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Diabetic Ketoacidosis

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Diabetic Ketoacidosis (DKA)

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Introduction

- Diabetic ketoacidosis (DKA) is a major complication of diabetes and often the initial admitting diagnosis in type I diabetes diagnosis (Sharma, Kumar & Yadav, 2017).
- The incidence of DKA has risen by 30% over the past decade, with more than 140,000 hospital admissions every year in the United States (Basam et al., 2017).
- DKA hospitalizations account for 2.4 billion dollars annually (Basam et al., 2017).
- DKA can be a life threating complication for any diabetic.
- As a future family nurse practitioner (FNP), managing patient's diabetes is a crucial aspect in my future job prospects.
- The best treatment for DKA is prevention. As a FNP, close monitoring and education is crucial in the prevention of DKA.
- DKA being a major complication, understanding the etiology and pathophysiology is imperative in treatment and educating patients in prevention.
- Over the past 20 years (1986-2006), there has been no reduction in the DKA mortality rates, which remained between 3.4% and 4.6% (Singh, Thangaraju, Kumar, Aravindan, Balasubramanian, & Selvan, 2014).
- 20% to 30% of cases of DKA occur in newly diagnosed diabetes mellitus patients (Singh, Thangaraju, Kumar, Aravindan, Balasubramanian, & Selvan, 2014)

Underlying Pathophysiology

- DKA begins with a state of little to no insulin.
- Often brought on by infections and increases in counter regulatory hormones such as catecholamines, cortisol, glucagon and growth hormone leading to increase metabolic demand.
- Insulin deficiency leads to cells inability to uptake glucose from the bloodstream resulting in hyperglycemia and lipolysis.
- Fatty acid formation from lipolysis of adipocytes leading to production of ketone bodies (acetoacetate, hydroxybutyrate, and acetone) by the mitochondria of the liver.
- Accelerated gluconeogenesis, glycogenesis, and ketongenesis that exceeds peripheral use.
- Hyperglycemia leads to solute diuresis.

Hirsch & Emmett, 2017

- Excess ketone bodies cause acidosis, and bicarbonate buffering HC03 <18 leading to anion gap acidosis pH <7.3 and additional hydrogen ions

Misra & Oliver, 2015

Significance of Pathophysiology

- No insulin results in ketoacidosis and hyperglycemia.
- Leading to Kussmaul respirations to treat acidosis and acetone excretion.
- Osmotic diuresis caused by glucosuria and preventing glucose from being greater than 800 vs hyperosmotic hyperglycemic nonketotic state which is usually has glucose level greater than 1000.
- Volume depletion leads to dehydration of the tissues.
- Hypovolemia leads to acute kidney injury and elevated creatinine.
- Acidosis from ketone bodies cause bicarbonate depletion and anion gap acidosis which is a disassociation of anions and cations.
- Hyperkalemia and potassium depletion within the cells from acidosis.
- Diuresis leads to electrolyte abnormalities of Sodium, magnesium, and phosphorus.

Hirsch & Emmett, 2017

Signs and Symptoms

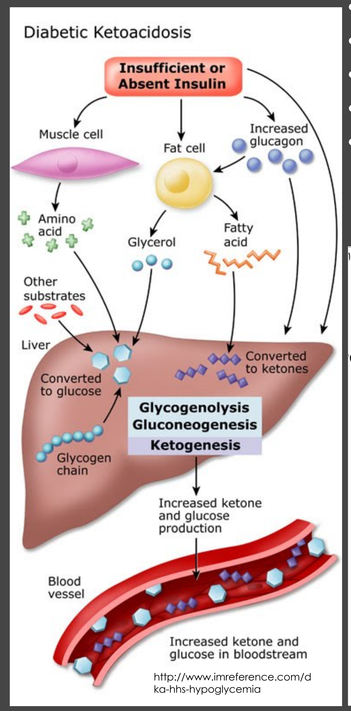
- Hyperventilation (Kussmaul respirations)
- Alter mental status (CNS depression)
- Anorexia
- Weight loss
- Nausea and vomiting
- Abdominal pain
- Ketonuria
- Polydipsia
- Polyuria
- Tachycardia
- Orthostatic hypotension

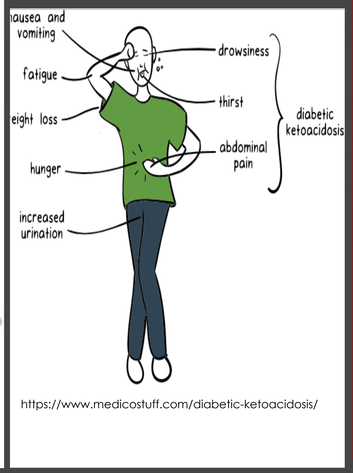
Westerberg, 2013

Risk Factors

- Infection
- Insulin pump malfunction
- Illicit drug use
- Non-compliant diabetic
- Antipsychotic agents:
- Acromegaly
- Arterial thrombosis
- Cushing disease
- Hemochromatosis
- Myocardial infarction
- Pancreatitis
- Pregnancy
- Psychological stress
- Shock/hypovolemia
- Trauma

Westerberg, 2013





Implications for Nursing

- Monitor mentation for S&S of cerebral edema.
- Monitor glucose closely with IV insulin.
- Monitor EKG due to electrolyte abnormalities.
- Monitor urine output, and trend lab values.
- Keep patient NPO until anion gap is less than 12, and long acting insulin administration and fast acting insulin sliding scale.
- Educate patients on DKA prevention.
- Sick day regimen such as closer glucose monitoring q2-3 hours, adequate fluid intake and q4 hours ketones urine strip tests.

Fusco, Gonzales, & Yeung, (2015)

Conclusion

- DKA is a major complication of DM.
- DKA is preventable with proper patient education.
- Although new diagnosis of DM presents as DKA, it's often not preventable.
- With a large financial burden of 2.4 billion annually and as a future advance practice provider this is directly link to my job.
- Educating patients and families for optimal DM management and DKA prevention and when to seek medical attention before DKA becomes profound in individuals.

Treatment

- Fluids- hydrates tissue, corrects dehydration and flush ketone bodies.
- Insulin- stops lipolysis, and allows glucose to enter the cell as optimal energy source.
- Electrolytes- needs replaces due to fluid shifts with rehydration Mg, Phos, K.
- Treat underlying cause of increases in counter regulatory hormones

Fusco, Gonzales, & Yeung, (2015)

References

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