Management of burns

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Abstract

Burns are common injuries that vary in severity from small superficial scalds to massive full-thickness flame burns with high morbidity and mortality. The purpose of this article is to review common burn presentations, the pathophysiology of these injuries and to give an overview of multidisciplinary burns management from the emergency department through to the specialist burns centre.

Keywords Burns; burns surgery; dressings; resuscitation; scalds; scar management; skin grafts

Introduction and epidemiology

Burns are traumatic injuries caused by coagulative destruction of the skin and are usually caused by thermal damage (heat and cold) but chemicals, electricity and radiation may also damage tissues in similar ways. In the United Kingdom approximately 250,000 people present to primary care and to hospitals with burns each year, though this is an underestimation of total numbers, as many people with small or innocuous burns manage their injuries themselves.[1] The number of patients treated by specialist burns services in the UK each year is increasing; admissions to such units numbered around 1000 patients in 2001, rising to 14,000 a decade later in 2011 and reaching over 19,000 in 2015. Mortality from these injuries is decreasing in the UK and across the Western world, with a mortality rate of around 1.4% of these cases or approximately 200 deaths from burn injuries per year in England and Wales.[2]

No age group or gender is immune from burns, though nearly two thirds of all burn injuries are sustained by males.[2] Whilst it used to be the case that scald injuries were most common in childhood and then gave way to increasing flame, flash and contact burns as age increased, this is now less apparent. In children, adults *and* elderly patients, scald injuries now account for the majority of all burns. The second most common cause of burns in adults and elderly patients are flame injuries, followed by contact burns (Figure 1).

Whatever the cause for the burn, there is often an underlying patient vulnerability that puts the patient at risk. The patient with diabetes with neuropathy who sustains an unnoticed contact burn to their foot is vulnerable due to their poorly controlled medical condition. The unsupervised toddler who pulls a cup of tea onto themselves is vulnerable due to lack of attentive supervision. The intoxicated person who falls asleep with a lit cigarette following drink or drugs and sustains burns is vulnerable due to their lack of cognitive awareness or addiction. Patients who are depressed and self-harm or self-immolate are vulnerable due to their mental illness. Such vulnerabilities are varied, but each requires investigation and management to reduce the burden of burns on individuals and on society.

Pathology and pathophysiology

Local effects

Following a burn, tissue destruction is proportional to the temperature of the burning agent and the duration it is applied to the body. For example, water at 48°C takes 5 minutes to cause a partial thickness burn, but when the water temperature is raised to 70° it takes just 1 second to cause a full thickness injury. Jackson described zones of burn injury related to the degree of tissue damage (Figure 2).[3] The inner zone of coagulative necrosis represents unsalvageable burnt tissue where the blood vessels are thrombosed and the skin is dead. The intermediate zone of stasis represents tissue affected by the burn with static blood flow. This area is amenable to first aid, resuscitation measures and good wound care and is therefore salvageable if cared for appropriately. The outer zone of hyperaemia represents red, hyperaemic tissue that surrounds any acute inflammatory process and will return to normal.

The tissue damage that results following thermal injury results in a marked increase in capillary permeability which is maximal within the first few hours following injury and resolves within 2–3 days. During this time, small protein molecules leak out of the circulation leading to oedema and significant fluid loss. The fluid loss is proportional to the size of the burn; however, when the size of the burn exceeds 30% body surface area (BSA), the leaking capillaries involve all body tissues and not just the skin, resulting in a systemic inflammatory response. Burn oedema is a result of circulating inflammatory mediators including histamine, prostaglandins, leukotrienes and kinins that result in increased capillary permeability. This oedema is exacerbated by increasing capillary hydrostatic pressure, decreasing tissue hydrostatic pressure, and decreased plasma oncotic pressure (due to loss of albumin from the circulation).

General effects

The local and systemic inflammatory mediators released following a burn (particularly injuries greater than 30% BSA) result in profound systemic effects. Due to ongoing fluid losses, cardiac output falls due to decreased venous return, inadequate preload and afterload, and decreased myocardial activity. Due to the 'fight or flight' effects, the patient experiences a catecholamine rush of sympathetic activity that contributes to increasing systemic vascular resistance. Pulmonary oedema develops because of the systemic increase in capillary permeability as well as increasing pulmonary vascular resistance, left sided heart failure, hypoproteinaemia, direct vascular injury, and sometimes the added insult of an inhalational burn.

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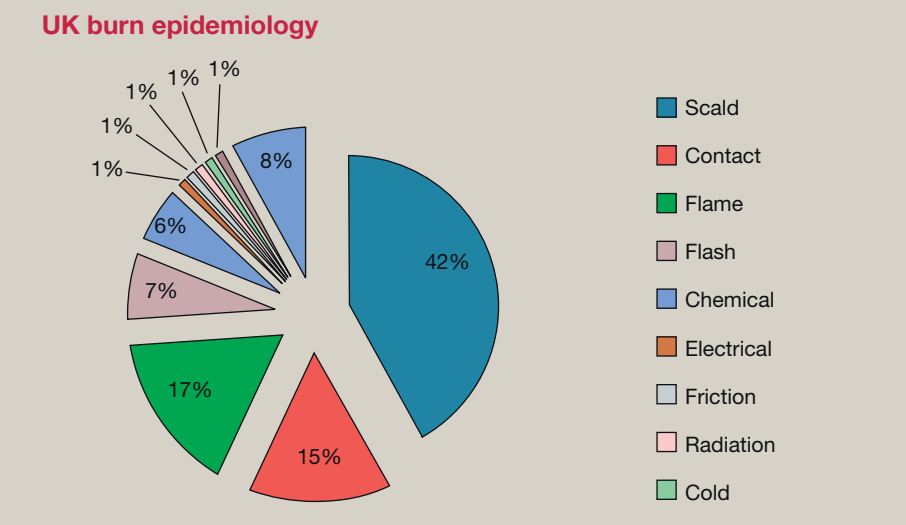

Figure 1

Other systemic effects following major burns include a significant increase in metabolism, nitrogen loss and poor temperature control due to loss of water and heat through the burnt tissue. The early cortisol rush following burn injury results in protein breakdown, gluconeogenesis, and impaired insulin release and glucose tolerance is seen. This catabolic state can last many weeks and months following burns and may result in ongoing weight loss in adults and impaired growth in children. The immunosuppressive effects of a burn are compounded by the weakened humoural and cellular responses following damage to the local circulation and the normal inflammatory process. This can cause increased risks of infection; the raw burn wound is an easy entry point for bacteria and yeasts. In addition, burns patients may lose the protective function of the gut following major injury, resulting in translocation of gut organisms into the circulation with increasing sepsis, morbidity and mortality.

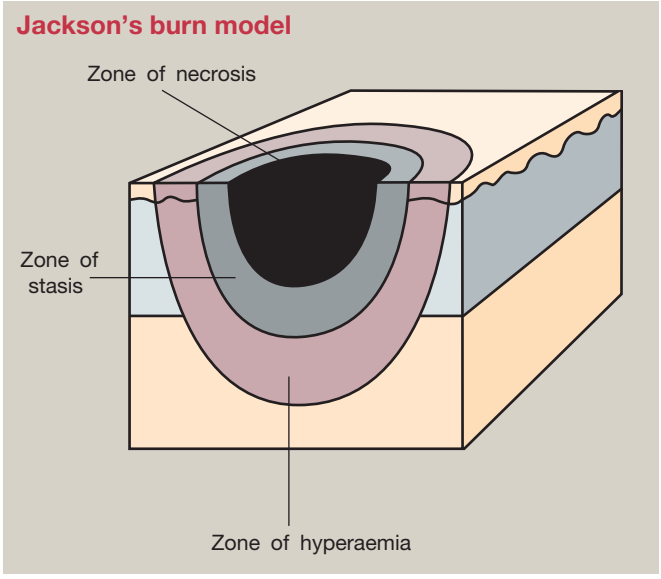

Figure 2

Diagnosis

History

The history of the injury and circumstances surrounding how the patient sustained their burn are important and can be crucial in predicting the depth of the injury and requirement for surgery. The time of the injury is also significant, as the appearance of the burn changes over subsequent hours and days. Knowing the exact cause of the burn is also key; scalding liquid, flame, explosion, contact, chemical and electric burns will all produce different injuries and this will need to be confirmed during the history and physical examination. The detail regarding the mechanism is important; freshly boiled water produces much deeper injuries compared to a cooled cup of tea. The location where the burn occurred can help predict severity; an explosion in a confined place such as a building or car, or if the patient required extraction from a burning house there will be a very high risk of inhalational injury. Information about first aid is useful, as appropriate first aid reduces the risk of needing a skin graft by 50%.[4] All burns should have cool running water applied for 20 minutes as soon as possible following their injury, but it is still effective up to 3 hours following the burn.[5] Medicated cooling gel sponges such as those carried by ambulances provide symptomatic pain relief to a burn but are not a substitute for running water first aid. Ice should not be applied as it can worsen the situation by causing a cold injury to the already damaged tissues.

Enquiries should be made regarding other injuries the patient may have sustained during the incident (be wary of road traffic accidents and fires where a patient may have jumped from a height to safety) and the presence of medical, surgical and psychiatric comorbidities. A drug history noting any allergies is required, as is tetanus status and tetanus toxoid should be given if unsure. A full social history should explore the patients' social and family circumstances, their employment, sports and hobbies along with any tobacco, alcohol and recreational drug use.

Examination, assessment, early management

The extent of the examination of the patient will depend on the size and severity of the burn. Where the history and initial

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