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### Diabetic Ketoacidosis in Pediatric Patients

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# Diabetic Ketoacidosis in Pediatric Patients

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## Topic

Diabetic Ketoacidosis (DKA) in Pediatric Patients

- Defined as hyperglycemia and metabolic acidosis with low serum bicarbonate, high serum ketones or urinary ketones.
- It is a potentially life threatening complication of diabetes characterized by hyperglycemia, ketosis, and acidosis resulting from insulin deficiency and increased levels of glucagon, catecholamine, cortisol, and growth hormone (Jensen et al., 2021).
- Type 1 Diabetes (T1D) results from destruction of insulin producing cells in the pancreas called beta cells (Giwa et al., 2020).
- DKA can be precipitated by infection, poor medication compliance, or insulin pump malfunction (Calimag et al., 2022).
- For the purpose of this poster DKA in type 1 diabetes will be discussed.

## Why DKA

- DKA is a common acute and severe complication of pediatric T1D, and is associated with mortality (Yuan et al., 2022).
- Epidemiological studies estimate a prevalence of 1 in 300 children in the United States with increasing incidence of 2-5% worldwide (Giwa et al., 2020).
- DKA in patients with T1D results in a cost burden of \$5 billion in the United States every year (Arendt article).
- Thirty percent of new onset type 1 diabetes present with DKA (Tzimenatos & Nigrovic, 2021).
- This occurs commonly at time of diabetes diagnosis with younger children at greatest risk (Jensen et al., 2021).
- Following the onset of the covid pandemic, children with new onset diabetes and covid positive status present with more profound acidosis, greater need for oxygen supplementation, and increased vasopressor requirements (Chambers et al., 2022).

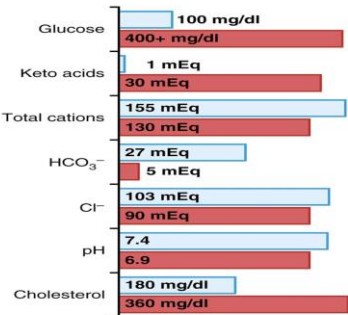
## Signs and Symptoms

- Early clinical signs can include polydipsia (increased thirst), polyuria (increase in amount of urine, weight loss, and fatigue. (Tzimenatos & Nigrovic, 2021).
- An untreated child with DKA can develop abdominal pain, vomiting, and headache (Tzimenatos & Nigrovic, 2021).
- Symptoms may be more difficult to recognize in younger children who are in diapers or before speaking (Tzimenatos & Nigrovic, 2021).
- Severe symptoms include Kussmaul respirations (fast, deep breathing), extreme lethargy, muscle stiffness, and generalized pain (Diabetic Ketoacidosis, 2021).
- DKA can be the first sign of diabetes in undiagnosed people (Diabetic Ketoacidosis, 2021).
- Signs of dehydration such as dry mucous membranes, tachycardia, or hypotension commonly occur as well (Calimag et al., 2022).

## Diagnostic Criteria

- Patients in DKA present with blood sugar levels > 250mg/dl, metabolic acidosis with arterial pH below 7.3, calculated anion gap > 10meq/L, and serum hyperosmolality > 300 mOsm/L ((Elisha et al., 2022, pg. 875).
- Classification of DKA
  - Mild- pH <7.3, serum bicarbonate <15, and glucose >200
  - Moderate- pH <7.2, serum bicarbonate <10, and glucose >200
  - Severe- pH <7.1, serum bicarbonate <5, and glucose >200 (Calimag et al., 2022).
- Beta hydroxybutyrate greater than 3 mmol/L (Calimag et al., 2022).
- Presence of urinary ketones positive nitroprusside reaction (Calimag et al., 2022).

Figure 1: Changes in blood components in DKA, Normal serum levels in blue and DKA levels in red.



Hall and Hall, (2020 pg. 985)

