

Clinical Pathway

Management of DKA <20 years of age

June 2010

Outcomes/Goals	<ol style="list-style-type: none"> 1. Initiate prompt, care of the pediatric patient presenting with DKA 2. Minimize / decrease risk of complications through slow decrease in blood glucose of 50-100mg/dl/hour until glucose <300 3. Correction of dehydration and hyperglycemia, resolution and anticipation of electrolyte abnormalities, identification of precipitating and co-morbid illnesses through coordinated care and close patient monitoring
NURSE documentation	ESI level II VS , onset of symptoms, extent of dehydration, level of consciousness, strict I/O, signs/symptoms of infection, IV access, cardiac and pulse oximetry monitoring, assess for cardiopulmonary and neurological stability including signs/symptoms of shock, respiratory distress, dehydration, subtle signs of cerebral edema
INTERVENTIONS Initiate on arrival	IV access (2 peripheral sites in separate extremities once identified as PICU admission) venous blood glucose cardiac/pulse oximetry monitoring with Q1 hour vitals and Q1 hour neuro checks urine ketone test NPO Strict I/O If unable to get PIV, consider central line or IO. No Arterial access.
DIAGNOSTICS	<ol style="list-style-type: none"> A. All patients: <ol style="list-style-type: none"> 1. Hourly capillary or venous glucose monitoring 2. Serum: glucose, CMS (includes phosphate, calcium, BUN, creatinine) then venous blood gas and electrolytes Q2-4 hours until acidosis resolved and transitioned to subcutaneous insulin 3. VBG 4. Micro urinalysis – culture if indicated 5. Urine ketones Q void until ketosis resolves B. All <u>new</u> diabetic patients consider: A1C
PHYSICIAN (LIP)	
Fluids (if indicated)	<ol style="list-style-type: none"> A. 10 ml/kg infuse over 30-60 minutes on a pump. Do not push fluids. Avoid excessive fluid administration or rapid osmotic shifts B. For patients not admitted to the PICU, add D5 or D10 when blood glucose <300 mg/dl – consult with endocrine for volume and rate. Keep glucose between 200-300 mg/dl until acidosis corrects.
Medication	<ol style="list-style-type: none"> A. Insulin - intravenous: start after consult with endocrine 0.05 unit/kg/hour Regular insulin. Titrate to prevent glucose from decreasing >100mg/dl/hour. <u>Do not</u> bolus insulin B. Potassium: after initial hydration/stability if K<5mmol and urine output established add KCL and KPhos. If no urine output established consult endocrine/PICU prior to adding replacement. If adequate urine output, NaCl 0.9% with 20 mEq KCL and 20 mEq KPhos IV at 1 ½ maintenance rate C. Bicarbonate Bicarb not indicated unless pH < 7 after initial hydration and consult with endocrine or PICU.
Diagnosis Criteria	Blood glucose >250mg/dl, venous pH <7.3, bicarbonate <15mEq/L, moderate ketonuria
Admission Criteria	PICU admission criteria (any one criteria): hemodynamically unstable, altered mental status, insulin drip, electrolyte abnormalities

Clinical Pathway Treatment Process

Diabetes with ketonuria (not DKA)

April 2012

Diabetes **with** moderate to large ketones
Blood sugar may be elevated **or** normal
Absence of acidosis (pH >7.30/HCO₃>20)
Normal/baseline mental status
Excludes new onset diabetes, <2 years of age, and pts must be followed by OHSU endocrine clinic

Ondansetron 0.1-0.2mg/kg
ODT (maximum dose 8mg)

Peripheral IV
Labs (BMS, VBG)
Urine ketones each void

Glucose < 250

Carbs+Insulin

Glucose > 250

Insulin

Initiate D10
at maintenance rate

Received
Basilar
(routine dose)
Insulin?

Yes

No

Ketone Correction Dose
Toddlers/young school age 0.1u/kg SC
Pubertal/High School/Adult 0.15u/kg SC

Regular Dose (Basilar) Dose
+
Correction Factor sliding scale
+
Ketone Correction Dose
Toddlers/young school age 0.1u/kg SC
Pubertal/High School/Adult 0.15u/kg SC

Recheck Blood Sugar / Ketones
in 2 hours

Ketones
mod/large?

