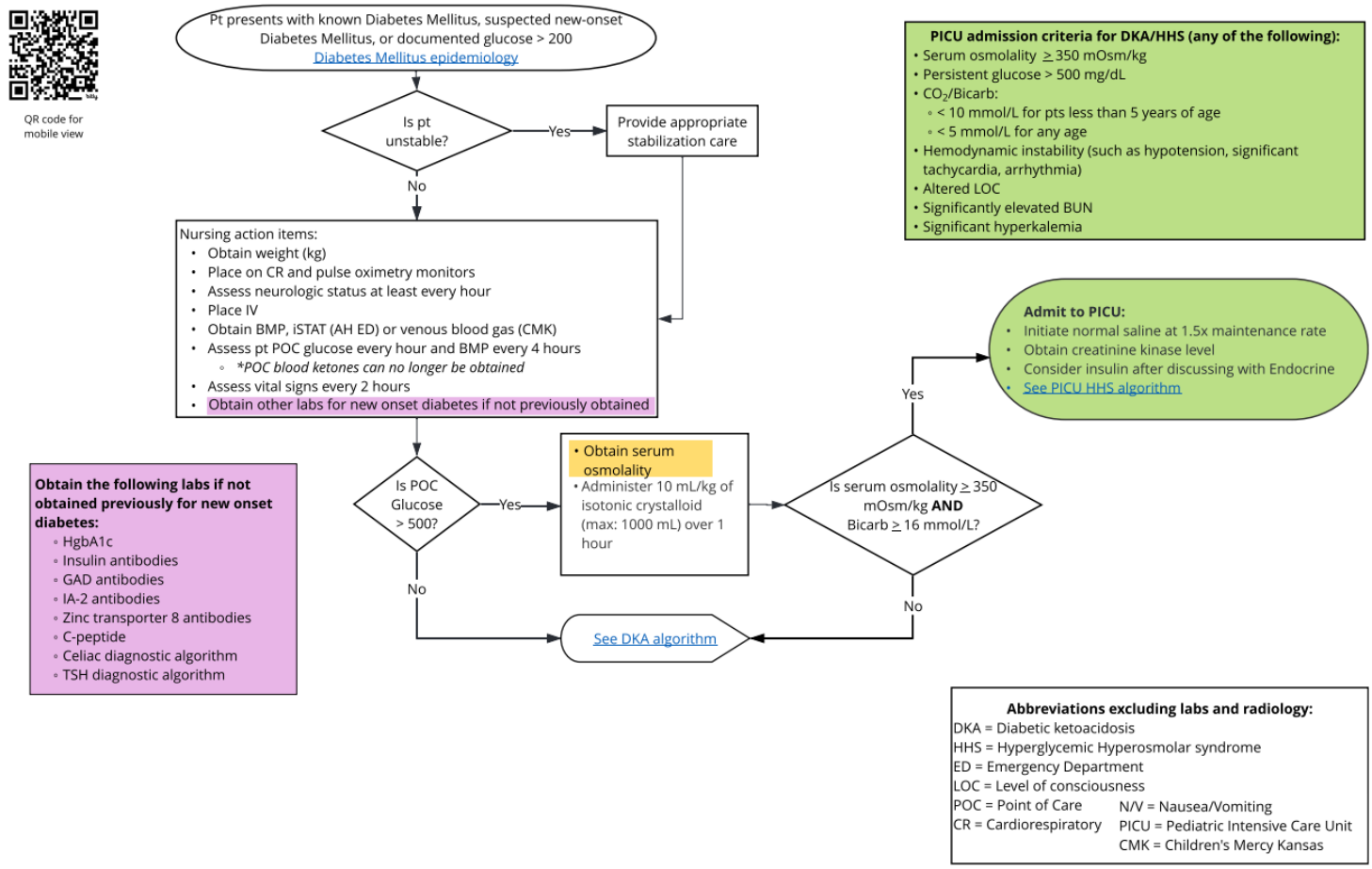
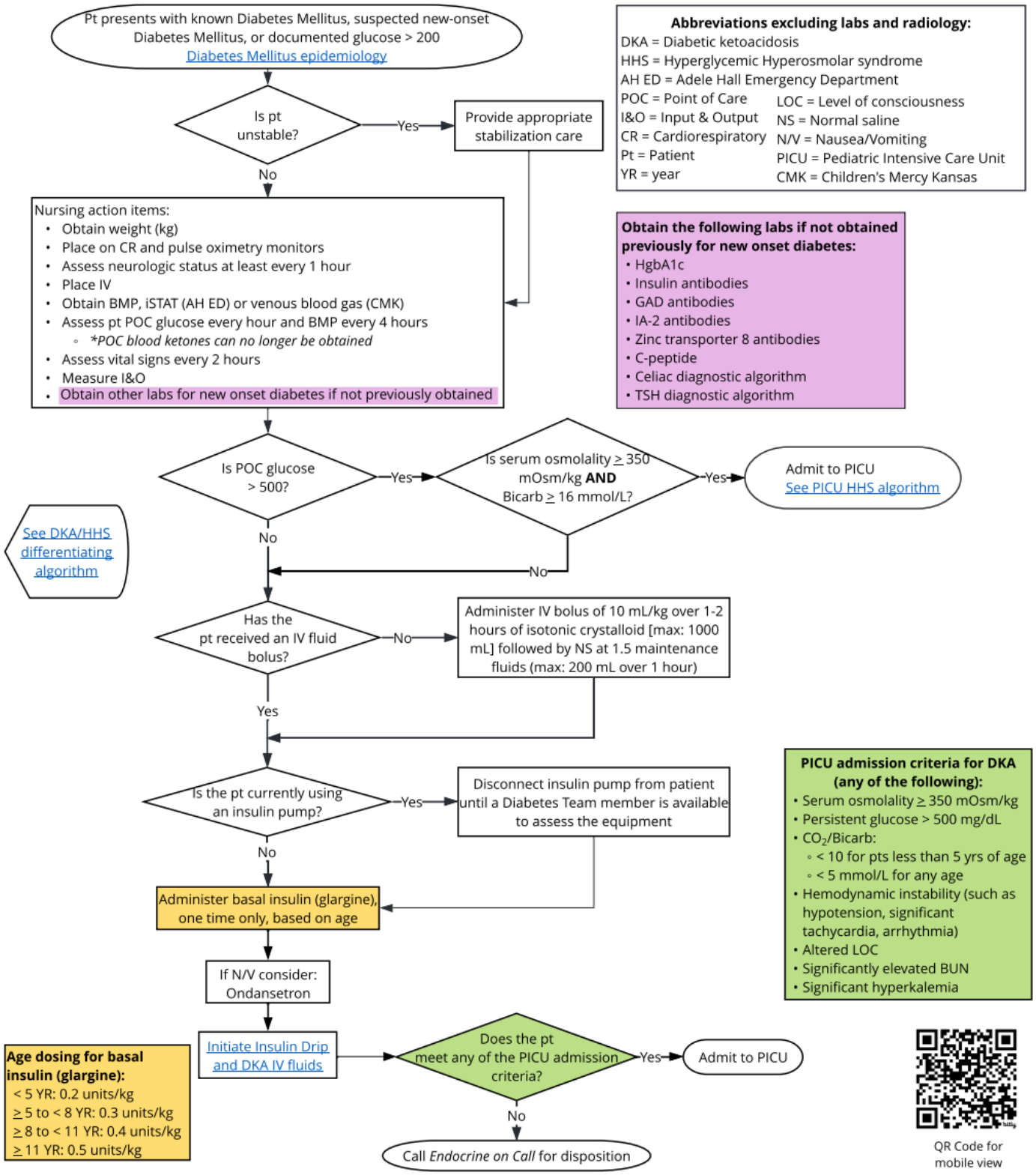
Diabetic Ketoacidosis (DKA)
Clinical Pathway Synopsis

Diabetic Ketoacidosis vs. Hyperglycemic Hyperosmolar Syndrome (HHS) Algorithm

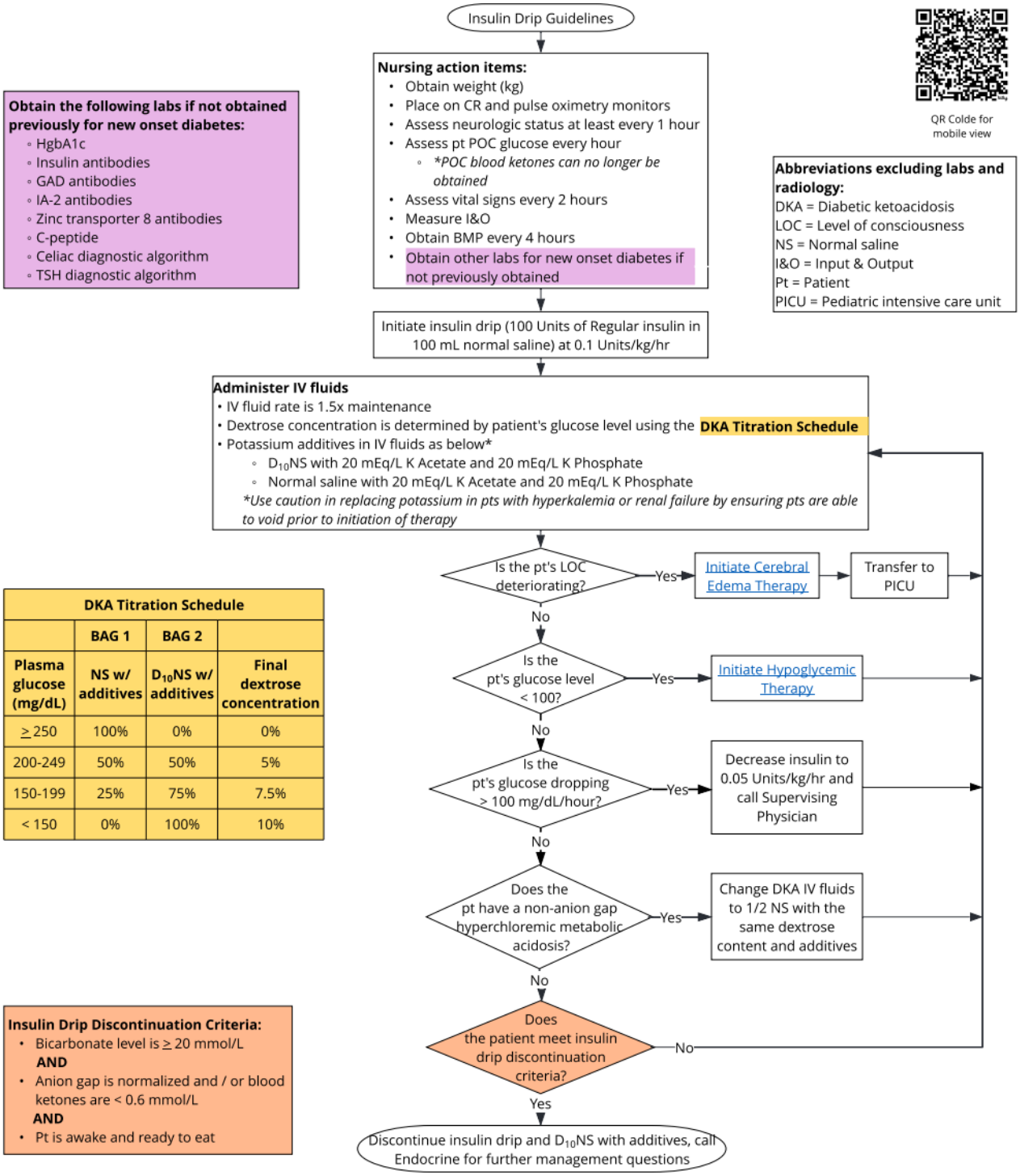


DKA Algorithm



** These products do not establish a standard of care to be followed in every case. It is recognized that each case is different, and those individuals involved in providing health care are expected to use their judgment in determining what is in the best interests of the patient based on the circumstances existing at the time. It is impossible to anticipate all possible situations that may exist and to prepare guidelines for each. Accordingly, these products should guide care with the understanding that departures from them may be required at times.*

Insulin Drip Algorithm



DKA Titration Schedule

	BAG 1	BAG 2	
Plasma glucose (mg/dL)	**NS w/ additives**	**D_{10}NS w/ additives**	**Final dextrose concentration**
≥ 250	100%	0%	0%
200-249	50%	50%	5%
150-199	25%	75%	7.5%
< 150	0%	100%	10%

Insulin Drip Discontinuation Criteria:
- Bicarbonate level is ≥ 20 mmol/L
  AND
- Anion gap is normalized and / or blood ketones are < 0.6 mmol/L
  AND
- Pt is awake and ready to eat

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Objective of Clinical Pathway

The objective of this pathway, besides standardizing care and the benefits associated with care standardization, is to identify and treat diabetic ketoacidosis (DKA) by correcting dehydration and acidosis, reversing ketosis, restoring blood glucose to near normal, avoiding complications of therapy (including cerebral edema), and identifying and treating any precipitating event(s) to prevent future DKA.

Background/Epidemiology

In most western countries, type 1 diabetes accounts for over 90% of childhood and adolescent diabetes. Type 2 diabetes is becoming more common in adolescents, particularly in the peri-pubertal period, and accounts for a significant proportion of youth onset diabetes in certain at-risk populations (International Diabetes Federation, 2010).

Type 1 diabetes incidence varies greatly between different countries, within countries, and between different ethnic populations. Annual incidence rate for childhood type 1 diabetes in the United States is 1 per 500-600 with the type 1 diabetes incidence doubling every 20 years (Rogers, Kim, Banerjee & Lee, 2017). There has been a well-documented rise in the incidence within the United States, with a disproportionately greater increase in those under the age of 5 years. A seasonal variation in the presentation of new cases is well described, with the peak being in the winter months (International Diabetes Federation, 2010).

Susceptibility to type 1 diabetes is associated with multiple genetic loci. Human leukocyte antigens (HLA) genes having the strongest known association account for approximately 40% of familial clustering of type 1 diabetes. There is also linkage to specific combinations of alleles at the DRB1, DQA1 and DQB1 loci, with both susceptible and protective haplotypes. Despite familial aggregation, which accounts for approximately 10% of cases of type 1 diabetes, there is no recognizable Mendelian pattern of inheritance. The concordance rates in the identical twin of a patient with type 1 diabetes is about 36%; for a sibling the risk is approximately 4% by age 20 years and 9.6% by age 60 years; compared with 0.5 % for the general population. Type 1 diabetes is 2-3 times more common in the offspring of fathers with diabetes (3.6–8.5%) compared with mothers with diabetes (1.3–3.6%) (International Diabetes Federation, 2010).

The environmental triggers (chemical and/or viral) which initiate pancreatic beta cell destruction remain largely unknown, but the process usually begins months to years before the manifestation of clinical symptoms. Enterovirus infection has been associated with development of diabetes-associated autoantibodies in some populations and enteroviruses have been detected in the islet cells of individuals with diabetes (International Diabetes Federation, 2010).

Target Users

- Emergency Medicine Providers
- Pediatric Hospitalists
- Endocrinologists
- Fellows
- Resident Physicians
- Pediatric Nursing Practitioners
- Staff nurses within Emergency Medicine, Pediatric Critical Care, and Inpatient Services

Target Population

Inclusion Criteria

- Patient has known or presumed diabetes mellitus (referred to as "diabetes") and CO_2 <16 mmol/L

Exclusion Criteria

- Hyperglycemia without acidosis

AGREE II

The International Diabetes Foundation/International Society for Pediatric and Adolescent Diabetes (IDF/ISPAD) guidelines that provided guidance to the DKA committee (International Diabetes Federation, 2010 & Wolfsdorf, Glaser, & Sperling, 2006). See Table 1 for AGREE II.
Table 1

** These products do not establish a standard of care to be followed in every case. It is recognized that each case is different, and those individuals involved in providing health care are expected to use their judgment in determining what is in the best interests of the patient based on the circumstances existing at the time. It is impossible to anticipate all possible situations that may exist and to prepare guidelines for each. Accordingly, these products should guide care with the understanding that departures from them may be required at times.*

AGREE II[a] Summary for the Guideline International Diabetes Federation (2010)

Domain	Percent Agreement	Percent Justification^
Scope and purpose	96%	The aim of the guideline, the clinical questions posed, and target populations **were** identified.
Stakeholder involvement	71%	The guideline **did not** include appropriate stakeholders (such as pediatric social workers, psychologists, dieticians) nor the viewpoints if the intended user.
Rigor of development	67%	The guideline developers **did no**t provide how the evidence was gathered and synthesized, how the recommendations were formulated nor how the guidelines will be updated.
Clarity and presentation	99%	The guideline recommendations **are** clear, unambiguous, and easily identified; in addition, different management options are presented.
Applicability	83%	Barriers and facilitators to implementation, strategies to improve utilization and resource implications **were addressed** in the guideline.
Editorial independence	92%	The recommendations **were not** biased with competing interests. However, Duality of Interests were identified by the Guideline Development Group.
Committee's recommendation for guideline use	Yes	

Note: Four EBP Scholars completed the AGREE II on this guideline.
^Percentage justification is an interpretation based on the Children's Mercy EBP Department standards.

Practice Recommendations

These recommendations are based on the degree of acidosis defined as:

- Severe: bicarbonate < 5 mmol/L, venous pH < 7.1
- Moderate: bicarbonate 5-10 mmol/L, venous pH < 7.2
- Mild: bicarbonate 11-15 mmol/L, venous pH < 7.3

Diagnostic evaluation:

History:

- Family history of diabetes
- Polyuria, polydipsia, polyphagia
- Weight loss
- Abdominal pain, nausea, vomiting
- Mental status
- Concurrent illness or infections
- Inadequate insulin therapy in a known diabetic such as non-adherence or inappropriate dosing or interruption of insulin delivery from insulin pump
- Steroid use
- Age 3 years or less (increased risk for cerebral edema)

Physical Exam (PE)/Monitoring: The degree of acidosis (mild, moderate, severe) is an important marker for determining the severity of DKA and is a risk factor for cerebral edema. PE/monitoring include:

- Assess dehydration (recognizing that clinical assessment of dehydration can be imprecise in the setting of DKA. Assume 5-10% dehydration in moderate to severe DKA)
- Assess level of consciousness
- Assess for Kussmaul respirations (deep labored breathing)

Mild DKA

- Heart rate, respiratory rate, and blood pressure should be obtained every 4 hours until resolution of DKA (normalization of bicarbonate or venous pH), then per routine
- Fluid balance input and output should be accurately measured

Moderate/Severe DKA

- Continuous cardiorespiratory and pulse oximetry monitors
- Hourly heart rate, respiratory rate, and blood pressure should be obtained
- Hourly fluid input and output measured
- Hourly neurologic assessment for warning signs of cerebral edema (headache, inappropriate slowing of heart rate, recurrent vomiting, change in neurologic status, rising blood pressure, decreased oxygen saturation) should be performed

Laboratory:

- Hourly capillary blood glucose for patients on insulin drips and those receiving every 2-hour correction doses of subcutaneous insulin should be obtained
- Obtain BMP every 4 hours
- Bicarbonate level (from BMP, iSTAT or venous blood gas) will assist emergency department clinicians in determining patient placement.
- Ongoing monitoring of BMP should be obtained every 4 hours

Obtain the following labs if not obtained previously for new onset diabetes:

- Hgb A1c
- Insulin antibodies
- GAD antibodies
- IA-2 antibodies
- Zinc transporter 8 antibodies
- C-peptide
- Celiac diagnostic algorithm
- TSH diagnostic algorithm

Criteria for Med/Surg and PICU admission stratified by age:

For Children **≤** *5 years of age*

Criteria for 6 Henson Admission:

- Bicarbonate level > 10

Criteria for PICU Admission:

- Patients at significant risk for cerebral edema (see risk factors under complications: cerebral edema section of the synopsis) or
- Neurologic signs that might indicate cerebral edema or
- Bicarbonate level ≤ 10 mmol/L or
- BUN level significantly elevated or
- Significant hyperkalemia

For Children **>** *5 years of age*

Criteria for 6 Henson Admission:

- Bicarbonate level > 5

Criteria for PICU Admission:

- Patients at significant risk for cerebral edema (see risk factors under DKA Complications: Cerebral edema section of the synopsis) or
- Neurologic signs that might indicate cerebral edema or
- Bicarbonate level ≤ 5 mmol/L or
- BUN level significantly elevated or
- Significant hyperkalemia

***Treatment*:**

Moderate/Severe DKA

- **If patient is exhibiting critical organ failure (such as shock, respiratory failure, cerebral edema, etc.) follow PALS resuscitation guideline until stable.**
- **Fluid and electrolyte replacement**
  - Administer 10 mL/kg IV bolus of isotonic crystalloid over 1-2 hours (maximum bolus 1 liter). In severe dehydration a second fluid bolus of 10 mL/kg over 1 hour may be necessary. After the fluid bolus is administered begin 1.5 maintenance IV fluid (IVF) of normal saline (maximum bolus 200 mL over 1 hour).
  - Patients in DKA are inherently potassium and phosphorus depleted and will require replacement therapy regardless of current levels of electrolytes on lab. If hypokalemic at presentation, begin potassium therapy at time of initial volume expansion. Use caution in replacing potassium in patients with hyperkalemia or renal failure. Verify these patients can void prior to initiation of therapy. Contact the Endocrinologist on Call.
  - 1.5 maintenance fluids should be changed to Normal Saline (NS) with 20 mEq/L K acetate and 20 mEq/L K Phosphate as soon as available.
  - An additional IVF bag of D_{10}NS, 20 mEq/L K Acetate and 20 mEq/L K Phosphate should be ordered to the bedside for use as indicated below under "continuous IV insulin infusion" and/or "rapid acting subcutaneous insulin".
  - NPO
- **Insulin initiation**
  - **Basal subcutaneous insulin:** Basal insulin should be given as soon as possible and may be administered while a patient is on continuous IV insulin infusion. This injection aids in the transition to subcutaneous dosing at DKA resolution, and only plays a small role in DKA treatment.
  - **Patients with new-onset diabetes–** administer subcutaneous insulin glargine:
    - 0-4 years = 0.2 units/kg
    - 5-7 years = 0.3 units/kg
    - 8-10 years = 0.4 units/ kg
    - 11 and up = 0.5 units/kg
  - **Known diabetes patient –** if patient is connected to an insulin pump, it should be disconnected. Then administer glargine insulin based on the above "new-onset diabetes" recommendations. If patient is on injections administer home dose of glargine or use above "new-onset diabetes" recommendations if home dose is unknown.
- **Continuous IV insulin infusion:**
  - Administer 0.1 units/kg/hour of IV regular insulin (100 units of regular insulin in 100 mL normal saline)
  - Monitor capillary blood glucose hourly. Follow the titration table below and adjust NS with additives and D_{10}NS with additives fluids based on blood glucose.
  - If blood glucose drops between 80 to 99 mg/dL, decrease IV insulin to 0.05 units/kg/hour and contact the supervising physician
  - If blood glucose drops < 80 mg/dL, stop IV insulin, follow hypoglycemia protocol located under supporting documents (*Appendix A*), and contact supervising physician.
  - Continue IV insulin infusion until bicarbonate level is ≥ 20 mmol/L AND 1) anion gap is normalized, or 2) blood ketones are <0.6 mmol/L.
  - Upon discontinuing IV insulin, discontinue D_{10}NS with additives if patient's blood glucose is stable.

DKA Titration Schedule			
	BAG 1	BAG 2	
	Given as Percentages		
Plasma Glucose	**NS w/ additives**	**D_{10}NS w/ additives**	**Final Dextrose Concentration**
> 250	100%	0%	0%
200-249	50%	50%	5%
150-199	25%	75%	7.5%
< 150	0%	100%	10%

Mild DKA

- **Fluid and electrolyte replacement**
  - Administer 10 mL/kg IV bolus of isotonic crystalloid over 1 hour (maximum bolus 1 liter) followed by 1.5 maintenance fluids of normal saline (International Diabetes Federation, 2010 & Wolfsdorf, Glaser, & Sperling, 2006).
  - Patients in DKA are inherently potassium and phosphorus depleted and will require replacement therapy regardless of current levels of electrolytes on lab. If hypokalemic at presentation, begin potassium therapy at time of initial volume expansion. Use caution in replacing potassium in patients with hyperkalemia or renal failure. Verify these patients can void prior to initiation of therapy and contact the Endocrinologist on Call.
  - 1.5 maintenance fluids should be changed to NS with 20 mEq/L K Acetate and 20 mEq/L K Phosphate as soon as available.
  - An additional IVF bag of D_{10}NS, 20 mEq/L K Acetate and 20 mEq/L K Phosphate should be ordered to the bedside for use as indicated below under "continuous IV insulin infusion" and/or "rapid acting subcutaneous insulin".
  - NPO with ice chips initially. If patient clinically appears well and would like to eat, they may do so, but they must receive the appropriate dose of insulin for carbohydrates consumed. This dose may be found in their diabetes clinic visit or inpatient consultation records.
- **Insulin initiation**
  - **Basal subcutaneous insulin:**
    Basal insulin should be given as soon as possible and may be administered while a patient is on continuous IV insulin infusion. This injection aids in the transition to subcutaneous dosing at DKA resolution, and only plays a small role in DKA treatment.
  - **Patient with new-onset diabetes –** administer subcutaneous insulin glargine:
    - 0-4 years = 0.2 units/kg
    - 5-7 years = 0.3 units/kg
    - 8-10 years = 0.4 units/kg
    - 11 and up = 0.5 units/kg
  - **Known diabetes patient –** if patient is connected to an insulin pump, it should be disconnected. Then administer glargine insulin based on the above "new-onset diabetes" recommendations. If patient is on injections administer home dose of glargine or use above "new-onset diabetes" recommendations if home dose is unknown.
- **Continuous IV insulin infusion:**
  - Preferred treatment for mild DKA is IV regular insulin. IV insulin may be utilized on the medical/surgical floors for mild DKA. However, staffing and bed availability may make this option unavailable in which case rapid acting subcutaneous insulin is an effective alternative.
  - Administer 0.1 units/kg/hour of IV regular insulin
  - Monitor capillary blood glucose hourly. Follow the titration table below and adjust NS with additives and D_{10}NS with additives fluids based on blood glucose.

DKA Titration Schedule			
	BAG 1	BAG 2	
	Given as Percentages		
Plasma Glucose	**NS w/ additives**	**D_{10}NS w/ additives**	**Final Dextrose Concentration**
> 250	100%	0%	0%
200-249	50%	50%	5%
150-199	25%	75%	7.5%
<150	0%	100%	10%

- If blood glucose drops to between 80 to 99 mg/dL, decrease IV insulin to 0.05 units/kg/hour, and contact Supervising Physician.
- If blood glucose drops to < 80 mg/dL, stop IV insulin, follow hypoglycemia protocol located under supportive documents (*Appendix A*), and contact supervising physician.
- If blood glucose decreased by > 100 mg/dL/hour, then decrease insulin infusion rate to 0.05 Units/kg/hr and contact supervising physician.
- Continue IV insulin infusion until bicarbonate level is ≥ 20 mmol/L AND either 1) anion gap is normalized, or 2) blood ketones <0.6mmol/L.
- Upon discontinuing IV insulin, discontinue D10 NS with additives if patient's blood glucose is stable.

- **Rapid acting subcutaneous insulin:**
  - Rapid acting insulin (lispro, aspart, glulisine) should be administered 0.2 units/kg subcutaneously every 2 hours
  - Monitor capillary blood glucose hourly. Follow the titration table below and adjust NS with additives and D_{10}NS with additives fluids based on blood glucose.

DKA Titration Schedule			
	BAG 1	BAG 2	
	Given as Percentages		
Plasma Glucose	**NS w/ additives**	**D_{10}NS w/ additives**	**Final Dextrose Concentration**
> 250	100%	0%	0%
200-249	50%	50%	5%
150-199	25%	75%	7.5%
< 150	0%	100%	10%

- If blood glucose drops < 80 mg/dL, follow hypoglycemic protocol located under supportive documents (*Appendix A*) and contact supervising physician. Do not administer additional subcutaneous insulin until speaking with the supervising physician.
- Continue every 2-hour subcutaneous rapid acting insulin until bicarbonate level is ≥ 20 mmol/L AND either 1) anion gap is normalized, or 2) blood ketones < 0.6mmol/L.

Complications of DKA

- **Cerebral edema**
  - Those at increased risk include younger age, new onset diabetes, and longer duration of symptoms.
  - Additional risk factors at diagnosis or during treatment – more severe acidosis, very elevated BUN at presentation, use of bicarbonate for treatment of acidosis, fluid bolus volume over 40 mL/kg given in the first 4 hours of treatment, and administration of insulin in the first hour of fluid treatment (International Diabetes Federation, 2010 & Wolfsdorf, Glaser, & Sperling, 2006).

- Signs and symptoms – headache, slowing or irregular heart rate, change in neurologic status (restlessness, irritability, increased drowsiness, and /or incontinence), cranial nerve palsies or other specific neurologic signs, increasing blood pressure, hypoxemia.
- One diagnostic criterion, 2 major criteria, or 1 major and 2 minor criteria have a sensitivity of 92% and a false positive rate of only 4% for detecting cerebral edema (Muir et al., 2004 & Rosenbloom, 2004).
    - Diagnostic criteria – abnormal motor or verbal response to pain, decorticate or decerebrate posture, cranial nerve palsy, abnormal neurogenic respiratory pattern (grunting, tachypnea)
    - Major criteria – altered mental status, fluctuating level of consciousness, sustained heart rate deceleration (decrease more than 20 beats per minute) not attributable to improved hydration or sleep, age-inappropriate incontinence
    - Minor criteria – vomiting, headache, lethargy, diastolic BP >90 mm Hg, age <5 years.
- Medical/Surgical Treatment:
    - Contact and begin immediate transfer to critical care unit:
        - Elevate head of bed
        - Reduce fluid rate by 1/3 to ½
        - Begin medication therapy with one of the two options:
            - Mannitol 0.5-1 g/kg IV over 20 minutes
            - Hypertonic saline (3%) 5-10 mL/kg over 30 minutes
        - Obtain CT head **after** treatment has been started to rule out other possible intracerebral causes of neurologic deterioration (thrombosis or hemorrhage). ***If cerebral edema is suspected treatment should precede imaging studies***!

- **Hypokalemia** – may precipitate cardiac arrhythmia, cardiac monitoring is recommended
- **Hypoglycemia** – increase dextrose in IVF to treat, additionally see hospital guidelines for treatment of hypoglycemia in diabetics under supporting documents (*Appendix A*)
- **Hypophosphatemia**
- **Pancreatitis**

Discharge Criteria for DKA

- **Resolution of DKA**: bicarbonate ≥18mmol/L OR venous pH >7.3, WITH normal (closed) anion gap, or blood ketones <0.6mmol/L.

Additional Questions Posed by the Clinical Pathway Committee

1. For pediatric patients experiencing diabetic ketoacidosis (DKA) is Lactated Ringer's (LR) versus Normal Saline (NS) superior in preventing hyperchloremic metabolic acidosis or cerebral edema?

   Recommendations from the DKA Clinical Pathway Committee
   No recommendation can be made to change the standard of care from use of NS to LR, based on expert opinion and review of current literature by the subject matter experts and the Department of EBP. The overall certainty in the evidence is very lowa. No studies from the literature search were identified comparing LR to NS in preventing hyperchloremic metabolic acidosis. The literature search identified two cohort studies (Bergmann et al., 2021; Hsia et al., 2015) addressing prevention of cerebral edema. The first study (Bergmann et al., 2021; Hsia et al., 2015) found LR superior to NS in preventing cerebral edema for patients with DKA. The second study (Hsia et al., 2015) found LR to be equivalent to ½ NS in prevention of cerebral edema for patients with DKA. When there is a lack of scientific evidence, standard work should be developed, implemented, and monitored.

2. In the pediatric patient with Type I diabetes experiencing diabetic ketoacidosis (DKA) are continuous glucose monitoring (CGM) system results as accurate as fingerstick (i.e., capillary) glucose results?

   Recommendations from the DKA Clinical Pathway Committee
   No recommendation can be made for or against use of CGMs in the inpatient setting instead of fingerstick, based on expert opinion and review of current literature by the subject matter expert and the Department of EBP. There was no evidence to support change from fingerstick glucose monitoring to continuous glucose monitoring for patients experiencing DKA. When there is a lack of scientific evidence, standard work should be developed, implemented, and monitored.

Children's Mercy Practice Recommendations and Reasoning

Children's Mercy adopted the practice recommendations made by the IDF/ISPAD Clinical Practice Guideline with no variations or additions (International Diabetes Federation, 2010).

Measures

- Time duration to resolution of DKA based on severity
  Number of PICU admissions
- Frequency of hypoglycemia
- Potential cost savings with decreased PICU admissions and lab utilization

Value Implications

- Potential cost savings with decreased PICU admissions and lab utilization

Potential Organizational Barriers and Facilitators

Potential Barriers

- Variability of acceptable level of risk among providers
- Variability of adherence to clinical pathways
- Patients with low socioeconomic status, limited access to medical care for social or economic reasons and patients without private insurance have an increased risk of developing DKA (Ehrmann et al., 2020)

Potential Facilitators

- Collaborative engagement across care continuum settings during clinical pathway development
- High rate of use of clinical pathway
- Standardized order set for Emergency Department, Hospital Medicine, and Pediatric Intensive Care

Power Plans

- Emergency Department: EDP Diabetes: DKA, Bicarb < 16 Pathway
- PICU: PICU DKA

- Inpatient: DKA- Diabetic Ketoacidosis Pathway

Associated Policies

- Insulin Pump
- Continuous Glucose Monitor

Clinical Pathway Preparation

This clinical pathway was prepared by the Evidence Based Practice (EBP) Department in collaboration with the DKA Clinical Pathway Committee composed of content experts at Children's Mercy Kansas City. The development of this clinical pathway supports the Quality Excellence and Safety initiative to promote care standardization that is evidenced by measured outcomes. If a conflict of interest is identified, the conflict will be disclosed next to the committee member's name.

DKA Clinical Pathway Committee Members and Representation

- Ryan McDonough, DO | Endocrinology | Committee Chair
- Tiffany Musick, DO | Endocrinology | Committee Member
- Asdis Wagner, DO | Intensivist | Committee Member
- Alyssa Stoner, MS, DO | Intensivist | Committee Member
- Kayla Heller, MD | Hospitalist | Committee Member
- Theodore Barnett, MD | Emergency Department| Committee Member
- Stephanie Assad, BSN, RN, CPN | 6 Henson Education Coordinator| Committee Member
- Charleen Cunningham, RN, BSN, CPN | Emergency Department | Committee Member
- Sally Fagan, RN, BSN, CCRN-K | PICU Education Coordinator | Committee Member
- Deanna Porter, MSN, RN, CPN | Director, Nursing 6 Henson | Committee Member
- Jana Wheeler, MSN, RN-BC, CPN | Manager, Clinical Practice and Quality | Committee Member

EBP Committee Members

- Kathleen Berg, MD, FAAP | Hospitalist | Evidence Based Practice
- Jarrod Dusin, PhD, CPHQ | Evidence Based Practice
- Megan Gripka, MT (ASCP) SM | Evidence Based Practice

Additional Review & Feedback

- The clinical pathway was presented to each division or department represented on the DKA Clinical Pathway committee as well as other appropriate stakeholders. Feedback was incorporated into the final product.

Implementation & Follow-Up

- Order sets consistent with clinical pathway recommendations were created for each care setting (Emergency Department, Pediatric ICU)
- Education was provided to all stakeholders:
    - Nursing units: Emergency Department, Pediatric ICU
    - Providers from Emergency Medicine, Endocrinology, Critical Care
    - Resident physicians
- Additional institution-wide announcements were made via email, hospital website, and relevant huddles.
- Metrics will be assessed and shared with appropriate care teams to determine if changes need to occur.

Clinical Pathway Development Funding

The development of this clinical pathway was underwritten by the following departments/divisions: EBP, Endocrinology, Emergency and Pediatric Intensive Care departments.

Approval Process

This clinical pathway was reviewed and approved by the DKA Clinical Pathway Committee, Content Expert Departments/Divisions, and the EBP Department; after which they were approved by the Medical Executive Committee. Clinical pathways are reviewed and updated as necessary every 3 years within the EBP Department at CMKC. Content expert teams are involved with every review and update.

Approval Obtained

Department/Unit	Date Approved
Emergency Medicine	December 2024
Hospital Medicine	December 2024
Endocrinology	December 2024
Critical Care Medicine	December 2024

Version History

Date	Comments
April 2013	Version One
January 2014	Version Two
September 2014	Version Three
December 2014	Version Four
December 2015	Version Five
December 2016	Version Six
November 2019	Version Seven
December 2022	Version Eight
December 2024	Version Nine

Date for Next Review

- December 2027

Disclaimer

When evidence is lacking or inconclusive, options in care are provided in the supporting documents and the power plan(s) that accompany the clinical pathway.

These clinical pathways do not establish a standard of care to be followed in every case. It is recognized that each case is different, and those individuals involved in providing health care are expected to use their judgment in determining what is in the best interests of the patient based on the circumstances existing at the time.

It is impossible to anticipate all possible situations that may exist and to prepare clinical pathways for each. Accordingly, these clinical pathways should guide care with the understanding that departures from them may be required at times.

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Appendix A- Supporting Documents

Diabetes Mellitus Epidemiology
Cerebral Edema Therapy
Hypoglycemia Therapy
DKA Fluids CAT
DKA CGM CAT