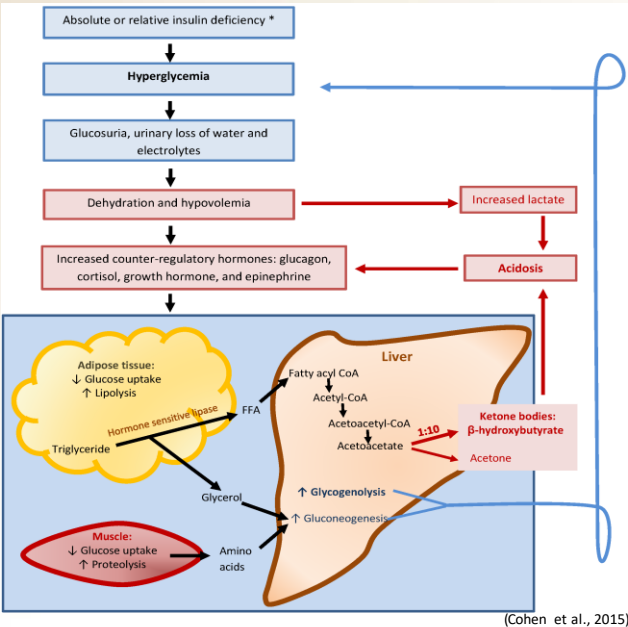
## Underlying Pathophysiology

- Primary pathophysiologic mechanism is caused by an imbalance in ratio of insulin and counter regulatory hormones such as glucagon, cortisol, catecholamines and growth hormone (Calimag et al., 2022).
- Absolute or relative insulin insufficiency leads to increased hepatic gluconeogenesis and glycogenolysis (Calimag et al., 2022).
- The hyperglycemia caused by lack of insulin leads to osmotic diuresis and glucosuria (Elisha et al., 2022).
- Type 1 Diabetes results from destruction of beta cells in the pancreas which secrete insulin (Giwa et al., 2020).
- Overexpression of human leukocyte antigen, HLA class molecules, or environmental factors lead to beta cell recognition as autoantigens and result in an autoimmune attack (Giwa et al., 2020).
- Insulin inhibits breakdown of triglycerides to free fatty acids. Without insulin, triglycerides breakdown into free fatty acid leading to increased ketogenesis in the liver resulting in acidosis (Elisha et al., 2022).
- Increased cortisol and catecholamines lead to protein catabolism which increases gluconeogenic amino acid precursors alanine, lactate, and glycerol (Calimag et al., 2022).

## Significance of Pathophysiology

- Blood glucose levels exceeding renal threshold of 180 mg/dL causes glycosuria (glucose in urine). The osmotic diuresis caused by large glucose molecules leads to profound volume depletion and dehydration (Jayashree et al., 2019).
- An elevated white blood cell count with a left shift often occurs due to hypercortisolemia, increased catecholamines, and acidosis. Leukocytosis doesn't always indicate acute infectious process but a full septic workup can help identifying the underlying cause (Calimag et al., 2022).
- Hypovolemia, acidosis, and raised blood urea nitrogen can lead to cerebral vasoconstriction. Vasoconstriction of cerebral vessels causes cerebral ischemia, increased permeability of the blood-brain barrier, and hypoxia ultimately culminating in cerebral edema (Jayashree, 2019).

Figure 2: Pathophysiology of Diabetic Ketoacidosis.



(Cohen et al., 2015)

## Nursing Implications

- Patients require close neurological monitoring with hourly measurements of a Glasgow Coma scale score for rapid identification of neurological deterioration (Tzimenatos & Nigrovic, 2021).
- Regular laboratory studies include hourly glucose draws, serum electrolytes and arterial blood gas every 2-3 hours (Tzimenatos & Nigrovic, 2021).
- According to Kichloo et al. (2021), between 2008 and 2018 there has been an increasing trend in hospitalizations for T1DM and DKA (pg. 2).
- Children are more likely to have symptomatic cerebral edema compared to adults especially with new onset. The mortality rate associated with cerebral edema is between 10-25% (Jayashree, 2019).

## Treatment

- Focus is to restore intravascular volume, correct electrolyte abnormalities, improve acid-base balance, and treat the insulin deficiency (Elisha et al., 2022 pg. 875).
- Initial fluid bolus of isotonic crystalloid of 10 mL/kg. If in severe shock, defined as weak and thready pulses with hypotension, 20 mL/kg fluid bolus is recommended (Calimag et al., 2022).
- Potassium replacement is typically needed because acidosis is resolved with insulin administration which drives potassium into cells and decreases serum potassium rapidly (Elisha et al., 2022, pg. 875).
- Routine use of Bicarbonate is not recommended to correct acidosis (Elisha et al., 2022, pg. 875).
- Intravenous regular insulin dosed at 0.05-0.1 unit/kg/hr until acidosis resolves (Calimag et al., 2022).
- Resolution of DKA is defined as pH greater than 7.3, serum bicarbonate greater than 15 mmol/L, beta-hydroxybutyrate less than 1 mmol/L, and closure of anion gap (Calimag et al., 2022).
- Use two bag system when serum glucose drops below 300 for maintenance fluids. One bag contains 10% dextrose with 0.9% sodium chloride and the other is 0.9% sodium chloride. Rates are titrated to keep blood glucose between 150-250 (Tzimenatos & Nigrovic, 2021).
- If cerebral edema is suspected, mannitol administration of 0.5-1g/kg or 3% NaCl at 5 mL/kg IV over 20 minutes is indicated (Tzimenatos & Nigrovic, 2021).
- If phosphate levels are below 1 mg/dL, 20-30 meq/L of phosphate should be added to maintenance intravenous fluids to correct electrolyte abnormality while simultaneously monitoring calcium levels (Calimag et al., 2022).
- Take medication for underlying illness that may have precipitated DKA such as antibiotics (Diabetic Ketoacidosis, 2021).

Figure 3: 2 bag fluid system with insulin on syringe pump.



(Tzimenatos & Nigrovic, 2021)

## Conclusions

- Children with type 1 diabetes do not produce enough endogenous insulin and must receive exogenous insulin to allow an influx of glucose into cells throughout the body to provide energy.
- Diabetic Ketoacidosis (2021) states that prevention of DKA is possible with the following actions for known diabetics:
  - Frequent blood sugar checks especially when sick.
  - Maintain glucose levels in target range.
  - Take medications as prescribed.
  - Discuss with primary care provider how to adjust insulin based on eating habits, activity level, and if sick.
- DKA is associated with acute complications, mortality, long term diabetic associated complications and co-morbidities, and significant health care costs (Jensen et al., 2022).

## References



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