



Case Report: Second-Degree Burns due to Hot Liquid Asphalt Cement Spillage in an Adult Patient with Comorbid Diabetes Mellitus

¹ Maha Umami Putri Qulzum, ² Sri Vitayani, ³ Widjoyo

¹ Faculty of Medicine, Universitas Muslim Indonesia, Makassar, Indonesia

² Department of Dermatology and Venereology, Ibnu Sina Hospital, Makassar, Indonesia

³ Department of Surgery, Siloam Hospital, Makassar, Indonesia

Corresponding Author Email: putraaputriq@gmail.com

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ABSTRACT

Burns represent a significant form of injury that leads to substantial tissue damage, necessitating complex management strategies tailored to the degree and total body surface area (TBSA) affected. The complexity of burn management is notably increased in patients with comorbidities such as diabetes mellitus. Diabetes mellitus can induce vascular disorders and peripheral neuropathy, which collectively impair the natural wound healing process. This report details the case of a 42-year-old male who presented to the Emergency Department of Makassar City Hospital with burns on his right and left legs. The injuries were sustained approximately 30 minutes prior from a spill of hot liquid asphalt cement at his workplace and were accompanied by a significant burning sensation. The patient, weighing 58 kg, reported no fever, headache, dyspnea, nausea, or vomiting, with normal bowel and bladder function. He had no history of similar injuries, allergies, or hypertension. However, he had a known history of diabetes mellitus, which was managed with metformin 500 mg three times daily. A family history of

diabetes was present on his maternal side. Burns, or *combustio*, are defined as tissue damage or loss resulting from contact with thermal sources like fire and hot water, or with chemicals, electricity, and radiation. Hot water and flames are the most frequent causes of burns. The primary principle of first aid is to halt the burning process and reduce the wound's temperature. For flame-related burns, this involves the "Stop, Drop, Cover (Face) & Roll" technique.

Keywords: burns, cement spills, asphalt liquid, diabetes mellitus

INTRODUCTION

Burns are a form of traumatic injury that can result in significant tissue destruction, requiring intricate management protocols that vary based on the burn's depth and the extent of the body surface area involved. Second-degree burns, also known as partial-thickness burns, affect the epidermis and a portion of the dermis. These burns are clinically characterized by the formation of blisters (bulla), severe pain, and erythema, often leading to a significant impairment of skin function. Management is contingent upon the depth of the injury and the patient's overall clinical status.¹

While uncommon, burns caused by construction materials like cement and hot liquid asphalt pose a high risk. Hot asphalt's high temperature and viscosity cause it to adhere firmly to the skin, resulting in deep and challenging-to-treat thermal injuries. Furthermore, the chemical composition of asphalt may introduce an additional irritant effect, thereby exacerbating tissue damage.¹

The management of burns is substantially more challenging in patients with pre-existing conditions such as diabetes mellitus. Diabetes can lead to vascular insufficiencies and peripheral neuropathy, both of which impede the wound healing cascade. Chronic hyperglycemia also negatively affects leukocyte function and the inflammatory processes essential for effective wound repair. Consequently, diabetic patients face a heightened risk of infection, delayed healing, and other burn-related complications.²

This case, involving a burn from a hot asphalt spill in an adult with diabetes, is clinically significant due to the rare etiology combined with a comorbidity that complicates the patient's condition. This report aims to illustrate the clinical challenges in diagnosing and managing second-degree burns from hot asphalt in a diabetic patient and to underscore the necessity of a multidisciplinary approach to care.

CASE REPORT

A 42-year-old male presented to the emergency department of Makassar City Hospital with burns on both legs. The injury occurred at his workplace approximately 30 minutes prior to arrival, caused by a spill of hot liquid asphalt cement, and was associated with a severe stinging pain. The patient denied other symptoms such as fever, headache, shortness of breath, nausea, or vomiting. His weight was 58 kg. His bowel and bladder functions were within normal limits. He had no history of previous similar injuries, allergies, or hypertension. He had a known diagnosis of diabetes mellitus, which was controlled with metformin 500mg taken three times a day. A family history of diabetes was noted in his mother.

On physical examination, the patient's vital signs and general status were within normal limits. Local examination of the right and left crural regions revealed spilled asphalt cement material on both legs, which were covered with gauze dressings. The examination identified the presence of bullae and hyperemia, with an estimated Total Body Surface Area (TBSA) of 15% and tenderness upon palpation.

Laboratory investigations revealed the following: White Blood Cell (WBC) count of $11.51 \times 10^3/\mu\text{L}$, Red Blood Cell (RBC) count of $4.60 \times 10^6/\mu\text{L}$, Hematocrit (HCT) of 38.7%, Hemoglobin (HGB) of 13.2 g/dL, Platelet (PLT) count of $343 \times 10^3/\mu\text{L}$, and a random blood glucose level of 210 mg/dL.

The patient was diagnosed with a grade 2A burn on the right and left crural regions, covering 15% of the TBSA. Pharmacological treatment included intravenous ranitidine (50 mg) and ketorolac (30 mg). The wounds were irrigated with water to remove the asphalt cement, lower skin temperature, and reduce the depth of injury. Topical gentamicin antibiotic was applied, and the wounds were dressed with sterile gauze. Debridement was subsequently performed.

DISCUSSION

A burn, or *combustio*, is a form of tissue damage or loss resulting from contact with a heat source, such as fire, hot liquids, chemicals, electricity, or radiation. The majority of burn cases are

caused by hot water and flames.¹ Burns are a common occurrence, with first and second-degree burns being the most frequent types encountered in domestic settings. These injuries often lead to significant morbidity and disability, with the degree of disability being relatively high compared to other types of injuries, resulting in substantial healthcare costs.³ Identifying the source of the burn is the initial and crucial step before evaluation and management. Burns can be classified into four main types: thermal, chemical, electrical, and radiation injuries.⁶

Globally, burns represent a significant public health issue. The World Health Organization (WHO) estimated that in 2016, burns caused 265,000 deaths annually, with nearly half of these incidents occurring in Southeast Asia. The majority of these cases are concentrated in low- and middle-income countries. In contrast, high-income nations have seen a steady decline in burn-related mortality rates. The mortality rate for children from burns is seven times higher in low- to middle-income countries compared to high-income countries.⁷ Most burn incidents occur in domestic and occupational settings. Surveys from Bangladesh and Ethiopia indicate that 80-90% of burns happen at home. Women are often at a higher risk than men due to their frequent involvement in household activities involving heat sources. Children are another high-risk group, often due to inadequate supervision during activities near heat sources.⁸

In Australia and New Zealand, approximately 1% of the population sustains a burn requiring medical attention each year, with 10% of these cases needing hospitalization and another 10% being classified as severe, life-threatening burns.⁷ A study at Cipto Mangunkusumo Hospital (RSCM) in 2011-2012 reported 257 burn patients with a mean age of 28 years and a male-to-female ratio of 2.7:1. The causes were predominantly flame burns (54.9%), followed by hot water (29.2%), electrical (12%), and chemical burns (3.1%).⁸ A retrospective study at RSUD Daya Makassar from 2018 to 2021 analyzed 145 burn patients, finding that thermal burns were the most common (74%), followed by electrical (11%), chemical (8%), and contact burns (4%). The most affected age group was young adults (20–45 years) at 42%, with a male predominance (58%). Although less frequent, chemical burns are clinically important due to their potential for deep tissue damage and systemic

complications.⁹

Burns are classified by depth into first, second, and third degrees. First-degree burns, or superficial burns, affect only the epidermis and typically result from prolonged sun exposure or brief contact with a hot object. They usually heal within a week without scarring. Second-degree burns, or partial-thickness burns, extend into the dermis. They are subdivided into superficial partial-thickness, which heals in under three weeks, and deep partial-thickness, which takes longer than three weeks to heal and often results in hypertrophic scarring. Third-degree burns, or full-thickness burns, destroy all skin layers, including subcutaneous tissue and hair follicles, and require surgical grafting for healing.¹⁰ The extent of a burn is estimated using methods like the "Rule of Nines," the Lund and Browder chart, or the hand-palm method. The severity of a burn is determined by its depth and extent, with mortality being strongly correlated with the patient's age and the TBSA burned.^{12,13}

Following thermal trauma, the extent of skin and underlying tissue damage is influenced by the duration of contact and the temperature. Jackson's model from 1950 describes three zones of injury: a central zone of coagulation (most severe damage), a surrounding zone of stasis, and an outer zone of hyperemia. The thermal injury also elicits a systemic response, including increased capillary permeability, which leads to plasma leakage into the interstitial space. This process peaks within the first 8 hours and can continue for up to 48 hours. After this period, capillary permeability may normalize, or thrombus formation can impede circulation.¹⁶ The release of inflammatory mediators and neural stimuli triggers systemic physiological changes. Hypovolemia results from this fluid and protein shift. In burns covering more than 20% TBSA, systemic capillary permeability increases, making aggressive fluid resuscitation a life-saving intervention in the initial hours. The systemic response also includes a hypermetabolic state driven by stress hormones and immunosuppression, which increases the risk of infection.¹⁷ Burn pain originates from the damaged tissue and the inflammatory response, which releases mediators like bradykinin and histamine, leading to both primary and secondary hyperalgesia.¹⁸

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia stemming from insulin deficiency, insulin resistance, or both. Long-term complications of DM affect multiple organ systems, including the vascular, nervous, and immune systems, all of which are crucial for wound healing. According to the International Diabetes Federation (2021), the global prevalence of diabetes is rising, and these patients are more susceptible to severe wound complications and delayed healing. In the context of burns, DM presents significant clinical challenges by slowing tissue regeneration, elevating infection risk, and increasing mortality and morbidity.¹⁹

The complex process of wound healing occurs in four overlapping phases: hemostasis, inflammation, proliferation, and remodeling. DM disrupts these phases through several mechanisms¹⁹:

- **Immune Cell Dysfunction:** Hyperglycemia impairs the chemotaxis, phagocytosis, and bactericidal activity of neutrophils and macrophages, prolonging the inflammatory phase and increasing infection risk.
- **Microcirculatory Dysfunction:** Diabetes-induced microangiopathy causes endothelial damage and reduced tissue perfusion, hindering the delivery of oxygen and nutrients essential for new tissue growth.
- **Oxidative Stress and Chronic Inflammation:** Hyperglycemia accelerates the formation of Advanced Glycation End Products (AGEs), which promote oxidative stress and chronic inflammation, thereby inhibiting epithelialization and collagen deposition.
- **Impaired Growth Factor Production:** DM reduces the levels and activity of crucial growth factors such as VEGF, TGF- β , and PDGF, which are vital for cell proliferation and angiogenesis.

Key principles in burn management include assessing the mechanism of injury and determining the burn's extent and severity. Burns can disrupt the skin's seven primary functions:

thermoregulation, sensory perception, immune response, protection from bacterial invasion, control of fluid loss, metabolic functions, and aesthetic/psychological well-being. The primary goal of first aid is to stop the burning process and cool the wound. This involves removing burning or hot, soaked clothing and cooling the area with running water, ideally at a temperature between 8°C and 25°C. Cooling helps to limit damage in the zone of stasis; however, ice water is not recommended as it can cause vasoconstriction.²²

Fluid resuscitation is indicated for patients with burns covering >10% TBSA. The initial fluid of choice is a crystalloid solution, such as Ringer's lactate, with its composition tailored to the patient's needs. The patient's weight and the burn extent, calculated using established charts, are used to determine the fluid requirements according to resuscitation formulas.²³

Burn complications can be local or systemic and may occur in acute or chronic phases. Acute complications include hypovolemic shock from fluid loss, which can lead to organ failure. Infection is a major risk as the compromised skin barrier allows pathogens like *Staphylococcus aureus* or *Pseudomonas aeruginosa* to enter, potentially leading to sepsis. Other serious acute complications include electrolyte imbalances, inhalation injuries, and acute kidney failure. Burns on the face or chest can cause airway obstruction due to edema or constricting eschar. Chronic complications commonly include hypertrophic scarring and contractures, particularly over joints, which can limit mobility and function. Patients also frequently experience chronic pruritus, body image disturbances, anxiety, and depression.²⁶

The prognosis for a burn patient depends on the extent and depth of the burn, the patient's age, and any comorbidities. In diabetic patients, the prognosis is more guarded. Even minor burns can progress to chronic wounds or systemic infections if not managed properly. The risk of limb amputation is elevated, especially in the presence of poor tissue perfusion or diabetic neuropathy. The prognosis for life (*ad vitam*) is generally good for localized second-degree burns but can become serious if systemic infection or metabolic disturbances occur. The prognosis for function (*ad functionam*) is highly dependent on the location of the burn, joint involvement, and the success

of rehabilitation therapies.²⁶

CONCLUSION

Burns are a form of thermal trauma that can lead to significant tissue damage and complex clinical consequences. This case was rendered more complex by the patient's comorbid diabetes mellitus. The presence of diabetes worsens the prognosis for wound healing, as chronic hyperglycemia impairs leukocyte function, inhibits angiogenesis, and prolongs the inflammatory phase. Therefore, strict glycemic control is a critical component of long-term management for burn patients with diabetes. The prognosis for this patient was assessed as good both for survival (*prognosis ad vitam: bonam*), given the absence of systemic infection or organ failure, and for function (*prognosis ad functionam: bonam*), as the injury was localized to the extremities and managed promptly. Nevertheless, continued evaluation of motor function and potential long-term mobility limitations is warranted.

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