

**"SODIUM AND POTASSIUM
DERANGEMENTS IN SEVERE
BURNS IN THE IMMEDIATE
POST-BURN PERIOD."**

**DISSERTATION SUBMITTED IN PARTIAL
FULFILMENT OF REQUIREMENTS FOR THE
AWARD OF THE DEGREE OF MASTER OF
MEDICINE IN GENERAL SURGERY OF THE
UNIVERSITY OF NAIROBI**

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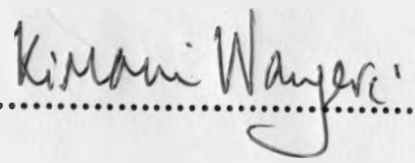
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DEDICATION

To my late father, Samuel, for being a beacon of hope throughout my formative years.

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LIST OF ABBREVIATIONS

AAS	-	Atomic absorption spectrophotometer
ABCDE		Airway, Breathing, Circulation, Disability and Exposure
ABSI	-	Abbreviated Burn Severity Index
ADH	-	Antidiuretic Hormone
TBSA	-	Body Surface Area burnt - expressed as a percentage
BU	-	Burns Unit
Cl ⁻	-	Chloride ions
CNS	-	Central Nervous System
CVP	-	Central Venous Pressure
ECF	-	Extra-cellular Fluid.
ECG	-	Electrocardiogram
FES	-	Flame Emission Spectrophotometry
GFR	-	Glomerular Filtration Rate
H ⁺	-	Hydrogen ion
HDU	-	High Dependency Unit
ICU	-	Intensive Care Unit
IL	-	Interleukin
ISE	-	Ion-Selective-Electrode
IV	-	Intravenous
K ⁺	-	Potassium ion
KNH	-	Kenyatta National Hospital
MRF	-	Mean Rehydration Factor
Meq/l	-	Milliequivalent per liter
Mmol/l	-	Millimoles per liter
Na ⁺	-	Sodium ion
NaCl	-	Sodium Chloride
RAAS	-	Renin- Angiotensin- Aldosterone system
SIADH	-	Syndrome of Inappropriate Antidiuretic hormone secretion
TBSA	-	Total Body Surface Area
UoN	-	University of Nairobi

DEFINITIONS

- Burn - coagulative destruction of body surface induced by physical heat or cold injury, chemical agents or electric current.
- Severe burn - a burn that is $\geq 15\%$ TBSA in children and $\geq 25\%$ in adults or $\geq 5\%$ TBSA full thickness burn and where there is inhalation injury irrespective of the TBSA burnt. High voltage electrical injuries are also defined as severe burns. In KNH, due to limitation of Unit capacity, TBSA of ≥ 20 and $\geq 30\%$ are the cut-off for children and adults respectively.
- Hypokalaemia - blood potassium less than 3.5mmol/L.
- Hyperkalaemia - blood potassium more than 5.0mmol/L.
- Hyponatraemia - blood sodium less than 135mmol/L.
- Hypernatraemia - blood sodium greater than 145mmol/L.

SUMMARY

This was a descriptive prospective study that aimed to assess the incidence of sodium and potassium derangements seen in severe burn patients in the first five days post-burn.

The study group comprised all patients with severe burns admitted to the Burns Unit (BU), High Dependency Unit (HDU) and Intensive Care Unit (ICU) of Kenyatta National Hospital (KNH) within 24 hours of injury.

60 patients who met the inclusion criteria and consented to be enrolled were interviewed, examined and serial blood analysis for sodium and potassium undertaken daily for 5 consecutive days. 53 patients survived the five days of investigation, with 4 patients dying on days 6 and 7.

Data on demographic characteristics, time of injury, time to admission, type of burn, percentage total body surface area burnt (%TBSA), predominant burn depth, amount of fluids given within 24 hours post-burn and presence of inhalation injury was collected. The mean rehydration factor (MRF) was calculated. The derangements observed were correlated with gender, age, mean rehydration factor, time delay to admission, type of burn and burn severity. Outcome at day 7 was assessed. This outcome was in terms of survival.

The male: female ratio was 1:1.08. Open flame burns were most common (75%), followed by scalds (18%). Electrical and chemical burns were seen in 5% and 1.7% respectively. Scalds caused mainly partial-thickness burns in contrast to full-thickness burns seen following open flame injuries.

Hypokalaemia and hyponatraemia were the predominant derangements seen. Hypokalaemia was noted in 33(62%) patients. Only 2 patients had episodes of hyperkalaemia. 45(85%) patients registered at least one episode of hyponatraemia as compared to 8 (15%) who did not. 8 patients registered hypernatraemia, all on

day 1. Age and mean rehydration factor were the most predictive of the two main derangements, hypokalaemia and hyponatraemia.

There were 11 deaths in week one, translating to a week 1 mortality of 18%. While those with hypokalaemia and hyponatraemia registered more deaths than those without the derangements, this did not attain statistical significance (p-values of 0.1397 and 0.9090 respectively). Electrolyte derangements did not seem to significantly influence survival. Presence of inhalation injury and high %TBSA were however associated with poor outcome (p-values 0.0329 and 0.0034 respectively).

It is very difficult to predict who will develop a certain derangement, but the young and those with low MRF should be closely scrutinised. Urea and Creatinine abnormalities were not seen in this study. The mean urea was 5.7 ± 2.6 mmol/L, while Creatinine was 76.8 ± 23.4 μ mol/L. There was no significant difference between those with hyponatraemia or hypokalaemia and those without in regard to urea and Creatinine.

LITERATURE REVIEW

Introduction

Burns are a major cause of preventable injury that is associated with significant morbidity and mortality^{1, 2, 3}. Burns can result from a wide range of insults to body surface. They are caused by extremes of temperature, corrosive chemicals or electric current¹. Depending on the offending agent, thermal burns are either scalds or open flame. The former may be splash injuries that tend to be superficial or immersion burns that are characteristically deeper and with clear demarcation from the normal tissue³.

The aetiology of burns varies with age. Scalds, usually from hot water, are the most common cause of burns.^{2, 4} Oil scalds tend to be deeper compared to hot water scalds. Mung'ara⁴ in his study on paediatric burns in KNH found scalds to account for 69.3% of burns. Gichuhi² found 56% and Okonjo⁵ 70.3% of burns in children to result from scalds. A striking contrast is the fact that scalding is not a significant cause of thermal injury in adults who suffer more of open flame and dry heat burns^{1,2,3,4,5}.

Exposed areas are less deeply burned compared to covered areas because clothing retains the heat and keeps the liquids in contact with the skin for a longer period. While most scalds are accidental, 5% of paediatric burn admissions are due to child abuse¹. These are identified when there is a discrepancy between the history by the care provider and the distribution and probable cause of the burn.

Burns may present as subtle injuries like sunburns or as life-threatening and dramatic injuries that demands expertise input to salvage life. In Kenyatta National Hospital (KNH), burn patients are managed as either in or out- patients depending on severity of the burns. Patients are either admitted to Ward 4D (General Plastic Surgery ward), Burns Unit (BU), High Dependency Unit (HDU) or intensive Care Unit (ICU).

Burn Pathophysiology

Our understanding of the pathophysiological characteristics of the burn injury has improved leading to better survival. More aggressive approaches to resuscitation are certainly one of the many factors that have contributed to this. Besides sepsis, multiple organ failure (MOF) is the most frequent cause of death and its prevention has played a major role in the declining mortality rate¹⁰.

Current clinical experience suggests that severe burn shock, shock refractory to all resuscitative efforts, and sepsis are the most important factors contributing to MOF during the early period post-burn^{1, 10}. The need for close monitoring of kidney, liver, lung and heart functions to detect slight irregularities that might indicate the development of MOF is thus crucial. It is important that hypovolaemia, inadequate perfusion, hypoxia, disorders of electrolytes, acid-base balance, anaemia and hypoproteinaemia are corrected or improved in order to prevent MOF^{1,3,7,8,10}.

The body's response to injury, including burns, is complex, integrated and designed to restore homeostasis and heal the wound as rapidly as possible. It is often a well co-ordinated process. Burns cause coagulative necrosis of the skin and underlying tissue to variable depth. Besides the skin, the effects of burns are manifested in virtually all the organ systems depending on the severity.

Perhaps the most dramatic effects of a burn are noted in the cardiovascular system. Achieving haemodynamic stability in this system thus assumes treatment priority to limit volume deficits and prevent the development of the dreaded burn shock.^{1,6,7} Direct thermal injury as well as liberation of vasoactive substances from the burn area induces changes in microvasculature in these areas and those remote to the burn.

The initial change is the increase in capillary permeability, which leads to loss of the normal compartmentalization of body into intravascular and extravascular spaces. There is massive shift of intravascular fluid to the extravascular space leading

to oedema. This further destabilises the Starling's forces that maintain normal equilibrium. The magnitude of these volume shifts is proportional to the extent of the burn and clinically evident in oedema.^{1, 7 and 8}

The shift results in significant shrinkage of the blood volume and the body reacts to counter this by causing redirection of the available blood with priority circulation going to the brain and the heart. All these changes in homeostasis are a result of a cascade of events largely mediated by products of inflammation and other neuro-humoral products that are released by inflammatory cells, injured tissues and hypothalamo-pituitary-adrenal axis among others.^{9, 11,12,13,14}

The most frequently incriminated products are histamine, arachidonic acid metabolites (thromboxane A2 and leukotrienes), substance P, activated proteases and platelet activating factor. They cause increased capillary permeability. Cells of the immune system are not spared either; they produce factors that activate complement, lysosomal enzymes, increased xanthine oxidase activity, free oxygen radicals, activated killer lymphocytes and lymphokines (Interleukin 1, 6 and 8).^{1, 11}

While these factors are meant to be beneficial, they are at times deleterious especially when produced in excess¹³. The micro-vascular derangements are maximal at 24 hrs and gradually regress thereafter. This is paralleled by the onset of oedema that starts immediately after injury, and reaches peak at 3-24 hrs post-burn.

Loss of normal skin integrity also leads to massive loss of fluids via the wounds. Evaporative loss is also significantly high. This is more severe in paediatric patients who have a large body surface area to body mass ratio. Besides, children have proportionately higher body water as compared to adults. A child's skin is also relatively thinner and hence tends to suffer deeper burns than young adults.^{1, 7, 8}

The diminished blood volume and cardiac output cause a post-burn decrease in renal blood flow and glomerular filtration rate (GFR). If this is uncorrected, acute renal failure sets in. The hypovolaemia may also impair flow to other organs as well

for example gastrointestinal tract accounting for the well-known Curling's ulcers and the malabsorption so common in acute burns.

All these changes in body fluid homeostasis affect the electrolytes balance that is delicately regulated by interplay of various organ systems. Key amongst these electrolytes is sodium and potassium. Thus all resuscitative efforts must be geared to restoration of not only adequate volume but also correct electrolyte concentration in plasma.

At the cellular level, there is loss of normal trans-membrane potential partly due to dysfunction of the $\text{Na}^+\text{-K}^+\text{-ATPase}$. Na^+ shifts to the intra-cellular space with water following. If progressive, there is swelling of the cell with eventual rupture. The sick cell concept attributes osmolality changes to isosmotic redistribution of solutes (from cells to extra-cellular space) caused by an abrupt increase in cell permeability, sustained dilution with no osmolality changes and to a widespread impaired capability of cells to maintain a normal content of non-diffusible solutes. This also occurs in areas remote to the burned tissues and accounts for some of the electrolyte derangements seen¹⁵.

Initial Burn Management

Over the years, there has been tremendous improvement in outcome for burn patients. In the last 10 years or so, the main factor that has helped improve outcome is care of burn patients before they arrive in burn care centres.

Like all the trauma patients, the initial step is removal of the patient from danger. This must be done with care to both the victim and the rescuers especially where electrical burns are involved.

The A-B-C-D-E of trauma is applicable even in burn patients.^{1, 16} The first priority must be maintenance of a patent airway, effective ventilation and support of the systemic circulation. Endotracheal intubation and liberal oxygen supply is offered to those who have suffered severe burns or where there is any suspicion of an

inhalational injury. An easy intubation may convert to a difficult or impossible intubation with development of burn oedema, creating a need for nasotracheal intubation or tracheostomy, both associated with a higher complication rate. With a secure airway, effective ventilation and adequate circulation, attention is focused on associated injuries, some of which might be life-threatening e.g. perforated viscus and closed head injuries. Fully exposing patient and doing a quick thorough examination is mandatory. This must be done with care to prevent hypothermia.

History of circumstances of the injury, pre-morbid conditions, medications, presence or absence of inhalation injury is recorded. At this time if properly trained personnel are available, a large IV access is fixed and fluid therapy initiated.^{16, 17} Referral to hospital or burn care centre must be by the quickest route possible. In advanced countries, it is recommended that distances greater than 200km are best covered by use of rotary blade or fixed wing planes whenever possible. Patients must be kept warm while on transit and wounds covered with clean sheets.

Single dose tetanus prophylaxis¹⁸ and pain management by intravenous opiates are recommended. Subcutaneous and intramuscular injections are absorbed variably. Poor management of pain may contribute to patient discomfort, dissatisfaction, delayed healing, and prolonged hospitalization. Although eliminating all pain is not a realistic expectation in acute burns, an aggressive approach to pain management may reduce such pain.

Pain relief is of paramount value in burn care. Besides pain, burn patients are very anxious and hence, every worker must bear this in mind. The hypovolaemia and its effect on peripheral circulation preclude oral and intra-muscular routes of analgesic administration in the immediate period. Patients are usually given opiates by intravenous route for effective pain management.

Once in a hospital, repeat assessment is performed and the patient referred to appropriate centre or wards. An assessment of burn severity, depth and extent is performed then. Escharotomies of circumferential limb burns or on the chest are performed as emergency procedures to aid distal circulation and breathing.¹⁹

Burn wounds require cleaning, removal of adherent clothing, dirt or foreign material by gently rinsing with warm water, preferably sterile. Small blisters may be left unbroken. If a full thickness burn encircles the trunk or limbs escharotomies are needed.

Antibiotics may be indicated in treatment of some burns, which are contaminated and severe. Use of topical antibiotic in wound management has definitely reduced the incidence of burn sepsis.²⁰ Silver sulphadiazine is widely used in Kenyatta National Hospital. The silver is an irritant to the eyes and mucous membranes and hence Tetracycline ointment is used for facial burns in KNH.

Some topical disinfectants like 1% povidone iodine have fallen from favour, being reserved for infected wounds, and even then only for cleaning and not dressing. They tend to absorb water (dehydrating the skin) and may impact negatively on fluid management inducing hypernatraemia.²⁰

Burn assessment and risk stratification are important in planning for management. This is influenced by factors such as age of patient, burn surface extent, burn depth, presence of inhalation injury and whether serious co-morbid medical conditions are present. In KNH, burns $\geq 20\%$ and $\geq 30\%$ TBSA in children and adults respectively are considered severe and admitted to specialised Burns Unit or HDU and ICU.

Assessment of Burn Depth

The skin is composed of an epidermis and dermis that rests on a subcutaneous tissue. The thickness of living epidermis is relatively constant throughout the body but the dead epidermis varies from place to place, being thickest in the palms and soles. The thickness of the skin is therefore determined by the thickness of the dermis.²¹ It is thinnest in the eyelids and genitalia but thick in inter-scapular region. Older patients and young children by virtue of their thin skin suffer relatively deeper burns as compared to adults when exposed to equal degree of thermal insult. Besides, this is compounded by their inability to escape or move away from fires.

Burn depth is the depth of necrosis into the skin or subcutaneous tissues. This is not constant and may change with time. There are many methods that can be used to assess burn depth including; Biopsy, staining with vital dyes, fluorescent fluorometry, laser doppler flowmetry, thermography, ultrasound, nuclear magnetic resonance and light reflectance¹.

The standard technique has been clinical observation of wound. However, it must be appreciated that a burn is a dynamic process for the first few days. Besides the kind of topical wound care can dramatically change the appearance of a wound. Most of the above listed techniques are expensive, not reproducible and time consuming. The surgeon's critical eye is thus important in determining burn depth and hence the decision for early excision and grafting.

Classification of Burn Depth

1. Superficial Burns

(a) 1st degree

Involve only epidermis. Clinically appears dry and red, blanches with pressure and heals by re-epithelization in 3-6 days with no scarring.

(b) 2nd degree superficial

Involve epidermis and upper dermis. Wound has blisters which when broken the burn appears red and moist, and blanches with pressure. They are exquisitely painful, heal in about 2 weeks with no scarring but pigmentation may delay or fail to take place fully.

2. Deep burns

(c) 2nd degree deep

Involve epidermis and much of the dermis (up to 2/3). Blistering may occur. Wounds appear wet or waxy dry, have patches of white to red and do not blanch. They heal with scarring if not excised and grafted early.

(d) 3rd degree burns

The whole of epidermis and dermis is involved. Skin appears white or leathery, grey to char black, dry and inelastic. No blanching occurs with pressure. They are relatively painless and should be grafted for healing to occur.

(e) 4th degree burns

Involve subcutaneous tissue, muscles, bones and / or organs. They are common in open flame burns in elderly and epileptics. Electrical burns also cause such severe damage. There is a potential risk for myoglobin production, which may clog the small tubules in the kidney predisposing to acute renal failure.

Burn Surface Extent

There are various charts or methods for the estimation of burnt surfaces. They are often expressed as a percentage of the total body surface area (%TBSA). They are only estimates and are not very accurate.

Wallace's rule of nine is often used in adults. It allocates 9% to the head and neck, and each upper limb, 18% to each lower limb, anterior trunk and posterior trunk. The perineum is estimated at 1%. This rule is not used for paediatric patients because they have relatively bigger surface area for the head as compared to the limbs. Due to this variation in body morphology with age, the Lund and Browder charts (appendix 1) are preferred for children. Kenyatta Hospital has adopted the Lund and Browder charts for estimation of %TBSA.

Inhalation injury must be sought and management initiated for it impacts negatively on survival²². Diagnosis of inhalation injury is largely clinical but this is augmented by laboratory work-up.

Features that are suggestive of inhalation injury include;

- History of burn in a confined space
- Facial burns or burns with burnt vibrissae
- Cough, wheezing and dyspnoea

- Excessive production of carbonaceous sputum
- High carboxy- haemoglobin level in arterial blood
- Bronchoscopic findings of laryngeal oedema and the cherry red appearance of carbon monoxide poisoning
- Loss of consciousness in the absence of head injury

A near accurate assessment of body surface area burnt (%TBSA) is essential as resuscitation is guided by this factor in most formulae. Besides, the use of %TBSA reflects the amount of damaged tissue, which directly correlates to the degree of fluid, and electrolyte demand that is imposed on the patient.

Burn Severity Assessment

Burns severity takes into account the age of the patient, total burnt surface area, burn depth, presence or absence of inhalation injury and whether there are co-morbid medical conditions. The American Burn Association grading is widely used for this purpose. Here burns are classified as minor, moderate and severe¹.

1) Minor Burns

- i. Involves less than 15% TBSA in adults and 10% in young or patients older than 50yrs.
- ii. Less than 2%TBSA full thickness burn

2) Moderate Burns

- i. 15-25% TBSA in adults
- ii. 10 – 15% TBSA in the young or old
- iii. 2-5% TBSA is full thickness burn
- iv. Suspected inhalation injury
- v. Co-morbid medical problem
- vi. Circumferential burns

3) Severe Burns

- i. 25% TBSA – adults
- ii. 15% TBSA in young and old
- iii. 5% TBSA full thickness
- iv. High voltage electrical injuries
- v. Known inhalation injury

Fluid Resuscitation

Fluid resuscitation is a cornerstone in burn management^{1, 8}. The marked improvement in burn patient survival over the years is a result of a sound understanding of the burn patho-physiology and attendant aggressive and timely fluid management. To achieve this goal, many workers have formulated many regimes, all geared to the provision of adequate fluid to produce acceptable urine output.^{1, 7,8,23}

The various formulae vary in the fluid composition, amount, rates of infusion and the timing of administration. However, most seem to take into account the severity of the burns (%TBSA), weight of the patient and for some, age of the patient. For purposes of standardization, the Parklands formula (Table 1) is adopted for use in KNH. Other formulae used in other centres include Evans, Brooke Amy, Modified Brooke and Slater (Table 2).

Much of the debate is on the choice between crystalloid and colloid fluids and their timing. Proponents of the Parkland formula in which Ringers Lactate is used in the first 24hrs see no benefit from colloids arguing that with the altered permeability, the colloids are unlikely to be retained in intravascular space any more than the crystalloids. The lactate is thought to be beneficial since on metabolism, bicarbonate is generated which is used by the body to correct the attendant metabolic acidosis. After restoration of normal capillary function, beyond 24 hrs, colloids are used to maintain plasma volume.^{1, 8, 23}

Colloids include albumin, plasma, starches, dextrose and gelatins. They are expensive (relative to crystalloids), may predispose to pulmonary and peripheral oedema. They may also induce anaphylactic reactions²⁴.

There are proponents for use of hypertonic solutions who think that these solutions help in reduction of total volume of infusate, which may be beneficial in cardiac patients, or patients with reduced pulmonary reserve. Indeed, addition of hypertonic saline and protein infusions along Ringers Lactate in the first 24 hrs may result in 30% decrease in fluid requirements^{1, 25, 26, 27}.

Patients with documented inhalation injury require additional fluids. In KNH, 10% is added to estimated BSA during calculation of fluid requirements. Some workers have estimated fluid requirements in patients with inhalation injury at 5.7mls / kg/ % TBSA.^{1,27,28,29} The burned child represents another special challenge, because resuscitation must be more precise than for an adult with similar burn. Children have a limited physiological reserve. They require proportionately more fluid for burn shock resuscitation than adults with similar thermal injury; fluid requirements average 5.8-mL/kg/%burn.^{1,23}

Fluid formulae act only as guidelines, and resuscitation must be guided by individual patient response. Factors like age, presence or absence of inhalation injury, time to intravenous access and start of fluids, pre-morbid patient status and institutional guidelines influence fluid management to a great degree. Indeed, some workers have faulted the Parklands formula for inadequate fluid resuscitation especially in children who need a relatively bigger maintenance volume.

Urine output monitoring provides a cheap and easy way of fluid monitoring. The target urine output is influenced by the age of the patient and the cause of burns. In children, a urine output of 1ml/kg/hr is considered adequate, while for adults it is 30-50ml/hr. In electrical burns, the fluid resuscitation should target double the urine output i.e. 2mls/kg/hr and 100mls/hr in children and adults respectively. This is because of potential deleterious effects of myoglobin on the kidneys following electrical injury^{1,8,19,23,27}. The use of diuretics precludes urine assessment as a means of assessing adequacy of resuscitation.

Where possible, other parameters like serial weight measurements and pulmonary capillary wedge pressure assessment can be used. The latter is only helpful if the patient does not have attendant cardiopulmonary disease. While the need for adequate fluid resuscitation is obvious, caution must be exercised not to over-hydrate patients. This may lead to pulmonary oedema and abdominal compartment syndrome. Indeed, some workers have suggested monitoring of intra-abdominal pressure to prevent abdominal compartmental syndrome³⁰.

Table 1**PARKLANDS FORMULA**

Time After Burn	Type of Solution Used	Administration Rate	Urine Output
First 24 Hours	Ringers' Lactate 4mls/Kg/%TBSA	½ in First 8 Hours Then ½ in the next 16 Hours	50-70mls/hr
Second 24 Hours	5% Dextrose; For replacing evaporative loss Colloid; Used to maintain plasma volume	Maintenance Fluid	30-100mls/hr

Table 2**OTHER FORMULAE USED IN FLUID RESUSCITATION ON DAY ONE¹²**

Formula	Crystalloid	Colloid	D5W
Parklands	Ringers' Lactate 4mls/Kg/%TBSA	None	None
Evans	Normal Saline 1ml/Kg/%TBSA	1ml/Kg/%TBSA	2000mls
Brooke Amy	Ringers' Lactate 1.5mls/Kg/%TBSA	0.5ml/Kg/%TBSA	2000mls
Modified Brooke	Ringers' Lactate 2mls/Kg/%TBSA	Not used	None
Slater	Ringers' Lactate 2 L/24hours	FFP 75mls/Kg/24 Hours	None

A mean rehydration factor can be calculated at 24 hours post-injury if the amount of fluids given is known. Thus;

Factor of rehydration = Amount of fluids given at 24 hours (mls)

Calculated fluid needs in mls ($4 \times \%TBSA \times \text{Weight in Kg}$)

It would be expected that if the Parklands formula is followed, the factor would be 1. Values less than this may be considered inadequate resuscitation.

Sodium And Potassium Metabolism

Sodium is the major cation of the extra-cellular fluid. Because it represents 90% of the approximately 154mmol of inorganic cation charge per litre of plasma, Na^+ is responsible for almost one half of the osmotic strength of plasma, its regulation thus is central in maintaining the normal distribution of water and the osmotic pressure in the ECF compartment.

The daily intake averages 8-15g (130-260 mmol/l) of sodium chloride (NaCl), all almost completely absorbed. The body requires only 1-2 mmol/day and the excess is excreted by the kidneys. Sodium is freely filtered in the glomerulus. 70-80% of the filtered sodium is reabsorbed in proximal tubules with chloride (Cl^-) and water passively following for electrical neutrality and iso-osmotic equilibrium respectively. Another 20-25% is reabsorbed in the loop of Henle along with Cl^- and more water.^{31, 32}

In the distal tubules, interaction of the adreno-cortical hormone aldosterone with the coupled Na^+/K^+ and Na^+/H^+ exchange systems directly results in Na^+ reabsorption of the remaining 5-10% of the filtered load. It is the regulation of this latter fraction of filtered Na^+ that determines the amount excreted in urine.

Disorders of Na^+ homeostasis can be due to;

- (a) Excessive loss, gain or retention of Na^+ or
- (b) Excessive loss, gain or retention of water

The delicate Na^+ balance by the kidneys is aided by the presence of receptors in the Juxtaglomerular cells that sense decreased arteriolar pressure or decreased Na^+ concentration in filtered fluid thus stimulating renin production. Through activation of the renin-angiotensin-aldosterone system (RAAS), the latter facilitates sodium reabsorption at the expense of K^+ ⁹. Water regulation in the kidney occurs from the distal tubule through the collecting duct where tubular permeability to water is under the influence of antidiuretic hormone (ADH).^{31, 32, 33}

ADH is secreted from the posterior pituitary under influence of baro-receptors in the aortic arch and hypothalamic osmoreceptors. When blood volume is reduced as occurs following burns, ADH helps in conservation of water by increasing reabsorption by the nephrons¹². The converse occurs in hypervolaemia or decreased osmolality (reduced Na^+) favouring water diuresis. The body's only other sodium and/or water regulation is by ingestion of water. Thirst is stimulated by either vascular collapse or hyperosmolar states.

One would therefore be correct to deduce that where one has no access to free water, the sodium levels may rise while the volume shrinks. This is typified by patients with severe burns and more so those with inhalation injury. These patients are often intubated precluding oral ingestion of free water. Some patients have burns on the upper limbs and may not have easy access to free water. Besides, health workers often bar burn patients from taking free water- and wrongly so³⁴.

Hyponatraemia manifests clinically at values below 120mmol/l. Central nervous system (CNS) symptoms are predominant. They are due to intracellular shifting of water to maintain osmotic balance, resulting in swelling of CNS cells. The rapidity of the development of hyponatraemia influences the levels of Na^+ at which symptoms develop. Possible causes of hyponatraemia include;

1) Extra- renal loss

- a) Vomiting and diarrhoea.
- b) Skin loss as in burns and sweating.

2) Renal loss

- a) Diuretics.
- b) Mineralocorticoid deficiency.
- c) Metabolic alkalosis.
- d) Proximal renal tubular acidosis.
- e) Carbonic anhydrase inhibitors.
- f) Polycystic kidney disease.

3) Syndrome of Inappropriate ADH Secretion (SIADH)

4) Others

- a) Hypothyroidism
- b) Congestive Cardiac Failure
- c) Cirrhosis
- d) Nephrotic syndrome

Hypernatraemia, plasma $\text{Na}^+ > 150\text{mmol/l}$, presents with tremors, irritability, ataxia, confusion and coma. This is due to intracellular dehydration. In many cases, underlying conditions that contribute to the development of hypernatraemia may mask the symptoms of hypernatraemia. Indeed, most cases of hypernatraemia occur in patients with altered mental status or infants; both of whom may have difficulty in rehydrating themselves despite a normal thirst reflex.

In general hypernatraemia occurs due to;

- (i) Excessive water loss or failure to replace normal water losses
- (ii) A net Na^+ gain in excess of water e.g. use of large volumes of sodium bicarbonate to correct acidosis^{35,36}.
- (iii) Decreased Na^+ excretion.

Thus, the assessment of total body water status by physical examination is important in establishing a diagnosis of hypernatraemia.

Total body potassium of a 70kg subject is 3.5 mol, (40-50 mmol / kg), of which only 1.5 – 2% is present in ECF. The body requirement of K^+ is satisfied by a dietary intake of 50-150 mmol/day. Almost all is absorbed and a little of it get to the cells, the rest being excreted by the kidneys. Aldosterone enhances K^+ secretion and

Na⁺ reabsorption in the distal tubules via the Na⁺/K⁺ exchanger. The tubular system responds almost immediately to K⁺ loading by increased renal loss. In contrast, the tubular response to conserve K⁺ in instances of depletion is very slow in the initial stages and may take up to a week to peak.

Fortunately, intracellular stores remain adequate to maintain near-normal K⁺ concentration in the ECF. For the above reason, K⁺ depletion is often an early consequence of restricted K⁺ intake or of unusual losses of K⁺ by extra renal routes. K⁺ retention occurs in chronic renal failure due to reduced tubular flow. Acid - base disturbances also affect K⁺ levels due to competition of Na⁺ and H⁺ for excretion in the distal tubules besides its effects on intracellular shift that occurs in acidosis³⁵.

Burns present the body with a big challenge on electrolyte balance via the interplay of several factors amongst them;

- a) Loss of water and electrolytes via wounds
- b) Shifts of fluid / electrolytes between various body compartments.³⁵
- c) Type, amount and rate of infusion of resuscitation fluids
- d) Hormonal response to shrunken plasma volume e.g. RAAS, catecholamines,
- e) Altered fluid intake e.g. patient's inability to normally respond to thirst reflex³⁴.
- f) Drugs used in burns e.g. K⁺ containing antibiotics, mannitol and other diuretics used.
- g) Extra renal electrolytes and water loss due to vomiting and diarrhoea that may follow severe burns³⁷.
- h) Increased insensible loss following pyrexia that is common in burns³⁵.

Electrolytes Disturbances In Burns

The world over, burns have been studied extensively. However, there appears to be paucity of literature on electrolyte disturbances in burn patients. There is no much literature that specifically addresses the incidence of electrolyte disturbances in burns. There are several studies done in KNH touching on various aspects like epidemiology and mortality⁵, childhood thermal injuries³⁸, burn infection³⁹, burn prevention² and post-burn contractures⁴⁰. Nthumba attempted to validate the ABSI.^{41,}

42

Among the latest studies was one by Mogire who looked into fluid resuscitation in severely burnt adult patients admitted to KNH⁴³. Among his key observation was that there was inadequacy of fluid resuscitation in the first 24 hrs-mean rehydration factor of 0.54 against the expected of 1. There were at least 4 deaths out of 46 patients (8.6 %) in the first 48 hrs⁴³. Gichuhi found a median time delay of 12 hours, mean of 5.4 hours, before fluids were started for patients admitted in KNH². Many deaths in the resuscitative phase are either due to fluid and electrolyte disturbances or inhalation injuries.

Mohammed et al looked at hypernatraemia in septicemia and non-septicemic patients and concluded that hypernatraemia may be used as a marker of sepsis in burn patients³⁴. Ramos noted that understanding of burn pathophysiology have resulted in improved patient survival due to appropriate and timely fluid resuscitation⁴⁴. He listed hypernatraemia, hyponatraemia, hyperkalaemia, hypokalaemia, hypocalcaemia, hypomagnesaemia and hypophosphataemia as possible complications of burns.

Electrolyte balance may be a useful indicator of clinical well being in trauma and post-operative patients and persistent derangements may represent emergent or continuing sepsis, tissue ischemia, or other complications other than primary disturbances of electrolyte balance; action must thus be directed to correcting the underlying disorder, not the imbalance¹⁵.

As earlier alluded, burns present a major challenge in fluid and electrolyte balance. One may consider electrolytes balance in burn patients in 3 periods⁴⁴.

(a) Initial resuscitation period – (between 0-36 hours)

Intravascular volume is lost in burned and unburned tissues due to; increased vascular permeability, increased interstitial osmotic pressure in burn tissue and cellular oedema^{1, 3, 7, 8}. A modest hyponatraemia ($\text{Na}^+ < 135\text{mmol/l}$) occurs in this period due to extracellular sodium depletion following changes in cellular permeability. This is compounded by the infusion of large volumes of lactated Ringers Solution, which is hypotonic with respect to sodium. It requires no specific treatment as evaporative water loss corrects the imbalance. It may persist or be exaggerated in patients with 0.5% silver nitrate soaks.²⁶ SIADH secretion is a rare cause of generally asymptomatic hyponatraemia and usually responds to reduction in amount of administered electrolyte free fluid.^{45,46}

Severe hyponatraemia that is symptomatic is not common^{44, 45, 46}. The symptomatology is largely neurological and occurs because of a shift of water intracellularly leading to brain cell swelling and cerebral oedema. It is characterised by altered consciousness, seizures, cerebral oedema, coma, anorexia, nausea and vomiting, cramps or weakness may be noted in neuromuscular system⁴⁵. Such hyponatraemia is addressed by infusion of normal saline and sodium lactate.

Release of potassium from red blood cells and other damaged tissues commonly produces a modest hyperkalaemia ($\text{K}^+ > 5.5 \text{ mol/l}$) during this period. This often requires no specific therapy. If acidosis due to inadequate resuscitation supervenes, the serum potassium concentration may be elevated to symptomatic levels. This is more common after electrical injuries. Hyperkalaemia particularly affects the cardiovascular system. The resting membrane potential is related to the ratio of ICF and ECF K^+ concentration. Hyperkalaemia partially depolarises the membrane. Prolonged depolarisation impairs membrane excitability that manifests as weakness. Amongst the changes it induces are; ECG changes of tall tented t-waves, widened QRS complex, depressed ST segment and disappearance of p waves, heart

block and diastolic heart arrest.^{1,32,45,46} Therapeutic options for treatment of hyperkalaemia include several steps;^{45, 46}

1. Reverse K^+ effects in cellular membrane with calcium chloride 10% (10 mls IV over 10 min)
2. Transfer extracellular potassium into cells with:
 - Glucose (250- 500 mls of 10% dextrose) + insulin (5-10 IU)
 - Sodium bicarbonate
 - Hyperventilation
3. Remove potassium from the body by:
 - Diuretics
 - Potassium exchange resins
 - Haemodialysis in severe cases

(b) Early Post-resuscitative Period (day 2-6)

During this period hypernatraemia, hypokalaemia, hypocalcaemia, hypomagnesaemia, and hypophosphataemia may be noted.

Hypernatraemia ($Na^+ > 155\text{mmol/l}$) is associated with dehydration due to inadequate replacement of insensible water loss.³⁴ The water vapour barrier of skin is destroyed in burns and evaporative water loss is markedly elevated. The amount of insensible loss may be estimated by a formula;

$$\text{Insensible loss (mls/hr)} = (25 + \%TBSA) \times TBSA (m^2)$$

Hypernatraemia in this period follows intracellular sodium mobilization, reabsorption of burn oedema and urinary retention of sodium (because of increase in renin, angiotensin and ADH).^{7, 23, 34} Patient presents with; peripheral oedema, ascites, pleural effusion and interstitial/ alveolar oedema or more significantly with excessive thirst. The degree of hypernatraemia is proportional to the %TBSA. Inhalation injury and pyrexia may induce hypernatraemia.³⁴

Administration of blood, plasma, and albumin, sodium bicarbonate to counter acidosis, Total Parenteral Nutrition solution and antibiotics may all increase sodium level. Other possible causes of hypernatraemia include; osmotic diuresis induced by glucosuria, urea diuresis, underlying diabetes mellitus, diabetes insipidus and sepsis.

Hypernatraemia is treated by administration of hypotonic fluids or even free water. Such fluids include NaCl 0.45%. The correction must be gradual not to exceed 1.5 mmol/hr because of the risk of inducing cerebral oedema.^{45, 46}

Hypokalaemia ($K^+ < 3.5$ meq / l) may occur as a consequence of:

- 1) Potassium losses.
 - a. Urinary - diuretics, kaliuretic effect of mafenide acetate
 - b. Gastric - diarrhoea due to enteral feeding
 - c. Transechar K^+ loss - exaggerated by 0.5% silver nitrate²³
- 2) Intracellular shift of K^+ e.g respiratory alkalosis due to hyperventilation for patients on mechanical ventilation
- 3) Use of K^+ depleted fluids.^{1,3,23,27}

Symptoms can affect several organs with greater effects on the heart. It enhances the arrhythmogenic effects of catecholamines, digoxin and calcium.^{32, 45, 46} Potassium deficits are corrected by intravenous administration of potassium chloride. This is best done under electrocardiogram (ECG) monitoring whenever possible.

(iii) Hypocalcaemia may be noted in this period. This is partially attributable to the attendant hypoalbuminaemia. Due to extra load of fluid infused there is a dilutional effect causing hypoalbuminaemia. There is also reduced hepatic synthesis of proteins. True hypocalcaemia however occurs due to increased renal excretion of calcium, trans-eschar losses and sustained metabolic acidosis^{35, 47}. Hypocalcaemia is corrected by administration of calcium chloride or calcium gluconate.

(iv) Hypophosphataemia is often common in burn patients during week one^{44, 47}. This occurs in association with elevated catecholamine levels, infusion of glucose containing fluids, respiratory alkalosis in hyperventilating patients, phosphate binding by antacids used to treat Curling ulcers, and initiation of high calorie nutritional support.

(C) Inflammation/ infection period (Hypermetabolic period)-beyond week one

During this period several imbalances may coexist. Each disturbance is addressed on its own merit. The most common is hypernatraemia associated with burn sepsis³⁴.

Laboratory Assay of Sodium And Potassium

Serum, plasma or whole blood may be used for Na^+ and K^+ determination.

Specimens may be obtained by venipuncture into evacuated tube, capillary samples, and heparinised arterial / venous samples. There are minor differences for values of these analytes between arterial and venous samples but only the differences between serum and plasma K^+ can be considered significant⁴⁸.

Use of plasma or whole blood is advantageous in expediting the assay because it is not necessary to wait for clotting to occur. Furthermore plasma or whole blood has a distinct advantage in determining K^+ concentration, which is higher in serum in a manner that is dependent on platelet count. Haemolysis causes release of K^+ resulting in elevated K^+ reading. Marked haemolysis may raise K^+ by as much as 30%. Platelet rupture during the coagulation process accounts for the higher K^+ in serum than plasma. This may be to a value of 0.1 - 0.7 mmol/l. This range is dependent on the platelet count^{48,49}.

Frozen specimens (whole blood) results in impaired glycolysis and thus affect Na^+ K^+ ATPase activity leading to shift of intracellular K^+ to the extracellular fluid. The increase of K^+ in serum is of the order of 0.2mmol/l in 1.5 hr at 25degrees Celsius and as much as 2mmol/l increase after 5hrs at 4deg.celsius. The converse is true with K^+ level decreasing with increasing temperature due to increased glycolysis. This is however biphasic for as glucose substrate gets exhausted, K^+ leaks from the cells. To counter these effects, K^+ determination is best done immediately specimens are taken and at standardized temperatures.

Sodium may be determined by Atomic Absorption Spectrophotometry (AAS), Flame emission spectrophotometry (FES), electrochemically with a Na^+ - ISE (ion selective electrodes) or spectrophotometrically. Of these methods, ISE is often used. Excellent accuracy and coefficient of variation of less than 1.5% are readily achieved with modern equipment, reliable calibrators and a good quality assurance programme⁴⁸.

Potassium is determined by spectrophotometry, AAS, FES, or electrochemically with a K^+ ISE. Again, ISE is widely used with variations of <2% being achievable^{48, 53}. The ISE machine is available and used in KNH.

Reference Values^{32, 48, 53}

The following are widely accepted as the reference values for sodium and potassium in blood.

Sodium: 135 – 145 mmol/l

: 128 – 145 mmol/l (for premature new- born)

Potassium: 3.5 – 5.0 mmol / l for serum

: 3.3 – 4.5 mmol/l for plasma

Burn Mortality

There has been a lot of research on burn mortality and its predictors. Indeed, an Abbreviated Burn Severity Index (ABSI)⁵⁰ has been developed (appendix 2). The major determinants of survival are age, burn severity, presence of inhalation injury and to a less extent, gender.^{22, 43, 50, 51} Generally, the higher the %TBSA and burn depth, the worse the prognosis. Children under 2years and adults over 50 years have a poorer prognosis.^{52, 53} Elderly patients have slow reflexes, physical debilitation and pre-existing conditions⁵². Their skin is relatively thin and hence they tend to suffer deeper burns with attendant grave outcomes. Surprisingly, female gender tends to do worse than their male counter parts even after adjustment for burn severity⁵⁴.

The overall mortality for burns varies from centre to centre- depending on the level of development of burn care centres⁶. Some centres have quoted values as low as 4% and others as high as 22%^{51, 55}. Locally, Nthumba⁴¹ found a mortality of 100% for burns greater than 60% TBSA, and 78.6% deaths for burns between 41 and 50%. Okonjo⁴³ found 59% mortality in Burns Unit while Mogire⁴³ found 94% death in patients with inhalation injury. Mung'ara found an overall 3% mortality in paediatric thermal injuries in a study at KNH. Wanjeri³⁹ found a mortality of 87.5% with burns greater than 50%.

The value of pre-hospital care, proper initial management especially for fluids cannot be overemphasized; its impact on mortality is significant⁶. However, the outcome of burns must not be viewed solely as death or survival without consideration of the quality of life. It is very difficult to predict survival in burns. However, there is general concession that age of patient, burn extent and presence of inhalation injury are key determinants of survival.^{41, 50, 51, 52, 53} Besides, the presence of cumulative risks impacts negatively on survival.

STUDY JUSTIFICATION

Burns are a common and serious cause of injury in our setting, and severe burns are associated with an unacceptably high mortality rate as reported in Kenyan studies.^{4,5,38,39,41,43} A significant number of these deaths occur in the acute setting and are largely a consequence of electrolyte and volume disturbances⁴³. This is in contrast to experience in many specialised burn centres where mortality rate averages 4% in many series.⁵¹

As many as 13% of burn patients present with shock at admission in KNH.⁴ Out of 46 adult patients with severe burns (%TBSA range of 30% to 110%, mean 52.3%) followed by Mogire⁴³ in a study in KNH, only 6 survived, with 20 dying within one week. Electrolyte disturbances might have contributed to this high mortality. Mogire also demonstrated that our patients may not be receiving adequate fluid resuscitation, probably due to delayed presentation. Our Patients present late and this may impact negatively on electrolyte and fluid management.^{2,43} There are no local studies that look into electrolyte disturbances in burn patients.

This study hoped to at least address this dearth of information and hopefully make recommendations on areas that need improvement.

OBJECTIVES OF THE STUDY

BROAD OBJECTIVE

Determine the type of sodium and potassium derangements in severe burns seen in KNH.

SPECIFIC OBJECTIVES

1. Determine the type of electrolyte derangements seen in severe burns.
2. Determine effect of time delay in admission on the derangements observed.
3. Assess the influence of degree of burn on the derangements seen.
4. Determine the relationship between mean rehydration factor to the electrolyte derangements seen.
5. Relate the electrolyte derangement seen to the severity of the burn.

MATERIALS AND METHODS.

Study Design.

This was a prospective descriptive cross-sectional study.

Study Location.

Kenyatta National Hospital's Burns Unit, Intensive Care Unit and High Dependent Unit where severe burns patients are admitted.

Study Population.

All patients admitted to KNH with a diagnosis of severe burns and who met the inclusion criteria and consented to be enrolled in the study.

Inclusion Criteria

1. All patients admitted to KNH with acute severe burns viz
 - a. Adults - $\geq 30\%$ TBSA
 - b. Children- $\geq 20\%$ TBSA
 - c. Inhalational injuries irrespective of TBSA
 - d. High voltage electrical injuries.
2. Severe burns arriving in KNH less than 24hrs after the burn.
- 3 Eligible patients who consented to be included in the study.

Exclusion Criteria

1. Patients with burns not classified as severe
 - a. $< 30\%$ TBSA for adults
 - b. $< 20\%$ TBSA for children
 - c. Low voltage electrical injuries.
2. Patient presenting more than 24 hrs after the event
3. Patients who declined to participate in the study in spite of been eligible

Methodology

Patients were recruited from the ICU, HDU and Burns Unit upon admission. An explanation of the nature of study was given to the patient and / or the guardian. An informed consent was obtained for eligible patients. Those who consented were interviewed and examined by the investigator. Data was collected on a prepared questionnaire (appendix 4). Data pertaining to patient demographics, circumstances of the burn injury and time of injury was recorded.

Patients were examined to determine the burn severity in terms of %TBSA, burn depth and presence or absence of inhalation injury. The Lund and Browder charts (appendix 1) and clinical examination by the investigator were used for this purpose. Amount of fluid required was worked out using the Parklands Formula (table1). At 24hours from time of burn; the amount of fluid administered was recorded. This helped to work out the mean rehydration factor (MRF).

$$\text{Mean rehydration factor} = \frac{\text{Calculated Amount of fluids needs for 24 hours (mls)}}{\text{Actual amount of fluid given in 24hours (mls)}}$$

The expected normal rehydration factor would therefore be one if the patient received the calculated fluid needs.

Heparinized arterial or venous blood specimens were taken while observing standard safety precautions by the investigator. They were then transported by the investigator in sealed small cartons to the ICU Laboratory for analysis within 10 minutes of removal. This was done for 5 consecutive days for each patient. The analysis was carried out by a qualified Medical technologist with special training in the use of an Ion-Selective-Electrode machine. The machine is able to analyse sodium, potassium and blood gases but does not entail chloride analysis. Urea and creatinine were analysed on day five.

This machine offers an excellent accuracy and coefficient of variation of <1.5 and 2% for Na^+ and K^+ respectively. The expedited analysis of specimens eliminates a possible source of error due to time-dependent haemolysis of cells that give a falsely elevated K^+ reading. Specimens were immediately disposed off upon analysis.

The ICU laboratory has a quality control programme where standardized specimens are used to assess accuracy of results emanating from the laboratory. Besides, to further strengthen this process the investigator took every tenth patient's second specimen to the Renal laboratory for comparison of results.

The obtained results were interpreted thus; for sodium, the reference values was the internationally accepted 135-145mmol/L (both inclusive). Any value outside this was considered hyper- or hyponatraemia. Normal potassium level was all values between 3.5-4.5mmol/L (both inclusive). Values outside this were read as hypo- or hyperkalaemia.

The results were correlated to several factors viz; burn severity, mean rehydration factor, type of burn, time delay to admission, gender and age of patients. The patients were then revisited on day 7 to assess outcome in terms of survival – dead or alive.

Sample Size Determination

There is no previous study in KNH that has looked into the incidence of electrolyte derangements in burn patients.

Given a confidence interval of 95% and a level of precision of 5%, the Kish and Leslie Formula was used during this study for sample size determination.

$$N = \frac{Z^2 \times p(1-p)}{d^2}$$

N = sample size to be determined

p = estimated prevalence of severe burns

Z = standard errors of the mean corresponding to 95% confidence interval

d = Absolute precision (0.05)

Severely burned patients constitute 2.05% (according to hospital records for the year 2003) of all surgical patients admitted to KNH (Appendices 3a, 3b and 3c)

Burns Unit = 197 patients

ICU and HDU Burns = 26 patients

Total General surgical admissions = 10,675

Total = 10,898

Severe Burns = 223 = 2.05%

N = 30

Besides, on average the burns unit admits 90 to 150 patients per year. With the study period intended to be three months, a sample size of 30 was reasonable. A total of 60 patients were recruited due to extension of study duration by 3 months.

Sampling procedure

Consecutive sampling of patients who met inclusion criteria and consented was done until the calculated sample size was achieved.

Data Analysis

Descriptive statistics such as means, frequency distribution and standard deviation were largely used. Data analysis was by Epi info package version 3.3.2. Tests for significance using χ^2 and Fishers exact test were performed where applicable, in answering the objectives of the study, with p-values of <0.05 considered significant.

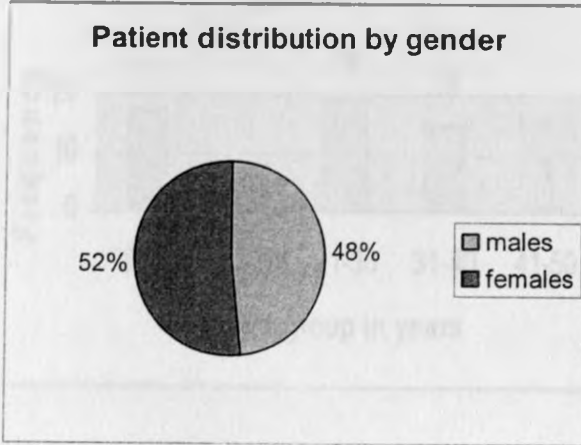
ETHICAL CONSIDERATIONS

- 1) Approval was sought from Kenyatta National Hospital Ethical Committee.
- 2) Informed written consent was obtained from patients or guardians before enrolment in the study.
- 3) Universal precautions were strictly observed while handling patients or the specimens.
- 4) Specimens were analysed immediately after removal and discarded upon analysis, making them inaccessible to any third parties.
- 5) All information obtained has remained confidential unless there is separate written approval from the patient or guardian for it to be shared.

RESULTS

A total of 60 patients with severe burns were recruited for the study. There were 29 males and 31 females, giving a male to female ratio of 1:1.08. 48 of the patients were admitted to the Burns Unit and the remaining 12 to the Intensive Care Unit. No patient was admitted to the High Dependency Unit.

Figure 1: Distribution of patients by gender.

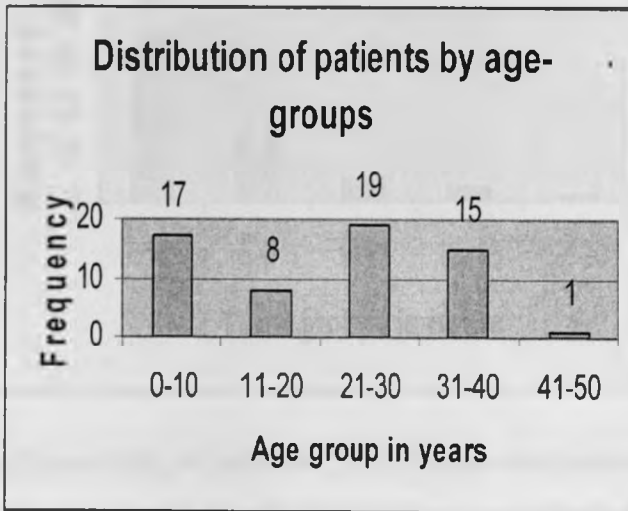


The patient's age ranged from 3 months (0.25 years) to 45 years with a mean age of 20years. The age distribution appeared bi-modal with peaks at 0-10 yrs and 21-30 yr age groups.

Table 3: Distribution of patients by age-groups

Age group in years	Frequency	Percentage
0-10	17	28.3
11-20	8	13.3
21-30	19	31.7
31-40	15	25.0
41-50	1	1.7
Total	60	100

Figure 2: Age-group distribution

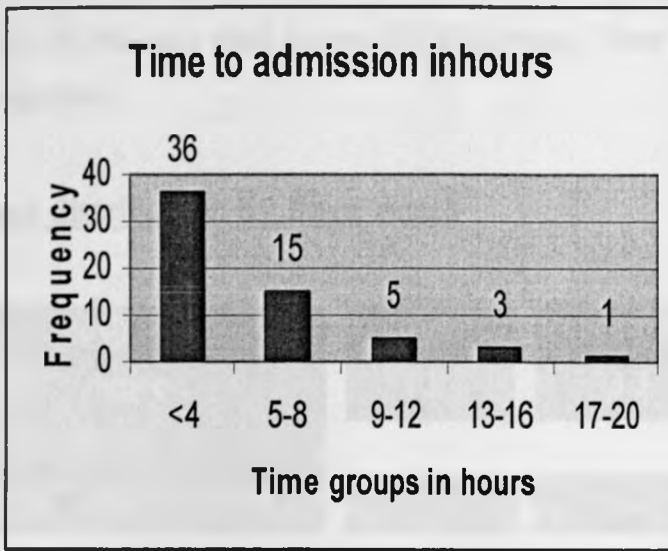


The time delay from injury to admission in Burns Unit / ICU ranged from 1-18 hrs mean 5.4 ± 5.8 hours, median 4hrs. Only 1 patient was admitted within the 'golden hour'. 48 patients (80%) were admitted within 8 hrs, with 12 (20%) coming in beyond 8 hrs.

Table 4: Time delay to admission in hours

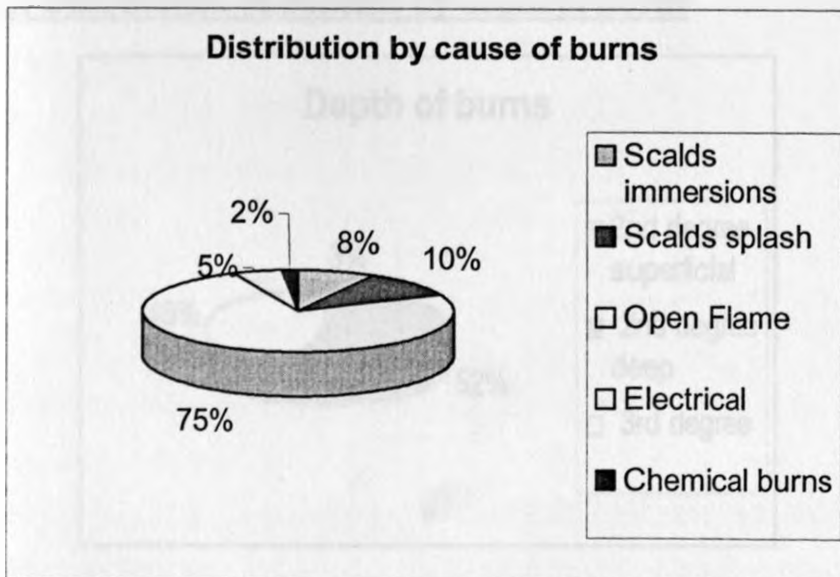
Time to admission in hrs	< 4hrs	5-8	9-12	13-16	17-20	Total
Frequency	36	15	5	3	1	60
Percentage	60	25	8.3	5	1.7	100%

Figure 3: Time delay to admission in hours



Open flames were the majority, 45 patients (75%). Scalds were seen in 11 patients (18.3%) of the burns, with electrical and chemical burns contributing 3 (5%) and 1 (1.7%) respectively.

Figure 4: Distribution of patients by Cause of Burns.



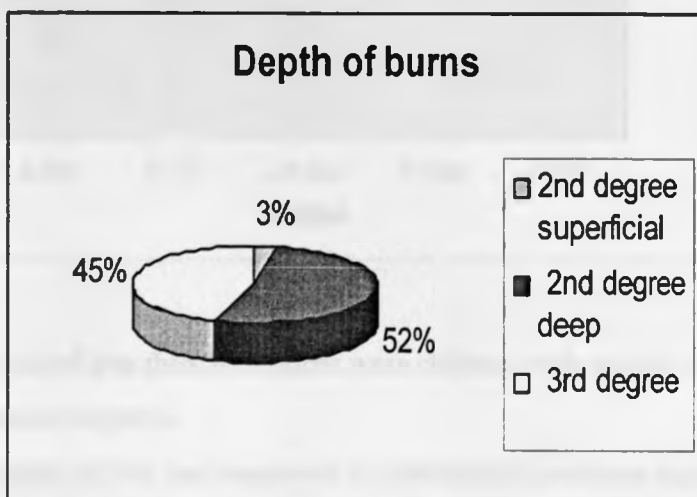
Partial thickness burns 33 (55%) outnumbered full thickness burns 27(45%).

Majority of the burns were second-degree deep 31(51.7%) compared to second-degree superficial 2(3.3%) and third degree 27(45%) burns. There were no fourth degree injuries registered.

Table5: Patient distribution by burn depth

Depth of Burns	Frequency	Percentage
a) 2 nd degree superficial	2	3.3
b) 2 nd degree deep	31	51.7
c) 3 rd degree	27	45
Total	60	100

Figure 5: Distribution of patients by depth of burns

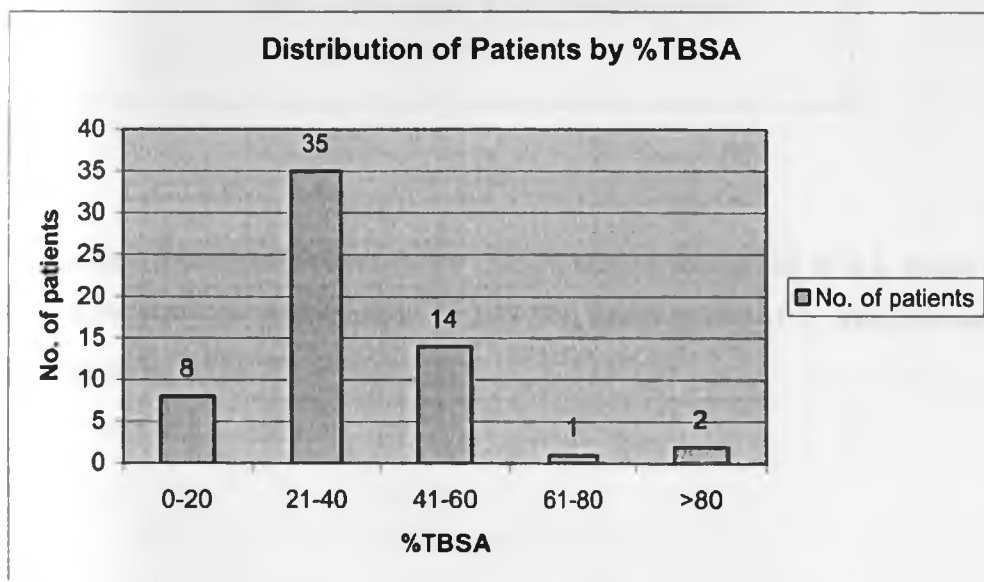


The total body surface area burnt (%TBSA) ranged from 14-90% with a mean of 38%. Most patients had less than 50% TBSA (86.6%).

Table 6: Distribution of Patients by %TBSA

%TBSA	0-20	21-40	41-60	61-80	>80	Total
No. of patients	8	35	14	1	2	60
Percentage	13.3	58.3	23.3	1.7	3.3	100

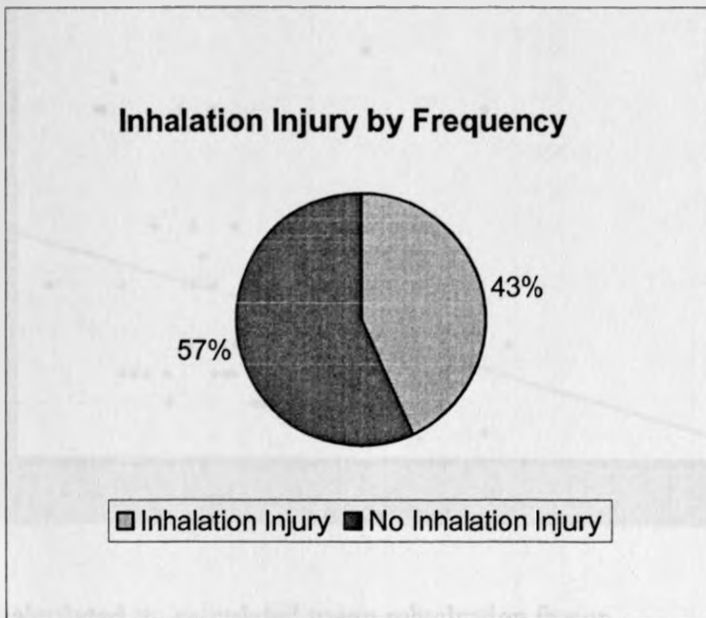
Figure 6: Distribution of Patients by %TBSA



Those who sustained less than 20% burns were children with scalds, electrical injuries or smoke inhalation injuries.

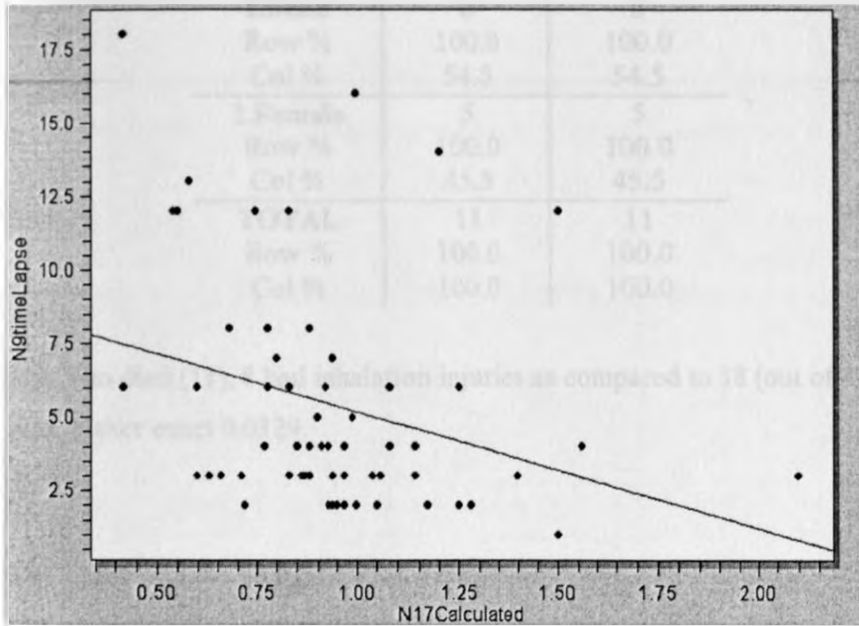
Twenty six patients (43%), had suspected or confirmed inhalation injuries as compared to 34(57%), who did not. All the 26 patients with inhalation injuries were intubated and supplemental oxygen given.

Figure 7: Incidence of Inhalation injury



The calculated mean rehydration factor (MRF) ranged from 0.42 to 2.1, mean 0.95 ± 0.29 . 66.7% (40) had MRF below 1, 2 (3.3%) had a factor of 1, with the rest 18 (30%) registering MRF > 1.

Figure 8: Scatter graph of Time delay to admission Versus Mean rehydration factor



Key

X-Axis – N17calculated = calculated mean rehydration factor

Y-Axis - N9timeLapse = time lapse in hours before admission

Only 9 patients (15%) had well kept urine out-put charts with an overwhelming 51(85%) having no proper output registers. Most of those with proper charts were in ICU as compared to Burns Unit. All the patients with known urine out-put were adults except a single child of 1 year.

All the 53 patients who were alive at day 5 had normal Urea and Creatinine levels (taken day 5). Vomiting and diarrhoea was reported in only 3(5%) of the patients and diuretics were used in only 2 (3.3%) of the patients. These 2 patients had their urine out-put known and succumbed on the day they were commenced on the diuretics.

Of the 60 patients recruited for the study, there were 11(18.3%) deaths in week one. The deaths were 3,1,1,2 and 4 in days 2 through 6 respectively. The mean age for those who died in week 1 was 24years compared to 19.1years for those who survived, t-statistic 1.1020, p-value 0.2750 DF=1. There were 6males and 5 females who died in week 1. This was found to be statistically insignificant, Fishers exact 0.4506, Odds ratio 0.7372, chi-square 0.0150.

Table 7: Patient outcome at day 7 by gender

OUTCOME AT DAY 7		
5.Sex	2. Dead	TOTAL
1.Male	6	6
Row %	100.0	100.0
Col %	54.5	54.5
2.Female	5	5
Row %	100.0	100.0
Col %	45.5	45.5
TOTAL	11	11
Row %	100.0	100.0
Col %	100.0	100.0

Of those who died (11), 8 had inhalation injuries as compared to 18 (out of 49) who survived, Fisher exact 0.0329.

Table 8: Distribution of patients by inhalation injury and outcome

Inhalation Injury	Week1 survivors	Week 1 mortality	TOTAL
Yes	18	8	26
No	31	3	34
TOTAL	49	11	60

Week 1 survivors had better MRF (mean 0.967 ± 0.30) as compared to those who died (0.87 ± 0.25). This translated to a P-value of 0.3259, T-statistic 0.9909.

Though not statistically significant, (p-value 0.084), those who died had a longer time delay to admissions (mean 6.1hr) as compared to the survivors (mean 5.2hrs).

The difference in % TBSA between survivors and the dead was highly significant (T-statistic 4.3573, p value 0.0034). The mean %TBSA for the survivors was 34.4% in contrast to 54.7% for those who succumbed.

Open flame burns had a slightly higher mortality (9) as compared to other causes of burns (2). This was however statistically insignificant (chi square 0.0371, fishers exact 0.4407).

Depth of burn was slightly different between those who survived and the dead; with the dead sustaining more full-thickness burns. It did not however attain statistical significance, Fisher exact 0.1494.

Table 9: Outcome by depth of burn.

Depth	Survivors	Dead	Total
<i>Full</i>	20	7	27
<i>Partial</i>	29	4	33
Total	49	11	60

Table 10: Summary of Burn Characteristics

	Parameter	Frequency	Percentage
Gender	<i>Males</i>	29	48
	<i>Females</i>	31	52
Cause of burn	<i>Scalds immersion</i>	5	8.3
	<i>Scalds splash</i>	6	10
	<i>Open flame</i>	45	75
	<i>Electrical</i>	3	5.0
	<i>Chemical</i>	1	1.7
Inhalation	<i>Inhalation injuries present</i>	26	43
Burn depth	<i>Full thickness burns</i>	27	45
	<i>Partial thickness burns</i>	33	55
Time delay	<i>Time delay < 8hr</i>	48	80
	<i>Time delay > 8hr</i>	12	20
Outcome	<i>Week 1 mortality</i>	11	18

POTASSIUM ANALYSIS

Of the 60 patients observed, only 2 patients had episodes of hyperkalaemia. Each of these registered one episode only. Both had severe burns, 80% TBSA with inhalation injury in one and electrical injury in the other. The episode occurred on day 1 and did not recur thereafter. Thus this group was too small to be analysed.