No

Yes

Home
Follow up with Endocrine
within 3 hours by phone

Glucose
<250 or
>250?

Pediatric DKA

Goals of Clinical Pathway

1. Initiate prompt, care of the pediatric patient presenting with DKA
2. Minimize / decrease risk of complications through slow decrease in blood glucose of 50-100mg/dl/hour until glucose <300
3. Correction of dehydration and hyperglycemia, resolution and anticipation of electrolyte abnormalities, identification of precipitating and co-morbid illnesses through coordinated care and close patient monitoring

Diabetic ketoacidosis (DKA) is a complex metabolic state of hyperglycemia, ketosis, and acidosis. Hyperglycemia causes an osmotic diuresis that leads to excessive loss of free water and electrolytes. Resultant hypovolemia leads to tissue hypoperfusion and lactic acidosis. Successful treatment of DKA includes correction of the dehydration and hyperglycemia, resolution and anticipation of electrolyte abnormalities, identification of precipitating and comorbid illnesses, and frequent patient monitoring.

Data Considerations	Rationale
Cerebral Edema and Fluid Resuscitation	DKA-associated cerebral edema has a low incidence, occurring in about 1% of all episodes of DKA. Overly rapid correction of serum hyperglycemia and osmolarity may create a large gradient between intracerebral and serum osmolarity. Free water then shifts into the brain and may cause cerebral edema with herniation. Therefore, fluid resuscitation and correction of hyperglycemia should be gradual and closely monitored. A study by Glaser et al suggests that the mechanism of cerebral edema is vasogenic and not oncotic. The authors also note that patients with greater dehydration and more profound hypocapnia had increased risk of symptomatic cerebral edema. (2001)
Potassium Replacement	Despite what may be severe total body potassium depletion, apparent serum hyperkalemia is often observed in patients with diabetic ketoacidosis prior to volume resuscitation. The risk of dangerous irregularities in the heart rate is increased, therefore, continuous observation (cardiac monitoring) of the heart rate is recommended.
BiCarb	Ketosis and lactic acidosis produce a metabolic acidosis; however, supplemental bicarbonate is not recommended. Acidosis usually resolves with isotonic fluid volume replenishment and insulin therapy. A pediatric trial of bicarbonate in severe metabolic acidosis during DKA (pH <7.15) showed no benefit when compared with placebo. ² Indeed, multiple studies suggest that bicarbonate therapy may cause paradoxical intracellular acidosis, worsening tissue perfusion and hypokalemia, and cerebral edema. (Young, 2009)

Table 4. Comparison Of Insulin Types.*

Insulin	Onset of Action	Peak Action	Duration of Action
Humalog Insulin lispro	10 minutes	1 hour	4 hours
Regular	30 minutes	2-5 hours	8 hours
NPH	90 minutes	4-12 hours	22-24 hours
Lente	2.5 hours	6-16 hours	24 hours
Ultralente	4 hours	8-18 hours	30 hours
70/30 70% NPH, 30% regular	blend 30 minutes	2-12 hours	24 hours
50/50 50% regular, 50% NPH	blend 30 minutes	2-6 hours	24 hours
Insulin Lantus	glargine 1.5 hours	Flat	24 hours
Insulin Aspart	Within 15 minutes	1-3 hours	3-5 hours

*Table derived from data furnished by Eli Lilly and Novo Nordisk pharmaceuticals in their product information. Supplemental information from Endotext.com. See Reference 76.

Note: The standard insulin concentration in the United States is U-100 insulin. There are several insulins not charted above. Buffered insulins from Eli Lilly and Novo Nordisk and special U-400 insulin from Hoechst of Germany are designed for use in insulin pumps. U-40 insulin is available in some foreign countries, and users need to be aware of the different volume needed to achieve the same dose. The duration of action and time of onset of these insulin concentrations do not differ from those above.

Note: Glargine insulin must not be mixed in the same syringe as any other insulin or solution, because this will alter the pH of the insulin and affect absorption rates. The flat biological activity of glargine insulin is due to its absorption kinetics and not due to different pharmacodynamic activity.

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Approved by: