DKA For Emergency Caesarean Section

Dr. Janaki Subhadra Peyyety
Associate Professor,
Department of Anaesthesiology and Critical Care
Sri Venkateswara Institute of Medical Sciences
Tirupati
Diabetic ketoacidosis is a medical emergency with serious consequences if not recognized and treated immediately. Its occurrence in pregnancy compromises both mother and fetus profoundly and is associated with a high mortality in both. Fetal mortality rates as high as 30 to 90% have been reported, although more recently, it has decreased to as low as 9% due to better prenatal care and diabetic management. The exact rate of maternal mortality due to this condition is unknown but previous reports suggest it to be around 4% - 15%.

The prevalence of gestational diabetes mellitus (GDM) in India varied from 3.8 to 21% in different parts of the country, depending on the geographical locations and diagnostic methods used. GDM has been found to be more prevalent in urban areas than in rural areas.

The rate of pregnancy associated with diabetes is rising especially with the rise in the obesity. More women of reproductive age have diabetes and more pregnancies are complicated by diabetes, diagnosed either before or during the pregnancy. At present as many as 60 million women of reproductive age have type 2 diabetes, and GDM, first recognised during pregnancy, affects up to 15% of pregnant women worldwide.

Despite improvement in its incidence rates and outcomes over the years, it still remains a major clinical problem since it tends to occur at lower blood glucose levels and more rapidly than in non pregnant patients often causing delay in the diagnosis.

Normal pregnancy is characterized by a state of decreased insulin sensitivity, as well as accelerated lipolysis and ketogenesis. The concentration of serum ketones has been estimated to be two to four times greater than in the non-pregnant state. In addition, pregnant women have a respiratory alkalosis, lowering the serum bicarbonate concentration, thus reducing the capacity to buffer hydrogen ions. Despite these changes, the incidence of diabetic ketoacidosis (DKA) in pregnant diabetic women is only 1 to 3%.

DKA in pregnancy usually occurs in the second or third trimesters because of increased insulin resistance owing to the physiological changes that can predispose a pregnant woman with diabetes to diabetic ketoacidosis.

The actions of normal regulatory and counter regulatory hormones influencing metabolism during pregnancy are as follows:

1. Insulin: Promotes glucose uptake in fat, liver, and skeletal muscle. Stimulates adipocytes to store free fatty acids and inhibits lipolysis, gluconeogenesis, and glycogenolysis.

2. Glucagon: Augments hepatic ketone production and increases glucose output by inducing glycogenolysis and gluconeogenesis. Reduces concentration of malonyl-coA by inhibiting synthesis (blocks acetyl-coA carboxylase) and by reducing 3 carbon precursors (suppress glycolysis and pyruvate kinase activity) with glucagon excess see a 300% enhancement in ketone production independent of free fatty acid availability.
3. Catecholamines: Excreted during conditions of stress, dehydration, or acidosis and stimulates free fatty acid release and glycogenolysis. Also stimulates cellular alpha receptors which blunt liver and tissue response to insulin.

4. Cortisol: Also released under conditions of stress and enhances ketone production.

5. HPL, Prolactin: Decrease glucose tolerance, increase insulin resistance, decrease storage of hepatic glycogen and increase glucose production.

**Basic Mechanism of DKA**

*Insulin antagonistic state*

Pregnancy is a state of insulin resistance. Insulin sensitivity has been demonstrated to fall by as much as 56% through 36 weeks of gestation. The production of insulin antagonistic hormones like human placental lactogen, prolactin and cortisol, all contribute to this. The insulin requirement, for this reason, progressively rises during pregnancy explaining the higher incidence of diabetic ketoacidosis in the second and third trimesters. In addition the physiological rise in progesterone with pregnancy decreases gastrointestinal motility that contributes to an increase in the absorption of carbohydrates thereby promoting hyperglycaemia.

Decreasing insulin/glucagon ratio interrupts normal production and disposal of glucose and causes hyperglycemia and ketosis. Gluconeogenesis in the liver adds to hyperglycemia and decreased liver amino acid stores.

*Accelerated starvation*

In pregnancy, there is a relative state of accelerated starvation, especially in the second and third trimesters. The fetus and the placenta use large amounts of maternal glucose as a major source of energy and this leads to decreased maternal fasting glucose. This, associated with relative insulin deficiency leads to an increase in free fatty acids, which are then converted to ketones in the liver.

*Lowered buffering capacity*

A state of respiratory alkalosis exists in the normal pregnancy due to the progesterone effects leading to increased minute alveolar ventilation. This is further contributed to by the inability to use glucose in pregnant diabetics leading to oxidative metabolism of ketones. This process yields b-hydroxybutyrate and acetoacetate which decrease pH, increase respiratory rate and cause a compensatory respiratory alkalosis. This is compensated by increased renal excretion of bicarbonate. The net result is a lowered buffering capacity when exposed to an acid load like ketones.

The clinical implication of these metabolic changes is not only that pregnant diabetics are at risk of developing ketoacidosis, but this can occur rapidly and at a much lower glucose level compared to non-pregnant diabetics.
**Effect of emesis**

Nausea and vomiting are common due to increased human chorionic gonadotrophin in early pregnancy and increased oesophageal reflux in later stages. The resulting stress and fasting state in turn increases insulin antagonistic hormones. This, along with the dehydration that ensues contributes to the development of ketoacidosis.

**Fluid and electrolyte imbalance :**

Increased osmotic pressure leads to fluid and electrolyte imbalance with increased water and electrolyte loss. Acidosis causes potassium to leave the intracellular space in exchange for hydrogen ions. The serum potassium concentration may be normal but total body stores are low. As dehydration continues there is a progression to decreased cardiac output, hypotension, shock, and death.

**Precipitating factors**

Though it is more commonly observed along with type I diabetes, type II diabetes and gestational diabetes can also be associated with DKA. Unrecognized new onset diabetes accounted for 30% cases of DKA during pregnancy in a study by Monro et all.

It is likely to be precipitated by specific factors during pregnancy such as :

- **Intercurrent illness**
- **protracted vomiting and dehydration**
- **hyperemesis gravidarum,**
- **starvation,**
- **infections especially of the urinary tract**
- **insulin non-compliance (major cause/ contributory factor )**
- **medications precipitating DKP :**
  - beta sympathomimetic agents used for tocolysis in cases of premature labour, steroid prophylaxis/steroid treatment used for fetal lung maturation,
  - insulin pump failure (as pumps deliver rapid-acting insulin, interruption for a few hours completely deprives the patient of insulin) and conditions such as diabetic gastroparesis
- **undiagnosed pregnancy**
Diagnosis of DKA in pregnancy: (DKP)

The main symptoms and signs include the following in any combination and in such cases DKA should be ruled out first before embarking on evaluation for other differential diagnoses. Sometimes, DKP may be first presentation of diabetes during pregnancy. The warning signs and symptoms are:

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea or vomiting</td>
<td>Change in mental status</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Hyperventilation (Kussmaul breathing)/pear drop odour</td>
</tr>
<tr>
<td>Polyuria or polydipsia</td>
<td>Tachypnoea</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>Coma</td>
</tr>
<tr>
<td>Lethargy</td>
<td>Shock</td>
</tr>
<tr>
<td></td>
<td>Abnormal fetal heart tracing</td>
</tr>
</tbody>
</table>

Laboratory Investigations

These are required to confirm the diagnosis of DKP, to assess the severity of the condition and also to find out the cause. The Joint British Diabetes Societies Inpatient Care Group guidelines state the following diagnostic criteria for DKP:

1. **Blood ketone** level more than or equal 3.0 mmol/l (or) urine ketone level more than 2+

2. **Blood glucose** level more than 11.0 mmol/l or known diabetes mellitus (It is important to remember that DKP can occur at lower glucose levels than 11 mmol/l (<250 mg/dl).

3. **Bicarbonate level** less than 15.0 mmol/l and/or venous pH less than 7.3 (The reduction in HCO3 is proportional to the increase in concentration of ketoacids.)

In addition, **anion gap of >12,**  
**elevated base deficit** > 4mEq/l are helpful to guide the management.
Dyselectrolytemia: A falsely normal or low potassium may be seen sometimes but frequently there will be rise in serum potassium levels which will trend down as DKA resolves. Even in presence of hyperkalemia, the actual total body potassium can be very low due to urinary and gastrointestinal losses.

Decrease in sodium (<135 mg/dL) is also commonly seen, despite dehydration and hyperosmolarity. Insulin deficiency and glucagon excess exacerbate the loss of sodium in the urine. Another major contribution for hyponatremia is the shift of intracellular water to the extracellular space, because without adequate insulin, cells are impermeable to glucose.

Renal and hepatic function: Pre renal azotemia and slight increase in the liver enzymes may be observed. Lactate should be normal or just minimally elevated.

Water Deficit: Secondary to osmotic diuresis can be excessive, on the order of 100-150 ml/kg or 4-12 liters.

Diabetes and Preterm Labor

As already discussed, drugs which are used in non-diabetic patients to arrest preterm labor can precipitate DKA. Hence magnesium sulfate is the preferred intravenous tocolytic agent for women in premature labor with diabetes mellitus. For the same reasons, corticosteroids should be used carefully. If they are to be used for fetal lung maturation, then a low dose intravenous regular insulin infusion should be initiated to prevent hyperglycemia and blood glucose monitoring should be done frequently.

Factors Contributing To Increased Fetal Loss

The exact mechanism by which maternal diabetic ketoacidosis affects the fetus is unknown. Ketoacids as well as glucose readily cross the placenta. Whether it is the maternal acidosis, hyperglycaemia, severe volume depletion, or electrolyte imbalance that has the most detrimental effect on the fetus is unclear. DKA may result in severe maternal complications such as acute renal failure, ARDS, cerebral edema, coma and even death. High fetal morbidity and mortality can also be contributed to by increased rate of preterm delivery.

During DKA, the fetal brain is susceptible to increased maternal ketones and lactate concentration, which lead to decreased glucose uptake by the fetal brain. These events may increase the chance of fetal brain injury and may have a long-term developmental impact. The long term effect of diabetic ketoacidosis episodes during pregnancy on surviving fetus is lacking. Some studies have shown a direct relationship between plasma ketone levels in pregnant diabetic women and a lower IQ in the child.

The high mortality rate (ranges between 9% and 36%) associated with diabetic ketoacidosis certainly suggests a hostile intrauterine environment. Possible mechanisms include:
• Decrease in uteroplacental blood flow due to: (a) osmotic diuresis leading to volume depletion and (b) maternal acidosis that can cause fetal hypoxic insult.

• Maternal acidosis could lead to fetal acidosis and electrolyte imbalance.

• Maternal hypokalaemia and fetal hyperinsulinaemia if severe could cause fetal hypokalaemia leading to fetal myocardial suppression and fatal arrhythmias.

• Maternal hypophosphataemia associated with diabetic ketoacidosis can cause decrease in 2,3-diphosphoglycerate leading to impaired delivery of oxygen to the fetus.

• Fetal hyperinsulinaemia resulting from maternal hyperglycaemia increases fetal oxygen requirement by stimulating oxidative metabolic pathway.

Management of DKP

DKP is considered as an emergency that needs to be managed in a tertiary care level hospital in an ICU or HDU where a multidisciplinary approach with team of obstetrician, endocrinologist, obstetric anaesthesiologist and trained nursing and other paramedical staff is available to treat such patients. Prompt and vigorous treatment is essential to reduce maternal and fetal morbidity and mortality.

Goals of therapy:

1. Rehydration: by intravenous fluid replacement
2. Normalization of blood glucose: insulin therapy
3. Restoration of electrolyte homeostasis: replacement of sodium and potassium
4. Correction of acidosis: evaluation of the need for bicarbonate administration
5. Identification and treatment of precipitating factors/underlying causes
6. Monitoring of maternal and fetal response
7. General supportive therapy
8. Decision about fetal viability, labor and delivery – anaesthetic considerations

Rehydration:

Total body fluid deficit will be in the range of 6 – 10 litres and it is higher than that seen in the non-pregnant diabetic ketoacidosis. Management of DKP starts with securing at least two large bore iv cannulas. Insertion of central venous catheter should be done at an appropriate time because it may technically difficult in a patient with huge body fluid deficit. Fluid replacement should be commenced promptly with isotonic saline infusion at 10 – 15 ml/kg/h in the first hour (or 400 mL/m²/h, or approximately 1 L/h for the first 2 h of the resuscitation) This may amount to one - two litres depending upon the blood pressure. Once
If blood pressure is above 90 mmHg, the saline infusion can be continued at 500 ml/h for the next 4 hours and thereafter about 250 ml/h over next 8 hours.

The infusion rate can then be continued at 150 ml/h. Close observation of vitals will guide the fluid replacement adjusting according to her response based upon blood pressure, urine output and central venous pressure measurement, if available. Care is needed in patients with impaired cardiac and/or renal function. Cerebral oedema is a theoretical risk of diabetic ketoacidosis, but its association with aggressive fluid replacement has not been consistently proven.

IV fluid therapy improves tissue perfusion, decreases stress hormone levels, and causes haemodilution; this, in turn, lowers the hyperglycaemia and increases the response to insulin therapy. Adequate perfusion should be ensured, taking into account fluid losses, through close monitoring of urine output.Urine output should be monitored using an indwelling catheter, and it should be more than or equal to 0.5 ml/kg/h to ensure that the patient is well hydrated.

In addition to this, 5% or 10% dextrose or 0.45 normal saline should be added at 125ml/h once the blood glucose level approaches 250 mg%.

The presence of acidosis at lower initial glucose levels than in non-pregnant patients may necessitate simultaneous dextrose infusion to enable insulin treatment.

**Normalization of Blood Glucose:**

IV insulin therapy not only corrects the hyperglycaemia but also inhibits the ongoing synthesis of keto acids. IV therapy with regular insulin should be commenced promptly in patients with serum potassium level more than or equal to 3.3 mmol/l.

However, **insulin administration should be postponed if serum potassium is low**, until it is corrected to more than or equal to 3.3 mmol/l, because the insulin pushes the potassium into the intracellular space, which aggravates the existing hypokalaemia and may precipitate fatal cardiac arrhythmias.

Regular insulin infusion should be commenced at a **fixed rate of 0.1 unit/kg/h**, and it is recommended not to initially exceed 15 units/h. If the metabolic targets especially ketone levels are not achieved in one hour or at least 25% fall from the initial values, rate of insulin can be increased up to double the initial values.

Insulin infusion is reduced once serum glucose level drops to 250 mg/dl or 14 mmol/l.

Initial bolus and/or a high rate of insulin infusion have been advocated earlier but current evidence did not show any difference or extra benefit from either. Hence current recommendation is that a low dose infusion is adequate. The fixed rate of insulin can be discontinued only after DKP resolution and after 30 – 60 minutes from the first dose of subcutaneous rapid acting insulin has been resumed.

The patient usual daily dose of insulin should also be given over and above the iv insulin therapy to ensure presence of insulin in the blood in case of interruption of insulin infusion.
Euglycemic DKP:

Euglycemic diabetic ketoacidosis is a rare situation where patient presents with normal or below normal rather than high blood glucose levels. It can present in Type I, type II or gestational diabetes. The likely pathophysiological changes are as follows:

- The use of glucose by the fetoplacental unit, with decreased maternal glycogenolysis and gluconeogenesis
- Increased renal loss of glucose as the renal blood flow increases with increased glomerular filtration of glucose without a corresponding increase in tubular glucose reabsorption
- Increase in estrogen and progesterone in pregnancy accompanied by increased maternal usage of blood glucose
- Dilutional effect on blood glucose because of the increased plasma volume during pregnancy
- Starvation, which is associated with increased ketone production, is also accompanied by depletion of glycogen stores and normoglycaemic DKP.

The management of euglycaemic DKP follows the same principles. However, IV fluid therapy should involve the concomitant administration of 5% dextrose with IV saline via a separate line from the start of treatment to avoid hypoglycaemia caused by IV insulin administration, which is necessary to stop the production of ketoacids.

Restoration of electrolyte homeostasis: replacement of sodium and potassium:

Sodium replacement is achieved once intravenous saline infusion is initiated. These patients frequently have a total potassium deficit of 3–5 mmol/kg but the measured serum potassium is usually normal or even high. This is related to the increased osmolality and insulin deficiency, which cause trans-cellular shift of potassium outside the cells.

In patients with good urine output (at least 0.5ml/kg/h), and serum potassium is < 3.5 mEq/l, replacement with higher strength potassium chloride should be initiated immediately preferably via a central venous catheter, even before IV insulin therapy is started.

The goal is to maintain serum potassium level in the range of 4 – 5 mEq/l as the potassium starts to return to the cells with the ongoing IV fluids and insulin therapy. Failure to replace potassium may result in hypokalaemia with life-threatening cardiac arrhythmias. In patients with oliguria, potassium dose should be reduced by 50%.

If however, potassium is > 5.5 mEq/l at the beginning, no replacement should be given until good urine output is established and potassium level begins to drop below the normal range.
With potassium level at 3.5 – 5.5 mEq/l, 40 mEq/l of potassium chloride is added to the iv normal saline at a rate of 150 – 250 ml/h. This will give approximately 5 – 10 mEq/l of replacement. The National Patients Safety Agency and Irish Medication Safety Network recommend not to infuse more than 20 mEq/l (mmol) potassium per hour.

*Phosphate replacement*: Although the whole body phosphate is decreased, replacement is not recommended, unless the serum level is < 0.32 mmol/l (1 mg/dl) or the patient develops cardiac impairment or respiratory depression. Serum *magnesium* levels also need monitoring though replacement is not immediately required.

Serum electrolytes should be monitored every 2 – 4 hours during treatment.

**Correction of acidosis**: evaluation of the need for bicarbonate administration

In most patients, the above measures are usually sufficient to correct the acidosis. Bicarbonate is rarely needed and is not recommended as there is no beneficial effect with it and it may be harmful to the mother and fetus.

Bicarbonate causes iatrogenic metabolic alkalosis which inhibits the compensatory hyperventilation that washes out carbon dioxide (CO₂), leading to an increment in CO₂ partial pressure (PCO₂), which may, in turn, decrease fetal oxygen delivery.

In addition, the patient may develop paradoxical cerebral acidosis, because the CO₂ diffuses through the blood brain barrier faster than the infused bicarbonate. Further, bicarbonate administration delays the wash out of ketones and can worsen hypokalaemia.

**Identification and treatment of precipitating factors / underlying causes**

Recognition of the condition that precipitates DKP is essential for its management, as any delay in the correction of the precipitating factor can worsen the prognosis and increase the risk of recurrence. Infections, usually of urinary tract, are common precipitating factors. A detailed history and through clinical examination will help to identify the precipitating factors and guide investigations for initiating specific treatment. Elevated white blood counts do not always result from infection but dehydration may be the cause. Broad spectrum antibiotics should be initiated pending results of sepsis work up and cultures, if indicated.

**Monitoring of maternal and fetal response**:

Continuous monitoring of many biochemical and clinical parameters is an essential component of management of DKP. Both the mother and the fetus should be monitored throughout the period of stabilizing the mother and thus the fetus so that timely interventions can be done and decisions be made. *Indwelling urinary catheter* should be inserted and urine output should be measured hourly to assess the adequacy of treatment.
Continuous monitoring of maternal hemodynamics and respiratory function with ECG (risk of arrhythmias), heart rate, blood pressure (invasive arterial blood pressure when frequent ABGs are required or noninvasive), pulse oximetry, respiratory rate in case of un-intubated patients are mandatory, in addition to clinical assessment. Continuous fetal heart rate monitoring is essential to assess fetal well being. Ultrasonography is another useful tool in this regard.

**Maternal response:**

*Blood glucose levels and ketone levels:* hourly for the first 6 hours in order to ensure that **ketone levels decrease at the required rate of at least 0.5 mmol /l.** If ketonometer is not available, calculation of anion gap helps in monitoring the patient’s response. The target for blood glucose level is decrease by **50mg /dl/h (3 mmol/h)**

*Serum electrolytes:* potassium **every 2 hours in the first 6 hours.** Blood urea nitrogen, creatinine should also be monitored to check for status of the kidney function.

*Blood gas analysis* to monitor changes in pH and bicarbonate especially if the patient is hypoxic or has altered sensorium. The target for improvement in acidosis is **rise in the bicarbonate level by 3 mmol/h (3 mEq/l).**

Changes in the bicarbonate levels are reliable to evaluate the treatment response only in the first 6 hours of management of these patients. Because with successful and aggressive rehydration using 0.9% sodium chloride could lead to the development of hyperchloremic acidosis associated with normal anion gap which tend to lower the bicarbonate level. However, hyperchloremic acidosis is usually corrected by the kidney and no intervention is required.

*Urinary ketone levels:* take time to clear as the body excretes through metabolism. Thus, ketonuria can persist for a significant period after ketones have been cleared and the metabolic acidosis has resolved.

*Others:* Complete blood counts, Liver enzymes (may be slightly elevated), cultures for sepsis work up and other radiological investigations are required to find out the cause and treat them. **Lactate concentration: should be normal or just minimally elevated**

**Recovery from DKP:** Defined by blood ketone level< 0.6 mmol/l,

\[ \text{pH} > 7.3 \]

serum bicarbonate >15 mmol/l (during first 6 hours,)

normalisation of the anion gap (≤ 12 mEq/l)

**Fetal Response:**

Continuous fetal monitoring is mandatory to assess fetal wellbeing. Cardiotocography done during diabetic ketoacidosis in pregnancy has shown **absence of baseline heart rate variability,**
persistent late deceleration, and non-reassuring biophysical profile all suggesting fetal distress representing the effects of maternal metabolic acidosis on the fetus, but they are not necessarily indications for immediate delivery.

