk stratification, early obstetric referral, triage decisions, and development of standardized emergency management pathways.

The present study was therefore conducted to determine the proportion of fetal mortality and describe maternal outcomes among pregnant women presenting with diabetic ketoacidosis to the Emergency Department of Lady Reading Hospital, Peshawar. The study further aimed to identify clinical and biochemical factors associated with fetal mortality, with the hypothesis that severe metabolic acidosis, reduced serum bicarbonate, altered mental status, and delayed hospital presentation would be associated with increased fetal loss during the same hospital admission.

MATERIAL AND METHODS

This prospective observational study was conducted in the Emergency Department of Lady Reading Hospital-Medical Teaching Institution, Peshawar, Pakistan, from 1st July 2025 to 30th December 2025. The study design was selected to allow consecutive real-time enrollment of pregnant women presenting with diabetic ketoacidosis and to minimize missing clinical information in an emergency-care setting. Ethical approval was obtained from the Institutional Ethical Review Board of Lady Reading Hospital-Medical Teaching Institution, Peshawar, under Ethical Review Letter No. 334/LRH/MTI, dated 15th December 2025. Written informed consent was obtained from each participant or, when the clinical condition prevented direct consent, from the legally authorized attendant before enrollment.

Pregnant women in any trimester who presented to the Emergency Department with diabetic ketoacidosis during the study period were eligible for inclusion. Diabetic ketoacidosis was diagnosed according to American Diabetes Association criteria using clinical presentation together with biochemical evidence of hyperglycemia or known diabetes, ketonemia or ketonuria, and metabolic acidosis, defined by arterial or venous pH below 7.30 and/or serum bicarbonate below 18 mmol/L (7). Because diabetic ketoacidosis in pregnancy may occur with relatively lower glucose levels, cases with compatible acidosis and ketosis were evaluated for euglycemic diabetic ketoacidosis when marked hyperglycemia was absent. Patients were excluded if records were incomplete for the primary outcome, if participation was refused, or if fetal death had already occurred before the onset of diabetic ketoacidosis. A non-probability consecutive sampling technique was used, and all eligible patients presenting during the defined six-month study period were enrolled.

Data were collected prospectively using a structured proforma completed at the time of emergency evaluation and during the same hospital admission. Recorded variables included maternal age, gestational age at presentation, parity, type of diabetes, presenting symptoms, precipitating factors, duration of symptoms before hospital arrival, clinical status at presentation, laboratory profile, treatment requirements, and maternal and fetal outcomes. Presenting symptoms included vomiting, dehydration, abdominal pain, polyuria, altered mental status, and shortness of breath. Precipitating factors were categorized as infection, poor insulin compliance, newly diagnosed diabetes, or other documented causes. Laboratory investigations included random blood glucose, arterial or venous blood gas analysis, serum bicarbonate, serum or urinary ketones, serum electrolytes, renal function tests, and glycated hemoglobin. Severe metabolic acidosis was operationally defined as arterial or venous pH below 7.10, low serum bicarbonate as bicarbonate below 10 mmol/L, and delayed presentation as arrival at hospital more than 24 hours after symptom onset.

All patients received standard emergency management for diabetic ketoacidosis according to institutional protocol, including fluid resuscitation, insulin therapy, electrolyte monitoring and correction, identification and treatment of precipitating factors, and multidisciplinary care involving emergency physicians, obstetricians, intensivists, and relevant medical specialists. Fetal status was assessed during admission through obstetric evaluation, fetal heart assessment, ultrasound confirmation where indicated, and delivery outcome when delivery occurred during the same hospitalization. The primary outcome was fetal mortality during the same hospital admission, defined as intrauterine fetal demise or fetal death confirmed after presentation with diabetic ketoacidosis and before discharge. Secondary outcomes included maternal mortality, intensive care unit admission, requirement for mechanical ventilation, emergency cesarean section, preterm delivery, duration of hospital stay, and resolution of diabetic ketoacidosis.

To reduce selection bias, participants were enrolled consecutively from the emergency department during the defined study period. To improve measurement consistency, clinical and laboratory data were recorded on a structured proforma, and fetal outcome assessment was confirmed through obstetric evaluation. Potential confounding was addressed analytically by examining clinically relevant maternal,

biochemical, and presentation-related variables in relation to fetal mortality. The sample size represented complete enrollment of eligible cases presenting during the six-month study period at the participating tertiary-care emergency department.

