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Clinical Nutrition in Diabetes Mellitus with Third Degree Burns and Foot Ulcers

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Abstract

Burns are complex injuries that lead to hyperglycemia as a stress response. Diabetes Mellitus (DM) is a hormonal and metabolic disorder categorized by impaired insulin secretion and yielding to elevate plasma glucose levels and therefore complicates the therapy of burns, it is also developed via uncontrolled stress reaction to the burn. Also, DM will impact on nutrition and lifestyle of burn patients. This review provides pathophysiology, difficulties and managements of burns related to the patients with DM to offer evidence-based care to seriously ill people. Performing a treatment for diabetic burn patient is difficult for healthcare professionals. Traumatic burn in non-diabetic people may develop stress-induced hyperglycemia which ends up with DM due to endocrine complications from their injury. The higher risk for burn injuries is found in pre-existing DM. Special medical plan should be employed to control burns. Additionally, treatments for present burn must be reformed to avoid diabetes mellitus, management of glucose level, and help healing process in diabetic foot ulcer.

Keywords: Burns, Diabetic Foot Ulcer, Diabetes Mellitus, Glucose, Insulin

1. Introduction

1.1 Burns

There are different treatment plans according to patient's personal profile which indicates the characteristics of wound with the previous history and coexisting condition. The physiological disorder of burns and the uncontrolled body reaction recognizes parts that should be identified and compared to past history of diabetes mellitus, including controlling levels of blood glucose and avoiding infection[1].

The aim of this review is to afford a pathophysiology, difficulties and managements of burns associated to diabetic patients to propose an evidence-based care to patients.

1.1.1 Pathophysiology

Local coagulation necrosis developed after burn[2]. An inflammatory response will be initiated by both vascular and cellular actions to burns. As a response to histamine secretion by injured cells arterioles vasoconstriction followed by vasodilation will arise temporarily. A clot formation will occur due to platelets and the fibrin that formed from the fibrinogen that released with albumin

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out of the cells due to high permeability[3]. Healing also, promoted by growth factors secreted by platelets while a chemotactic response including movement of neutrophils and monocytes to the injured area, where neutrophil started phagocytosis of the bacteria and debris within six hours causing pus formation[4].

In addition, after 3 to 7 days of burns monocytes that reached the injured area converted to macrophages and initiating a phagocytic process and a non-defined protection and stay there for weeks while precise immune reaction developed by lymphocytes[5]. Repairing of wounds started after 6-12 hours of burns and new tissue formation started by fibroblasts and collagen fibrils[6].

Damaged cell membranes end with formation of chemical mediators from arachidonic acid. Prostaglandins lead to vasodilation that increases blood flow and edema while thromboxanes lead to vasoconstriction that causes accumulation of platelets[7]. Also, leukotrienes may be a reason of edema because it raises the capillary wall permeability and this is serious especially around the air passages or in compartment syndrome[5]. In severe burns, the systemic inflammation cause fluid and electrolyte shift. Also burns may lead to shock (Figure 1)[8].

1.1.2 Classification

Classification of burns could be done depending on nature, deepness, size and location. According to that burns are divided into thermal, chemical, electrical burns and smoke and inhalation injuries[4]. Previously, burns classify according to depth into 4th degrees. Nowadays they are classified according to the depth of the affected tissue. The damage in partial-thickness burns classified as superficial in the epidermis and deep in the dermis the damage in full-thickness burns affect fat, muscle and bone tissue under the dermis[7] (Table 1, Figure 2).

The Total Body Surface Area (TBSA) measures the area of a burn injury comparing to the whole area of the body surface. The body could be divided into nine sections according to the rule of Nines chart[9].

1.2 Diabetes Mellitus

1.2.1 Pathophysiology

Beta cells in the islets of Langerhans of the pancreas secreted the peptide hormone Insulin due to high blood glucose level. The action of insulin includes glucose uptake

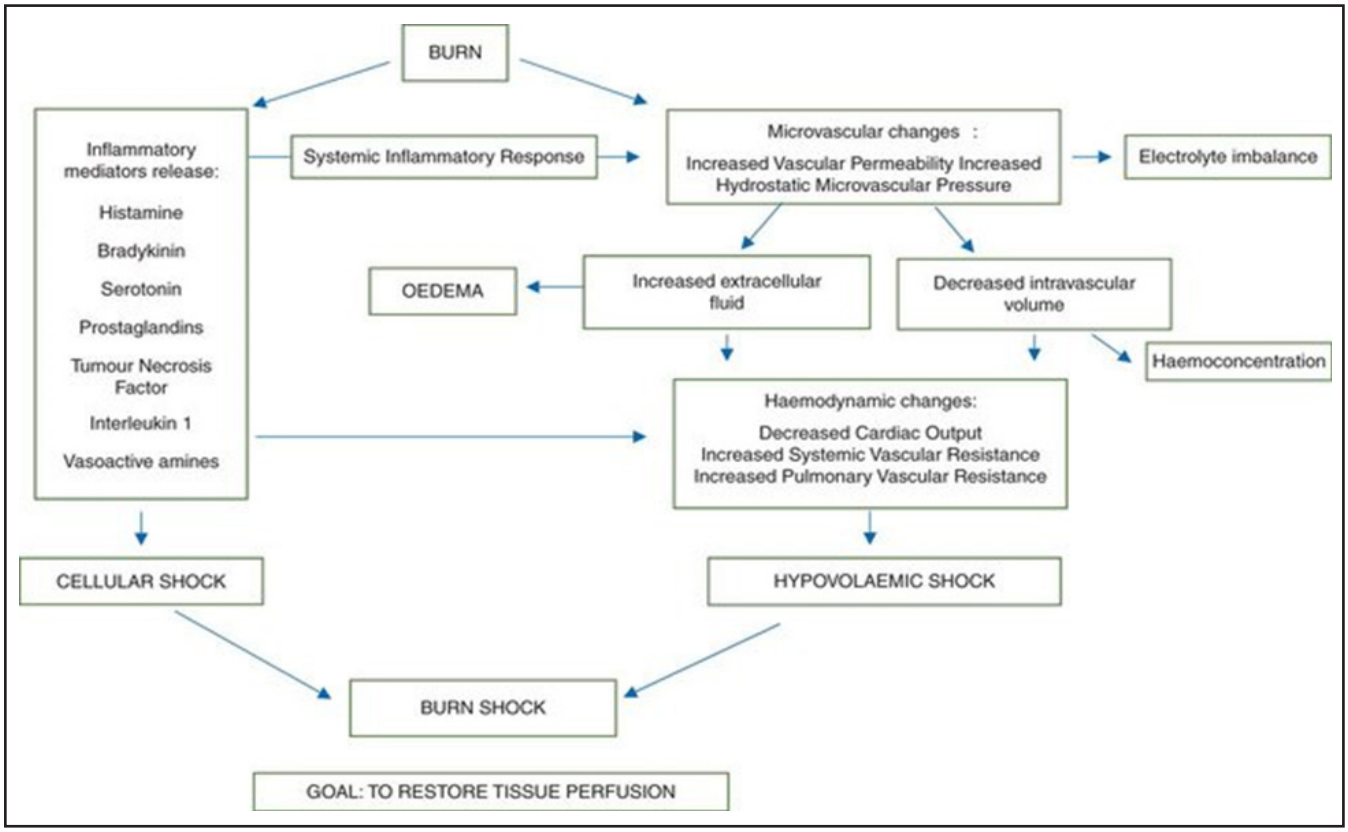

Figure 1. Burn shock pathophysiology[8].

Table 1. Burns divided according to depth

Class	Depth	Color	Sensation
Superficial	Epidermal	Red	Present
	Superficial Dermal	Pale Pink	Painful
	Mid Dermal	Dark Pink	+/-
Deep	*Deep* Dermal	Blotchy Red	Absent
	Full Thickness	White	Absent

by cells and storage as glycogen in liver and muscles, also, storing the rest as lipid in adipose tissue and enhancing amino acid uptake by cells increasing protein synthesis[9].

Insufficient insulin production leads to hyperglycemia; which causes burn injury or complication during healing at long term[11].

DM-type 1 occurred because of deficiency of insulin due to autoimmune destruction of β-cells, it's managed by insulin administration to control blood sugar[12]. DM-type 2 occurred because of insulin abnormal production or resistance by hepatocytes, myocytes and adipocytes leads to lack of glucose uptake due to genetic or environmental factors as obesity and a sedentary lifestyle[11], it's managed by medications (insulin) and lifestyle modifying.

1.2.2 Diagnosis

Diagnosis of DM could be done by many tests include; Fasting Blood Glucose (FBG) test measures in which blood glucose level tested after fasting of 8 hr. Results are: Normal (70-100 mg/dL), impaired fasting glucose (100-125 mg/dL), Dinettes Mellitus (DM, over 125 mg/dL). Second test is the Oral Glucose Tolerance Test (OGTT) that measures blood glucose after ingestion of 75 g of glucose. Results are taken after two hours and could be: Normal if levels lower than 140 mg/dL, impaired glucose tolerance if levels between 140-199 mg/dL, and DM if levels greater than 200 mg/dL[13].

Hemoglobin A1C (HbA1c) test measures glycated hemoglobin that reflects the patient's control of diabetes over the past three months, it is DM when HbA1c > 6.5%[9].

1.2.3 Symptoms

The big three diabetes signs are polyuria and polydipsia (due to the osmotic effects of glucose, hyperglycemia causes elimination of glucose with water from the kidney, causing feeling of thirst), the 3rd sign is Polyphagia due inability of the cells to take up glucose[14].

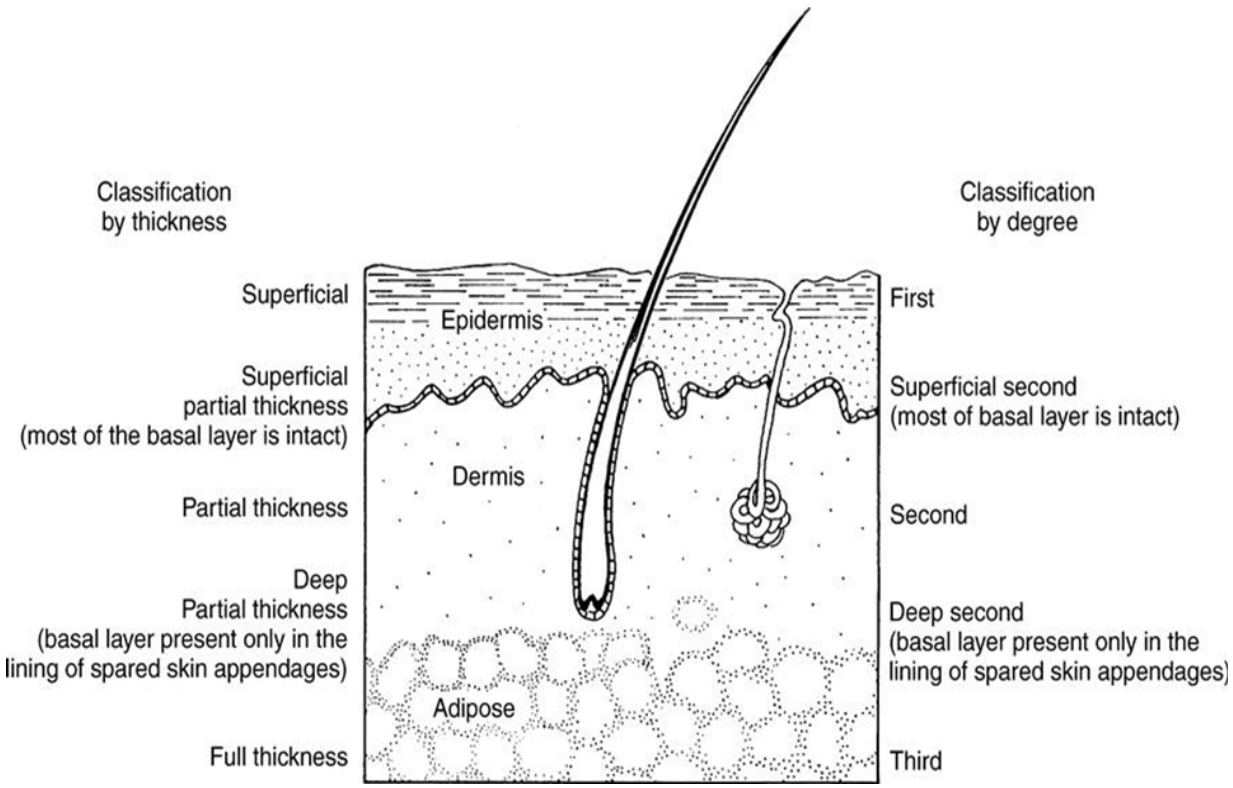

Figure 2. Classification of burns according to the depths of burn injury and the degree[10].

1.2.4 Consequences of Hyperglycemia

Results of chronic hyperglycemia are: Microvascular (retinopathy, neuropathy and nephropathy), macro vascular (hypertension and hyperlipidemia which lead to CHD) and immune dysfunction[14]. Overall, DM led to reducing phagocytosis, chemotaxis and the bactericidal capacity of the inflammatory response and therefore, hyperglycemic burn patients have a high risk for infections[15].

1.2.5 Hormonal Influences

In a stress condition, some hormones are released, Epine phrine which inhibits glycogenesis and stimulates glyco genolys is in the liver. The growth hormone inhibits insulin action. And cortisol promotes gluconeogenesis[16]. Hyper-production of these hormones can lead to DM in non-diabetic people[12].

1.2.6 Complications of Burns Associated with DM

Difficulties in burns healing due to the pre-existing comorbidity of Diabetes Mellitus comparing to the absence of DM. The burn itself can develop a metabolic disorder[2].

1.2.7 Diabetes Mellitus with 3rd Degree Burns

The 3rd degree burns in DM lead to 1. Hyperglycemia which developed to Insulin Resistance (If not controlled glucose levels), 2. Long hospitalizations, recently, a significance interaction between burns and DM was reveled[11]. A 30,997 patients admitted with an index burn between January 1, 1980 and June 30, 2012 enrolled were compared with 123,399 patients without any serious injuries in the same time. 3. Increase risk for a metabolic disorder[17].

1.2.8 Diabetic Foot Ulcer

Diabetic Foot Ulcers (DFUs) are chronic complication of Diabetes Mellitus. It represents 50% of all diabetic hospitalization cases, it is an asymptomatic disease, which impedes ulcers healing, so the annual diabetic foot screening helps for early detection[15]. The time needed to develop a foot ulcer in diabetic could 25% higher than non-diabetic[2]. The tissue engineering techniques and regenerative medicine are recently applied in treatments of DFUs[18].

1.3 Wound Care

The comorbidity involves additional care because of hyperglycemia, reduced circulation, neuropathy and a failure in proper healing[15]. Impaired skin integrity by 3rd burn degree will increase vulnerability to pathogens even with patient's bacterial flora, in this case the hospital should increase caring and monitoring.

Pillows or tight bandages shouldn't be used in case of 3rd degree burns around the ears, face and neck[19].

Insulin can be used during Total Parenteral Nutrition (TPN) for burned diabetic patient in order to control DM, to treat endocrine defects or to maintain levels of blood glucose[20]. Multiple Organ Dysfunction Syndrome (MODS) can be treated with insulin-ethyl pyruvate[17]. MODS can be developed by post-burn infection, severe inflammation, oxidative stress, hyperglycemia and insulin resistance. The addition of antioxidant/anti-inflammatory agent next to Insulin helps to treat burns in diabetics[3].

1.4 Nutrition

Burns cause a hyper metabolism (due to the action of catecholamine) this will increase calories for healing. Severity and position of the 3rd degree burn in some patients may necessitate TPN treatment starting with 20-40 mL/hour and increased to reach the desired level within 24-48 hours, this procedure may alter carbohydrate metabolism diseases[17]. The result of a study with 605 patients getting TPN showed that 50.9% of them glucose level greater than 180 mg/dL. Though, 71.6% of the patients treated with insulin. HgbA1c was performed to monitor glucose levels and conclude that insulin must be monitored during receiving TPN to avoid hyperglycemia risk[16].

In a study included 52 severely burned patients enteral nutrition was applied to feed patients: 28 patients were given enteral nutrition within 3 days of injury and 24 patients received enteral nutrition after three days. Results showed that the earlier use of the enteral nutrition improved pre-albumin[20,21].

2. Conclusion

Third degree burns are complicated wounds that necessitate a convention strategy of care for each patient. As a result of their vascular, neuropathic and immune anomalies diabetics face high risk for burns and their complications. For that, evidence based-practice is

essential when preparing the strategy of care. Diabetics with 3rd degree burn require special standard treatment procedures. In which a special attention must be applied for insulin and antibiotic treatment during using topical treatment that indorse curing and defend the injury. Also, special consideration must be taken to avoid infection that may appear due to moisture or extra glucose administration

Patient should be aware about their blood sugar control and protection from infection. These procedures are evidence based and must be applied to be the lowest normal for diabetic burn patients.

3. Conflicts of Interest

There are no conflicts of interest.

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