Evidence-Based Pediatric Diabetic Ketoacidosis Education

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Walden University
2020
Abstract
Evidence-Based Pediatric Diabetic Ketoacidosis Education
by
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MSN, California State University, Fullerton, 2013
BSN, California State University, Fullerton, 2012

Project Submitted in Partial Fulfillment
of the Requirements for the Degree of
Doctor of Nursing Practice

Walden University
August 2020
Abstract

Pediatric patients with diabetic ketoacidosis (DKA) are a unique subset of patients who arrive to the emergency department (ED) for care, and differ from adults regarding symptomology, treatment, and adverse effects. Nurses who will encounter this patient population must have adequate knowledge and confidence to care for them, yet research continues to show a gap in practice. The purpose of this doctoral project was to determine if a staff education project would increase ED nurse knowledge and confidence in caring for pediatric emergency patients with DKA. Utilizing Kirkpatrick’s evaluation model, an evidence-based staff education program was formulated, reviewed by an expert panel, administered via a self-learning packet, and completed by 35 ED nurses. Sources of evidence used to develop the education program were from the International Society for Pediatric and Adolescent Diabetes, the American Diabetes Association, and the Emergency Nurses Association and the Society of Critical Care Medicine. Evaluation of learning outcomes was completed using a paired-samples $t$-test. Findings from this project demonstrated that a self-learning module on pediatric DKA was successful in improving both knowledge ($p < .001$) and confidence levels ($p < .001$) of ED registered nurses with 100% recommending the course to peers. Recommendations are to provide additional staff education in clinical management of pediatric patients with high risk and low volume diagnoses. The staff education project contributed to positive social change by improving nurse knowledge, which may improve patient care and reduce adverse outcomes for pediatric DKA patients.
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Thank you to all my family and friends who have supported me throughout my life both academically and professionally. You have been there for me since the beginning of my love of nursing, encouraging, and supporting me all the way. Your love and support have helped make this all possible and you motivate me to be better. To my parents, who took their 3 young children from England and moved all the way to California, I am thankful; I hope my life is what you hoped for when you moved to provide us a better future. I promise to continue to use my voice to help others and promote positive changes for the nursing profession and all those who need care.

2020 is marked as “Year of the Nurse”; I think it is safe to say that no one expected that to mean we would have to rise to the challenge of facing a pandemic during this year. To all the frontline nurses caring for patient, I appreciate you leaving your families to come to work to care for patients while potentially placing yourself at risk. To the nursing leaders who are trying to put on a brave face while planning for a surge of patients and trying to ensure your staff are safe, I applaud you and know I will look back proud that I am one of you. We will get through this together and we will show the world how valuable nurses really are.
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Section 1: Nature of the Project

Introduction

The emergency department (ED) nurse treats an array of patients with multiple chief complaints, comorbidities, and complexities in care. Pediatric patients are a specialized group that frequent the ED and have a risk of being assessed inappropriately. This is especially true for pediatric patients with diabetic ketoacidosis (DKA) as most patients with diabetes are adults, and children differ from adults in the areas of epidemiology, pathophysiology, response to therapy, and with developmental considerations (Chiang et al., 2018). Since 2000, there has continued to be a rise in the number of pediatric patients diagnosed with diabetes (Centers for Disease Control and Prevention, 2018). For the pediatric patients who are newly diagnosed with diabetes, approximately 25%-30% present to the ED with an initial diagnosis of DKA and begin initial treatment at that time (Dabelea et al., 2014; Long & Koyfman, 2017; von Oettingen, Rhodes, & Wolfsdorf, 2018).

There is an increased risk in younger patients, those with lower incomes, and those who lack health insurance to present with DKA prior to a diabetes diagnosis (Dabela et al., 2014; Long & Koyfman, 2017; von Oettingen et al., 2018). In addition, these patients may present to community EDs where educational resources for staff may be limited (Zee-Cheng, Webber, & Abu-Sultaneh, 2017). A sustained presence of alertness of healthcare providers to signs and symptoms of diabetic complications is required, especially in patients with no prior diagnosis of diabetes (Dabela et al., 2014). There is a practice gap regarding the management of pediatric DKA despite
internationally recognized guidelines for treatment (Clark & Dalabih, 2014; Zee-Cheng et al., 2017). The International Society for Pediatric and Adolescent Diabetes (ISPAD) 2018 clinical practice guidelines recommend that healthcare providers who have the potential to care for pediatric DKA patients complete training in the assessment and management of the disease process in diabetes (Wolfsdorf et al., 2018). My Doctor of Nursing Practice (DNP) project utilized the DNP Staff Education Manual to develop training that would improve staff understanding of the complexities of pediatric DKA and increase comfort in the assessment and management of these patients. My project facilitated social change at the project site by improving emergency nurse knowledge of evidence-based guidelines for the emergent care and management of children with diabetes.

**Problem Statement**

The project site was a nonprofit urban county ED in the Western region of the United States. There had been a recurrent request from the registered nurses (RNs) in the project site ED for supplementary education on the treatment and care of pediatric patients with DKA. The leadership team recognized the demand for more education for ED RNs on evidence-based management of pediatric patients who present in DKA. Lack of knowledge or complete understanding of the pathophysiology, symptomology, and treatment of pediatric DKA could lead to delays in identification, delays in care, and poor patient outcomes and complications (Brink, 2014). In addition, missed or late diagnosis, delayed or inappropriate treatment, and inappropriate monitoring can be associated with increased morbidity and mortality and increased costs to hospitals (Brink, 2014). This doctoral project addressed the need for formal education for ED nurses related to the
management of pediatric DKA. It was necessary to improve staff knowledge of the pathophysiology, symptomology, and treatment for this patient population in order for the ED RNs to effectively advocate for their patients and ensure the highest quality of care to be provided. This doctoral project holds significance for the field of nursing practice in that it promotes the education of frontline nursing staff in the identification and care of a specialized patient population.

**Purpose**

Pediatric patients who present to the ED in DKA require timely and evidence-based care (Chiang et al., 2018; Wolfsdorf, et al., 2018). The gap in practice that was addressed in this DNP staff education project was the need for staff nurse education to improve knowledge and confidence in caring for pediatric patients in DKA. The practice-focused question for this project was:

PFQ: For RNs working in a county ED, did a staff education program on evidence-based treatment for pediatric patients who present with DKA improve knowledge and confidence of the pathophysiology and treatment of DKA in pediatric patients?

This education assisted in closing the gap by increasing the knowledge base of the ED RN caring for this patient population.

**Nature of Doctoral Project**

I completed a literature review of peer-reviewed articles using Medline, CINAHL, Cochrane Review, and PubMed databases to identify evidence-based practice recommendations for emergency nurses who are treating pediatric patients with diabetes.
The search was limited to publications from the past 5 years and included the following combination of key words: emergency, emergency department, pediatric, diabetic ketoacidosis, clinical practice guideline, and evidence. Primary sources of evidence included expert opinion, current clinical practice guidelines, and position statements from relevant professional organizations including, but not limited to, the Emergency Nurses Association, Society of Pediatric Nurses, American Academy of Pediatrics, American Colleges of Emergency Physicians, and the American Diabetes Association. Examples included the Emergency Nurse Core Curriculum and the ISPAD 2018 clinical practice guidelines. Material from the Society of Critical Care Medicine’s Pediatric Fundamental Critical Care Support textbook was also utilized.

This staff education project was delivered via a self-learning module (see Appendix A) with a pre- and posttest (see Appendix B). The gap-in-practice that was addressed was the nurse’s knowledge and level of comfort in regard to the assessment and care of the pediatric DKA patient.

This project included education on the assessment, pathophysiology, and treatment of pediatric DKA. The education included

- the pathophysiology of pediatric DKA,
- the initial and delayed signs and symptoms of DKA in the pediatric patient,
- treatment of DKA specific to the pediatric population,
- potential complications of pediatric DKA and treatment of pediatric DKA, and
- signs of resolution of pediatric DKA.
The primary source of evidence for this project came from the results of the pre- and posttest. A pretest was created based on the topics listed above from the sources of evidence listed above. The pretest was vetted through the project team, as was the education. This pretest was completed prior to the educational program to assess baseline knowledge. Immediately following the education program, the test was taken again to evaluate learning outcomes from the course. Participants assigned themselves a number to write on their pre- and posttest, which allowed comparison of scores. A paired $t$ test was completed to compare pre- and posttest scores to determine whether the educational program was effective. Findings from the before and after education scores answered the project question related to whether education on the management of pediatric patients with DKA improved knowledge and understanding of the pathophysiology and treatment of DKA in pediatric patients.

**Significance**

Members of the project team included the clinical educator of the ED, an ED physician specialized in pediatric care, a pediatric intensive care unit (PICU) physician, and a clinical educator from the PICU. Stakeholders included ED RNs, pediatric emergency patients with DKA, and patients’ families. The significance of this project was that a successful implementation of an educational program for the ED RNs regarding pediatric DKA may lead to earlier identification, appropriate care, and increase in quality of care for the pediatric patient. Proper understanding of the pathophysiology of DKA allows for healthcare providers to provide the best treatment for patients who arrive in DKA (Brink, 2014). This care will then continue to be provided upon admission,
potentially limiting any complications from this disease process and decreasing length of stay due to appropriate and rapid treatment of DKA in the pediatric patient.

This doctoral project holds significance for the nursing field because the education can be applied in other EDs, especially those EDs that have a low pediatric patient volume or have newly graduated RNs who may be unfamiliar with pediatric DKA. This education could also be adapted for different settings such as pediatric medical offices, inpatient pediatric departments, and county education. Staff education related to pediatric DKA may result in positive social change by improving the care of patients with pediatric DKA and reducing adverse outcomes.

**Summary**

As the pediatric population diagnosed with diabetes continues to rise with a significant percentage first presenting with DKA, clinicians who are knowledgeable in the symptomology and treatment of DKA can advocate for appropriate treatment prior to serious complications occurring, which would result in improved patient outcomes (Brink, 2014; Dabela et al., 2014). Education that increases ED RNs clinical knowledge in the care of pediatric DKA patients has a positive impact on social change by improving outcomes for this patient population. In addition, the success of this education could be utilized as a guide for future education on other high-risk patient education topics. The hope was that this education allowed for better outcomes for pediatric patients, including potential decreased need for hospitalization and decreased length of stay. By applying the strategy of education for ED RNs to promote the health of the community pediatric diabetic population, this project supported the mission of Walden
University to promote positive social change. In the next section I discuss the theoretical approaches, relevance to nursing practice, and local background and context of the identified problem.
Section 2: Background and Context

Introduction

As stated in the above section, there has been a rise in the diabetic pediatric population with a large percentage of these patients initially being identified with DKA when being treated in the ED. The practice-focused question for this doctoral project was:

PFQ: For RNs working in a county ED, did a staff education program on evidence-based treatment for pediatric patients who present with DKA improve knowledge and confidence of the pathophysiology and treatment of DKA in pediatric patients?

The purpose of this project was to aid in closing the gap between ED RN knowledge and the appropriate care of a pediatric DKA patient. In this section, I discuss concepts and models used for this project, relevance to nursing practice, local background, the role of the DNP student, and the role of the project team.

Concepts, Models, and Theories

Kirkpatrick Evaluation Model

The model used for the evaluation of this training course was the Kirkpatrick evaluation model. This model has four levels of evaluation: reaction, learning, behavior, and results, and the updated model applies more emphasis on behavior and results than the previous model (Kirkpatrick & Kirkpatrick, 2016). The first level, reaction, is defined as the extent to which the learners deem the education to be constructive, engaging, and pertinent to them (Kirkpatrick & Kirkpatrick, 2016). The course included a postcourse survey that included questions on how satisfying, engaging, and relevant the education
was to aid in the evaluation of the level of reaction. The second level is learning, or the extent to which learners acquire the intended comprehension, proficiencies, attitudes, assurance, and commitment based on their participation in the training (Kirkpatrick & Kirkpatrick, 2016). For this project, this was evaluated by reviewing the posttest results to determine that there had been a change in the knowledge level of the ED RNs.

Behavior, the third stage, is defined as the extent to which the education is applied to the work setting (Kirkpatrick & Kirkpatrick, 2016). Kirkpatrick & Kirkpatrick (2016) discussed that a key concept in this stage is on-the-job learning, which creates the expectation that employees are responsible for their learning and performance. As this course was voluntary, the staff were empowered to complete the course because of their desire to learn and improve their knowledge and skills. Behavior is broken down into critical behaviors and required drivers; specific actions that lead to the desired results and processes that promote critical behaviors (Kirkpatrick & Kirkpatrick, 2016). In this intervention, the required driver was the education provided to the learners in addition to references available to the learner; critical behaviors would be determined via engagement and understanding of the education in addition to the use of the knowledge gained when working with pediatric patients.

The last stage is results, the extent to which measurable outcomes occur due to the education provided (Kirkpatrick & Kirkpatrick, 2016). The updated model states that for nonprofit organizations, this can also be described as accomplishing goals while using resources responsibly (Kirkpatrick & Kirkpatrick, 2016). This result, although not being measured for this project, would be that providing the most evidence-based, highest level
of quality care to pediatric DKA patients would be a successful outcome. Average
admission rates and length of stay decreases, along with fewer ICU admissions, would
show that the project was a success.

Clarification of Terms

*Emergency department nurse:* Any registered nurse currently working in the
emergency department that was the setting of this project.

*Learner/staff/nurse:* The participant and receiver of the project education. This
was any ED RN who completed the course.

Relevance to Nursing Practice

The epidemiology, pathophysiology, and response to treatment for diabetes
differs in the pediatric patient population with the added consideration of developmental
stages (Chiang et al., 2018). Approximately 20%-30% of newly diagnosed diabetic
pediatric patients have this disease process diagnosed after they present to the ED with a
diagnosis of DKA (Dabelea et al., 2014; Long & Koyfman, 2017; von Oettingen et al.,
2018). Additionally, pediatric diabetes patients are increasing in population with each
passing year (Centers for Disease Control and Prevention, 2018). When reviewing this
data concurrently, the conclusion can be made that there will be an annual increase in
pediatric DKA patients presenting to the ED, without a prior history of diabetes.

Thorough knowledge of the pathophysiology, assessment, and treatment of this patient
population is required to provide the most evidence-based and high-quality care. If
knowledge and confidence by ED RNs are lacking, delays in identification and care can
lead to complications and poor patient outcomes including increased morbidity and mortality with increased length of stay and costs (Brink, 2014).

The current evidence includes internationally recognized guidelines for the treatment of pediatric DKA; however, there continues to be a practice gap (Clark & Dalabih, 2014; Zee-Cheng et al., 2017). The 2018 clinical practice guidelines published by the ISPAD recommend that healthcare providers who have the potential to care for pediatric DKA patients have education on the management of this patient population (Wolfsdorf et al., 2018).

Lack of following established protocols contribute to preventable DKA morbidity and mortality (Brink, 2014). Mortality rates from pediatric DKA have not changed since the 1970s (Brink, 2014); there are continued concerns that these rates will not change in the future either (Clark & Dalabih, 2014). DKA continues to be the leading cause of death for children aged 15 years of age or less with diagnosed diabetes mellitus type 1 (Wolfdorf et al., 2018). DKA complications can occur due to errors in initial or subsequent orders and a lack of appropriate monitoring to the dynamic patient changes that can occur with this disease process and its treatment (Brink, 2014). Despite established guidelines for treatment of pediatric DKA, there continues to be a practice gap that occurs at all organizations, not just rural or nonpediatric organizations (Zee-Cheng et al., 2017). Furthermore, even in organizations with standardized order sets, compliance to the order sets can be subpar, leading to suboptimal care of these critical patients (Clark & Dalabih, 2014; Koves et al., 2014; Ronsley, Islam, Ronsley, Metzger, & Panagiotopoulos, 2018). The use of standardized order sets with a systematic review of
literature and dissemination of education to the staff can lead to decreased incidents of adverse effects and a decreased length of stay (Koves et al., 2014). Teaching health care professionals to understand the pathophysiology of DKA helps them provide better patient education and better treatment when DKA occurs (Brink, 2014). Additionally, appropriately trained healthcare professionals may aid in improving patient outcomes and decreasing length of stay (American Diabetes Association, 2019).

This doctoral project addressed the need for formal education for ED nurses related to the management of pediatric DKA. It was necessary to improve staff knowledge of the pathophysiology, symptomology, and treatment for this patient population to enable ED RNs to effectively advocate for their patients and ensure the highest quality of care to be provided.

This doctoral project advanced nursing practice by promoting the education of frontline nursing staff in the care and management of the specialized patient population of pediatric patients with DKA. Current evidence suggested that this patient population requires judicious evidence-based care (Chiang et al., 2018; Wolfsdorf, et al., 2018); at this organization, there was a gap in this practice. This gap was addressed in this DNP staff education project by providing education to ED RNs to improve their comprehension and confidence in caring for pediatric DKA patients. The goal of this DNP project was to contribute to the closing of this gap by increasing the number of ED RNs who had adequate knowledge in the care for this patient population.
Local Background and Context

The institution in which this project took place was an urban, county nonprofit ED in the Western region of the United States that sees an average daily census of 300-325 patients a day. This institution sees a moderate number of pediatric patients, with the organization providing primary care for those without private insurance or without insurance at all. ED staff had discussed concerns regarding comfort level assessing and treating pediatric DKA patients in addition to monitoring for complications; in discussion with the leadership team, this practice focused question was consequently developed.

Mission Statement and Nursing Values

This DNP project aligned with the organization’s mission statement to provide high-quality health care services and to improve the health of the community. Core nursing values that this project aligned with included patient-centered care and excellence as I hoped this project would lead to improved care of pediatric patients in addition to educating nurses in evidence-based care.

State Context

As stated in the previous chapter, approximately 20%-30% of pediatric patients newly diagnosed with diabetes present to the ED with an initial diagnosis of DKA; in addition to there being an increased risk with younger patients, the risks are also greater for lower socioeconomic status groups and those with a lack of health insurance (Dabela et al., 2014; Long & Koyfman, 2017; von Oettingen et al., 2018). For the county in which this hospital provides care, about 15% of the population had no health insurance, with half of these being children; for those with insurance, less than half had employer-based
health care while others had direct-purchase health insurance, Medicare, or Medicaid (U.S. Census Bureau, 2017). Data on the number of pediatric patients with diabetes were not available.

**Role of the Doctor of Nursing Practice Student**

My role in the doctoral project was all-encompassing regarding planning, implementing, and evaluating the program. I completed all research concerning the current evidence and the organization-specific guidelines, and contacted the project team for feedback and approval of the course and course material prior to the start of the intervention. Planning included the formation of the education curriculum and the pre- and posttest assessments. I completed a thorough literature review and analysis in addition to a review of current clinical practice guidelines, position statements, and material from textbooks, and I created a robust PowerPoint educational course. The project objectives included:

1. developing and implementing an educational course using PowerPoint to educate on pediatric DKA,
2. educating ED RNs on the pathophysiology and treatment of DKA in pediatric patients, and
3. evaluating the effectiveness of the educational course upon review of the pre- and posttest scores.

One of my motivations for this doctoral project was that as a clinician I have always taken it upon myself to stay up to date on evidence-based practice to provide the best care for my patients. As a clinical educator, this practice evolved into wanting to
teach others so that they could also provide the best care for their patients. The majority of ED RNs, especially those who do not work in a pediatric ED, can be unfamiliar and uncomfortable with the care of the pediatric patient population; too often they are assessed and treated as adults; however, this is not appropriate per evidence-based guidelines. Providing this education to the ED RNs could aid in increasing the ED RNs’ skill and comfort in assessing and caring for pediatric patients. A potential bias that I may have had for this project was that as the sole researcher and developer of the education project, I may have been unable to see any gaps in the education or weaknesses in the course and course material. Steps that I took to prevent this was to utilize a project team that provided feedback and signed off on the course and course materials prior to the start of the course.

**Role of the Project Team**

The project team consisted of the ED clinical educator, a pediatric emergency medicine provider, the PICU clinical educator, and a PICU physician. The members of the team reviewed the educational content and materials and approved them while I assumed all responsibilities as the leader of the project team. All material was shared with the team via e-mail, and I requested feedback within 1-2 weeks with reminders sent out after the first week. The ED and PICU medical team taught a separate pediatric course, and one of the approved textbooks was recommended and used for this project.

**Summary**

In summary, this doctoral project was designed to educate ED RNs on the pathophysiology and treatment of DKA in the pediatric patient population. The project
was reviewed utilizing the Kirkpatrick evaluation model. I have presented relevance to nursing practice, local context, and relevant background. I have also discussed the role of the doctoral student and the project. In the next section, I discuss the practice problem, sources of evidence, and analysis methods.
Section 3: Collection and Analysis of Evidence

Introduction

At a nonprofit urban county ED, there had been recurrent requests from the staff and leadership for additional education regarding the treatment of pediatric patients with DKA. Delays in the identification and care along with poor patient outcomes and complications can be caused by a deficit in knowledge of the pathophysiology, symptomology, and treatment of pediatric DKA. This can lead to increased morbidity, increased mortality, and increased costs to hospitals (Brink, 2014). The purpose of this doctoral project was to educate ED RNs on the identification and treatment of pediatric DKA. In the previous section, I discussed the extent of the problem at the project site by describing the background and context as well as the roles of those involved in the project. In this section, I discuss the practice-focused question and design for compilation and analysis of data.

Practice-Focused Question

The gap in practice at the location of this project was the need for improvement in staff nurse knowledge and confidence in caring for pediatric patients with DKA. The practice-focused question for this doctoral project was:

PFQ: For RNs working in a county ED, did a staff education program on evidence-based treatment for pediatric patients who present with DKA improve knowledge and confidence of the pathophysiology and treatment of DKA in pediatric patients?
The purpose of the project, educating ED RNs on pediatric DKA, aligned with the practice-focused question as the education was provided to the staff and then the staff were assessed for knowledge and confidence gained with a pre- and posttest, in addition to a course evaluation.

**Sources of Evidence**

The sources of evidence included clinical practice guidelines from the ISPAD, a consensus statement and standards of care from the American Diabetes Association, and textbooks created by and endorsed by the Emergency Nurses Association and the Society of Critical Care Medicine. To answer the practice-focused question, I used research evidence to educate the nurses and to formulate the pre- and posttest questions and answers (see Appendix C), in addition to a course evaluation that was used by the organization. The collection and analysis of this information allowed for the course material to be evidence-based and from vetted and reliable sources to provide a comprehensive education for the nurses. The collection of the pre- and posttest allowed for analysis of knowledge and confidence gained from the course regarding the care of a pediatric patient with DKA. The staff education program plan was be based on evidence from the literature as outlined below.

**Assessment and Monitoring**

The assessment of pediatric DKA includes assessment of dehydration via capillary refill, abnormal skin turgor, dry mucous membranes, sunken eyes, absent tears, weak pulses, and cool extremities; severe dehydration may be associated with weak or
absent peripheral pulses, hypotension, and oliguria (Wolfsdorf et al., 2018; Wolfsdorf, Glaser, & Sperling, 2006;).

Clinical signs of DKA include polydipsia, polyuria, nocturia, enuresis, dehydration, tachypnea, Kussmaul respirations, nausea, vomiting, abdominal pain, confusion, drowsiness, decreasing level of consciousness, and loss of consciousness (Bruzzi et al., 2013; Logee, 2020; Wolfsdorf et al., 2018). Level of consciousness is assessed via the Glasgow Coma Scale, and if the patient does not have normal airway protective reflexes, the airway should be secured and a nasogastric tube placed to prevent aspiration (Brink, 2014; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Cardiac monitoring should be initiated to monitor cardiac rhythm, specifically assessing T-waves for signs of hyper- or hypokalemia (Wolfsdorf et al., 2018). Additionally, accurately measured weight is vital, as all treatment is weight-based; a stated or historical weight is not appropriate (Wolfsdorf et al., 2006). During treatment, at least hourly vital signs, blood glucose, intake and output, and neurological status should be assessed, along with electrolytes every 2 hours (Wolfsdorf et al., 2018).

**Orders and Diagnostics**

Orders to anticipate include serum glucose, electrolytes, blood urea nitrogen, creatinine, serum osmolality, venous pH and pCO2, a complete blood count, urinalysis, and appropriate cultures if the patient is febrile (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006;). Hourly, or more frequently if indicated, assessment and monitoring should include vital signs, neurologic status, administered insulin, fluid intake and output, and capillary blood glucose testing (Wolfsdorf et al., 2006). Laboratory testing, including
serum electrolytes, glucose, calcium, magnesium, phosphorous, and blood gases should be repeated every 2-4 hours or more frequently if clinically indicated (Wolfsdorf et al., 2006)

DKA is diagnosed when there is a presence of hyperglycemia (blood glucose more than 200 mg/dl), a venous pH of less than 7.3 or a serum bicarbonate level less than 15 mmol/L, and with the presence of ketonemia or moderate to large ketonuria (Logee, 2020; Wolfsdorf et al., 2018). Although DKA is characterized by depletion of water and electrolytes both from intra- and extracellular fluid compartments, most pediatric patients will present with normal or even high blood pressures just as urine output will be present until renal blood flow decreases to a critical level (Wolfsdorf et al., 2018). An anion gap is normally 10-14 mmol/L; however, in DKA it is more likely to be 20-30 mmol/L or higher if accompanied by lactic acidosis (Wolfsdorf et al., 2018). The formula is Na - (Cl + HCO3) for calculating an anion gap (Wolfsdorf et al., 2006; Wolfsdorf et al., 2018). The severity of DKA is defined by the degree of acidosis in the venous pH: a pH of 7.2-7.3 is mild, 7.1-7.2 is moderate, and a pH < 7.1 is severe DKA (Wolfsdorf et al., 2006).

Pathophysiology of Pediatric Diabetic Ketoacidosis

Pediatric DKA occurs when there is a deficiency in the circulating insulin along with increased levels of the following counterregulatory hormones: catecholamine, glucagon, cortisol, and growth hormone (Chiang et al., 2018; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). This combination causes an accelerated catabolic state evidenced by an increase in glucose formation via the liver and kidneys and impairs peripheral glucose utilization, both leading to hyperglycemia and hyperosmolality (Wolfsdorf et al.,
The combination also causes lipolysis and ketogenesis, in turn causing the ketonemia and ketonuria and metabolic acidosis (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). The hyperglycemia and hyperketonemia lead to osmotic diuresis, dehydration, and loss of electrolytes that are also exacerbated with vomiting that can occur with severe ketosis (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006;). The cycle then increases as stress hormone is further produced, leading to more severe insulin resistance, worsening hyperglycemia and hyperketonemia, lactic acidosis from hypoperfusion, potential sepsis, and finally fatal dehydration and metabolic acidosis (Wolfsdorf et al., 2018).

Treatment of Pediatric Diabetic Ketoacidosis

As with every patient, the treatment is individualized; some patients may have mild dehydration while others present with severe dehydration (Wolfsdorf et al., 2018). Goals of treatment include to correct the acidosis and reverse ketosis, correct dehydration, normalize blood glucose levels, monitor for complications, and identify and treat precipitating events (American Diabetes Association, 2019; Wolfsdorf et al., 2018). Emergent treatment should follow the guidelines of Pediatric Advanced Life Support and include immediate diagnostic testing, including blood work and urine, assessment of dehydration and level of consciousness, and the insertion of two intravenous (IV) access points (Wolfsdorf et al., 2018). Central venous access should be avoided due to an increased risk of thrombosis (Wolfsdorf et al., 2018).

Fluid replacement is advised to occur prior to the start of insulin therapy to expand the blood volume and restore peripheral circulation with the goal to restore fluid
deficit over 24-48 hours (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Fluid replacement should start with isotonic fluid at a rate of 10ml/kg over 30-60 minutes, or more rapidly if poor tissue perfusion is present, and this may be repeated based on the calculated fluid replacement requirements (Bruzzini et al., 2013; Wolfsdorf et al., 2018). If the patient arrives from another facility and received IV or oral fluids, the amount of that fluid should be factored into the fluid replacement calculations (Wolfsdorf et al., 2018). If the patient presents in hypovolemic shock, fluid replacement should be increased to 20ml/kg of isotonic saline administered as rapidly as possible via large bore cannulas with reassessment after each bolus (Wolfsdorf et al., 2018). Once blood glucose levels are less than 300 mg/dl or if the blood glucose levels decrease by more than 90 mg/dL, IV fluids can be changed to 0.45% Normal Saline and glucose is to be added to the fluids to prevent hypoglycemia (Bruzzini et al., 2013; Wolfsdorf et al., 2018). Maintenance fluids are required as the vascular volume will decrease as plasma glucose concentrations normalize (Wolfsdorf et al., 2018). Oral fluids are typically started within 24 hours of treatment (Wolfsdorf et al., 2018).

Insulin therapy should be started at a rate of 0.05-0.1 unit/kg/hour via an IV infusion at least 1 hour after the start of fluid replacement (Bruzzini et al., 2013; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006;). An IV bolus of insulin is contraindicated in children as it may increase the risk of cerebral edema, can precipitate shock, and can promote hyperkalemia (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Once the ketoacidosis is resolved and the patient is tolerating oral fluids, subcutaneous insulin is to
be started in conjunction with the IV infusion, which is then slowly tapered off (American Diabetes Association, 2019; Wolfsdorf et al., 2018).

Intracellular potassium is lost because increased plasma osmolality draws potassium and water out of cells, in addition to the acidosis and glycogenolysis occurring due to insulin deficiency; if the patient presents with vomiting, this also causes potassium loss, in addition to urinary loss of potassium due to hyperaldosteronism (Brink, 2014; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Insulin therapy will push the potassium back into the cells, decreasing serum potassium levels (Brink, 2014; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). If the patient is hypokalemic, potassium replacement should occur at a rate of 20mmol/L, at the same time as initial fluids and prior to insulin therapy, as the insulin will cause further hypokalemia (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). If the patient is hyperkalemic, potassium treatment is deferred until urine output is documented and then replaced at 20-40 mmol/L (Wolfsdorf et al., 2018).

Assessment of the electrocardiogram may aid in determining if the patient is hypo- or hyperkalemic. Prolonged PR-interval, flattened T-wave, ST-depression, prominent U-waves, and long QT-intervals are signs of hypokalemia, while tall, peaked T-waves and shortened QT-intervals are signs of hyperkalemia (Brink, 2014; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). The maximum rate of potassium replacement is 0.5mmol/kg/h (Wolfsdorf et al., 2018).

Acidosis is reversed with the treatment of fluids and insulin; insulin stops additional ketoacid production and aids in the ketoacids being metabolized leading to bicarbonate generation, and IV fluids improve perfusion and renal function allowing for
the excretion of acids (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). The treatment by administration of bicarbonate is not recommended unless the patient presents with life-threatening hyperkalemia or acidosis with a venous pH less than 6.9 and signs of compromised cardiac contractility (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). If bicarbonate is to be given, the rate is 1-2 mmol/kg over 60 minutes (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006).

Serum sodium levels can be unreliable as glucose in the extracellular fluid causes osmotic movement of water into the extracellular fluid (ECF) causing dilutional hyponatremia (Wolfsdorf et al., 2018). To verify the corrected sodium level, the formula is $\text{Na} + 2(\text{plasma glucose} - 100) / 100 \text{mg/dL}$ and as patient improves the gap between the corrected sodium and the serum sodium levels should narrow (Bruzzini et al., 2013; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). As plasma glucose concentration decreases the serum sodium should increase; a failure of measured serum sodium levels to increase or to decrease more can be seen as a sign of impending cerebral edema (Wolfsdorf et al., 2018).

Serum phosphate is lost due to osmotic diuresis and can continue to fall with insulin therapy as phosphate is pushed into the cells (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Hypophosphatemia may also occur if IV therapy is continued without any oral intake of food after 24 hours of therapy (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Clinical signs include metabolic encephalopathy (irritability, paresthesias, confusion, seizures, and coma), impaired myocardial contractility and respiratory failure due to a weakened diaphragm, dysphagia, ileus, and thrombocytopenia (Wolfsdorf et al., 2018).
Treatment via potassium phosphates is recommended with monitoring of calcium levels as the administration of phosphate may induce hypocalcemia (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006).

Oral replacement and transition to subcutaneous (SQ) insulin - Oral intake is recommended to be started when the patient has clinically improved and only mild acidosis and ketosis is present, or when the venous pH is greater than or equal to 7.3 and the bicarbonate level greater than or equal to 18 mEq/L (Logee, 2020; Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Once ketoacidosis has resolved, and oral intake is tolerated, both long-acting and short-acting insulin should be administered subcutaneously, with the first dose being just before mealtime (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Treatment and type of insulin given are patient-specific; the first dose of SC insulin will be given concurrently with IV infusion but the time of administration varies on the type of insulin. Before stopping the insulin infusion, rapid-acting insulin will be given 15-30 minutes prior, regular insulin will be given 1-2 hours prior, and long-acting insulin will be given the evening before the infusion is stopped the next morning (Wolfsdorf et al., 2018). Strict and frequent blood glucose monitoring is continued to prevent hypo- or hyperglycemia (Wolfsdorf et al., 2018).

**Adverse Effects**

Symptomatic cerebral edema occurs in 0.5%-1% of pediatric DKA patients (Wolfsdorf et al., 2006). However, cerebral injury is the major cause of mortality and morbidity with cerebral edema accounting for 60%-90% of all DKA deaths and with 10%-25% of survivors of cerebral edema having significant residual morbidity.
(Wolfsdorf et al., 2018). Cerebral edema typically develops within the first 12 hours of
treatment but can also occur prior to treatment and is rarely seen 24-48 hours after
treatment has started (Wolfsdorf et al., 2018). Cerebral edema will present with a
headache or worsening headache after the initiation of treatment, decreased level of
consciousness (restlessness, irritability, increased drowsiness, confusion, incontinence),
cranial nerve disturbances, decreasing oxygen saturation, and a late sign of Cushing’s
triad (increasing blood pressure, bradycardia, and respiratory depression) (Wolfsdorf et
al., 2018; Wolfsdorf et al., 2006). Patients who are at high risk for cerebral edema include
those with elevated serum urea nitrogen concentration, severe acidosis, and severe
hypocapnia; those patients with a higher degree of dehydration and hyperventilation are
at more risk than those with higher blood glucose levels or osmotic changes during
treatment (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006;). Demographics of patients with
increased risk include younger pediatrics, new-onset diabetes, and longer duration of
symptoms (Logee, 2020; Wolfsdorf et al., 2018).

Diagnostic criteria for cerebral edema include abnormal motor or verbal responses
to pain, decorticate or decerebrate posture, cranial nerve palsy, and abnormal neurogenic
respiratory pattern (grunting, tachypnea, Cheyne-Stokes respirations) (Wolfsdorf et al.,
2018). Major criteria for cerebral edema include altered mentation, confusion, fluctuating
level of consciousness, sustained heart rate deceleration (decrease of more than 20 beats
per minute) not caused by improved intravascular volume or sleeping, and age-
inappropriate incontinence (Wolfsdorf et al., 2018). Minor criteria for cerebral edema
include vomiting, headache, lethargy or not easily awakening, diastolic blood pressure
>90 mm Hg, and age <5 years (Wolfsdorf et al., 2018). Cerebral edema can be correctly diagnosed with a 92% sensitivity if a patient has one diagnostic criterion, two major criteria, or one major and two minor criteria (Wolfsdorf et al., 2018).

If a pediatric patient is showing signs of cerebral edema, IV therapy should be decreased to a rate that maintains blood pressure without causing excessive fluid administration that may increase cerebral edema (Wolfsdorf et al., 2018). Treatment should be hypertonic intravascular fluid, such as mannitol or hypertonic saline; mannitol, at a rate of 0.5-1 g/kg IV over 10-15 minutes and hypertonic saline, at a rate of 2.5-5 ml/kg over 10-15 minutes (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). The effects of mannitol should be seen within 15 minutes and should last 120 minutes; if there is no response to the initial dose of mannitol, a subsequent dose or hypertonic saline can be administered 15-30 minutes after the initial dose (Wolfsdorf et al., 2018). The head of the bed should be elevated to 30 degrees and head should be kept midline and supplies for intubation should be readily available for those patients with impending respiratory failure (Wolfsdorf et al., 2018). Treatment should not be delayed obtaining cranial imaging; clinical symptoms should be utilized to identify cerebral edema, and imaging should occur after treatment is complete to determine if there are lesions such as a hemorrhage or thrombosis (Wolfsdorf et al., 2018).

**Prevention of Future Diabetic Ketoacidosis Episodes**

DKA management is not complete until the cause has been identified and treated (Brink, 2014; Wolfsdorf et al., 2018). In known diabetics, if the DKA is not brought on by a febrile illness or gastroenteritis, the most common cause is psychosocial or failure to
appropriately administer insulin; in new-onset diabetes, a delay in diagnosis is the most common case (Wolfsdorf et al., 2018). There may be a failure to monitor blood glucose levels or a failure to administer adequate insulin (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Psychosocial concerns include insulin omission in an attempt to lose weight in conjunction with an eating disorder, a way of escaping a difficult home situation, or depression (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). A psychiatric social worker or clinical psychologist should be consulted to identify any psychosocial contributions to the development of DKA (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006). Education should be provided on the recognition and treatment of DKA and when to seek medical help; if patients and parents are appropriately educated, there is a 10-fold decrease in DKA recurrence (Wolfsdorf et al., 2018; Wolfsdorf et al., 2006).

**Diabetic Ketoacidosis or Hyperglycemic Hyperosmolar State**

Hyperglycemic hyperosmolar state (HHS) is diagnosed with a blood glucose more than 600 mg/dL, venous pH greater than 7.25, serum bicarbonate greater than 15mmol/L, absent to mild ketonemia, serum osmolality greater than 320mOsm/kg, and altered consciousness (obtundation/combative) or seizures (Wolfsdorf et al., 2018). In HHS, treatment includes IV fluid replacement to gradually expand volume, restore renal perfusion, and correct serum sodium and osmolality (Wolfsdorf et al., 2018). Insulin treatment should only occur once glucose levels are decreasing less than 50 mg/dL an hour with fluid replacement only and should be started at a rate of 0.025-0.05 units/kg/hr (Wolfsdorf et al., 2018).
Evidence Generated for the Doctoral Project

Participants

Participants included the ED RNs working within the organization; Nurse participants volunteered to independently complete the course with there being no selection from the project team leader or team. Nurses were notified about the educational program through flyers posted in the departments, emails, and huddle announcements at each shift; participation was voluntary. The practice-focused question was relevant to emergency nurses as they all have the potential to care for a pediatric DKA patient.

Procedures

A pre- and posttest was created to collect evidence on knowledge and confidence gained from the course material. The pre- and posttest was the same and tested the learner’s knowledge using multiple choice questions and answers. These tools were vetted through the project team before implementation and drew upon the information from the evidence discussed above. A program evaluation rubric was used by the project team to score the education program (See Appendix D). A course evaluation (See Appendix E) was be given to the learners; this evaluation was organization-specific and a requirement from the Department of Education. Originally this course was proposed to be an in-person lecture with a PowerPoint didactic, given at different times and dates for maximum participation. The mode of delivery was changed to self-directed learning because a global pandemic, novel corona virus disease (COVID-19) occurred and guidelines included to restrict social gatherings to less than five (5) persons. The
organization chose to cancel all in-person courses; therefore, the staff education was
changed to an independent self-learning module with all forms, tests, and materials in a
packet that had course instructions written on it.

**Protections**

Participation in this project was strictly voluntary and this was presented as an
option to the staff. All pre- and post-tests, along with course evaluations, were
anonymous. Copies of the Consent Form for Anonymous Questionnaires from the DNP
Staff Education Manual were provided to participants. The education program
participants were asked to identify their course evaluations and pre- and post-tests with a
number unique to them so that data could be evaluated from before and after the course
without the ability to identify the individual. These documents were reviewed by the team
leader only and were stored in a locked drawer that only the team leader could access
using a key. This key was kept on the team leaders’ person to ensure no other individuals
had access to confidential records. The participants were notified of these safeguards and
that course data would not be shared with other nurses, leadership, or the organization.
Additionally, the Institutional Review Board (IRB) at Walden University approved the
project, in addition to the protections, prior to the start of the course and data collection
(IRB approval number 03-06-20-0979807). Signature on the site approval form from the
DNP Staff Education Manual were obtained prior to IRB approval.

**Analysis and Synthesis**

Data were recorded and tracked in an Excel spreadsheet that was password
protected; this spreadsheet was located on the team leaders’ laptop which was also
password protected. Data on this spreadsheet were organized per unique number and included the pre- and post-test scores, in addition to the course evaluation ratings. Data were statistically analyzed using SPSS version 25 to determine the effectiveness of the program and to answer the practice-focused question. To assure the integrity of the evidence, missing information, such as pre- or post-test scores was omitted. Statistical analysis included a paired $t$ test comparing the pre- and post-test results to answer the practice focused question.

**Summary**

The practice-focused question for this project is: For RNs working in a county ED, does a staff education program on evidence-based treatment for pediatric patients who present with DKA improve knowledge and confidence of the pathophysiology and treatment of DKA in pediatric patients? The evidence to support this question is biphasic; evidence from leaders in the care of pediatric DKA, in addition to organization-specific information, will be utilized to create the education but also to create the pre- and post-test for the staff. The pre- and post-test, in addition to the course evaluation, will be evaluated statistically to determine the effectiveness of the project. Section 4 will discuss the findings and recommendations of this project.
Section 4: Findings and Recommendations

Introduction

At the project site, a nonprofit county ED, there has been a need for supplementary education for RNs on the treatment and care of pediatric patients with DKA. Delays in identification and care and poor patient outcomes and complications leading to increased morbidity and mortality can be caused by a lack of understanding of the pathophysiology, symptomology, and treatment of pediatric DKA (Brink, 2014). The gap in practice is that despite internationally recognized guidelines for treatment, there are still disparities in the delivery of care and a lack of integration of evidence-based practice (Clark & Dalabih, 2014; Zee-Cheng et al., 2017). The practice-focused question for this doctoral project was:

PFQ: For RNs working in a county ED, does a staff education program on evidence-based treatment for pediatric patients who present with DKA improve knowledge and confidence of the pathophysiology and treatment of DKA in pediatric patients?

The purpose of this project was to educate ED RNs on pediatric DKA and improve their knowledge and confidence regarding this disease process and its’ treatment.

I completed a literature review of peer-reviewed articles using Medline, CINAHL, Cochrane Review, and PubMed databases to identify evidence-based practice recommendations for emergency nurses who are treating pediatric patients with DKA. The search included the following combination of key words: emergency, emergency department, pediatric, diabetic ketoacidosis, clinical practice guideline, and evidence.
Primary sources of evidence included clinical practice guidelines from the ISPAD, a consensus statement and standards of care from the American Diabetes Association, and textbooks created by and endorsed by the Emergency Nurses Association and the Society of Critical Care Medicine. These sources were utilized to create the staff education and the pre- and posttest; all these items were vetted through the project team prior to initiation of the staff education. Analysis of knowledge and confidence gained from the course regarding the care of a pediatric patient with DKA was achieved with the collection and analysis of the pre- and posttest results. This data were recorded and tracked in an Excel spreadsheet and was organized per unique number and included the pre- and posttest scores, in addition to the course evaluation ratings. I conducted statistical analysis, including a paired $t$ test comparing the pre- and posttest results, utilizing SPSS version 25 to determine the effectiveness of the program and to answer the practice-focused question. Any data that was missing was omitted to assure the integrity of the evidence.

### Findings and Implications

A total of 50 packets were sent out to the ED RNs and a total of 35 RNs participated in the study by returning the packet. One RN did not complete the posttest, three RNs did not complete the course evaluation, and 16 did not complete the pre- and postcourse confidence levels. Data (see Appendix F) were compared between the 34 RNs who did fully complete the pre-and posttests and the 19 RNs who completed the pre- and postcourse confidence levels. As shown in Table 1, the mean pretest scores (9.97) were lower than the mean posttest scores (13.29). On average, the RNs improved their scores
by at least three (3) points, meaning they answered three (3) more questions right on the posttest. The \( p \) value (.000), shown on Table 2, demonstrated a statistically significant improvement in posttest scores. Table 3 shows the mean precourse confidence levels (3.11 or somewhat confident) were lower than the mean postcourse confidence levels (4.11 or fully confident). On average, the RNs increased their confidence level by 1 point. Additionally, as shown on Table 4, the \( p \) value (.000) leads to the conclusion that there would be an increase in confidence levels for any RN taking this course.

Table 1

*Pre- and Posttest Scores Statistics*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>N</th>
<th>Std. deviation</th>
<th>Std. error mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest scores</td>
<td>9.97058823529</td>
<td>34</td>
<td>1.71420613515</td>
<td>.293983926821</td>
</tr>
<tr>
<td>Posttest scores</td>
<td>13.2941776470</td>
<td>34</td>
<td>1.60547192655</td>
<td>.275336163889</td>
</tr>
</tbody>
</table>

Table 2

*Pre- and Posttest Scores Paired Differences*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std. deviation</th>
<th>Std. error mean</th>
<th>95% confidence interval of the difference</th>
<th>Lower</th>
<th>Upper</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest score – Post-test score</td>
<td>-3.3235294</td>
<td>2.04080477</td>
<td>.349995131</td>
<td>-4.0355998 -2.614589</td>
<td>-9.496</td>
<td>33</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3

*Pre- and Posttest Confidence Statistics*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>N</th>
<th>Std. deviation</th>
<th>Std. error mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest confidence</td>
<td>3.11</td>
<td>19</td>
<td>.809</td>
<td>.186</td>
</tr>
<tr>
<td>Post-test confidence</td>
<td>4.11</td>
<td>19</td>
<td>.567</td>
<td>.130</td>
</tr>
</tbody>
</table>
Table 4

*Pre- and Posttest Confidence Paired Differences*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std. deviation</th>
<th>Std. error mean</th>
<th>95% confidence interval of the difference lower</th>
<th>Upper</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest confidence – Posttest confidence</td>
<td>-1.000</td>
<td>.882</td>
<td>.202</td>
<td>-1.425</td>
<td>-.575</td>
<td>-4.943</td>
<td>18</td>
<td>.000</td>
</tr>
</tbody>
</table>

The course evaluation asked participants to review the course on a numeric scale from 4-excellent, 3-good, 2-fair, and 1-poor. Table 5 shows the mean results of 3.91 for clarity of content, relevance to clinical practice, support materials, and overall evaluation, with a mean of 3.81 for my understanding of the content. Additionally, when asked if they would recommend this course to a colleague, all answered “yes” (see Appendix G). Specific course evaluation comments (Table 6) determined that the course was considered easy to understand, detailed, and informative.

Table 5

*Course Evaluation Data*

<table>
<thead>
<tr>
<th></th>
<th>Clarity of content</th>
<th>My understanding of content</th>
<th>Relevance to clinical practice</th>
<th>Support materials</th>
<th>Overall evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>3.91</td>
<td>3.81</td>
<td>3.91</td>
<td>3.91</td>
<td>3.91</td>
</tr>
<tr>
<td>N</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
</tr>
<tr>
<td>Std. deviation</td>
<td>.296</td>
<td>.397</td>
<td>.296</td>
<td>.296</td>
<td>.296</td>
</tr>
<tr>
<td>Std. error mean</td>
<td>.052</td>
<td>.070</td>
<td>.052</td>
<td>.052</td>
<td>.052</td>
</tr>
</tbody>
</table>
Table 6

Course Evaluation Comments

<table>
<thead>
<tr>
<th>Participant number</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Very well presented, easy to follow and understand</td>
</tr>
<tr>
<td>13</td>
<td>Easy to understand</td>
</tr>
<tr>
<td>17</td>
<td>More detailed than I expected; it provided great refresher info, as well as new info that I was not aware of</td>
</tr>
<tr>
<td>25</td>
<td>Informative</td>
</tr>
<tr>
<td>221</td>
<td>Excellent course, very clear and to the point</td>
</tr>
<tr>
<td>1010</td>
<td>Very informative!</td>
</tr>
<tr>
<td>1503</td>
<td>Very informative</td>
</tr>
<tr>
<td>8912</td>
<td>Information was clear, concise, and well presented</td>
</tr>
<tr>
<td>51501</td>
<td>Great! Informative!</td>
</tr>
<tr>
<td>74324</td>
<td>Great presentation, info was presented in clear flow, easy to learn and retain</td>
</tr>
<tr>
<td>1000000</td>
<td>Great information. Easily understood.</td>
</tr>
</tbody>
</table>

The major unanticipated limitation in the implementation of this project was that during this time the world is facing a pandemic due to an outbreak of novel coronavirus (COVID-19). Due to this outbreak the original format for this course, an in-person PowerPoint presentation, could not occur. The course was then changed to a handout with written material that was returned by the RN. The packet did ask the nurses to work independently and to complete the pretest without looking at the material; the pretest was also placed before the course content in an effort to prevent the RNs from reviewing the material first. However, as the RNs anonymously completed the packets, there is no way of determining if this actually occurred; findings from data analysis demonstrated an improvement in every respondents scores, which could be correlated to them completing the pretest without reviewing the material first. Another limitation is that there is no way to determine that the RNs retain the information the course provided and will utilize it the next time a potential pediatric DKA patient presents to the ED. Ideally there should be a decrease in admission rates and length of stays for these patients; however, the evaluation
of patient outcomes is not a part of this project. An unanticipated outcome of this course was that out of the questions, the most common posttest questions RNs answered incorrectly were regarding diagnostic testing prior to treatment for cerebral edema and psychologic causes of DKA. The impact of this finding is that the RNs did not fully understand these concepts and in practice this could lead to a delay in treatment of cerebral edema and a relapse into DKA if staff do not address the psychological causes of DKA.

These findings discussed above have individual, community, institutional, and systems level implications. For the RN as an individual, the findings determine that RNs can increase their knowledge base and raise their confidence level in caring for pediatric patients with DKA using a self-directed education program. At an individual and a community level these findings would correlate to earlier identification of pediatric DKA by ED RNs, resulting in improved care and decreased complications. At an institutional level, this increased knowledge base has the potential for earlier recognition and treatment of pediatric DKA, leading to fewer patients being admitted and a decrease in length of stay for those patients admitted. At a system level, these findings determine that refresher courses on critical elements such as complex patient events such as DKA lead to improved knowledge and confidence in caring for patients, leading to better care and decreased complications. This project led to increased ED RN knowledge and confidence with pediatric DKA patients, which has the potential to lead to early recognition and treatment of pediatric DKA patients presenting to the ED, decreased admission rates,
reduced lengths of stay, and improved outcomes. All this may lead to a healthier diabetic pediatric population and potentially decrease morbidity and mortality for this population.

**Recommendations**

Recommended solutions that will potentially address the gap-in-practice stated above revolve around continued education of the staff. Although the results demonstrated that an educational packet aids in closing the gap as evidenced by improved knowledge and confidence scores, an interactive in-person lecture using the course material may lead to even more improved confidence levels and knowledge retainment. Using the course material along with the pre-/posttest and pre-/posttest answer key would be an ideal way to continue to improve ED RNs knowledge and confidence in caring for pediatric DKA patients. Staff should be given enough time to sign up for the course, and multiple date and time offerings will improve course attendance. Administering only the pretest prior to the start of the course would be beneficial in obtaining an accurate pretest result. A posttest administered immediately after the completion of the course would evaluate knowledge gained; however a posttest administered at least 1 week later would evaluate knowledge retained. As pediatric patients with DKA in the ED is common but not common enough that RNs are able to obtain and maintain a solid knowledge and confidence base, frequent reeducation on this topic is necessary to fully decrease the gap-in-practice.

**Contributions of the Doctoral Project Team**

The doctoral project team consisted of myself, a pediatric emergency medicine provider, an ED clinical educator, a PICU physician, and a PICU clinical educator. My
Responsibilities included everything in this project from research of evidence, development of the educational program, and collection and analysis of data. The responsibilities taken on by the other team members included reviewing all the course content and materials and providing feedback. Based on feedback received (Appendix F) some changes were made to the final presentation that was given to the ED RNs. Plans to extend this project beyond the DNP doctoral project include teaching the course as it had originally been planned, an in-person interactive lecture. An additional plan is to publish this DNP project as a quality improvement project in the *Journal of Emergency Nursing*.

**Strengths and Limitations of the Project**

Strengths of this project include that the project was successful in increasing staff knowledge and confidence in the care of pediatric DKA patients in addition to receiving positive feedback via the course evaluations. The information was provided concisely and clearly, and the RNs found it to be very informative. Additionally, this topic is one that is important to ED RNs as these patients often present to the ED, and staff must be knowledgeable in the recognition and care of these patients. Limitations of this project include that the course was not able to be presented in the format originally intended, and due to this, there is no way to ensure that the data is completely accurate or if there would be a change in the course evaluations. Recommendations for future projects include continued education on pediatric specific clinical presentations of diseases and disorders in either an in-person or self-learning teaching modality. Additionally, education on any high-risk, low-frequency topic, such as eclampsia or care of patients with
ventriculostomies, would also be beneficial for ED RNs who may not feel comfortable caring for these patient populations.
Section 5: Dissemination Plan

**Dissemination Plan**

Dissemination of this project at the current practice site in addition to other practice sites will lead to advancing nursing practice and confidence when caring for pediatric patients with DKA, resulting in improved patient outcomes and decreased adverse effects and reoccurrences of DKA. Plans to disseminate this work to the institution staff include providing an in-person lecture at a later time to allow for diverse learning styles and needs. As this project is in regard to pediatric patients with diabetes, this course would be appropriate for anyone who cares or will care for these patients including nurses working in the ED, inpatient pediatric departments, outpatient pediatric clinics, and residents and nursing students. Appropriate dissemination to the broader nursing profession includes providing the course at different organizations so more nurses can receive the material, in addition to publishing the educational material to a journal such as the *Journal of Emergency Nursing*.

**Analysis of Self**

My career as an RN has always been about progression and challenging myself. I started as a new graduate RN training and working in the ED. During this time, I had self-doubts that made me think the ED may not be the right setting for me, and perhaps I would not succeed. As my experience grew, my confidence also grew, and I tried to foster learning in my coworkers and new nurses coming into the ED. After 5 years as a bedside RN, I decided to challenge myself by applying for an ED clinical educator position at a different organization, which allowed me to grow as a learner and leader,
learning the results of effective and ineffective leadership. Leaving this position, I briefly worked at an organization that also had some ineffective leadership; however, my position in this organization allowed me to find my voice and speak up in a professional manner. In my position now at a different organization, I feel that I have stagnated in the role of a clinical educator but am excited to try a different challenge in a different position. Additionally, as a young leader, I found there are always individuals who challenge my knowledge and ideas; however, I do not let this stop me from ensuring my voice is heard, and it motivates me to grow into different positions. I feel that complacency leads to stagnation, and it is my goal to never be stagnant.

As a scholar prior to any type of nursing education I was one who was not the most motivated, and though I would be distracted in lectures, I did quite well in school. Once I started my RN education, I strived to be a nurse who tried her very best instead of one who chose to get by. I had the benefit of attending a program that allowed me to achieve both my bachelor’s and master’s degree simultaneously, and I was benefited by having professors who truly cared about my growth as a scholar and learner. As an inexperienced leader, however, I feel the material I was taught was difficult to grasp as I was still working at the bedside. This doctoral program was different, as I have years of leadership experience and can reflect on my leadership style in addition to apply concepts learned. Although I am at the end of this program, I will strive to continue to grow as a scholar and keep my learning alive.

As a clinical educator I have created staff development material before. In this aspect, this project was familiar to me. However, the use of an expert review panel and
the data collection and analysis was something that was new to me, and I was excited for this challenge and growth. Time management has always been a challenge as I am a tenured procrastinator; however, with this project I have had only minor delays and setbacks. Challenges included the need for me to change the setting of my project due to the inability to obtain IRB approval; however, this was obtained at my clinical setting in a very timely manner. The biggest and most unexpected challenge for this project was the arrival of novel Coronavirus (COVID-19) to California. The social distancing demands forced me to change my project from an in-person lecture to a self-learning module. Fortunately, the RNs given the self-learning module promoted my project completion by returning the data needed in a very timely manner.

I have gained several insights during my scholarly journey. Firstly, I have learned a lot about the topic of my project, pediatric DKA; one of the reasons why I chose this topic was that it was something that I also was not comfortable or fully knowledgeable about. I have also gained insight into how to write in a scholarly manner and how to present scholarly material in a clear and organized way. As self-doubt is present in any individual, I have learned that I am capable of anything I set my mind to, and I have knowledge and ideas that are valuable to the nursing profession. I look forward to the future projects and endeavors this leads to.

**Summary**

ED nurses often are the first clinician a patient sees in addition to being ever present during their treatment in the ED. Pediatric patients present a challenge in that they are a specialized patient population with different responses to disease processes,
clinical presentations, treatment plans, and adverse reactions. Additionally, with complex patient situations, especially ones that may not be seen commonly, it is important that ED RNs be up to date on their knowledge of these disease processes in a pediatric patient. DKA is a complex disease process which, although is seen in the ED, is not a common enough that ED RNs feel comfortable in their knowledge of or care for. As many have never diagnosed pediatric diabetic patients presenting to the ED in DKA, it is critical that ED RNs be knowledgeable in all facets surrounding this disease process and its’ treatment, in addition to feeling confident in caring for this patient. Increased knowledge and confidence in caring for pediatric DKA patients can lead to early recognition, promote appropriate treatment, identify adverse effects, and decrease length of stays and negative outcomes.
References


doi:10.1542/peds.2013-2795


OBJECTIVES

- Upon completion of this course, the learner will:
  - Understand the pathophysiology of pediatric diabetic ketoacidosis (DKA)
  - Verbalize the initial and delayed signs and symptoms of DKA in the pediatric patient
  - Understand treatment of DKA specific to the pediatric population
  - Recognize potential complications of pediatric DKA
  - Verbalize the treatment of pediatric DKA
  - Recognize the signs of resolution of pediatric DKA
The material for this course is compiled from the following:

- Clinical Practice Guidelines from the International Society for Pediatric and Adolescent Diabetes (ISPAD)
- Consensus statement and standards of care from the American Diabetes Association (ADA)
- Textbooks created by, and endorsed by, the Emergency Nurses Association (ENA) and the Society of Critical Care Medicine (SCCM)

Since 2000 the number of pediatric patients diagnosed with diabetes has continued to rise. For those pediatric patients newly diagnosed with diabetes, 25-30% present to the Emergency Department with an initial diagnosis of DKA and begin the initial treatment at that time.

For these patients, there is an increased risk of presenting with DKA if:

- The patient is younger
- Comes from a family with lower incomes,
- From a household with a lack of health insurance.

ISPAD 2018 clinical practice guidelines recommend that healthcare providers who have the potential to care for pediatric DKA patients complete training in the assessment and management of the disease process.
ASSESSMENT AND MONITORING

Clinical Signs\textsuperscript{3,7,9}

- Polydipsia
- Polyuria
- Nocturia
- Enuresis
- Tachypnea/Kussmaul respirations
- Nausea/Vomiting
- Abdominal pain
- Confusion/drowsiness/decreased level of consciousness or loss of consciousness
- Dehydration

Assessment of Dehydration\textsuperscript{9,10}

- Prolonged capillary refill
- Abnormal skin turgor
- Dry mucous membranes
- Sunken eyes
- Absent tears
- Weak pulses
- Cool extremities
- Severe dehydration:
  - Weak/absent peripheral pulses, hypotension, oliguria

ASSESSMENT AND MONITORING (CONT.)

- LOC- If the patient cannot protect their airway (loss of gag reflex), the airway should be secured and a nasogastric tube placed to prevent aspiration\textsuperscript{2,9,10}
- Cardiac monitoring: Assess T-waves for signs of hyper- or hypokalemia\textsuperscript{9}
- Most pediatric patients will present with normal or high blood pressures and urine output until renal blood flow decreases to a critical level\textsuperscript{9}
- An accurate weight in kilograms is vital as all treatment is based on weight-based calculations\textsuperscript{10}
- At least hourly assessment of: vital signs, blood glucose, intake and output, and neurologic status\textsuperscript{9,10}
- Lab testing every 2 hours (electrolytes, glucose, calcium, magnesium, phosphorous, blood gases)\textsuperscript{9,10}
ORDERS AND DIAGNOSTICS

Orders to anticipate include:
- Serum glucose, electrolytes, blood urea nitrogen, creatinine, serum osmolality, venous pH and pCO2, a complete blood count, urinalysis, and appropriate cultures (if febrile).\(^7,9,10\)

DKA is diagnosed when:
- Hyperglycemia (blood glucose >200mg/dL) \(^7,9\)
- Venous pH < 7.3 or serum bicarbonate < 15 mmol/L \(^7,9\)
- Ketonemia or moderate to large ketonuria \(^7,9\)
- Anion gap 20-30 mmol/L or higher if accompanied by lactic acidosis (normal 10-14 mmol/L) \(^9,10\)
  - Calculation for anion gap is Na-(Cl + HCO3) \(^9,10\)

The severity of DKA is defined by the degree of acidosis in the venous pH \(^10\)
- Mild- 7.2-7.3, moderate- 7.1-7.2, and severe-pH<7. \(^10\)

PATHOPHYSIOLOGY OF DKA

DKA occurs when there is a deficiency in the circulating insulin and counterregulatory hormones (catecholamine, glucagon, cortisol, & growth hormone) \(^5,9,10\)

- This causes the body to go into an accelerated catabolic state with an increase in glucose production by the liver and kidneys with an impairment of peripheral glucose utilization, causing hyperglycemia and hyperosmolality \(^9,10\)
- This also causes lipolysis and ketogenesis, that causes ketonemia and ketonuria. \(^9,10\)
- This also causes metabolic acidosis. \(^9,10\)

The hyperglycemia & hyperketonemia cause osmotic diuresis, dehydration, and a loss of electrolytes (this can be more severe if the patient is vomiting due to severe ketosis) \(^9,10\)

  - This cycle increases as stress hormone is further produced, leading to more insulin resistance, worsening hyperglycemia & hyperketonemia, lactic acidosis due to hypoperfusion, and potential sepsis. \(^9\)
- This can all lead to fatal metabolic acidosis and fatal dehydration. \(^9\)
TREATMENT- OVERVIEW

- Treatment is dependent on the severity of dehydration.\textsuperscript{9}
- The end goals of treatment are:
  - Correct the acidosis and reverse ketosis.\textsuperscript{1,9}
  - Correct dehydration.\textsuperscript{1,9}
  - Normalize blood glucose levels.\textsuperscript{1,9}
  - Monitor for complications.\textsuperscript{1,9}
  - Identify and treat precipitating events.\textsuperscript{1,9}
- Any emergent presentations should follow Pediatric Advanced Life Support (PALS) guidelines.\textsuperscript{9}
  - 2 IV access points but central line should be avoided due to increased risk of thrombosis.\textsuperscript{9}

TREATMENT- FLUID REPLACEMENT

- Fluid replacement must start prior to the start of insulin therapy to expand blood volume and restore peripheral circulation. Fluid deficit should be restored over 24-28 hours.\textsuperscript{9,10}
- Isotonic fluid at 10ml/kg over 30-60 minutes or faster if poor tissue perfusion (can be repeated).\textsuperscript{3,9}
  - Hypovolemic Shock: 20ml/kg.\textsuperscript{3,9}
  - Pre-hospital fluids should be included in overall fluid calculations.\textsuperscript{9}
- Once blood glucose <300, or there is a decrease in more than 90 mg/dL/hour, change to 0.45% NS and add glucose.\textsuperscript{3,9}
- Vascular volume decreases as plasma glucose normalizes.\textsuperscript{3,9}
- Oral rehydration should start within 24 hours of treatment.\textsuperscript{9}
TREATMENT- 2 BAG METHOD

- Many organizations utilize this method for maintenance fluids for pediatric patients.
- Both bags started concurrently and have the same electrolytes, but 1 bag also has 10% dextrose added.
- Aids in a gradual decrease in blood glucose levels via titration of the fluids, and less titration of the insulin.

<table>
<thead>
<tr>
<th>Blood Glucose</th>
<th>Bag 1 (No Dextrose)</th>
<th>Bag 2 (D 10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greater than 300</td>
<td>100% of total IV fluid rate</td>
<td>0% of total IV fluid rate</td>
</tr>
<tr>
<td>250-300</td>
<td>50% of total IV fluid rate</td>
<td>50% of total IV fluid rate</td>
</tr>
<tr>
<td>200-249</td>
<td>25% of total IV fluid rate</td>
<td>75% of total IV fluid rate</td>
</tr>
<tr>
<td>Less than 200</td>
<td>0% of total IV fluid rate</td>
<td>100% of total IV fluid rate</td>
</tr>
</tbody>
</table>

TREATMENT- INSULIN

- Start at a rate of 0.05-0.1 unit/kg/hr at least 1 hour after start of fluids.\(^3,9,10\)
- IV bolus of insulin increases risk of cerebral edema, can precipitate shock, and promote hyperkalemia.\(^9,10\)
- When starting the IV insulin, flush the tubing with 20 ml of insulin to saturate the binding sites and discard.
- Once ketoacidosis is resolved, and the patient can tolerate PO fluids, start subcutaneous insulin while slowly tapering off intravenous insulin.\(^1,9\)
- The type of insulin will be dependent on the insulin the patient takes at home and per the medical team.
TREATMENT - POTASSIUM

- Hypokalemia causes
  - Acidosis, gluconeogenesis, and increased plasma osmolality pulls potassium and water out of cells.2,9,10
  - Urinary loss due to hypoaldosteronism and vomiting2,9,10
  - Insulin therapy that pushes potassium back into cells2,9,10
  - Replace potassium at a rate of 20mmol/L at the same time as fluids before insulin.9,10
  - Patients can also be hyperkalemic, if so treat after urine output is documented and give fluids with 20-40mmol/L.9
- Assess EKG
  - Hypokalemic: prolonged PR-interval, flattened T-wave, ST-depression, U-waves, long QT-interval2,9,10
  - Hyperkalemic: tall, peaked T-waves and shortened QT-interval2,9,10

TREATMENT - ACIDOSIS

- Acidosis will be corrected during treatment (fluids and insulin).9,10
  - Insulin stops ketoacid production and aids in its metabolism which generates bicarbonate.9,10
  - IV fluids improve perfusion and renal function allowing for acid excretion.9,10
- Bicarbonate is only to be given if there is life-threatening hyperkalemia or acidosis with venous pH <6.9 and signs of compromised cardiac contractility.9,10
  - Administer at 1-2mmol/kg over 60 minutes.9,10
TREATMENT - SODIUM

- Dilutional hyponatremia is seen as water shifts into the extracellular fluid due to the elevated glucose levels.  
  - Corrected Sodium formula is $Na + 2([\text{plasma glucose}-100]/100) \text{ mg/dL}$.  
  - As patient improves the serum sodium and corrected sodium levels should become more similar.  
  - If there is no increase in serum sodium levels or there is a larger gap, this can be a sign of impending cerebral edema.

TREATMENT - PHOSPHATE

- Loss due to osmotic diuresis and insulin therapy that pushes phosphate into cells.  
  - Also occurs with IV therapy without oral intake of food within 24 hours of therapy.  
- Signs of hypophosphatemia
  - Metabolic encephalopathy (irritability, paresthesias, confusion, seizures, coma), impaired myocardial contractility and respiratory failure d/t a weakened diaphragm, dysphagia, ileus, or thrombocytopenia.  
  - Treat with potassium phosphates but monitor for signs of hypocalcemia.
TREATMENT WEANING

- Oral intake should be started when mild acidosis and ketosis is present, or when venous pH is >7.3 and bicarbonate is >18mEq/L. 
- If oral intake is tolerated, long and short acting insulin should be administered with the first dose just before mealtime.
- Patient specific on which type of insulin (short or long) depending on what patient takes in the past.
- If giving rapid-acting insulin, Insulin infusion should be stopped 15-30 min after SQ administration, 1-2 hours after SQ administration of regular insulin, and in the am after a long-acting insulin was administered SQ the evening prior.
- Patient requires strict and frequent blood glucose monitoring for hypo- or hyperglycemia.

ADVERSE EFFECTS: CEREBRAL EDEMA

- Symptomatic cerebral edema occurs in 0.5-1% of pediatric DKA patients.
- In that percentage, 60-90% patients die and 10-25% of the survivors have significant residual morbidity.
- Typically occurs within the first 12 hours of treatment but can also occur prior to treatment and rarely 24-48 hours after treatment.
- High risk: those with elevated serum urea nitrogen concentrations, severe acidosis, severe hypcapnia.
- Patients with higher degrees of dehydration and hyperventilation at more risk than those with higher blood glucose levels.
- Demographics: younger pediatrics, new-onset diabetes, and longer duration of symptoms.
- Symptoms:
  - Headache or worsening headache after the initiation of treatment, cranial nerve disturbances, decreasing oxygen saturation.
  - Decreased level of consciousness (restlessness, irritability, increased drowsiness, confusion, incontinence).
  - Cushing's triad (increasing blood pressure, bradycardia, and respiratory depression)- late sign.
DIAGNOSTIC CRITERIA

- **Diagnostic criteria (needs 1)** for cerebral edema include:
  - Abnormal motor or verbal responses to pain, decorticate or decerebrate posture, cranial nerve palsy, and abnormal neurogenic respiratory pattern (grunting, tachypnea, Cheyne-Stokes respirations).\(^9\)

- **Major criteria (need 2 or 1 major & 2 minor):**
  - Altered mentation, confusion, fluctuating level of consciousness, sustained heart rate deceleration (decrease of more than 20 beats per minute) not caused by improved intravascular volume or sleeping, and age-inappropriate incontinence.\(^9\)

- **Minor criteria (2 minor with 1 major):**
  - Vomiting, headache, lethargy or not easily awakening, diastolic blood pressure >90 mm Hg, and age <5 years.\(^9\)

Cerebral edema can be correctly diagnosed with a 92% sensitivity if a patient has:

- One diagnostic criterion.\(^9\)
- Two major criteria.\(^9\)
- One major and two minor criteria.\(^9\)

TREATMENT

- Decrease IV therapy to a rate that maintains BP without causing excessive fluid administration.\(^9\)

- Hypertonic fluid (mannitol or hypertonic saline).\(^9,10\)
  - Mannitol- 0.5-1g/kg IV over 10-15 minutes. Works in 15 min and lasts 120 min.\(^9,10\) May repeat or give hypertonic saline 15-30 min subsequently.\(^9\)
  - Hypertonic Saline- 2.5-5ml/kg over 10-15 min.\(^9,10\)

- Elevate head of bed to 30 degrees and keep head midline.\(^9,10\)

- May need to be intubated.\(^9,10\)

- Do not delay treatment for cranial imaging; clinical symptoms should be utilized for diagnostics.\(^9,10\)
  - Imaging after treatment is complete to determine if lesions are present (hemorrhage or thrombus).\(^9,10\)
PREVENTION OF FUTURE EPISODES

- DKA management is not complete until the cause has been identified and treated.2,9
  - In known diabetics if not due to febrile illness or gastroenteritis the most common cause is psychological or failure to appropriately administer insulin.9
  - In new onset diabetics the most common cause is a delay in diagnosis.9
- Causes:
  - Failure to monitor blood glucose levels or failure to administer adequate insulin.9,10
  - Psychological concerns: insulin omission to lose weight in conjunction with an eating disorder, a way of escaping difficult home situation, depression.9,10
  - Psychiatric Social Worker/Clinical psychologist should be consulted.9,10
- Education of patient and family on recognition and treatment of DKA and when to seek help.9,10
  - Appropriate education decreases DKA recurrence 10-fold.9,10

DKA OR HYPERGLYCEMIC HYPEROSMOLAR STATE (HHS)

- HHS presents with:9
  - Blood glucose > 600 mg/dL
  - Venous pH > 7.25
  - Serum bicarbonate > 15 mmol/L
  - Absent to mild ketonemia
  - Serum osmolality > 320 mOsm/kg
  - Altered consciousness (obtundation/combativeness) or seizures

- Treatment:9
  - IV fluids to expand volume, restore renal perfusion, and correct serum sodium/osmolality
  - Insulin once glucose levels are decreasing < 50 mg/dL an hour with fluid replacement only
  - Start insulin at a rate of 0.025-0.05 units/kg/hr


Appendix B: Pre-/Posttest

Unique ID Number __________________

Pre-Test or Post-Test

Pediatric Diabetic Ketoacidosis Pre/Post Test

Please circle if this is your PRE or POST test. Please identify tests with a number unique to you.

Do NOT write your name on this document.

My current confidence level with assessment, knowledge, and treatment of pediatric diabetic ketoacidosis is (circle one):

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<th>Not Confident at All</th>
<th>Slightly Confident</th>
<th>Somewhat Confident</th>
<th>Fairly Confident</th>
<th>Extremely Confident</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

1. Clinical signs of diabetic ketoacidosis include
   a. Polydipsia, polyuria,
   b. Confusion, abdominal pain
   c. Dizziness, headache
   d. All of the above
   e. A & B only

2. True/False: Most pediatric patients will present with normal or high blood pressures and urine output until critical decreases in renal blood flow.
   a. True
   b. False

3. DKA is diagnosed with:
   a. Blood sugar greater than 200 mg/dl
   b. Venous pH less than 7.3 or serum bicarbonate less than 15 mmol/L
   c. Ketonemia or moderate/large ketonuria
   d. Anion gap 20-30 mmol/L
   e. All of the above

4. True/False: Pre-hospital IV fluids should not be included in fluid calculations
   a. True
   b. False

5. True/False: Fluid replacement must start prior to the start of insulin therapy
   a. True
   b. False

6. Treatment goals for fluid replacement are to restore fluid deficit over
   a. 4-8 hours
   b. 8-16 hours
   c. 24-28 hours
   d. 24-48 hours

7. Insulin therapy for pediatric DKA is dosed as follows:
a. IV bolus of 10 units, and then infusion at 0.05-0.1 units/kg/hr
b. Subcutaneous bolus of 10 units, and then infusion at 0.05-0.1 units/kg/hr
c. No bolus, IV infusion of 0.05-0.1 units/kg/hr
d. No bolus, IV infusion of 0.5-1 units/kg/hr

8. True/False: Hypokalemia should be corrected with initial fluid administration prior to insulin.
   a. True
   b. False

9. True/False: Sodium Bicarbonate should be given to any pediatric DKA patient who is acidic.
   a. True
   b. False

10. Those at higher risk for cerebral edema include
    a. During the first 12 hours of treatment
    b. Patients with more severe dehydration and hyperventilation
    c. Younger pediatrics, new-onset diabetes
    d. Higher blood glucose levels
    e. All except D

11. A diagnostic criterion for cerebral edema is
    a. Tachycardia
    b. Abnormal neurogenic respiratory pattern
    c. Vomiting
    d. None of the above

12. Major criteria for cerebral edema include
    a. Fluctuating level of consciousness
    b. Sustained heart rate deceleration
    c. All of the above
    d. None of the above

13. True/False A CT must be obtained prior to initiating treatment for cerebral edema
    a. True
    b. False

14. In known diabetics causes of pediatric DKA include
    a. Febrile illness
    b. Gastroenteritis
    c. Psychological
    d. Failure to appropriately administer insulin
    e. A, B, & D
    f. All of the above

15. Psychological concerns include
    a. Insulin omission to lose weight
    b. Depression
    c. Escaping a difficult home situation
    d. All of the above
Appendix C: Pre-/Posttest Answer Key

Pediatric Diabetic Ketoacidosis Pre/Post Test Answer Key

1. E
2. A
3. E
4. B
5. A
6. C
7. C
8. A
9. B
10. E
11. B
12. C
13. B
14. F
15. D
Appendix D: Completed Expert Panel Review Form

<table>
<thead>
<tr>
<th>Program Element</th>
<th>Evaluation Question</th>
<th>1 Strongly disagree</th>
<th>2 Disagree</th>
<th>3 Neutral</th>
<th>4 Agree</th>
<th>5 Strongly Agree</th>
</tr>
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<tr>
<td>Objectives</td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td>The program objectives are aligned with the program presentation and posttest.</td>
<td></td>
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<tr>
<td></td>
<td>Comments (Optional):</td>
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<tr>
<td>Presentation Materials</td>
<td>The presentation adequately addresses the program objectives.</td>
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<tr>
<td></td>
<td>The presentation integrates current evidence and scholarly literature.</td>
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</tr>
<tr>
<td></td>
<td>The presentation is professionally developed, well organized, and free of grammatical/spelling errors.</td>
<td></td>
<td></td>
<td></td>
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<td>X</td>
</tr>
<tr>
<td></td>
<td>Comments (Optional): Slide 5: add moderate to large ketones in the urine, sweet(fruity) odor to breath, Slide 6: rephrase or add “normal” to urine output, Slide 6: Mention CO2, I saw you included causes towards the end of the presentation, add traumatic stress on the body and a pump insertion coming out or not functioning.</td>
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<td>Participant Posttest</td>
<td>The program pre and posttest is clearly written and addresses the key elements addressed in the presentation.</td>
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<tr>
<td></td>
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<tr>
<td>Program Element</td>
<td>Evaluation Question</td>
<td>1 Strongly disagree</td>
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<td>3 Neutral</td>
<td>4 Agree</td>
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<tr>
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<td>Comments (Optional): emphasizing a conservative fluid bolus, potassium dose should be weight based</td>
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## Appendix E: Course Evaluation Form

### Course Evaluation Form

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1. **Program Evaluation**

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2. **GENERAL COMMENTS**

   A. Would you recommend this program to your colleague? Yes No

   B. Suggestions for improvement:

   ______________________________________________________

   ______________________________________________________

   C. General Comments: __________________________________

   ______________________________________________________

   D. Other topics of interest you would like to learn:

   ______________________________________________________

   ______________________________________________________
### Appendix F: Pre- and Posttest Data

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