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## The Impact of Diabetes Mellitus on Burns and Standard Burn Treatments

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### Abstract

Burns are complex injuries that trigger the stress response and result in increased blood glucose. Diabetes mellitus, an endocrine disorder with abnormal glucose regulation, can significantly alter the risk for burns and the treatment of burns. Pre-existing diabetes mellitus complicates burn treatments and healing ability. Diabetes mellitus may also develop directly from an unregulated stress response to the burn. The consideration of diabetes mellitus affects wound care, medications, and nutrition for burn patients. As patient advocates, nurses are responsible for caring for burn patients with awareness of endocrine complications. This paper provides a detailed overview of the pathophysiology, complications, and treatments of burns in relation to the adult population with diabetes mellitus to provide evidence-based care to critically ill patients.

### Burns and Diabetes Mellitus

The treatment of burn injuries in patients with the comorbidity of diabetes mellitus creates a difficult case for healthcare professionals, specifically nurses. The two conditions may cause one another or occur simultaneously and require attentive care. Individuals who are otherwise healthy and suffer from a traumatic burn may develop stress-induced hyperglycemia and subsequent diabetes mellitus due to endocrine complications from their injury (Mecott, Al-Mousawi, Gauglitz, Herndon, & Jeschke, 2010). Meanwhile, individuals with pre-existing diabetes mellitus are at a higher risk for burn injuries and complications during the wound healing process (Lazear, 2014). Despite numerous variations in the development of burns and diabetes mellitus together, the combination requires exceptional nursing care to control and prevent complications. In addition, current burn treatments should be adapted to prevent diabetes mellitus, control blood glucose, and promote healing. The purpose of this paper is to discuss the pathophysiology of burns and diabetes mellitus, complications of the combination, and appropriate treatments in relation to evidence-based practice of nurses.

#### **Burns**

Burns are diverse injuries that significantly vary in location, size, depth, and severity. As a result, each patient requires a unique treatment plan that incorporates the characteristics of his or her wound with his or her past history and comorbidities. The pathophysiology of burns and the body's stress response identifies areas that require more attention with a past history or acute development of diabetes mellitus, including blood glucose control and infection prevention.

**Pathophysiology**

**Local.** Histologically, coagulation necrosis occurs at the site of injury after a burn (Knighton, 2014). The body produces an inflammatory response to the burn, through both vascular and cellular actions. Arterioles temporarily vasoconstrict before vasodilating in response to histamine released by damaged cells (Lewis, 2014). Cell permeability increases and allows fluid to escape the vessels with albumin and fibrinogen. The fibrinogen converts to fibrin and works to create a clot with platelets. The platelets also promote healing by secreting growth factors. The cellular response consists of chemotaxis, which transports neutrophils and monocytes to the area of injury (Lewis, 2014). Neutrophils perform phagocytosis within six to twelve hours and may accumulate with bacteria and debris to create pus. Monocytes arrive in three to seven days and turn into macrophages to further perform phagocytosis and provide non-specific immunity. They remain for weeks while lymphocytes arrive to develop specific immunity (Lewis, 2014). Wound repair begins six to twelve hours after its occurrence. Fibroblasts and collagen fibrils repair and develop new tissue (Knighton, 2014).

In addition, chemical mediators further facilitate the inflammatory response. These mediators are created by conversion of arachidonic acid from the membranes of damaged cells. Prostaglandin causes vasodilation, increased blood flow, and edema. Thromboxane causes vasoconstriction to promote platelet aggregation. Leukotrienes increase capillary permeability leading to edema, which can become a critical concern around the airway or in compartment syndrome (Lewis, 2014).

**Systemic.** Severe burns may result in systemic inflammation that cause widespread fluid and electrolyte shifts. Increased capillary permeability results in a

movement of sodium and protein particles to the extracellular area. The loss of these particles, especially albumin, reduce the colloidal osmotic pressure and result in a movement of fluid to the interstitial spaces, called second spacing. Third spacing then occurs when fluid moves further into areas that typically do not contain fluid and creates blisters, exudate, and edema (Knighton, 2014). Extreme movements of fluid to interstitial areas and fluid loss through the wound cause low intravascular volume. Low vascular volume causes hypotension, tachycardia, and a reduction in oxygen distribution and tissue perfusion. The loss of vascular fluid increases the hematocrit and viscosity of the blood which increases the risk of clot formation. Hemolysis, the destruction of red blood cells (RBCs), may also occur due to the release of oxygen free radicals and trauma. The combination of increased capillary permeability, edema, decreased blood volume, increased hematocrit, and increased blood viscosity causes peripheral resistance and results in burn shock (Knighton, 2014). If the fluid shift is not corrected, the lack of perfusion may lead to hypovolemic shock and death.

The sympathetic nervous system (SNS) responds to a burn injury with a systemic flight-or-flight response. This stress response incorporates the respiratory, circulatory, endocrine, and nervous systems to protect the body and compensate for the perceived stressor. The respiration rate increases, heart rate increases, cardiac output increases, vessels constrict, and blood glucose rises (Lewis, 2014). The hypothalamus releases corticotropin-releasing hormone and stimulates the pituitary gland to release adrenocorticotrophic hormone (ACTH). ACTH causes the adrenal cortex to release cortisol and some aldosterone, which increase blood glucose and blood pressure respectively. The SNS also causes the release of epinephrine and norepinephrine from the

adrenal medulla (Lewis, 2014). The release of these hormones and physiological reactions are key to understanding the severity of a traumatic burn and the incorporation of diabetes mellitus.

Measurement of certain enzymes, byproducts, and inflammatory mediators can indicate the severity of damage throughout the body. Alanine aminotransferase (ALT), Creatine (CRE), and Creatine kinase (CK) are measurements of liver, kidney, and heart function (Wang, Chen, Zhu, Zhang, & Wang, 2016). High-mobility group box 1 (HMGB-1) is a protein released either actively by immune cells or passively by injured and dying cells. TNF- $\alpha$  and interleukin 6 are proinflammatory mediators and cause organ dysfunction in sepsis (Lewis, 2014). An increase in HMGB-1, TNF- $\alpha$ , and interleukin 6 indicate inflammation. Malondialdehyde (MDA) is a byproduct of the breakdown of polyunsaturated fatty acids in tissue membranes and is a measurement of damage by oxygen free radicals. The total antioxidant capacity (TAC) is a measure of the body's ability to resist oxidative stress. An increase in MDA and decrease in TAC indicates oxidative stress (Wang et al., 2016). Oxidative stress is "a pathological state in which reactive oxygen/nitrogen species (ROS/RNS) overwhelm antioxidative defenses of the organism, leading to oxidative modification of biological macromolecules (i.e., lipid, protein, DNA), tissue injury, and accelerated cellular death as the foundation of many diseases" (Apak, Ozyurek, Guclu, & Capanoglu, 2016, p. 997). The actions of neutrophils and other defense mechanisms increase ROS, decreases natural enzymatic antioxidant effects in the body, and results in oxidative stress (Silveira et al., 2014). These markers of inflammation are necessary for understanding the treatment options available for burns when considering diabetes mellitus.

**Classification**

Burns are classified according to type, depth, extent, and location. Treatment is based on these characteristics. Types of burns include thermal burns, chemical burns, smoke and inhalation injuries, and electrical burns (Knighton, 2014). Thermal injuries occur from contact with a hot substance or surface and vary based on temperature of the source of heat and the length of time in contact. Chemical burns occur after contact with acidic, alkali, or organic compounds. Smoke and inhalation burns result from inhaling hot air or chemicals and can damage the upper and lower airways (Knighton, 2014). Electrical burns are caused by electrical currents that easily pass through nerves and blood vessels and vary based on the current strength, the location of injury, and the length of exposure to electricity. Electrical burns can be insignificant on the surface but have widespread effects underneath the skin, thus creating an “iceberg effect” (Knighton, 2014, p. 452).

In the past, medical professionals classified burn depth as first through fourth degree. The depth of a burn is now classified based on the level of tissue thickness impacted. Superficial partial thickness burns include only the epidermis, the nonvascular top layer of the skin (Knighton, 2014). The epidermis consists of keratinocytes, melanocytes, merkel cells, and Langerhans cells which produce cytokines and assist in immunity (Grossman, 2014a). Deep partial thickness burns include damage to the dermis which consists of connective tissue, vessels, hair follicles, nerves, and glands. Full thickness burns include damage to fat, muscle, and bone tissue below the dermis (Knighton, 2014). Depending on the injury, some burns may encompass areas of multiple depths.



The extent of a burn is expressed as a measurement of the total body surface area (TBSA) affected by deep partial and full thickness burns. Multiple methods are available to assess the extent. The Rule of Nines chart divides the body into sections that are divisible by nine. The head, each arm, and the front and back of each leg are considered 9% of the TBSA. The front and back of the trunk are considered 18% each and the genital area is 1% of the TBSA. This method is adequate for an initial and quick assessment (Knighton, 2014). The Lund-Browder chart is a more precise calculation of the percentage of TBSA affected because it factors in the patient's age and body proportions at their developmental stage (Knighton, 2014). Documenting the Lund-Browder chart on a sticker that attaches to patient records reduces the incidence of losing the initial assessment (Adedokun, McInerney, Buckley, & Clover, 2016). In addition to these two conventional methods, a new software application, called "Peregrine," is available for Apple devices. Peregrine performs burn size assessment and calculates fluid resuscitation in less time and with the same accuracy as with pen and paper (Godwin, Tan, Bockhold, Ma, & Tran, 2015). The FDA has not approved the software yet, but a tool that may remove human error should be seriously considered in this area.

### **Acute Symptoms**

In addition to the stress response, manifestations of burns vary based on the type, cause, and severity. Superficial and partial thickness burns are often excruciatingly painful due to the nerve endings located in the dermis. Full thickness burns may not be as painful due to complete destruction of nerves (Knighton, 2014). Fluid shifts often cause blisters in partial thickness burns. Larger burn areas may result in absent bowel sounds and a paralytic ileus. The patient's mental status may vary due to fluid loss, a head injury,

smoke inhalation, drug and alcohol use, or medications (Knighton, 2014). Patients can shiver due to heat loss, pain, or stress. Burns can be traumatic injuries that impact social interaction and may require additional psychological and emotional support (Din, Shah, Asadulah, Jamal, & Bilal, 2015).

## **Diabetes Mellitus**

### **Pathophysiology**

Diabetes mellitus is an endocrine disorder caused by an absolute deficiency of insulin, inadequate insulin, or insulin resistance that results in disturbed glucose metabolism. Insulin is a hormone produced by beta cells in the islets of Langerhans in the pancreas. Insulin causes glucose uptake and storage as glycogen in muscle or adipose tissue, prevents fat and glycogen breakdown, and increases protein synthesis by promoting transport of amino acids into cells (Gerard, 2014). Ineffective insulin creation or secretion results in hyperglycemia. Ultimately, diabetic patients cannot take up blood glucose into cells without the assistance of insulin. Long-term hyperglycemia damages the body, which may result in a burn injury or complicate the healing process of a burn.

Diabetes mellitus occurs in two broad types, determined by insulin characteristics (Gerard, 2014). Type I diabetes mellitus is an absolute deficiency of insulin caused by autoimmune destruction of beta cells or idiopathic causes. Type I diabetes mellitus requires insulin administration to control blood sugar. Type II diabetes mellitus includes insulin resistance, abnormal insulin secretion, and hyperglycemia (Gerard, 2014). Type II diabetes mellitus can be caused by a combination of genetic and environmental factors, such as obesity and a sedentary lifestyle. As blood glucose increases, the beta cells attempt to increase insulin production. The increased demand on the pancreas and beta

cells eventually causes beta cell failure. Insulin resistance by muscle, liver, and fat tissues results in a lack of glucose transport to cells. Postprandial and fasting hyperglycemia can occur despite hyperinsulinemia. Insulin resistance may be caused by systemic inflammation due to a widespread burn. In addition, the liver continues to produce glucose due to insulin resistance and compromises healing (Gerard, 2014). Treatment for Type II diabetes mellitus includes medications, such as insulin, and lifestyle changes.

### **Diagnosis**

Diabetes mellitus is diagnosed with blood tests and symptom manifestation. The Fasting Blood Glucose (FBG) test measures the amount of glucose in the blood after an 8 hour fast. Normal blood glucose levels are between 70-100 mg/dL. A level between 100-125 mg/dL identifies impaired fasting glucose. A level over 125 mg/dL is diagnosed as diabetes mellitus. A two hour oral glucose test measures blood glucose after the ingestion of 75 grams of glucose. A normal result is a blood glucose level below 140 mg/dL after two hours. A level between 140-199 mg/dL represents impaired glucose tolerance. A level over 200 mg/dL two hours after the glucose ingestion or at any point in time is characteristic of diabetes mellitus (Gerard, 2014). Hemoglobin A1c (HbA1c) measures the amount of hemoglobin that is glycosylated, or covered in sugar, and represents the patient's control of diabetes over the past three months (Mayo Clinic Staff, 2018). Diabetes mellitus is diagnosed when the HbA1c is over 6.5% (Gerard, 2014). Stress-induced hyperglycemia is considered a blood glucose level over 200 to 240 mg/dL (Mecott et al., 2010).

**Symptoms**

The most common signs and symptoms of diabetes are polyuria, polydipsia, and polyphagia (Gerard, 2014). The osmotic effects of glucose cause polyuria and polydipsia. High blood glucose causes the kidneys to excrete excess glucose along with large amounts of water. The excess loss of water causes increased thirst. Polyphagia is caused by a lack of cellular access to glucose without insulin. Diabetes mellitus can also cause hypoglycemia or hyperglycemia conditions due to the dysregulation of glucose (Gerard, 2014).

**Consequences of Hyperglycemia**

Chronic hyperglycemia associated with diabetes mellitus causes microvascular, macrovascular, and immune dysfunction. Microvascular effects include retinopathy, neuropathy, and nephropathy (Holt, 2014). High levels of blood glucose cause vision impairment due to hyperosmolar effects on the lens and retina (Gerard, 2014). Vision impairment increases the risks of burns due to the inability to safely navigate around dangerous heat or make safe decisions. Neuropathy, a loss of feeling and sensation, increases the risk of damaging extremities by leaving the patient open to potentially burning themselves without noticing. Macrovascular effects include hypertension and hyperlipidemia which can result in heart disease, peripheral vascular disease, or stroke (Gerard, 2014). An overall decrease in adequate perfusion reduces healing capability. In general, diabetic patients have an increased risk of infections, such as cellulitis or a urinary tract infection, due to their increased blood sugar and decreased perfusion (Lazear, 2014). Diabetes mellitus also reduces phagocytosis, chemotaxis, and the

bactericidal capacity of the inflammatory response (Holt, 2014). As a result, burn patients who are struggling with hyperglycemia are at a higher risk for infection.

### **Additional Hormonal Influences**

Several additional hormones impact blood glucose levels, especially during a systemic response to a burn (Gerard, 2014). Epinephrine, a catecholamine released by the adrenal medulla, prevents insulin release while promoting glycogenolysis in the liver during stress. Growth hormone is released during a stressor, such as trauma, and also counteracts the action of insulin. Glucocorticoids, mainly cortisol, are also released during times of stress and cause gluconeogenesis in the liver (Gerard, 2014). Long-term hypersecretion of these hormones in response to a burn may lead to diabetes mellitus. As a result, if a patient did not previously have diabetes mellitus, stress, blood sugar, and insulin must be closely monitored after a burn to prevent the development of diabetes mellitus (Gerard, 2014).

### **Complications of Burns Associated with Diabetes Mellitus**

Combining the pathophysiology of burns, the stress response, and diabetes mellitus places the patient in a difficult position. In some cases, the pre-existing comorbidity of diabetes mellitus makes healing from a burn difficult and can also be exacerbated by the stress response. Other times, the burn itself can cause the body to develop the metabolic disorder of diabetes mellitus.

### **Pre-existing Diabetes Mellitus**

A pre-existing comorbidity of diabetes mellitus alters the human body's compensation for a burn by the cardiovascular, renal, respiratory, musculoskeletal, and immune systems. The compensation for a burn includes major fluid shifts that put

extensive strain on the cardiovascular and renal systems, which are already compromised by diabetes mellitus. The location of a burn injury combined with diabetes mellitus significantly influences the potential complications and nursing considerations during care (Knighton, 2014).

Fluid loss secondary to a burn places stress on the cardiovascular system by causing hypovolemia, electrolyte abnormalities, and potential arrhythmias. The increase in blood viscosity and damage to capillaries in the dermis cause sludging which may be treated with fluid replacement. Patients with increased age, obesity, or immobility are at risk for thrombus formation after a burn (Knighton, 2014). Diabetes mellitus further impairs circulation by increasing blood viscosity with glucose and damaging blood vessels, thus contributing to fluid loss and decreased perfusion with burns (Holt, 2014). Fluid loss and replacement can also cause electrolyte abnormalities and fluid overload in heart failure patients (Knighton, 2014).

Fluid shifting out of the vasculature may also damage the kidneys due to a decrease in perfusion. A loss of blood flow to the kidneys results in acute kidney injury (Grossman, 2014b). In addition, myoglobin and hemoglobin may disrupt kidney filtration and can result in altered sodium and potassium levels (Knighton, 2014). Patients with pre-existing diabetes already have nephropathy and poor renal function so fluid resuscitation is critical (Holt, 2014).

Burns can affect the respiratory system both directly and indirectly. Inhalation burns directly impact the upper airway, lower airways, or both (Knighton, 2014). Burns in the airways may occur without burns to the skin and may be assessed with bronchoscopy, arterial blood gas (ABG) measurements, or carboxyhemoglobin levels.

Indirectly, burns around the face, neck, and chest may result in obstruction of the airway due to edema from fluid shifts. Pulmonary edema may also occur after fluid resuscitation. Previous lung conditions increase the risk of infection, such as pneumonia. Pneumonia is the most common cause of death in patients with an inhalation burn (Knighton, 2014). The risk for a pulmonary infection is once again increased for diabetic patients with a compromised immune system (Holt, 2014).

The musculoskeletal system can be severely impacted by burn injuries, especially full thickness burns. Circumferential burns around a limb can result in edema that blocks circulation to an area of the body and eventually compartment syndrome (Knighton, 2014). Compartment syndrome is a serious phenomenon that occurs when “increased pressure in a limited space compromises the circulation and function of the underlying tissues” (Smith, 2013, p. 48). Contractures can result from limited movement and a lack of range-of-motion exercises (Knighton, 2014).

Immune responses to burns are also affected by diabetes mellitus with increased blood glucose and decreased inflammatory ability. Infection and sepsis are serious complications due to breakdown of the body’s first defense and exposure to the body’s own flora (Knighton, 2014). Deaths from burns have been reduced by over 50% in the past 40 years but “up to 75% of morbidity in burn patients is related to infection” (Heo et al., 2013, p. 511). Burn location also impacts the risk of infection. For example, burns around the face can lead to infections around the eyes or ears. In addition, burns in the perineal area or buttocks are at risk for infection due to exposure to urine and feces (Knighton, 2014). Normal flora is typically beneficial but causes infection in the immunocompromised patient with no skin integrity and diabetes mellitus. Extreme

precautions should be taken to prevent the spread of bacteria for these immunocompromised patients (Holt, 2014).

The ability to provide self-care and independence may also be significantly altered by burns. Burns to the hands, feet, and eyes hinder the ability to take care of oneself, which is essential to maintenance of diabetes mellitus. Burns to hands and feet may result in damage to numerous superficial vessels and nerves which are already compromised by diabetes mellitus (Knighton, 2014). Coping skills, previous mental health, and personality heavily impact the ability of the patient to adjust both psychologically and socially (Attoe & Pounds-Cornish, 2015).

### **Diabetes Mellitus Second to Burns**

Endocrine complications from burns may result in diabetes mellitus, even if the patient had never experienced the disease previously. Burns, a physical stressor, cause hyperglycemia due to the epinephrine and cortisol released during the stress response (Mecott et al., 2010). When these glucose levels are not controlled, insulin resistance may occur and the endocrine disorder develops (Gerard, 2014). Similar to other long term complications, diabetes mellitus can be prevented with attentive nursing care and appropriate fluid and drug administration which will be discussed under treatments.

Another consequence of developing diabetes mellitus with a burn is an increase in hospitalizations and the length of hospital stays. A recent study performed in Western Australia identified the significance of the interaction between burns and diabetes mellitus (Duke et al., 2016). Researchers assessed 30,997 people admitted with an index burn between January 1, 1980 and June 30, 2012. The patients with burns were compared with 123,399 people who did not experience any serious injuries in that time. They were



matched based on gender and birth year. The Western Australia Data Linkage System tracked the number of hospital admission and length of hospital stays for diabetes mellitus after the date of discharge from the burn injury. The organization adjusted for socio-demographic and previous health factors to find that the patients who experienced burns were two times more likely to be admitted to the hospital or experience longer hospital stays for diabetes mellitus than their uninjured counterparts. In addition, burn patients with pre-existing diabetes mellitus were three times more likely to be hospitalized than uninjured patients with pre-existing diabetes mellitus. These higher rates of hospitalization for diabetes mellitus in burn patients were especially significant in the first five years post-burn (Duke et al., 2016). Ultimately, burns significantly increase the risk for the metabolic disorder or can worsen a pre-existing condition. After looking at this data, blood sugar control and prevention or control of diabetes mellitus should be a high priority for nurses when treating patients after a burn (Duke et al., 2016). Nurses that are mindful of diabetes mellitus in burn patients will reduce the complications of infection and fluid imbalances.

### **Treatments**

Nursing care for burns occurs in four phases: prehospital, emergent, acute, and rehabilitation (Knighton, 2014). Each step has a broad goal and standard but the individual treatments and medical decisions are based on the patient's unique history, symptoms, and wound. To treat burn patients adequately, the pathophysiology of diabetes mellitus must be considered when choosing treatments to either prevent or control the metabolic disorder. Considering the pathophysiology will promote the use of evidence-based practice and support patient safety and health. Nurses are heavily involved in most

standard burn treatments and therefore have the power and ability to make a significant difference in the correlation between burns and diabetes mellitus.

### **Prehospital**

The prehospital phase launches the initial care directly at the site of the injury. Personal protection of the rescuer should be addressed before attempting to assist the patient. Removing the patient from the source of the burn is essential no matter the cause. Removal includes the hot surface, electrical current, or chemical substance and then any burnt clothing, if possible. Immediate action after the burn should include an assessment of the airway, breathing, and circulation of the patient. After these vital functions are stabilized, an assessment for other injuries that could be more severe than the burn itself should be noted (Knighton, 2014).

Burns that cover less than 10% of the TBSA should be covered with a damp, cool cloth until they can receive medical care. Burns that cover more than 10% of the TBSA should be cooled for no more than 10 minutes and then simply covered with a clean sheet or blanket until medical care is available (Knighton, 2014). Covering the wound is especially important for diabetes mellitus patients who are prone to infection (Holt, 2014).

### **Emergent**

After the prehospital phase, the emergent phase occurs until life-threatening issues are fully addressed and fluid mobilization results in diuresis (Knighton, 2014). At this time, an appropriate assessment of the airway, breathing, and circulation must be established once again to identify life-threatening conditions. The burn should be assessed to according to the characteristics referred to previously. A comprehensive

assessment of this baseline enables the nurse and other medical staff to identify positive or negative changes in the patient's status.

**Airway.** Airway management may be performed in the prehospital phase to treat a life threatening status or as a safety measure in the acute phase. For example, patients with burns to the face, neck, or chest may be intubated to prevent an emergency tracheostomy later (Knighton, 2014). If the burn victim has diabetes mellitus, special considerations should be made when using intubation. In a preoperative study of 60 diabetic patients and 60 nondiabetic patients, several anesthesiologists identified difficult intubation in the diabetic population:

This is due to nonenzymatic glycosylation of collagen and its deposition in joints resulting in Limited Joint Mobility Syndrome (LJMS). It occurs in 25 to 45% of patients with long-standing diabetes mellitus due to involvement of atlanto-occipital joint, which limits adequate extension of head and neck during laryngoscopy leading to difficult intubation. (Gondane, Kudalkar, Padmanabha, & Raut, 2017, p. 523)

The Palm Print Index, an assessment of the hand print, was the most specific predictor of intubation difficulty and second in sensitivity to the Mallampati Test, an assessment of the distance between the tongue and hard palate, for the 60 diabetic patients studied (Gondane et al., 2017). If possible, these assessments may prepare nurses, respiratory therapists, or anesthesiologists for possible difficult intubation.

**Fluid.** Fluid resuscitation is based on the TBSA affected by burns. If 15% of the TBSA is affected, two large bore IVs are required in locations that allow for infusion of large volumes of fluid. If 30% of the TBSA is affected, the patient may require a central

line. Goals for fluid resuscitation include urine output of approximately 0.5-1 mL/kg/hr and the “mean arterial pressure should be greater than 65 mmHg, systolic blood pressure greater than 90 mmHg, and heart rate less than 120 beats/minute” (Knighton, 2014, p. 461). If central or peripheral venous pressures are used to measure fluid therapy, three standards must be met for evidence-based practice. The data must be accurate, the data must be relevant to the patient, and changes in the data must be noted (Athina, 2015).

Fluid resuscitation is regulated by fluid requirements based on the Parkland-Baxter Formula. Four milliliters of lactated Ringer’s solution per kilogram body weight per percent of TBSA burned is the total fluid requirement over the first 24 hours after a burn (Knighton, 2014). Crystalloids administered based on this formula should be distributed by half of the total in 8 hours, then a quarter of the total over the next two sets of 8 hours. The Parkland-Baxter Formula may cause “fluid creep” or excessive resuscitation (Sun et al., 2013, p. 478). Therefore, resuscitation should be closely monitored by the patient’s urinary output and hemodynamics (Knighton, 2014).

The type of fluids used to resuscitate is a controversial topic, especially in relation to diabetic patients. Lactated Ringer’s solution is an isotonic crystalloid that contains less chloride than normal saline and includes lactate to combat acidosis (Reddy, Weinberg, & Young, 2016). Researchers compared 20 diabetic and 20 nondiabetic patients post-carotid endarterectomy who were given lactated Ringer’s solution and normal saline. No clinically significant difference was found in blood glucose levels was noted (Billiodeaux, Samuelson, Willett, Arulkumar, & Thomas, 2014). Further study of lactated Ringer’s solution in 115 burn patients revealed an inadequacy in the management of hypoalbuminemia and hyponatremia. Albumin or other colloids and sodium could be

used to supplement the lactated Ringer's solution 12-24 hours post injury (Habib, 2017). Researchers also compared Ringer's acetate to lactated Ringer's solution in eighty burn patients. The group who received Ringer's acetate experienced an improved Sequential Organ Failure Assessment score in patients with a TBSA of 20-70% (Gille et al., 2014). A fourth study performed on 56 rats compared fluid resuscitation with lactated Ringer's and hypertonic saline. The hypertonic saline was shown to reduce intestinal edema, systemic inflammatory response, and serum measurements of interleukin and HGMB-1, thus reducing multiple-organ dysfunction syndrome (MODS) caused by intestinal bacteria (Sun et al., 2017). Hypertonic saline also combats oxidative stress in the lungs and intestine (Sun et al., 2013). Ultimately, an isotonic crystalloid is recommended and may be accompanied with a colloid or hypertonic saline (Yuichiro et al., 2016). No evidence definitively confirms if hypertonic saline or lactated Ringer's solution has a more significant effect on the systemic inflammatory response. After the airway and circulation are stabilized, wound care, drug therapy, and nutrition therapy become key nursing priorities.

**Wound Care.** Wound care varies significantly depending on the location and extent of the burn injury. The comorbidity further requires extra care due to hyperglycemia, reduced circulation, neuropathy, and a resulting inability to heal properly (Holt, 2014). A burn leaves diabetic patients even more vulnerable to pathogens due to impaired skin integrity (Knighton, 2014). As a result, nurses must pay special attention to hand hygiene between patients and even during the care of a single patient. For example, performing a dressing change after performing oral or perineal care would significantly increase the patient's risk of infection from his or her own bacterial flora (Knighton,

2014). Microvascular damage from diabetes mellitus and decreased perfusion should also be considered (Holt, 2014). Treatment of burns around the ears, face, and neck must include no use of pillows or tight bandages due to lack of blood flow and increased swelling (Knighton, 2014). Literature varies and the best treatment is always unique to the patient's past history and current status.

**Dressings.** Wound care in the acute phase may be open or closed. Open treatment is the application of antimicrobial agents with no dressing. Closed treatment includes covering the antimicrobial agent with a sterile dressing or the application of a dressing with antimicrobials. Dressing changes can occur anywhere between every 12 hours or every 14 days (Knighton, 2014). The choice of dressings and topical agents are based on the burn, the patient's condition, risk of infection, available resources, and physician's preference based on experience. A preexisting comorbidity of diabetes mellitus should also be considered when dressing burns with modern medicine.

***Dressing materials.*** Wound-dressings materials can be divided into two categories: modern and classic. Modern materials create a moist environment, cover the wound, and promote wound healing. Moist wound healing "retains multinucleated leukocytes, macrophages, enzymes and cell growth factors contained in effusion on the wound surface. Such an environment promotes autolysis and removal of necrotic tissues and does not interfere with cell migration" (Yuichiro et al., 2016, p. 991). The moist environment also reduces scarring and promotes granulation (Abedini, Ahmadi, Yavari, Hosseini, Mousavi, 2012). However, the moist environment should be carefully considered and monitored in diabetic patients due to an increased risk of infection. Classic materials allow the wound to dry and will not maintain a moist environment.

Conventional gauze is not considered in either category (Yuichiro et al., 2016). Each material should be carefully considered by the physician and nurse during dressing changes.

The development of hybrid dressings combines the benefits of both dry and moist materials and includes medications to promote healing. A study on 20 female guinea pigs with deep partial thickness burns to their backs and sides was used to explore a hybrid dressing that released gentamicin, an antibiotic (Zilberman et al., 2015). The dressing consisted of a top layer of porous poly(dl-lactic-co-glycolic acid) with gentamicin and a collagen sublayer. The first group of four guinea pigs received a Melolin® dressing which includes “a low adherent perforated film, a highly absorbent cotton/acrylic pad and a hydrophobic backing layer” (Zilberman et al., 2015, p. 157). The next group received an Aquacel® Ag dressing with ionic silver, a common dressing used for control. The third group received a hybrid dressing without antibiotics. The last two groups received the hybrid dressing with gentamicin, both slow and fast release. After 12 days, the burn tissue was assessed. The hybrid dressing with slow release of gentamicin caused 28% reepithelization of the burns, in comparison with 11% by Aquacel®. The slow release hybrid dressing also lowered contractions and inflammation (Zilberman et al., 2015). This dressing provides a moist environment for healing with infection prevention for patients with diabetes and should be considered in the future.

**Topical agents.** Disinfection of burns is a debated topic, with opinions for and against the process. Some topical agents used include silver sulfadiazine, chlorhexidine, sterile saline, and povidone iodine (Yuichiro et al., 2016). Silver sulfadiazine is a conventional antibacterial agent used to treat burns. The broad-spectrum antimicrobial is

effective against both *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Silver itself is used in multiple forms to treat burns and diabetic wounds. The silver is incorporated into newer forms of dressings that perform better than traditional silver sulfadiazine. Agicoat® is “a woven silver-coated nylon fabrics in which silver is deposited on nylon fibres by autocatalytic electroless deposition techniques” that releases silver over an extended period of time (Abedini et al., 2012, p. 574). The material is then covered by an occlusive dressing, dampened with sterile water, and changed every seven days until reepithelialization is achieved. A study of 69 patients with partial-thickness burns were randomly assigned to be treated with silver sulfadiazine or Agicoat®. The patients treated with Agicoat® experienced less dressing changes, less pain, and reduced risk of infection (Abedini et al., 2012). The study did not address full thickness burns or a large sample but the positive response to Agicoat® is an encouraging development.

Silver sulfadiazine is often applied as a cream to the wound with sterile gloves or on a dressing. In a study of 12 rats with 4 dorsal full thickness burns each, silver sulfadiazine was added to a blend of polymers nanofiber scaffolds with polyurethane and gelatin. The substances acted as a base for skin regeneration while releasing the antibiotic which reduced infection and accelerated healing better than gauze or nanofiber alone within 3 weeks (Heo et al., 2013). Once again, this was a very small sample size with only full thickness burns and could be explored further. While silver sulfadiazine may still be a standard treatment, other forms of the agent should be considered. Other options include a polyhexanide/betaine gel that provides the same antimicrobial characteristics with better pain control than silver sulfadiazine (Wattanaploy, Chinaronchai, Namviriyachote, & Muangman, 2017).



Chlorhexidine is also a broad spectrum antibacterial that may be diluted to 0.5% for wound cleansing or 2-4% for surgical skin preparation. An integrated review by the Wound Healing and Management Node Group revealed that chlorhexidine acetate 0.5% is effective against *Acinetobacter baumannii* and methicillin-resistant *Staphylococcus aureus* (MRSA) infected burns (2016). However, chlorhexidine failed to remove bacteria from eschar or prevent muscle invasion by MRSA in four studies on full-thickness burns in rats. Instead, nanocrystalline silver dressings combined with fusidic acid prevented deep muscle invasion by MRSA and removed MRSA from eschar (Wound Healing and Management Node Group, 2016). The further proof of infection prevention with silver products is crucial for burn patients who are immunocompromised due to diabetes mellitus (Holt, 2014).

Povidone iodine is a topical agent conventionally used to disinfect burns. Recently, research revealed that povidone iodine should be carefully reconsidered, especially in diabetic patients. Povidone iodine may actually cause “iodine poisoning” after widespread application on patients with burns and a past history of kidney or thyroid dysfunction (Yuichiro et al., 2016). Diabetic patients are at a higher risk for contrast-induced acute kidney injury (CI-AKI) due to nephropathy. CI-AKI is a 25% or 0.5 mg/dL increase in serum creatinine within three days of iodine administration. The iodine can cause renal ischemia and tubular toxicity. Previously, CI-AKI was associated with intravascular administration of iodine-based contrast medium for diagnostic studies. However, recent reports have noted a population of patients developing CI-AKI through absorption of iodine through mucosa or burned tissue. A review of thirty-three reported cases revealed that a third of those cases were due to the absorption of povidone iodine

after topical treatment of a burn. The chance of developing CI-AKI is proportional to the size of the area of application. When treating patients with burns and diabetes mellitus, the use of povidone iodine should be reconsidered, especially widespread application in the diabetic population (Perrin, Hemett, Menth, & Descombes, 2012).

**Debridement.** Debridement is the removal of necrotic tissue that hinders healing. Debridement can be performed by nurses during dressing changes or by physicians in the operating room, depending on the severity of the necrosis (Knighton, 2014). An escharectomy removes necrotic tissue until viable tissue is found in order to place an autograft (Rosenburg et al., 2014). Some less invasive methods are currently available to reduce the risks of infection associated with surgery and diabetes mellitus.

Debridement may be performed with a hydrocolloid dressing that liquefies necrotic material from burn lesions in diabetic patients. A case report of a 60-year-old man with diabetes and a fully occluded foot ulcer concluded that hydrocolloid dressings can be effective in debridement and maintained healing. Use of the hydrocolloid dressings over 10 weeks completed granulation, epithelization, and eventually a closed wound for 12 months (Cuschieri, Debosz, Miiller, & Celis, 2013).

Another alternative to surgical debridement is a debriding enzyme called NexoBrid™. NexoBrid™ is mixed with a carrier gel and placed on a dressing for four hours. The enzyme, retrieved from pineapple plant stems, reduces the need for surgical debridement or autografting and reduces the risk of elevated compartment pressures (Rosenburg et al., 2014). These noninvasive debridement methods should be considered for diabetic patients.

Hydrotherapy uses water to cleanse and remove necrotic tissue from a burn. This method is not well researched, especially in relation to diabetes mellitus. Reusing equipment for hydrotherapy increases the risk of spreading MRSA and does not significantly improve the healing of burns (Yuichiro et al., 2016). In addition, when a past history or current development of diabetes mellitus is involved, hydrotherapy could further increase the risk of infection. Ultimately, all treatments that may reduce infection should be considered for diabetic burn patients due to their increased blood glucose levels and compromised immunity that lead to infection (Holt, 2014).

**Medications.** Drug therapy for burn patients includes analgesics, a tetanus immunization or immunoglobulins, antibiotics, and heparin for deep vein thrombosis prophylaxis if allowed (Knighton, 2014). Analgesics should be administered on a fixed schedule and before dressing changes. Proper timing reduces pain and the stress response which further worsens hyperglycemia. Tetanus toxoid (Tt) or human tetanus immunoglobulin (TIG) is recommended to prevent infection with *Clostridium tetani*, especially for those with unknown immunity or ten years since their last vaccination (Yuichiro, 2016). Diabetic patients are considered immunocompromised and are therefore candidates for early antibiotic administration after a burn. Bacterial cultures from the wound should be considered when choosing the antibiotic. Antibiotics reduce mortality and improve skin graft adherence (Yuichiro et al., 2016). All medications should be considered for side effects of hyperglycemia, such as some diuretics and glucocorticoids (Gerard, 2014).

Some patients may also require insulin after experiencing a burn to control diabetes mellitus, to treat endocrine abnormalities, or to maintain blood glucose levels

during total parenteral nutrition (TPN) administration. Researchers performed a study on the effects of insulin combined with ethyl pyruvate in the treatment of MODS, a possible occurrence after burns. MODS is defined as “the acute, simultaneously or successively progressive dysfunction or disorder of and damage to multisystem organs due to loss of control over systemic inflammatory responses and oxidative stress that are caused by trauma, shock, and severe infection” (Wang et al., 2016, p. 2154). MODS differs from organ failure because organs may still function. Infection post-burn, severe inflammation, oxidative stress, hyperglycemia, and insulin resistance lead to the development of MODS. Ethyl pyruvate is a “simple ester of pyruvic acid” that contains reductive ketones from pyruvate molecules (Wang et al., 2016, p. 2154). The addition of this anti-inflammatory and antioxidant agent should be considered to treat burn patients in addition to insulin.

To measure the effects of ethyl pyruvate combined with insulin, 80 rats were anesthetized and given third degree burns, which covered approximately 30% of the TBSA. Endotoxins and saline were injected after the burn. The rats were divided into four groups that received an injection of Ringer’s solution intraperitoneally six hours after the burn and then once a day for seven days (Wang et al., 2016). The control group received no other substances. The second group received 0.2 U/mL of insulin at 2.0 U/kg. The third group received ethyl pyruvate at 8.0 mg/mL at 40 mg/kg. The fourth group received both insulin and ethyl pyruvate based on their weight. Blood samples were drawn before the burn and on days 1, 3, 5, and 7. The levels of alanine aminotransferase (ALT), creatine (CRE), creatine kinase (CK), tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ), high-

mobility group box 1 (HMGB-1), malondialdehyde (MDA), and total antioxidant capacity (TAC) were measured (Wang et al., 2016).

The results displayed better outcomes when insulin was combined with ethyl pyruvate (Wang et al., 2016). The control group experienced increased glucose, ALT, CK, CRE, TNF- $\alpha$ , HMGB-1, MDA and low TAC. The group that received insulin and ethyl pyruvate experienced the lowest levels of glucose, ALT, CK, CRE, TNF- $\alpha$ , HMGB-1, MDA and highest level of MDA. The insulin alone and ethyl pyruvate groups were better than the control group but not as improved as the combination of both substances. Insulin has better anti-inflammatory properties than ethyl pyruvate, as evidenced by lower amounts of TNF- $\alpha$  and HMGB-1. This effect of insulin also correlates with the control of glucose levels provided by insulin. Ethyl pyruvate has better antioxidant properties, demonstrated by lower MDA and higher TAC. As a result, the combination of insulin and ethyl pyruvate optimizes potential anti-inflammatory and antioxidants effects, thus reducing MODS in burn patients, controlling blood sugar, and prevent diabetes mellitus (Wang et al., 2016). Therefore, the nurse's goal should be to reduce ALT, CK, CRE, HMGB-1, TNF- $\alpha$ , MDA, and increase TAC and ultimately promote appropriate regulation of glucose. The incorporation of ethyl pyruvate with insulin could be a key treatment for burn patients with diabetes mellitus.

**Nutrition.** Burns cause a hypermetabolic state that may increase the resting metabolism by 50-100% due to the action of catecholamines. Catabolism also occurs with protein breakdown and gluconeogenesis (Knighton, 2014). Nutrition therapy at this stage includes meeting the increased caloric needs of the patient by mouth if possible or through a parenteral feeding. An increase in metabolism requires increased calories to

heal and function. Depending on the severity and location of the burn, some patients may require TPN. TPN should be begun at 20-40 mL/hour and increased to the goal rate within 24-48 hours (Knighton, 2014). The use of TPN may lead to or worsen carbohydrate metabolism disorders. In a study of 605 patients receiving TPN, 50.9% of patient had a glucose over 180 mg/dL at some point. 71.6% of the patients received insulin. This study used HgbA1c to evaluate blood glucose control and found that close blood glucose and insulin monitoring should be performed in patients receiving TPN due to the higher risk of hyperglycemia (Oliveira et al., 2015). Attentive monitoring is even more crucial for burn patients with an exaggerated stress response and endocrine abnormalities.

Another option is enteral nutrition through a feeding tube. In a study of 52 severely burned patients, 28 patients were given enteral nutrition on post-injury day three and 24 patients received enteral nutrition past post-injury day three. Beginning the enteral nutrition early increased the energy intake necessary and improved prealbumin levels (Wu, Liu, Jin, Liu, & Wu, 2018). However, an increase in calories in diabetic patients also increases their blood sugar and requires increased supplemental insulin as well.

### **Acute**

After the prehospital and emergent phases, the acute phase occurs after diuresis of excess extracellular fluid and ends when the wound is healed or covered by a skin graft (Knighton, 2014). Healing continues as the area sloughs necrotic tissue and fibroblasts set up collagen for granulation tissue. Wound care continues with debridement, cleansing, dressing, and potentially a skin graft. Superficial partial thickness wounds may heal by themselves if infection and dryness are appropriately prevented. Deep partial and full

thickness burns may need a skin graft due to a lack of viable dermis with re-epithelializing cells (Knighton, 2014). Some grafts may come from porcine skin, cadaveric skin, the patient's own skin, collagen, or other dermal matrix. Excision of eschar is performed first. Once clean, viable tissue is maintained, the graft may be placed (Knighton, 2014). Diabetic patients experience longer hospital stays and require more grafts than patients without diabetes mellitus (Lawrence & Li, 2015).

### **Rehabilitation**

The rehabilitation phase begins when the burns have healed and the patient can begin some self-care. Prevention of skin and joint contractures by practicing range-of-motion exercises is key to rehabilitation. In addition, skin care with moisturizers and sun protection is necessary for recovery (Knighton, 2014). Patients and their caregivers need to understand how to perform appropriate, safe wound care at home. In addition, proper pain management, nutrition, and skin care are essential to recovery. Some patients may require more surgeries and occupational and physical therapy to recover and maintain their mobility. Encouragement is crucial at this time because burn patients may experience many psychological, sexual, cultural, or spiritual difficulties as they process their injuries and the implications on their life (Knighton, 2014).

### **Discussion on Nursing Implications**

**Prevention.** The first step in managing burns is to prevent their occurrence with education and evidence-based practice, especially in the diabetic population. Nurses can promote the use of items that prevent the risk of a burn such as child-resistant lighters, nonflammable clothing, anti-scald devices, fire-safe cigarettes, strict building codes, smoke detectors, and fire sprinklers (Knighton, 2014). For diabetic patients, nurses

should educate on the consequences of neuropathy and subsequent loss of sensations that indicate heat and danger. Meticulous attention to detail around hot water, stoves, irons, and other sources of heat is a high priority for patients struggling with diabetes mellitus. Nurses can personally reduce burns by educating diabetic patients with creative handouts, videos, or practice activities. Education could include testing smoke detectors, using a thermometer to check the temperature of water, or turning handles in on the stove to prevent spilling scalding liquids (Knighton, 2014). Burn prevention is not a topic specific to diabetes mellitus pathophysiology itself, but plays a crucial role to promoting patient health and safety.

**Safety.** After a burn occurs, the nurse's first priority is to maintain safety for himself or herself and the patient at all times (Knighton, 2014). The next priority of care is to stabilize the patient's airway, breathing, and circulation and securing any potential cervical spinal fracture. After the emergent phase, infection prevention, activity maintenance, and adherence to treatments become important priorities in nursing care.

**Evidence-based practice.** The decisions on health care treatments and methods made by both nurses and physicians should be based on recent and credible research that is applicable to the patient's history and presentation (Chan, 2013). As the patient advocate, the nurse should be familiar with standard treatments and systematic application of new research on safer or more effective techniques (Buckwalter et al., 2017). For example, while silver sulfadiazine cream and surgical debridement may be common and conventional treatments, nurses and physicians should be willing to try silver impregnated dressings and noninvasive debridement due to the evidence supporting the efficacy and safety of these methods. In addition, nurses are responsible for close



assessment of burn patients and any development of endocrine disorders. For patients with preexisting diabetes, nurses should prioritize infection prevention and blood glucose control. Once again, a vast amount of current research on burns is general and not specific to the diabetic population. However, due to the immune and endocrine complications of diabetes mellitus, strict adherence to the most effective methods is crucial for the healing process of diabetic burn victims.

The Iowa Model, a framework for research and implementing evidence-based practice, includes steps to promote the development of health care: identifying a triggering issue, stating a question, forming a team, synthesize the body of evidence, design a practice change, integrate the change, and disseminate results (Buckwalter et al., 2017). Following the Iowa Model, the triggering issue was identified as adult burn patients with diabetes mellitus and the question was to find treatments unique to the pathophysiology of these two disease processes. After reviewing and synthesizing current research on both burn treatments and diabetes, recommendations can be made for nursing staff regarding the care of burn patients with diabetes. Despite the lack of specific research or protocols dedicated to this population, an appropriate approach to airway management, fluid resuscitation, wound care, infection prevention, and blood sugar control can provide better outcomes for these patients.

1. Airway – Assess the need or future need for intubation. If intubation is necessary for cardiac or respiratory complications, use the Palm Print Index or Mallampati Test to assess for possible difficulty (Gondane et al., 2017).
2. Fluid resuscitation – Assess vital signs and labs to establish the patient's fluid status. Use the Parkland-Baxter Formula to calculate initial fluid resuscitation

with lactated Ringer's solution in the first 24 hours after a burn injury (Knighton, 2014). Consider adding hypertonic saline or albumin after 24 hours (Yuichiro et al., 2016).

3. Wound care – Perform debridement with hydrocolloid dressings (Cuschieri et al., 2013). NexoBrid™ may also be considered (Rosenburg et al., 2014). Hybrid dressings with slow release of antibiotics should also be considered (Zilberman et al., 2015). Disinfect burns with products containing silver, such as Agicoat® or nanocrystalline silver dressings with silver products. Avoid the use of povidone iodine (Perrin et al., 2012).
4. Infection prevention – Perform hand hygiene before, during, and after patient interactions (Knighton, 2014). Begin antibiotics as soon as cultures are drawn (Yuichiro et al., 2016).
5. Blood glucose control – Assess the ability of the patient to eat and the quality of their nutrition. An increase in metabolism may require TPN or enteral nutrition (Knighton, 2014). If TPN is required, monitor blood glucose closely (Oliveira et al., 2015). If enteral nutrition is considered, begin within 3 days of the burn injury to provide maximum nutritional benefits (Wu et al., 2015). Give insulin with ethyl pyruvate to reduce inflammation and promote healing, in addition to treating diabetes mellitus (Wang et al., 2016).

The evidence found throughout this research included a range of levels of evidence, from levels one through five, and included case reports through randomized control trials (Boswell & Cannon, 2017). This assembly of research may be used to

conduct further research or design a practice change according to the Iowa Model for evidence-based practice (Buckwalter et al., 2017).

**Psychological factors.** Burn patients also often need increased emotional and psychological support (Knighton, 2014). In addition, a past history of chronic diabetes mellitus increases the patient's risk for depression (Katon et al., 2010). Understanding the impact of diabetes mellitus should also drive nurses to prevent the development of diabetes in burn patients without past endocrine diseases. Nurses have the opportunity to encourage and assist patients through one of the hardest physical struggles they may experience. Unfortunately, the task of assisting patients that are dealing with significant emotional issues can also take a toll on healthcare workers. Working in this area requires immense wisdom and discernment to maintain the patient's life with compassion and love for their circumstances and attitude (Knighton, 2014).

### **Conclusion**

In summary, burns are complex injuries that require a custom plan of care based upon each patient. Patients with diabetes are at an increased risk for burns and complications from burns due to their vascular, neuropathic, and immune abnormalities. As a result, evidence based-practice is crucial when making decisions on the plan of care. Standard treatments for burn patients should be modified specifically for diabetic patients. Treatment should focus on insulin and antibiotic therapy while using topical agents that promote healing and protect the wound. Any risk of infection through moisture or extra glucose administration should be avoided. Patient education should be centered on blood sugar control and infection prevention. These practices are evidence based and should become a minimum standard for diabetic burn patients.

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