Data were analyzed using Statistical Package for the Social Sciences version 27.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were summarized as mean ± standard deviation when approximately normally distributed and as median with interquartile range when distributional assumptions were not met. Categorical variables were presented as frequencies and percentages. Comparisons between fetal survival and fetal mortality groups were performed using the Chi-square test or Fisher's exact test for categorical variables, and the independent samples t-test or Mann–Whitney U test for continuous variables, as appropriate. Binary logistic regression was used to identify independent predictors of fetal mortality. Variables with clinical relevance and variables showing significant bivariate association were considered for multivariable analysis, while model interpretation was performed cautiously because of the limited number of fetal mortality events. Adjusted odds ratios with 95% confidence intervals were reported for independent predictors. A p-value of ≤0.05 was considered statistically significant.

RESULTS

During the six-month study period, 62 pregnant women presenting with diabetic ketoacidosis were enrolled. The mean maternal age was 28.9 ± 5.4 years, with an age range of 18–40 years. The mean gestational age at presentation was 29.6 ± 6.8 weeks. Multiparous women constituted 56.5% of the sample, while primigravida women constituted 43.5%. Type 1 diabetes mellitus was present in 38.7% of patients, followed by type 2 diabetes mellitus in 33.9% and gestational diabetes in 27.4%.

Table 1. Baseline Demographic, Obstetric, and Diabetes Characteristics of Pregnant Women With Diabetic Ketoacidosis

Variable	Value
Total sample, n	62
Age, years, Mean ± SD	28.9 ± 5.4
Age range, years	18–40
Gestational age, weeks, Mean ± SD	29.6 ± 6.8
Primigravida, n (%)	27 (43.5)
Multiparous, n (%)	35 (56.5)
Type 1 diabetes mellitus, n (%)	24 (38.7)
Type 2 diabetes mellitus, n (%)	21 (33.9)
Gestational diabetes mellitus, n (%)	17 (27.4)

SD: standard deviation.

The baseline profile showed that most patients presented in the third trimester range on average, with a mean gestational age of 29.6 weeks. Multiparity was more frequent than primigravida status, and the diabetes profile was distributed across type 1 diabetes mellitus, type 2 diabetes mellitus, and gestational diabetes mellitus, with type 1 diabetes mellitus representing the largest subgroup.

The most frequent presenting symptom was vomiting, observed in 79.0% of patients, followed by dehydration in 74.2%, abdominal pain in 61.3%, polyuria in 54.8%, altered mental status in 30.6%, and shortness of breath in 25.8%. Infection was the most common precipitating factor, identified in 38.7% of patients, while poor insulin compliance was reported in 29.0%. Newly diagnosed diabetes and other causes each accounted for 16.1% of presentations. The mean random blood glucose level was 418.5 ± 96.4 mg/dL, mean arterial pH was 7.18 ± 0.09, mean serum bicarbonate was 10.8 ± 3.4 mmol/L, and mean HbA1c was 9.8 ± 1.9%.

The clinical profile reflected a predominantly gastrointestinal and dehydration-related presentation, with vomiting and dehydration affecting more than two-thirds of patients. Infection and poor insulin compliance together accounted for the majority of identifiable precipitating factors. The biochemical profile was consistent with clinically significant diabetic ketoacidosis, as reflected by elevated random

blood glucose, reduced arterial pH, low serum bicarbonate, and poor preceding glycemic control indicated by mean HbA1c of 9.8%.

Table 2. Clinical Presentation, Precipitating Factors, and Laboratory Profile

Variable	Value
Vomiting, n (%)	49 (79.0)
Dehydration, n (%)	46 (74.2)
Abdominal pain, n (%)	38 (61.3)
Polyuria, n (%)	34 (54.8)
Altered mental status, n (%)	19 (30.6)
Shortness of breath, n (%)	16 (25.8)
Infection, n (%)	24 (38.7)
Poor insulin compliance, n (%)	18 (29.0)
Newly diagnosed diabetes, n (%)	10 (16.1)
Other precipitating causes, n (%)	10 (16.1)
Random blood glucose, mg/dL, Mean ± SD	418.5 ± 96.4
Arterial pH, Mean ± SD	7.18 ± 0.09
Serum bicarbonate, mmol/L, Mean ± SD	10.8 ± 3.4
HbA1c, %, Mean ± SD	9.8 ± 1.9

SD: standard deviation; HbA1c: glycated hemoglobin.

Fetal mortality occurred in 13 patients, representing 21.0% of the study population, while fetal survival during the same hospital admission was documented in 49 patients. Maternal mortality occurred in 2 patients. Intensive care unit admission was required in 33.9% of patients, and mechanical ventilation was required in 12.9%. Emergency cesarean section was performed in 17.7%, and preterm delivery occurred in 24.2%. The mean hospital stay was 5.8 ± 2.4 days.

Table 3. Fetal, Maternal, and Obstetric Outcomes During Hospital Admission

Outcome	Value
Fetal survival, n (%)	49 (79.0)
Fetal mortality, n (%)	13 (21.0)
Maternal mortality, n (%)	2 (3.2)
Intensive care unit admission, n (%)	21 (33.9)
Mechanical ventilation, n (%)	8 (12.9)
Emergency cesarean section, n (%)	11 (17.7)
Preterm delivery, n (%)	15 (24.2)
Hospital stay, days, Mean ± SD	5.8 ± 2.4

SD: standard deviation.

Fetal mortality represented the major adverse outcome, occurring in approximately one-fifth of pregnancies complicated by diabetic ketoacidosis. Maternal mortality was comparatively lower at 3.2%, but the need for intensive care admission in 33.9% and mechanical ventilation in 12.9% reflected substantial maternal illness severity. Obstetric complications were also clinically relevant, with emergency cesarean section and preterm delivery occurring in 17.7% and 24.2% of patients, respectively.

Fetal mortality was significantly higher among patients with severe metabolic acidosis, defined as arterial pH <7.10, compared with those with pH ≥7.10. Fetal mortality was also higher among patients with serum bicarbonate <10 mmol/L, altered mental status at presentation, and delayed hospital presentation more than 24 hours after symptom onset. Maternal age, parity, and type of diabetes were not statistically associated with fetal mortality. On multivariable binary logistic regression, severe metabolic acidosis and altered mental status remained independent predictors of fetal mortality.

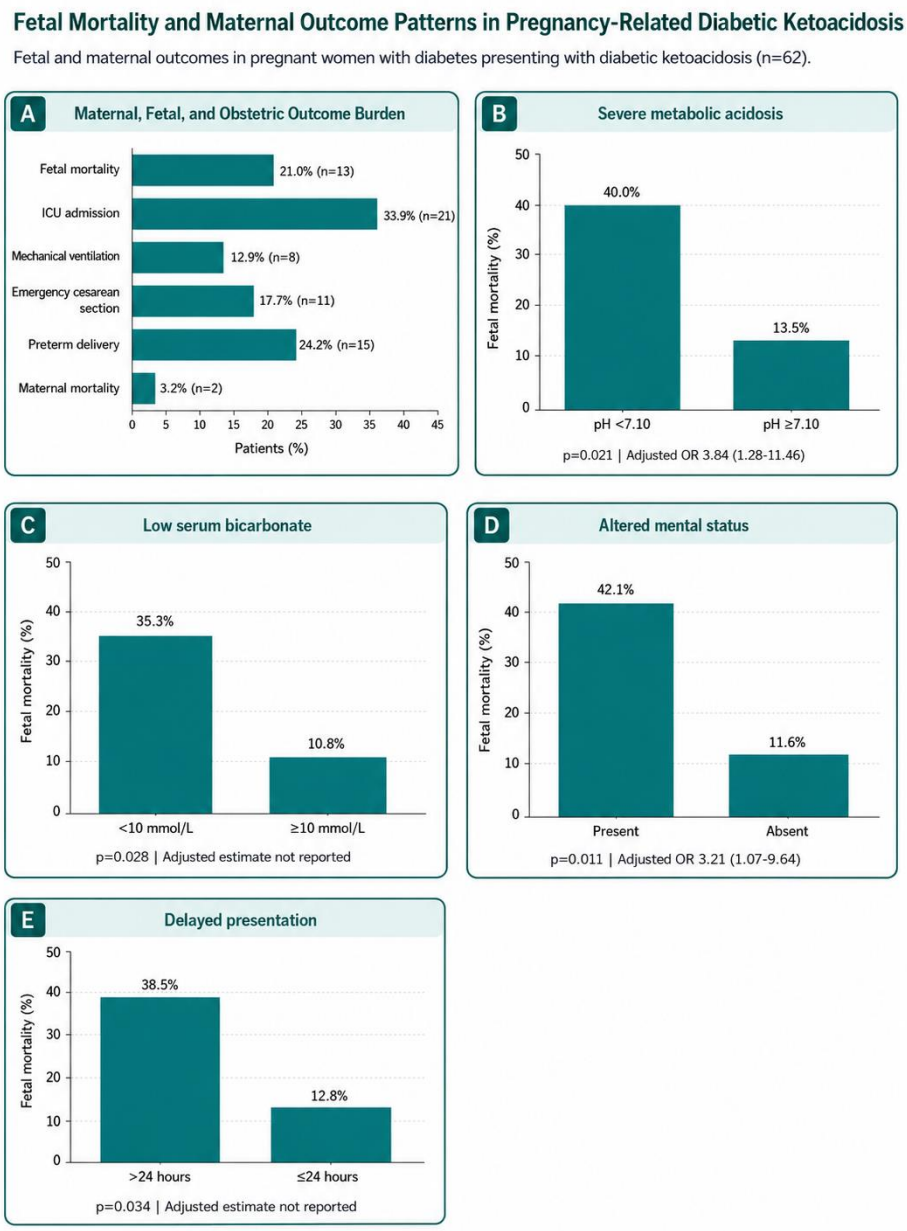

Figure 1. Maternal, fetal, and clinical risk patterns among pregnant women presenting with diabetic ketoacidosis. The panelled figure summarizes the overall burden of adverse outcomes and fetal mortality gradients across key clinical and biochemical risk strata. Fetal mortality occurred in 21.0% of patients, while ICU admission, preterm delivery, emergency cesarean section, mechanical ventilation, and maternal mortality occurred in 33.9%, 24.2%, 17.7%, 12.9%, and 3.2%, respectively. Fetal mortality was higher among patients with severe metabolic acidosis compared with those without severe acidosis (40.0% vs 13.5%; p=0.021), low serum bicarbonate compared with higher bicarbonate levels (35.3% vs 10.8%; p=0.028), altered mental status compared with preserved mental status (42.1% vs 11.6%; p=0.011), and delayed presentation compared with earlier presentation (38.5% vs 12.8%; p=0.034). In adjusted analysis, severe metabolic acidosis and altered mental status remained independently associated with fetal mortality, with adjusted odds ratios of 3.84 and 3.21, respectively.

DISCUSSION

The present prospective observational study found that diabetic ketoacidosis during pregnancy was associated with substantial fetal mortality despite comparatively low maternal mortality. Fetal mortality occurred in 13 of 62 patients, representing 21.0% of pregnancies complicated by diabetic ketoacidosis during the same hospital admission, whereas maternal mortality was observed in 3.2% of patients. These findings reinforce that fetal outcome remains highly vulnerable to maternal metabolic instability even when maternal survival is achieved. The observed fetal mortality is within the internationally reported range of fetal loss following diabetic ketoacidosis in pregnancy, which has commonly been reported

between approximately 10% and 35%, depending on illness severity, gestational age, delay in presentation, and access to intensive obstetric and metabolic care (3–5).

The clinical profile of the study population reflects the typical emergency presentation of diabetic ketoacidosis in pregnancy, with vomiting, dehydration, abdominal pain, polyuria, altered mental status, and shortness of breath being common presenting features. Vomiting and dehydration were particularly frequent, occurring in 79.0% and 74.2% of patients, respectively, while infection and poor insulin compliance were the most common precipitating factors. These findings are clinically important because pregnancy-related diabetic ketoacidosis may be difficult to recognize early, particularly when gastrointestinal symptoms are misattributed to pregnancy or intercurrent illness. The mean random blood glucose level of 418.5 mg/dL, mean arterial pH of 7.18, mean serum bicarbonate of 10.8 mmol/L, and mean HbA1c of 9.8% indicate a population presenting with clinically significant metabolic disturbance and poor preceding glycemic control.

A major finding of this study was the strong association between fetal mortality and markers of severe maternal metabolic compromise. Fetal mortality was higher among patients with severe metabolic acidosis, defined as pH <7.10, compared with those with pH ≥7.10. Severe metabolic acidosis also remained an independent predictor of fetal mortality on multivariable logistic regression, with an adjusted odds ratio of 3.84. This association is biologically plausible because maternal acidemia reduces uteroplacental perfusion, impairs oxygen delivery, and contributes to fetal hypoxia. In diabetic ketoacidosis, maternal dehydration, ketonemia, electrolyte imbalance, and reduced circulating volume may collectively compromise fetal oxygenation and cardiac function, increasing the likelihood of fetal distress or intrauterine fetal demise (3,5).

Altered mental status was another clinically important predictor of fetal mortality. Patients presenting with altered mental status had substantially higher fetal mortality than those without altered mental status, and altered mental status remained independently associated with fetal mortality after adjustment. Altered consciousness in diabetic ketoacidosis usually reflects advanced metabolic derangement, severe dehydration, hyperosmolarity, acidosis, infection, or delayed treatment. In an emergency department setting, altered mental status should therefore be considered a high-risk clinical marker requiring immediate multidisciplinary escalation, intensive metabolic correction, and urgent obstetric assessment.