Subjecting a patient in diabetic ketoacidosis to emergency caesarean section could cause further maternal deterioration while offering minimal, if any, benefit to the fetus. Interestingly, once hyperglycaemia and acidosis is reversed and maternal stabilization achieved, fetal compromise may no longer be evident. Normalization of fetal heart rate tracing after correction of DKP may require 4 - 8 hours.

In the majority of cases of DKP, the aim should be to monitor the fetus until the maternal metabolic state is stabilized, without any immediate plans for delivery, and to continue the pregnancy with complete resolution of DKP. There is no consensus on further fetal monitoring after complete resolution of DKP, especially when the fetus is preterm. The frequency of fetal monitoring is unknown and no definite recommendations are currently available.

**Supportive Therapy:**

Admission into a high dependency unit (HDU) or an ICU - where the staff, medical, obstetric and intensivists and nursing staff who are trained in taking care of such patients, is essential to ensure continuous monitoring and interventions as necessary. These patients should always be nursed in a left (sometimes right, as per patient's convenience) lateral position to avoid aorto-caval compression.

Oxygen therapy with face masks with or without venturi should be instituted immediately. Depending on the arterial blood gases, pH and clinical presentation, some of these women may require mechanical ventilator support till maternal stabilization is achieved.

**Decision about fetal viability, labor and delivery – anaesthetic considerations:**

If the fetus is not nearer term and maternal stabilization is in progress, it is better to wait because the effects of DKP on the fetus revert back with treatment of the mother. Magnesium sulfate is the tocolytic agent of choice in DKP.

The decision to deliver should be individualized and should be primarily based on evaluation of the maternal clinical status to ensure a safe labor and delivery, fetal gestational age and the results of fetal investigations such as fetal heart tracing. Fetal biophysical profile and Doppler studies may also reflect the fetal acidotic status. All these factors should be considered together with a multidisciplinary approach while making a decision regarding delivery. However, in the majority of cases of DKP, the aim should be to monitor the fetus until the maternal metabolic state is stabilised, without any immediate plans for delivery, and to continue the pregnancy with complete resolution of DKP.

Delivery of a compromised fetus should be undertaken ONLY after the mother is metabolically stable. It can be an induction of labor and delivery per vaginum. In the event that the baby
cannot be delivered but by an abdominal delivery, this should be done only when the mother has satisfactory metabolic and biochemical parameters and DKA is resolved adequately.

Patient should be given general anaesthesia where airway, breathing and circulation are under the control of the anaesthesiologist totally. GA can be problematic because of gastroparesis, limited atlanto-occipital joint extension, increased haemodynamic response to intubation, and impaired counter regulatory hormone responses to hypoglycaemia during sleep. All the standard measures to administer a general anaesthetic should be followed diligently: left lateral position/wedge, anti-aspiration prophylaxis, difficult intubation cart, trained assistant and rapid sequence induction and so on.

Every possible care should be taken to avoid episodes of hypoxia, hypercarbia and hypotension should be strictly avoided. Placental insufficiency can be influenced by the anaesthetic technique and can directly impact neonatal well being.

Placental abnormalities have been observed even in association with mild, well-controlled gestational diabetes. Choice of drugs should be based on the effects of the anaesthetic agents on hemodynamics, utero-placental blood flow, time of onset and metabolism and presence of renal dysfunction, if any. Postoperatively mechanical ventilator support may be needed, especially if DKP could not be totally corrected before anesthetizing the patient.

Central neuraxial blockade including graded epidural anaesthetic in a hypotensive and acidotic mother could only worsen maternal condition and further aggravate fetal acidosis because these fetuses tolerate episodes of hypotension and hypoxia very poorly when compared to those of non ketotic hypotensive mothers.

Is there any indication for emergency Caesarean section in a mother with DKP?

If the mother is in active labor and decision is made for an abdominal delivery and DKP is suspected/diagnosed, then, fetal heart rate monitoring should be continued and all the above steps to manage the DKP should be initiated immediately and aggressively. Stabilization of the metabolic condition of the mother for as long as possible before subjecting her to general anaesthesia is essential to prevent high mortality of both the mother and fetus.

Conclusion:

DKP is a life-threatening condition; therefore, prompt diagnosis along with rapid initiation of acute care management involving an experienced multidisciplinary team could help to reduce maternal and fetal mortality, and morbidity.

While the outcomes of diabetic ketoacidosis in pregnancy have improved over the years, significant maternal and fetal mortality still remains. Prevention, early recognition and hospitalization, and aggressive management remain the cornerstones to minimize the outcomes of this dreadful complication.
Suggested Reading:


