ED Legal Letter™

The Essential Monthly Guide to Emergency Medicine Malpractice Prevention and Risk Management

From the publishers of *Emergency Medicine Reports* and *ED Management*

Thomson American Health Consultants Home Page—http://www.ahcpub.com For more information, call (800) 688-2421. CME for Physicians—http://www.cmeweb.com

Diabetic ketoacidosis: Don't miss this potential killer

By **James E. Szalados, MD, MBA, MHA, FCCP, FCCM,** Associate Professor of Anesthesiology and Medicine, University of Rochester; Medical Director of Respiratory Care, Director of Quality, and Attending in Critical Care and Medicine; Attending Anesthesiologist, Westside Anesthesia Associates of Rochester, NY; Unity Health System Adjunct Clinical Professor, Rochester Institute of Technology, Rochester, NY; Student, University of Buffalo School of Law, Buffalo, NY.

***Editor's note**: Emergency practitioners must be well versed in the management of diabetic emergencies. New-onset diabetes discovered on presentation to the emergency department (ED) is not an infrequent occurrence. The recognition and management of this condition can be lifesaving. Whether the patient is newly diagnosed or has longstanding diabetes mellitus, prompt recognition and rapid treatment of complications of diabetic ketoacidosis (DKA) will reduce adverse outcomes and shorten hospital stays. Numerous cases in which physicians encountered litigation for mismanagement of diabetic emergencies are presented in this issue of* ED Legal Letter.

Introduction

DKA is the most serious metabolic complication of diabetes mellitus Type 1, formerly known as insulin-dependent diabetes mellitus (IDDM). DKA accounts for more than 160,000 hospital admissions in the United States per year. The incidence of DKA is 4.6-8 per 100,000 person-years among patients with diabetes.[1] The estimated mortality for DKA is 4-10%. The highest rates of DKA occur among teenagers and in the elderly. DKA-related complications are the most common cause of death in children, teen-agers, and young adults with diabetes. DKA accounts for approximately 50% of all deaths in diabetics younger than 24 years of age.[2]

Case :#1: Hospital Policies and Procedures

In *Lyons v. Walker Regional Medical Center,*[3] the administrator of Kenneth Cook's estate brought a wrongful-death suit against the treating hospital and the ED charge nurse. Mr. Cook, a county jail detainee, was taken to a regional

hospital with complaints of lower abdominal pain, nausea, and hematemesis that had lasted two weeks.

At 4 p.m., Mr. Cook's triage vital signs were taken by an employed registered nurse, Ms. Evans, who did not regularly work in the ED. Hospital policy required triage nurses to have two years' of ED experience. Mr. Cook's blood pressure was 159/106 mmHg, and his pulse was "a little fast."[3] Nurse Evans drew blood for a complete blood count at 4:15 p.m., the results of which were reported at 5:37 p.m. and revealed a "slightly elevated white blood cell count." She then turned the care of the patient over to another nurse, Nurse Hunter, who at 5:08 p.m. requested a blood analysis for electrolytes, prothrombin time (PT), and partial thromboplastin time (PTT). Blood work was collected at 5:26 p.m.

The attending physician, Dr. Boone, first saw Mr. Cook at 5:45 p.m. and ordered a nasogastric tube placed to check for blood in Mr. Cook's stomach. Nurse Evans attempted to place the tube but encountered some difficulty. Mr. Cook then refused further attempts. Thus, at 6:05 p.m. Nurse Hunter had Mr. Cook sign a form indicating that he was refusing medical treatment. Almost simultaneously, at 6:07 p.m., the computer reported the results of Mr. Cook's electrolyte panel. The blood urea nitrogen (BUN) was slightly elevated at 26 mg/dL, and chloride level was normal at 101 mEq/L. The computer highlighted, with the words "PANIC VALUES EXCEEDED," the markedly elevated potassium of 7.2 mEq/L, carbon dioxide content of 7 mEq/L, the serum glucose of 599 mg/dL, and the creatinine of 2.6 mg/dL.

The hospital had a policy and procedure manual that required markedly abnormal laboratory results immediately be reported by telephone to the charge nurse. She in turn would record the results on the front page of the chart and inform the physician immediately. In this case, there was no follow-up to the laboratory results because, in Nurse Hunter's words, "once the patient is discharged . . . the patient is no longer the hospital's responsibility, and there's no need to report panic levels." The final entry in the chart was completed at 6:10 p.m.

Nurse Hunter did not inform Mr. Cook or his accompanying guard of Mr. Cook's elevated blood pressure, pulse rate, and serum potassium; that important other laboratory results still were pending; or that he may have a potentially life-threatening condition. In fact, neither Mr. Cook nor his guard received any instructions whatsoever prior to Mr. Cook's discharge back to the county jail. Mr. Cook died on May 10, 1994. Defendants won a summary judgment, which later was reversed and remanded by the appellate court.[4]

Discussion

In addition to underscoring the life-threatening electrolyte disorders that accompany DKA, this important case highlights three important process-related issues: 1) the need for an effective system for communication of aberrant test results in acute care areas to the physician in charge; 2) adherence to hospital policies; and 3) the process of obtaining informed consent/refusal of treatment.

Frequently, DKA is the first presentation of new-onset diabetes. The mortality from DKA is usually the result of hypovolemic shock, profound metabolic acidosis, or other electrolyte derangements. Hypovolemic shock results in widespread tissue underperfusion and subsequent end-organ dysfunction. Acidosis and the

ED Legal Letter™, ISSN 1087-7341, is published monthly by Thomson American Health Consultants, 3525 Piedmont Road N.E., Bldg. 6, Suite 400, Atlanta, GA 30305.

Vice President/Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
Production Editor: Nancy McCreary
GST Registration Number: R128870672.
Periodicals postage paid at Atlanta GA 30304.
POSTMASTER: Send address changes to ***ED Legal Letter,*** P.O. Box 740059, Atlanta, GA 30374.

Back issues: $82. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

THOMSON
AMERICAN HEALTH CONSULTANTS

Subscriber Information

Customer Service: (800) 688-2421
Customer Service E-Mail Address: customerservice@ahcpub.com
Editorial E-Mail Address: allison.mechem@thomson.com
World Wide Web: http://www.ahcpub.com

Subscription Prices
United States: $489 per year
Multiple Copies:
2-9 additional copies: $391 each.
10+ copies: $293 each.
Canada: $519 per year plus GST
Elsewhere: $519 per year

Accreditation

Thomson American Health Consultants is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide CME for physicians. American Health Consultants (AHC) designates this educational activity for up to 18 credit hours in Category 1 credit toward the AMA Physician's Recognition Award. ***ED Legal Letter™*** is also approved by the American College of Emergency Physicians for 18 hours of ACEP Category 1 credit. This CME activity was planned and produced in accordance with the ACCME Essentials.

This continuing education program is sponsored by Thomson American Health Consultants, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation. Provider approved by the California Board of Registered Nursing for approximately 18 contact hours. Provider #CEP10864.

Questions & Comments

Please contact **Allison Mechem,** Managing Editor, at allison.mechem@thomson.com or (404) 262-5589.

Now available on-line at www.ahcpub.com/online.html

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Hubler (executive editor); Advisory Board members Ball, Bitterman, Blaylock, Gibbs, Lawrence, Montgomery, Moore, Severson, Sullivan, and Weaver have reported no relationships with companies having ties to the field of study covered by this CME program. Dr. Szalados (author) has no disclosures.

accompanying hyperkalemia acutely depress cardiovascular function, sometimes resulting in cardiac arrest.

Ketoacidosis represents one type of increased anion-gap metabolic acidosis (others include renal tubular acidosis, lactic acidosis, and poisoning with salicylate, paracetamol, acetaminophen, or with methanol, ethylene glycol, or other toxic alcohols).

Ketoacidosis is the result of insulin deficiency and hyperglucagonemia. In the absence of insulin, fatty acids are oxidized in the liver to produce ketoacids, primarily aceto-acetate and beta-hydroxybutyrate. The buildup of ketoacids causes metabolic acidosis, manifested as a decrease in serum bicarbonate and pH. Insulin deficiency also causes hyperglycemia, which results in an osmotic diuresis once the filtration threshold for glucose is exceeded in the renal tubules. The osmotic diuresis then results in intravascular volume depletion. Intravascular volume depletion leads to diminished cardiac output and tissue perfusion, which worsens the metabolic acidosis via lactic acidosis. In the case of *Lyons v. Walker,* such diminished perfusion may have manifested itself as stress gastritis within the gastric mucosa, causing hematemesis.

Electrolyte abnormalities are the hallmark of DKA.[5] Typically, there are predictable electrolyte derangements that occur in a biphasic pattern: pretreatment and post-treatment. As the pH serum becomes more acidic, paralleled by low serum bicarbonate, potassium moves from the intracellular space to the intravascular space. Therefore, patients with DKA may present with acidosis and hyperkalemia, which can be acutely life threatening. Hyperkalemia depresses the excitability of tissues that rely on electrical potentials, such as nerve and muscle. Acute hyperkalemia may arrest the heart. Later, as DKA resolves, hypokalemia, hypomagnesemia, and hypophosphatemia predominate.

The dehydration is reflected in an elevated ratio of serum BUN to creatinine; however, following protracted dehydration and diminished renal perfusion, the creatinine itself may be elevated because of acute renal failure. Diabetics especially are predisposed to acute renal failure because diabetic microangiopathy is a sequela of long-standing hyperglycemia, thus causing a diminished renal reserve over time. Where renal failure has occurred, a renal tubular acidosis may complicate the assessment of the DKA patient's response to therapy using acid-base status alone. In such a case, the rising potassium and worsening acidosis constitute a life-threatening emergency that would result in death if left untreated.

When appropriate aggressive therapy is initiated and the intravascular volume is restored using appropriate volumes of isotonic fluid (preferably normal saline), insulin is given simultaneously and glucose is driven intracellularly, where it can be used in metabolism. Therefore, both ketoacidosis and lactic acidosis respond to hydration and insulin therapy. As the acidosis resolves, potassium moves intracellularly, resulting in lowered serum potassium levels. In fact, although serum potassium may be elevated prior to treatment, total body potassium stores are severely depleted in the setting of DKA. Serum potassium must be monitored carefully during treatment of DKA to avoid hypokalemia. In addition, there often is depletion of magnesium and phosphate, which are essential to prevent myocardial dysrhythmias such as torsades de points and to facilitate energy storage, respectively. Up to 90% of DKA patients will develop acute hypophosphatemia within six to 12 hours of beginning therapy for DKA.[6] However, unlike other electrolyte abnormalities, lack of phosphate has not been shown to cause any detriment.

Since the abnormalities in serum chemistries generally are very predictable, regular and frequent "stat" laboratory testing is fundamental to appropriate medical care of DKA. Obviously, in the busy ED or intensive care unit (ICU) setting, protocols for obtaining lab specimens, reporting abnormal results, and immediate notification of the treating physician are vital to good patient care. Acute care settings are characterized by their intensity and complexity. Therefore, where hospital policies define the criteria for staffing based on training and experience, deviation from these policies can result in errors of commission or omission.

Finally, the processes of "refusal to consent to treatment" and "discharged against medical advice" are based on an informed decision. Therefore, not only must the patient have the competence and capacity, but the risks, benefits, and the providers must disclose alternatives. Moreover, the above should be documented clearly in the record; a failure to do so would fall below the standard of care in obtaining refusals. In this case, a refusal of nasogastric intubation might not necessarily constitute a refusal of all treatment, especially after appropriate disclosure using the "reasonable patient" standard.

In this case, it could be argued that insertion of the nasogastric tube represented a nonessential intervention. A patient frequently may refuse one level of

therapeutic intervention but reasonably expect that all other reasonable options be offered and provided. For example, patients may refuse cardiopulmonary resuscitation but can nonetheless expect appropriate medical care during their hospitalization, or patients may refuse blood transfusions but may nonetheless remain candidates for surgery. Limitations of therapy such as do not resuscitate orders (DNRs) do not constitute a directive of "do not treat." Health care providers are obligated to communicate with patients or their proxies to define the parameters of therapy and to document such communications.

In this case, the charge nurse or the treating physician did not inform the patient of his abnormal lab results. Even if he already had left the ED, he should have been contacted by phone or other means. The knowledge that the patient had a potentially life-threatening condition revealed in his lab results makes the providers not only medically/legally culpable, but also morally responsible to make reasonable attempts to have the patient return to the ED. Appropriate patient contact and service recovery in this case may have saved the patient's life.

Case #2: Electrolyte Disorders in DKA

In *Metcalf v. United States*,[7] plaintiff Thomas O. Metcalf, an insulin-dependent diabetic diagnosed in 1977 at the age of 34, was admitted to the Washington, DC, Veterans Administration Medical Center at approximately 3 p.m. on July 18, 1983, with severe abdominal pain, nausea, and vomiting, and was later diagnosed as having appendicitis. Dr. E. Pendleton Alexander, the admitting surgeon, erroneously recorded in his admitting note that Metcalf was a noninsulin-dependent diabetic (diabetes mellitus Type II). The preoperative blood glucose was approximately 272 mg/dL. The plaintiff underwent an appendectomy that same day at 7:52 p.m.

Postoperatively, Mr. Metcalf's serum glucose was recorded as 333 mg/dL, requiring treatment with six units of regular insulin, which were administered at 10:20 p.m. At the time, the patient was taking nothing by mouth and was receiving only IV fluids containing 5% glucose with potassium at the rate of 125 cc per hour. Results of blood work drawn at 7:30 a.m. the following morning were: serum glucose of 112 mg/dL, potassium 4.3 mEq/L, and BUN 9 mg/dL (all essentially normal). No insulin was ordered or administered that day.

On the following day at 7 a.m., a urine test revealed 3+ glucose but no acetone. However, by 1 p.m. that same day, the patient's urine was remarkable for only 1+ glucose but a large amount of acetone. A progress note written at 8:30 p.m. described Mr. Metcalf to be "feeling terrible," vomiting dark-green liquid, and perspiring profusely. At 9 p.m., a urinalysis revealed 3+ glucose and large amounts of acetone. The evening shift's nursing notes described Mr. Metcalf to be "confused and breathing in short, rapid respirations" and his skin to be "warm and dry." In order to treat his anxiety, Mr. Metcalf received 8 mg morphine at 1:20 a.m.

At 7 a.m., Dr. Alexander discovered his patient in a state of disorientation and hyperventilation. Shortly thereafter, Mr. Metcalf became apneic and required cardiopulmonary resuscitation. Blood tests at that time revealed a serum glucose of 1161 mg/L, potassium of 8.0 mEq/L, BUN of 36 mg/dL, and a blood pH of 6.85.

Mr. Metcalf was resuscitated, then transferred to the medical ICU with the diagnosis of DKA. He was comatose and febrile (with temperatures as high as 105.3° F), and he also was diagnosed as having pneumonia. Since he continued in a stuporous mental state, an electroencephalogram (EEG) was performed on July 28, which was read as "extremely abnormal." Mr. Metcalf remained hospitalized until Nov. 10, 1983, and a neurologist testified that he lived with the sequelae of "significant permanent organic brain damage, with difficulties in the areas of mental status functioning and cerebellar functioning."

However, the court disagreed with the contention that Mr. Metcalf had suffered significant and permanent brain damage as result of his hospitalization and treatment. The court found that Mr. Metcalf's contributory negligence precluded any recovery; this was based on the documentation that he had described his diabetes as "stable and controlled" when he first presented for treatment. The court used the reasonable person standard to determine that patients similarly situated in the plaintiff's position normally would be expected to submit complete and truthful information about their medical history. On these bases, the court found on behalf of the defendant.

Discussion

The fundamental principles of the management of DKA are: 1) restoration of intravascular volume and tissue perfusion; 2) correction of hyperglycemia and

ketoacidosis by the administration of insulin; and 3) aggressive monitoring and correction of serum electrolyte abnormalities. The release of catecholamines, glucagons, and cortisol are the hallmark of the neuroendocrine response to surgical stress in the perioperative period. This response increases the state of insulin resistance, potentially predisposing or exacerbating DKA.[8]

The tort of negligence requires proof of the elements of an established duty, breach of that duty, a causation of harm, a sufficient causal connection to support proximal cause, and damages. Where a plaintiff can demonstrate that the care rendered did not meet the standard of care that would be rendered by like professionals and resulted in damages, recovery in litigation is likely.

In this case, the medical staff had a duty to monitor and treat the patient for reasonably likely metabolic derangements that could occur in the postoperative period. In addition, abrupt decompensation in high-risk patients sometimes can be unpredictable. However, when the therapeutic interventions are appropriate, the adverse outcomes may be unpreventible. There appears to have been a delay in treatment of this patient's DKA. However, for some reason that is not apparent in the record; this delay did not satisfy the threshold of negligence in this case.

Patients also have a responsibility to disclose material facts in their history that may impact their medical care. Since patients cannot be expected to foresee which facts might be important, detailed questioning during medical history taking is designed to prompt patients.

This case also illustrates just how rapidly DKA can progress. Clinicians need to be aware that diabetics are significantly more susceptible to infection and that such infection may precipitate DKA.

Case #3: Documentation

In *Howard v. St. Edward Mercy Medical Center*,[9] the patient's widow brought suit against both the medical center and the ED physician for medical malpractice in the death of her husband. Mr. Howard, age 52, presented to the ED at St. Edward Mercy Medical Center at approximately 4 p.m. complaining of sore throat, fever, a "burning sensation in his chest," and blurred vision. The triage nurse recorded an admission heart rate of 139 beats per minute.

Dr. Crabtree, a board-certified emergency medicine physician with six years of experience, who was working as an independent contractor to the hospital, then examined the patient. Dr. Crabtree did not document the tachycardia, and found the patient's mucous membranes to be pink and moist. The triage nurse made no mention of polyuria and polydipsia. Dr. Crabtree also denied knowledge of such complaints. However, the patient's eldest daughter testified that she had to provide the details of the medical history because "her father was too weak and sick" to answer questions.

Dr. Crabtree consulted a gastroenterologist, and an endoscopy was scheduled for the following morning. When Mr. Howard was discharged from the ED at 4:54 p.m. that day, he was so weak that he had to be taken by wheelchair to his car. Mrs. Howard testified that her husband rested on the couch that evening but made frequent trips to the bathroom; and as the evening progressed, her husband became increasingly disoriented and confused.

The family took Mr. Howard back to the St. Edward Mercy Medical Center ED at 11:30 p.m. and told the triage nurse that Mr. Howard had been vomiting for the past week and that he complained of blurred vision, weakness, and excessive sleeping. Dr. O'Mara, an ED physician who was certified in family practice, examined Mr. Howard on this return visit. Dr. O'Mara's physical examination revealed a drowsy, confused patient with "very dry mucous membranes." Laboratory testing revealed serum glucose of 1504 mg/dL, and the patient was admitted to the ICU. By 8:15 the following morning, Mr. Howard was in a coma; and shortly after 9 a.m., he suffered a cardiopulmonary arrest from which he could not be resuscitated. He was pronounced dead at 9:35 a.m.

At trial, Dr. O'Mara testified that Dr. Crabtree's treatment fell below the standard of care because he did not address Mr. Howard's elevated heart rate, which indicated dehydration. On cross-examination, the defense challenged Dr. O'Mara's testimony based on the notations in the medical record wherein Dr. Crabtree documented pink and moist mucous membranes.

The defense introduced expert testimony from Dr. Liebovich, a board-certified emergency medicine physician, who testified that Dr. Crabtree had indeed met the standard of care for ED physicians in Fort Smith, AR. The expert witness opined that it was "not unusual for a patient presenting to the ED to have an elevated heart rate, which subsequently

returns to a normal rate," and that the referral for endoscopy the next day was appropriate.

The defense also presented expert testimony from Dr. Fonseca, a professor of medicine and endocrinology at Tulane University, who testified that the most classic symptoms of diabetes are excessive thirst and frequent urination, and that according to the medical records these symptoms were not present on presentation to Dr. Crabtree. In addition, Dr. Fonseca testified that "an elevated pulse rate is not necessarily a symptom of diabetes, and there was no way, without laboratory tests, to know what Mr. Howard's blood sugar was . . ." The jury rendered a 10-2 verdict in favor of Dr. Crabtree.

Discussion

This case reinforces the importance of careful documentation in the clinical record. Documentation refers not only to a reiteration of clinical data such as physical examination, vital signs, radiology reports, and laboratory data, but also to the process of clinical reasoning and decision making. The latter is very important and often unrecognized by physicians who prefer objective documentation. However, since medical errors are not the same as negligence, the distinction often hinges on the documentation found in the clinical record.

During the initial phases of a lawsuit, the plaintiff's attorney obtains all applicable medical records and obtains expert review to determine whether the allegations of medical malpractice have merit. The level of documentation within the record is a key factor in determining whether to commence suit. The documentation should reflect both objective and subjective data obtained through history and physical examination, vital signs, and references to supporting radiographic and laboratory test results.

The documentation also should reflect clinical impressions, reasons for assigning different weight to various data elements, or reasons for discounting various data. A differential diagnosis with supportive reasoning may help in the defense of a poor outcome. Moreover, documentation also should reflect aspects of communication such as the parties involved, the relationships, the permission of the patient for their involvement, the discussions underlying consent or refusal of consent, and discharge instructions given to the patient and caregivers.

The final element of good documentation is appropriate dating and timing of a contemporaneous entry. There are no circumstances that can justify altering the medical record. Where alterations occur and are discovered, the credibility of the defendant will be destroyed.

Patients presenting with tachycardia are not necessarily dehydrated. Tachycardia may be a primary cardiogenic problem such as arrhythmia, or a secondary problem due to pain, anxiety, medications, drugs, hypoxia, or other causes. A compensatory tachycardia in response to decreased preload and cardiac output is common in hypovolemia. However, since diabetic patients, especially those with long-standing diabetes mellitus, are prone to dysautonomias, there may be an absence of compensatory tachycardia in the setting of acute hypovolemia.[10,11] Documentation of other physical findings such as blood pressure, pulse pressure, jugular venous distention, capillary refill, and urine output is important to help support the diagnosis of hypovolemia. In the absence of the historical details of polydipsia and polyuria, or prior history of diabetes mellitus, the index of suspicion for DKA is reasonably low. In this case, the patient may or may not have been clinically hypovolemic on his first presentation. DKA is known to evolve and progress rapidly.

One could argue that a documented heart rate of 139 beats per minute in a 52-year-old man presenting with weakness should not be discounted without a reasonable period of observation, an electrocardiogram, or further testing.

Case #4: Cerebral Edema as a Cause of Death

In *Bourgeois v. Bailey*,[12] the plaintiff, mother of 6-year-old Aaron Bourgeois, testified that her son complained of headache and began to have a fever on March 10, 1997. Despite treatment with acetaminophen on the following evening, Aaron began to vomit.

When Aaron awoke the next morning, he complained that his knee hurt. That day, his parents took him to their family physician, Dr. Collin Bailey, who prescribed ibuprofen and encouraged increased fluid intake. However, Aaron continued to vomit and began to have diarrhea that night. The following morning, he looked worse, and his mother returned him to Dr. Bailey's office, where he was found to be "limp."

An IV line of 5% dextrose in water (D5W) was initiated in the office, and he was transported by ambulance to River Parishes Hospital, where he arrived at 11:45 a.m. His initial vital signs revealed a temperature of 96.4° F, pulse 136 beats per minute, and

respirations of 32 per minute. Aaron's urine output was only 10 cc during the four hours he was in the ED.

Daphne Caldwell, the nurse who cared for Aaron in the ED, testified that in accordance with the ED policy for the care of children younger than 12 years of age, Aaron's blood pressure was taken only once, at admission. In addition, she testified that although it was not noted in the medical records, she examined the patient and found capillary refill to be normal, skin warm and dry to the touch, oral mucosa pink and moist, and skin turgor normal. However, it was noted that the nurses had difficulty drawing Aaron's blood, and warm towels were necessary.

Dr. Richard Roberts III, the ED attending physician, acknowledged that it was unusual to have a child come to the ED from a physician's office in an ambulance with an IV line. However, he also testified that although the patient's vital signs were normal, the child was "talking and had no signs of severe dehydration and was not in hypovolemic shock." Dr. Roberts ordered a chest x-ray and laboratory tests that indicated a very elevated glucose of 495. Dr. Roberts diagnosed the child as having diabetes and made a decision to transfer him to another hospital.

However, Aaron suffered a cardiac arrest at 3:20 p.m., before he could be transferred, and was pronounced dead at 3:56 p.m. Autopsy results revealed evidence of cerebral edema as well as gastroenteritis.

Dr. Paul Perchonock provided expert witness testimony on behalf of the plaintiff and openly criticized the ED attending physician, Dr. Roberts, for failing to properly hydrate a dehydrated patient. Aaron arrived in the ED with an IV infusing D5W, which was changed to 5½% normal saline, which Dr. Perchonock testified was improper, in his opinion. In addition, he felt that the volume of fluid boluses was inadequate given the extent of the patient's dehydration. Moreover, he opined that Aaron should have received sodium bicarbonate intravenously to treat his acidosis. Dr. Perchonock also testified that he felt that the type of IV fluids administered contributed to the elevated blood glucose.

Dr. Karl Hanson, accepted by the court as an expert witness in family medicine, testified that it would not be possible for the amount of dextrose in the IV fluids to raise Aaron's blood sugar to 495 mg/dL. In addition, the autopsy report revealed that the IV catheter had been placed in the patient's left arm and the phlebotomy puncture wound was on the right. Thus the physical evidence could not substantiate the argument by the plaintiff that laboratory values were tainted by IV fluid infusing distal to a phlebotomy site.

Dr. Charles Preston, accepted by the court as an expert in emergency medicine, testified for the defense. He concluded, based on the evidence, that Dr. Roberts' treatment had met the required standard of care.

The jury found the ED physician, Dr. Roberts, individually liable by a 9-3 verdict. On the other hand, the court found no breach of the standard of care or basis for comparative fault by either Dr. Bailey or the hospital.

Dr. Roberts filed an appeal on the grounds of a challenge for cause based on juror bias, and for failure to conduct a Daubert hearing. The appellant argued that Dr. Perchonock should have been prohibited from testifying because he was not currently practicing medicine and that he was not board-certified in emergency medicine since he had let his time-limited certification lapse. The trial judge concluded that Dr. Perchonock was indeed qualified, since he had been practicing emergency medicine for 20 years and was currently teaching and supervising residents in emergency medicine. The appellate court affirmed and awarded damages limited to $500,000 pursuant to the state malpractice act.

Discussion

This case illustrates the cornerstone of therapy for DKA — hydration and electrolyte management, the urgency for initiating hydration and serum glucose control in a timely fashion, and the risks of rapid hydration. In addition, this case highlights the typical timeline for decompensation in pediatric patients with evolving shock.

Patients with DKA require fluid resuscitation with isotonic fluids to produce intravascular volume expansion to maintain tissue perfusion. Normal saline is the crystalloid solution of choice because it is retained within the intravascular space for a longer duration than fluids containing free water. Although normal saline can produce hyperchloremic metabolic acidosis, this is rarely a clinically significant issue. Lactated Ringer solution contains lactate, which under normal circumstances is metabolized by the liver to bicarbonate and water. Therefore, lactated Ringer solution carries a potentially significant free water load. Lactated Ringer solution also contains a small potassium load.

Solutions containing dextrose, such as D5W or D5½ NS, are isotonic on infusion but contribute significant volumes of free water after the dextrose is metabolized. The dextrose-containing solutions must be avoided during the acute management of DKA because: 1) additional dextrose exacerbates hyperglycemia; 2) free water is not retained intravascularly and is ineffective for intravascular volume expansion in shock; and 3) administration of free water can cause a hypo-osmolar state that is implicated in cerebral edema.[13,14]

Since children have a relatively good end-organ system reserve, there typically is a protracted period during which physiologic compensation occurs. Decompensation in such patients, when it occurs, is usually abrupt and difficult to reverse because of the extent of prior deterioration. All the physicians who testified agreed that Aaron's vital signs on presentation were signs of dehydration and could be signs of impending shock although they disagreed about whether the dehydration was moderate or severe.

In patients with normal renal function who have not been medicated, the urine output is an exceptionally reliable guide to intravascular volume status and cardiovascular function. Oliguria is widely recognized to be an early sign of hypovolemia. Furthermore, a minimum urine output of 0.5-1.0 cc/kg/hr is considered adequate and persistent oliguria puts the patient at risk for acute renal failure due to acute tubular necrosis. Confirmatory laboratory evidence for hypovolemia include an elevated BUN / creatinine ratio, hemoconcentration, and increased urine osmolarity and specific gravity, low urine sodium, and a fractional excretion of sodium (FE_{Na}) < 1.0. Since these tests are widely available and the results can be obtained rapidly, they may be considered a standard of care in the determination of intravascular volume status. In addition, serial vital signs are the cornerstone of acute care. Patients with abnormal vitals or those who are at high risk for decompensation must have more frequent assessment of their vital signs.

Severe hyperglycemia increases the serum osmolarity in a manner not unlike severe hypernatremia. The elevated serum osmolarity may then shift intracellular and interstitial fluid out of the brain, causing a state of relative cerebral dehydration. Rapid correction of serum osmolarity with hypotonic fluids or aggressive glucose reduction has been implicated in the development of cerebral edema, which causes life-threatening, irreversible brain injury.[2,15] It appears that although the syndrome is not restricted to the pediatric population, infants and children are at significantly higher risk. Although the likelihood of developing cerebral edema during an episode of DKA is 0.7-1%,[15] the probability of brain herniation is approximately 5.8%,[15] and the cumulative morbidity and mortality from cerebral edema is 24-31%.[2,16]

The use of sodium bicarbonate to reverse the metabolic acidosis in DKA is controversial. First, sodium bicarbonate can increase serum osmolarity acutely. Second, the metabolic acidosis usually reverses rapidly when hypovolemia and insulin deficiency are treated. The administration of sodium bicarbonate may be restricted to those cases where the acidosis or hyperkalemia are imminently life-threatening. [2,17,18]

The "lost chance of survival" doctrine is important in cases in which plaintiffs can prove that there was some level of lost opportunity for recovery. Typically, this leads to recovery of damages in litigation. Therefore, critically ill patients who present for treatment before they are *in extremis* must receive aggressive interventional measures to provide them with every reasonable chance of survival.

Case #5: Gestational Diabetes

In *Rajnowski v. St. Patrick's Hospital*,[19] Nancy Rajnowski consulted Dr. Floyd Guidry, an obstetrician and gynecologist, on Aug. 30, 1982. Mrs. Rajnowski was approximately 16 weeks pregnant at the time and apparently was previously healthy except for slightly elevated blood pressure. However, the patient was 5'2" tall and weighed 200 lbs.

Mrs. Rajnowski's pregnancy progressed normally until Dec. 31, 1982, when at a routine office visit her urine test revealed 2+ glucose. Dr. Guidry followed up the urine test with glucose tolerance and postprandial glucose testing, which revealed Mrs. Rajnowski's fasting blood glucose to be 209 mg/dL and that she had 2+ ketones in her urine.

At the next regularly scheduled office visit, Dr. Guidry informed Mrs. Rajnowski that she had gestational diabetes, and he placed her on an 1800-calorie-per-day diabetic diet restriction. He also informed her that her diabetes could cause the death of her fetus. He discussed with her his opinion that it would be necessary to induce labor early because of the relatively greater risk to the fetus if the pregnancy was allowed to progress to full term. Dr. Guidry also requested a second series of lab tests. The lab, however, invalidated these additional tests because Mrs.

Rajnowski had not fasted, as required, prior to taking the tests. These tests were not repeated.

Office urine tests at subsequent visits continued to reveal 1+ urine glucose. Dr. Guidry interpreted this to suggest that the dietary restriction was effective. Dr. Guidry did not check the urine for ketones because the urine glucose was less than 2+.

On Jan. 31, Mrs. Rajnowski, now in her 38th week of gestation, complained of decreased fetal movement, which Dr. Guidry generally attributed to her diabetes. He admitted Mrs. Rajnowski to St. Patrick's Hospital the subsequent morning, Feb. 1, for the routine induction of labor. At approximately 12:30 p.m. that same day, the fetal monitor indicated fetal distress and Dr. Guidry decided to perform a cesarean.

At section, Dr. Guidry found the umbilical cord had a true knot and was wrapped around the baby's neck four times. The infant was in a depressed state upon delivery, with no heart rate and an Apgar score of 2, but the score improved to 7 at five minutes. The male infant weighed 6 lbs., 5 oz., and although there were no immediate signs of complications such as seizures or congestive heart failure, the infant remained in intensive care until Feb. 16.

The infant developed normally until October 1983, when at 8½ months of age he sustained a seizure. Dr. Kim, the infant's pediatrician, and Dr. Bell, a pediatric neurologist, both examined the child and found no specific cause for the seizure.

In early 1985, Mr. and Mrs. Rajnowski viewed a television program that discussed the possible connection between diptheria-pertussis-tetanus (DPT) vaccine and infant seizures, and they subsequently consulted an attorney for advice. A pediatric neurologist, Dr. Robert Summers, examined the child, and in August, he informed Mr. and Mrs. Rajnowski that the child had brain damage localized to the right temporal lobe of the brain, which, in his opinion, could have occurred at any time, including pregnancy or delivery.

The attorney, Mr. Pulsaki, then informed the Rajnowskis that they did not have a cause of action based on the DPT vaccinations. Thereafter, the Rajnowskis sought the advice of a second attorney, Mr. Pignatelli, in the summer of 1986, and under his advice, they subsequently filed suit in September 1986, contenting that Dr. Guidry had failed to disclose material information to them regarding the risks of the pregnancy.

The court found that Dr. Guidry discussed all his material findings with the plaintiffs and did not hold any information back, and the case was dismissed.

Discussion

The importance of *Rajnowski v. St Patrick's Hospital* is in the importance of the treating physician's duty to disclose all material facts and their associated implications to the patient. In this case, the physician diagnosed gestational diabetes and not ketoacidosis. Therefore, the level of disclosure becomes important, as noted by the two dissenting judges who raised the question of deliberate concealment of material elements and misrepresentation and fraud sufficient to meet the application of the doctrine of *contra non valentum*. The doctrine of *contra non valentum* states that limitations or prescriptive periods do not begin to run against the plaintiff who is unable to act, usually in situations where the cause for action is concealed from the plaintiff by the defendant.

Gestational diabetes is a relatively uncommon, but nonetheless important, cause of DKA.[20] DKA in diabetes usually is related to: 1) undiagnosed pre-existing type I diabetes; 2) complicated gestational diabetes; or 3) the use of steroids or beta-agonists.[20-22] However, there may be no obvious precipitating cause.[22] In this case, the patient reasonably could be suspected to have been at risk given her body mass index being consistent with obesity. Although the obstetric management may have been within the limits of the standard of care, the documentation of the communications and disclosures within the patient-physician relationship were clearly determinative. *(Editor's note: The patient may have had complications related to delivery or other causes. It is unclear from the facts presented in this particular case that the patient actually was in DKA.*)

Hypovolemia due to hyperglycemia and its resultant osmotic diuresis typically causes vasoconstriction in most vascular beds. Therefore, patients with hyperglycemia are at risk for splanchnic ischemia, renal hypoperfusion, mental status changes, and, in the case of pregnancy, uteroplacental insufficiency. In addition, diabetic mothers often give birth to high birth-weight infants. Finally, infants born to diabetic mothers must be observed closely for the development of precipitous postnatal hypoglycemia.

Case #6: Airway Control and Gastroparesis

In *Gray v. Brock*,[23] plaintiff's estate brought suit against ED physicians for failure to prevent aspiration

of gastric contents.

Mr. Gray was a longtime patient of Dr. Brock, who managed his diabetes by diet, weight loss management, and interval serum glucose testing. On Jan. 11, 1982, Mr. Gray began vomiting. On Jan. 18, he became confused and disoriented, and his wife took him to the ED of the Independence Sanitarium and Hospital, where he arrived at 10:23 a.m. The physician in charge of the ED, Dr. Robert Carrillo, administered IV fluids, oxygen, and insulin, which reduced his serum glucose from 1265 mg/dL to approximately 900 mg/dL.

Mr. Gray then was transferred to the ICU under the care of Dr. Lewis; his condition had been upgraded from poor to fair. Dr. Lewis entered a note into the chart diagnosing Mr. Gray with nonketotic hyperosmolar diabetic coma based on the laboratory results and the prior three-day history of polydipsia and polyuria. However, Mr. Gray subsequently developed an aspiration pneumonia, pulmonary edema, and septicemia, and died. Autopsy evidence confirmed that aspiration pneumonia had occurred.

At trial, the jury returned a verdict that assigned fault as follows: 13% to the ED physician, 5% to the general physician, and 82% to the decedent. On appeal, appellants argued that there was no substantial evidence that the patient knew that his diabetes was out of control, and the court agreed, reversing the judgment in favor of the decedent.

Discussion

The key issue in this case is the need for airway protection and aspiration prevention in patients with diabetes. Gastroparesis occurs as a complication of long-standing diabetes, presumably related to generalized neurological dysautonomia. The gastroparesis predisposes diabetics to delayed gastric emptying and, therefore, aspiration of gastric contents in the setting of a loss of protective airway reflexes. For this reason, in diabetics with central nervous system injury, metabolic encephalopathy, or those patients administered sedation, the induction of general anesthesia should be treated as a potential aspiration risk. Airway protection should be a strong consideration. Although a nasogastric tube decreases the liquid contents of the stomach, larger particles may not be removed by nasogastric suction. Consideration of the use of prokinetic agents such as metoclopramide as therapeutic or prophylactic adjuncts is appropriate.

The aspiration of gastric contents may result in Mendelsohn's syndrome, aspiration pneumonitis, which is dependent on the pH and the volume of aspirated material. Aspiration of acidic stomach contents results in acute respiratory distress syndrome, characterized by hypoxemia and pulmonary vascular congestion. A bacterial superinfection frequently results either from primary aspiration of pathogenic bacteria, or from opportunistic invasion of damaged airways. Aspiration pneumonitis may take up to 24-48 hours to manifest itself on chest radiographs, depending on the severity of the aspiration.

The indications for endotracheal intubation in the ED include respiratory failure, the need for hyperventilation, airway burns, and airway protection. Although an endotracheal tube with an inflated cuff does not definitively protect the airway from aspiration of material in the oral pharynx, it can significantly limit the volume of aspirate.

Although DKA and nonketotic hyperosmolar state (KHS) share similar pathophysiology, they differ in presentation **(see Table, p. 131)**. However, the treatment of both conditions relies on correction of dehydration, shock, hyperglycemia and hyperosmolarity, acidosis, and/or electrolyte abnormalities.[24]

Conclusion

DKA is a potentially reversible life-threatening emergency that requires a high index of suspicion and confirmatory laboratory data for its diagnosis. Patients with DKA must be monitored closely for potential decompensation and treated aggressively in an appropriate setting. The appropriate setting for the treatment of DKA depends on the severity of the presentation, the intensity of therapy required, and the resources of the health care system. Patients with severe hypovolemia, hyperglycemia, electrolyte abnormalities, or associated comorbidities best are treated in the ICU after initial stabilization in the ED.[25] Hydration, glucose control, and electrolyte management constitute the mainstay of therapy. Frequent assessment of vital signs, physical examination, and laboratory evaluation is necessary. Infants, children, and pregnant women are especially challenging, and consultation with intensivists or endocrinologists should be considered strongly.

As in all areas of legal medicine, the need for careful and complete documentation of objective data and subjective basis for decision making in assessment and management cannot be overemphasized.

TABLE: Risk Management Tips

1. Diabetic ketoacidosis (DKA) is a life-threatening medical emergency with a mortality of approximately 5% when recognized and treated.
2. The diagnostic hallmarks of DKA are hyperglycemia, metabolic acidosis, and ketosis.
3. The cornerstones of therapeutic intervention are volume replacement with isotonic fluid (normal saline), insulin replacement, and correction of electrolyte abnormalities.
4. Appropriate therapy is guided by information: physical examination, frequent serial laboratory testing, finger-stick blood glucose, assessment of urine output, and invasive monitoring if indicated.
5. Legible and complete documentation is the basis of medical evidence.
6. Adjunctive therapeutic considerations must include identification and management of infection, airway management, and consideration of the sequelae of hypovolemic shock such as myocardial infarction, cardiovascular accident, visceral or limb ischemia, and acute renal failure.
7. Pregnancy testing should be considered in all critically ill females of childbearing age. DKA as the first manifestation of gestational diabetes must be considered. Potential impact on the fetus also must be considered.
8. Hospital policies and guidelines should be followed closely; where a deviation from standard practice occurs, the medical reasoning must be annotated.

References

1. Chiasson JL, Aris-Jilwan N, Belanger R, et al. Diagnosis and treatment of diabetic ketoacidosis and the hyperglycemic hyperosmolar state. *CMAJ* 2003;168:859-866.
2. White NH., Diabetic ketoacidosis in children. *Endocrinol Metabol Clin NA* 2000;29:657-681.
3. *Lyons v. Walker Reg'l Med Ctr*., 791 So.2d 937 (2000).
4. *Lyons v. Walker Reg'l Med Ctr*., Ala. LEXIS 104 (2003).
5. Gomez Diaz RA, Rivera Moscoso R, Ramos Rodriguez R, et al. Diabetic ketoacidosis in adults: Clinical and laboratory features. *Arch Med Res* 1996;27:177-181.
6. Miller DW, Slovis CM. Hypophosphatemia in the emergency department therapeutics. *Am J Emerg Med* 2000;18:457-461.
7. *Metcalf v. United States,* U.S. Dist. LEXIS 2236 (1990).
8. Grimaud D, Levraut J. Acute postoperative metabolic complications of diabetes. *Miner Anesthesiol* 2001;67:263-270.
9. *Howard v. St. Edward Mercy Med. Ctr*., Ark App. LEXIS 81 (2002).
10. Ewing DJ, Campbell IW, Clarke BF. Assessment of cardiovascular effects in diabetic autonomic neuropathy and prognostic implications. *Ann Intern Med* 1980;92:308-311.
11. Lloyd-Mostyn RH, Watkins PJ. Defective innervation of the heart in diabetic autonomic neuroparthy. *BMJ* 1975;3:15-17.
12. *Bourgeois v. Bailey*, 817 So.2d 240 (2002).

United States Postal Service

Statement of Ownership, Management, and Circulation

1. Publication Title	2. Publication No.	3. Filing Date
ED Legal Letter	1 0 8 7 - 7 3 4 1	10/01/03
4. Issue Frequency	5. Number of Issues Published Annually	6. Annual Subscription Price
Monthly	12	$489.00

7. Complete Mailing Address of Known Office of Publication *(Not Printer) (Street, city, county, state, and ZIP+4)*
3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, Fulton County, GA 30305

Contact Person: Robin Salet
Telephone: 404/262-5489

8. Complete Mailing Address of Headquarters or General Business Office of Publisher *(Not Printer)*
3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, GA 30305

9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor *(Do Not Leave Blank)*

Publisher *(Name and Complete Mailing Address)*
Brenda Mooney, 3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, GA 30305

Editor *(Name and Complete Mailing Address)*
Allison Mechem, same as above

Managing Editor *(Name and Complete Mailing Address)*
Valerie Loner, same as above

10. Owner *(Do not leave blank. If the publication is owned by a corporation, give the name and address of the corporation immediately followed by the names and addresses of all stockholders owning or holding 1 percent or more of the total amount of stock. If not owned by a corporation, give the names and addresses of the individual owners. If owned by a partnership or other unincorporated firm, give its name and address as well as those of each individual. If the publication is published by a nonprofit organization, give its name and address.)*

Full Name	Complete Mailing Address
Thomson American Health Consultants	3525 Piedmont Road, Bldg. 6, Ste 400 Atlanta, GA 30305

11. Known Bondholders, Mortgagees, and Other Security Holders Owning or Holding 1 Percent or More of Total Amount of Bonds, Mortgages, or Other Securities. If none, check box → ☐ None

Full Name	Complete Mailing Address
Thomson Healthcare, Inc.	Five Paragon Drive Montvale, NJ 07645

12. Tax Status *(For completion by nonprofit organizations authorized to mail at nonprofit rates.) (Check one)*
The purpose, function, and nonprofit status of this organization and the exempt status for federal income tax purposes:
☐ Has Not Changed During Preceding 12 Months
☐ Has Changed During Preceding 12 Months *(Publisher must submit explanation of change with this statement)*

PS Form **3526**, September 1998 *See instructions on Reverse)*

13. Publication Name: ED Legal Letter
14. Issue Date for Circulation Data Below: October 2003

15. Extent and Nature of Circulation			Average No. of Copies Each Issue During Preceding 12 Months	Actual No. Copies of Single Issue Published Nearest to Filing Date
a. Total No. Copies *(Net Press Run)*			671	600
b. Paid and/or Requested Circulation	(1)	Paid/Requested Outside-County Mail Subscriptions Stated on Form 3541. *(Include advertiser's proof and exchange copies)*	369	379
	(2)	Paid In-County Subscriptions *(Include advertiser's proof and exchange copies)*	4	3
	(3)	Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Non-USPS Paid Distribution	3	4
	(4)	Other Classes Mailed Through the USPS	19	7
c. Total Paid and/or Requested Circulation *(Sum of 15b(1) and 15b(2)*			395	393
d. Free Distribution by Mail *(Samples, Complimentary and Other Free)*	(1)	Outside-County as Stated on Form 3541	22	21
	(2)	In-County as Stated on Form 3541	1	1
	(3)	Other Classes Mailed Through the USPS	0	0
e. Free Distribution Outside the Mail *(Carriers or Other Means)*			46	25
f. Total Free Distribution *(Sum of 15d and 15e)*			69	47
g. Total Distribution *(Sum of 15c and 15f)*			464	440
h. Copies Not Distributed			207	160
i. Total *(Sum of 15g, and h.*			671	600
Percent Paid and/or Requested Circulation *(15c divided by 15g times 100)*			85	89

16. Publication of Statement of Ownership
Publication required. Will be printed in the November 2003 issue of this publication. ☐ Publication not required.

17. Signature and Title of Editor, Publisher, Business Manager, or Owner: Publisher — Date: 9/30/03

I certify that all information furnished on this form is true and complete. I understand that anyone who furnishes false or misleading information on this form or who omits material or information requested on the form may be subject to criminal sanctions (including fines and imprisonment) and/or civil sanctions (including multiple damages and civil penalties).

Instructions to Publishers

1. Complete and file one copy of this form with your postmaster annually on or before October 1. Keep a copy of the completed form for your records.
2. In cases where the stockholder or security holder is a trustee, include in items 10 and 11 the name of the person or corporation for whom the trustee is acting. Also include the names and addresses of individuals who are stockholders who own or hold 1 percent or more of the total amount of bonds, mortgages, or other securities of the publishing corporation. In item 11, if none, check the box. Use blank sheets if more space is required.
3. Be sure to furnish all circulation information called for in item 15. Free circulation must be shown in items 15d, e, and f.
4. Item 15h., Copies not Distributed, must include (1) newsstand copies originally stated on Form 3541, and returned to the publisher, (2) estimated returns from news agents, and (3), copies for office use, leftovers, spoiled, and all other copies not distributed.
5. If the publication had Periodicals authorization as a general or requester publication, this Statement of Ownership, Management, and Circulation must be published; it must be printed in any issue in October or if the publication is not published during October, the first issue printed after October.
5. In item 16, indicate date of the issue in which this Statement of Ownership will be published.
6. Item 17 must be signed.

Failure to file or publish a statement of ownership may lead to suspension of second-class authorization.

PS Form **3526**, September 1999 *(Reverse)*

CE/CME Objectives

[For information on subscribing to the CE/CME program, contact customer service at (800) 688-2421 or e-mail customerservice@ahcpub.com.]

The participants will be able to:

- identify high-risk patients and use tips from the program to minimize the risk of patient injury and medical malpractice exposure;
- identify a "standard of care" for treating particular conditions covered in the newsletter;
- identify cases in which informed consent is required;
- identify cases which include reporting requirements;
- discuss ways in which to minimize risk in the ED setting.

13. Zornow MH, Scheller MS, Shackford SR. Effect of hypertonic lactated Ringer's solution on intracranial pressure and cerebral water in a model of traumatic brain injury. *J Trauma* 1989;29:484-488.
14. Zornow MH, Prough DS. Fluid management in patients with traumatic brain injury. *New Horiz* 1995;3:488-498.
15. Bohn D, Daneman D. Diabetic ketoacidosis and cerebral edema. *Curr Opin Pediatr* 2002;14:287-291.
16. Edge JA, Hawkins HM, Winter DL, Dunger DB. The risk and outcome of cerebral oedema developing during diabetic ketoacidosis. *Arch Dis Child* 2001;85:16-22.
17. Graf H, Arieff AI. The use of sodium bicarbonate in the therapy of organic acidosis. *Intensive Care Med* 1986;12:286-288.
18. Umperierrez GE, Kitabacki AE. Management strategies for diabetic ketoacidosis. *J Crit Illness* 1996;11:437-443.
19. *Rajnowski v. St. Patricks Hosp.,* 564 So.2d 671 (1990).
20. Ramin KD. Diabetic Ketoacidosis in Pregnancy. *Obstet Gyn Clin North Am* 999;26:481-488.
21. Chauhan SP, Perry KG Jr., McLaughlin BN, et al. Diabetic ketoacidosis complicating pregnancy. *J Perinatol* 1996;16: 173-175.
22. Pitteloud N, Binz K, Caulfield A, Phillipe J. Ketoacidosis during gestational diabetes. *Diabetes Care* 1998;21:1031-1032.
23. *Gray v. Brock*, 750 S.W.2d 696 (1988).
24. Delaney MF, Zisman A, Kettyle WM. Diabetic ketoacidosis and hyperglycemic hyperosmolar nonketotic syndrome. *Endocrin Metab Clin North Am* 2000;29:683-705.
25. Freire AX, Umpeierrez GE, Afessa B, et al. Predictors of intensive care unit and hospital stay in diabetic ketoacidosis. *J Crit Care* 2002;17:207-211.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. There is no need to complete and return a Scantron form. To clarify confusion surrounding any questions answered incorrectly, please consult the source material.

At the conclusion of this semester, you must complete the evaluation form that will be provided at that time, and return it in the reply envelope that will be provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

CE/CME Questions

17. All of the following are hallmarks of acute severe established DKA *except:*

A. hypovolemia.
B. acidosis.
C. hyperkalemia.
D. polyuria.

18. The mainstay of therapy for acute severe DKA include all of the following *except:*

A. volume resuscitation with isotonic fluid such as normal saline.
B. volume resuscitation with hypotonic dextrose containing fluids such as D5W.
C. glucose control with insulin therapy.
D. frequent assessment of serum pH, glucose, and electrolyte status.

19. Which of the following is *false?*

A. Urine output is generally a reliable indicator of intravascular volume status.
B. An elevated BUN/creatinine ratio usually implies hypovolemia.
C. The fractional excretion of sodium can be used to diagnose hypovolemia.
D. The urine specific gravity is generally decreased in the setting of intravascular volume depletion.

20. Cerebral edema in the setting of DKA is associated with:

A. rapid correction of a hyperosmolar state
B. administration of too much insulin
C. volume overload
D. acidosis

Answer key: 17.D; 18.B; 19.D; 20.A.

In Future Issues:

EMTALA Update