Low serum bicarbonate and delayed presentation were significantly associated with fetal mortality in bivariate analysis. Patients with serum bicarbonate below 10 mmol/L had higher fetal mortality than those with bicarbonate ≥10 mmol/L, while patients presenting more than 24 hours after symptom onset also had higher fetal mortality than those presenting earlier. These findings are clinically coherent because bicarbonate depletion reflects reduced buffering capacity and severity of acidosis, while delayed presentation prolongs fetal exposure to maternal dehydration, ketonemia, acidosis, and impaired uteroplacental perfusion. However, because adjusted estimates were not reported for these variables, they should be interpreted as significant bivariate associations rather than confirmed independent predictors.

The maternal outcome pattern also demonstrates the severity of diabetic ketoacidosis in pregnancy. Although maternal mortality was low, one-third of patients required intensive care unit admission and 12.9% required mechanical ventilation. Emergency cesarean section was performed in 17.7% of patients, and preterm delivery occurred in 24.2%. These findings indicate that diabetic ketoacidosis in pregnancy is not only a metabolic emergency but also an obstetric and critical-care emergency requiring coordinated management. Current diabetes-in-pregnancy recommendations emphasize prompt recognition, fluid resuscitation, insulin therapy, electrolyte replacement, treatment of precipitating causes, and fetal monitoring after maternal stabilization (7,15).

The findings have practical implications for emergency departments in Pakistan and comparable low- and middle-income settings. Pregnant women with diabetes or suspected hyperglycemia who present with vomiting, dehydration, abdominal pain, respiratory symptoms, altered mental status, or infection should be screened promptly for ketones, metabolic acidosis, serum bicarbonate, electrolytes, and blood glucose. Because diabetic ketoacidosis in pregnancy may occur at lower glucose levels than in non-pregnant adults, reliance on marked hyperglycemia alone may delay diagnosis. Emergency physicians should treat severe acidosis, altered mental status, low bicarbonate, and delayed presentation as high-risk markers for fetal compromise and should involve obstetric, endocrine, and intensive-care teams early.

The study has several strengths. Its prospective design allowed real-time data collection in an emergency department and reduced the likelihood of missing key clinical and laboratory information compared with retrospective chart review. It also provides locally relevant data from a tertiary-care Pakistani hospital, addressing an important evidence gap in the emergency management of diabetic ketoacidosis during pregnancy. The study further identifies clinically recognizable markers associated with fetal mortality, which may support triage and early escalation of care.

Several limitations should be acknowledged. This was a single-center study with a relatively small sample size, and only 13 fetal mortality events occurred, which limits the stability of multivariable regression estimates. The use of non-probability consecutive sampling may limit generalizability to other hospitals and regions. Long-term neonatal outcomes after discharge were not evaluated. Some potential confounders, including socioeconomic status, antenatal care quality, pre-pregnancy glycemic control, diabetes treatment adherence, infection severity, and timing of obstetric intervention, were not comprehensively assessed. In addition, subgroup denominators for several bivariate comparisons were not reported, and future analyses should provide complete two-by-two data, crude odds ratios, adjusted odds ratios, and sensitivity analysis where possible.

Future multicenter prospective studies with larger samples are needed to determine the national burden of diabetic ketoacidosis in pregnancy and to validate predictors of fetal mortality in Pakistan. Further research should evaluate standardized emergency management pathways, time-to-treatment indicators, fetal monitoring protocols, and long-term neonatal outcomes. Integration of diabetes care, antenatal surveillance, emergency triage, and obstetric critical care may help reduce preventable fetal loss associated with diabetic ketoacidosis in pregnancy.

CONCLUSION

Diabetic ketoacidosis during pregnancy remains a serious emergency associated with considerable fetal mortality despite comparatively low maternal mortality. In this prospective study, fetal mortality occurred in 21.0% of pregnant women presenting with diabetic ketoacidosis, while maternal mortality occurred in 3.2%. Severe metabolic acidosis and altered mental status were independent predictors of fetal mortality, whereas low serum bicarbonate and delayed hospital presentation were significant bivariate risk markers. These findings highlight the need for early recognition of diabetic ketoacidosis in pregnancy, rapid correction of maternal metabolic abnormalities, prompt treatment of precipitating factors, and coordinated emergency, obstetric, endocrine, and intensive-care management to improve fetal survival.

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