In contrast, hypokalaemia was observed in 33 patients (62%). There were 65 episodes in 265 observations for the 53 patients who survived to day 5. If any episode of hypokalaemia registered during the 5 day observation period, a patient was labelled as hypokalaemic, and those who did not as non-hypokalaemic. A similar exercise was undertaken with every day separately; therefore creating two groups of patients, hypokalaemia and non-hypokalaemic.

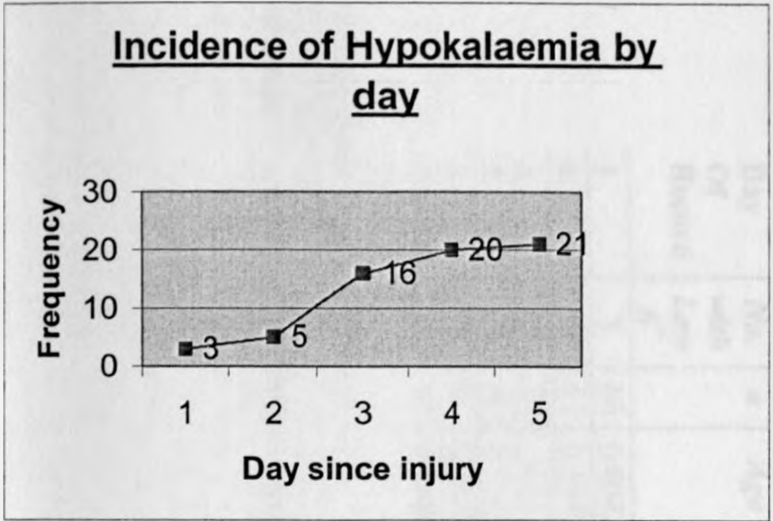
These two groups were analyzed for statistical differences with respect to;

- a) Gender
- b) Age in years
- c) Calculated mean rehydration factor(MRF)
- d) Depth of burn- Full thickness versus partial thickness
- e) Mean time delay to admission in hours
- f) Type of burn- open flame versus others
- g) Presence of inhalation injury
- h) Outcome at day 7- dead or alive

The influence of these variables was analyzed for statistical significance by way of chi-square, 2-tailed p-value, or T-statistic and its equivalent p-value and Fisher exact p-value where applicable and the results were as tabulated in table 11.

The number of hypokalaemic patients increased as days progressed from the day of admission.

Figure 9: Incidence of hypokalaemia by day since the injury



The daily mean potassium level declined as days progressed.

Figure 10: Pattern of change of daily K⁺ means by day since injury

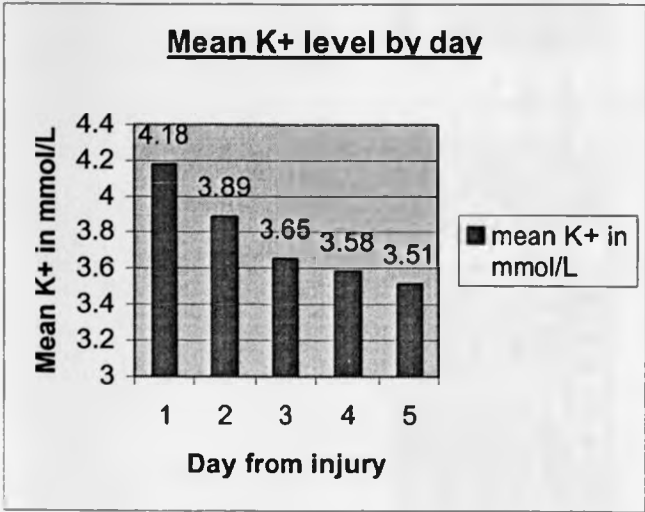


Table 11: P-values for Comparison of patients with hypokalaemia against those with no hypokalaemia for various variables

<i>Day Of Record</i>	<i>No. with Low K⁺</i>	<i>n</i>	<i>Age</i>	<i>sex</i>	<i>Mean rehydration</i>	<i>Inhalation Injury</i>	<i>%BSA</i>	<i>Burn type</i>	<i>Time delay</i>	<i>outcome</i>
1	3	60	0.0082	0.1314*	0.0181	0.076*	0.1471	0.4156*	0.3527	0.5384*
2	5	57	0.5941	0.187*	0.3368	0.317*	0.519	0.604*	0.380	0.545*
3	16	56	0.24	0.321*	0.0308	0.556*	0.789	0.4345*	0.0205	0.0937*
4	20	55	0.7121	0.4893*	0.7295	0.02576*	0.5049	0.4832*	0.002	0.3769*
5	21	53	0.5602	0.589*	0.6592	0.595*	0.2098	0.6050*	0.4037	0.166*
Overall	33	53	0.6549	0.241**	0.7519	0.978**	0.2369	0.4816*	0.0119	0.1397*

*Fishers exact

**2-tailed p-values

Others are T-statistic's equivalent p-value

Age had a very significant influence on hypokalaemia episodes. On day 1, there were 3 patients with low potassium (mean age 0.42 ± 0.29 years) as compared to 57 (mean age 21.06 ± 13.0 years) with no hypokalaemia. This was highly significant, T-statistic 2.7365, p-value 0.0082. All the three patients were females (100%) as compared to 49% (28 out of 57) who were non-hypokalaemic. This however did not reach statistical significance (Fisher exact 0.1314).

The mean rehydration factor was higher (1.33) in the hypokalaemic as compared to non-hypokalaemic (0.93). This translated to a T-statistic 2.4331 and p-value 0.0181.

Though there was a higher proportion of inhalation injuries (100%) among those with hypokalaemia than those with no hypokalaemia (23 out of 57, 40.4%) this did not attain statistical significance- chi-square 2.0576, 2-tailed p 0.1514 and Fisher exact 0.076.

There was no significant difference between the 2 groups on day one as regards gender, type of burn, %TBSA and mean time delay to admission- p-values 0.1314, 0.4156, 0.3527, and 0.1471 respectively. All the 3 hypokalaemic patients in day 1 survived week one as compared to 46(99%) who were non-hypokalaemic, Fisher exact 0.5384.

When similar comparisons were performed for day 2, there appeared no significant differences with respect to the afore-mentioned variables (table 11).

However, on day 3, the mean rehydration factor and mean time delay to admission were significant, p-values 0.0308 and 0.0205 respectively. The calculated MRF for those in group with hypokalaemia was 1.09 as compared to 0.91 for group without hypokalaemia, t-statistic 2.218 and p-value of 0.0308. Likewise, former had longer mean time delay to admission 7.4 ± 5.16 hours compared to 4.75 ± 3.13 hours for the latter group. This translated to a t-statistic 2.3883, and p-value 0.0205.

Inhalation injured patients had higher episodes of hypokalaemia on day 4 as compares to those without, p-value 0.0258.

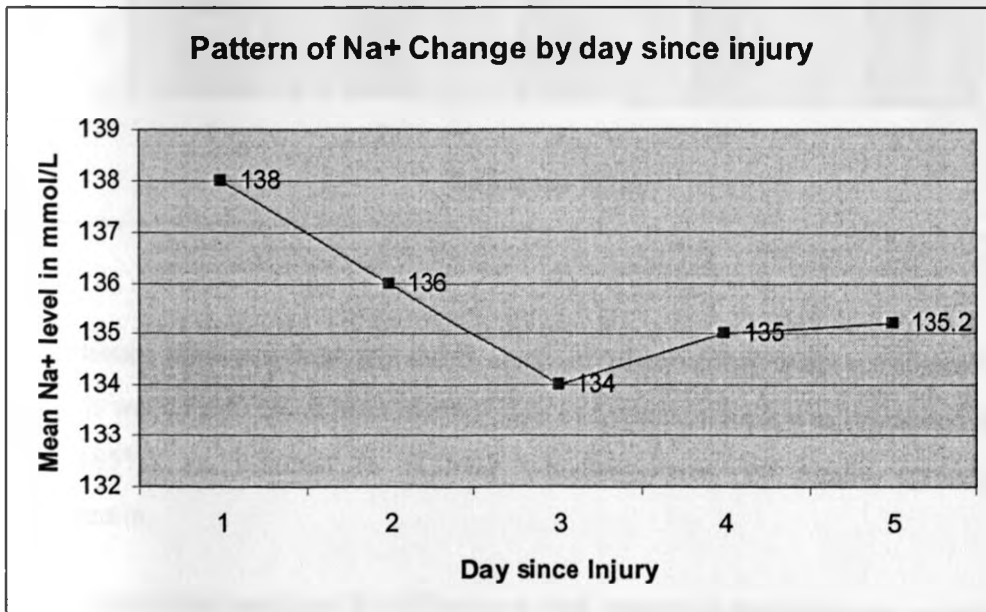
On the overall, when those with 5 readings were considered, hypokalaemia was registered in 33 patients as compared to 16 who did not. Only time delay was found to

be significantly different, $p=0.0119$. The hypokalaemia group had mean delay of 6.04 ± 4.25 hours while group with no hypokalaemia had delay of 3.70 ± 2.45 hours.

SODIUM ANALYSIS

The mean sodium levels for each day were analyzed and found to be highest on day 1 (138 mmol/L) and lowest on day 3 (134 mmol/l). The mean of sodium was below normal on day 3, while other days recorded normal means. The pattern was as shown in the figure below.

Figure 11: Pattern of change of Na⁺ means by day since injury

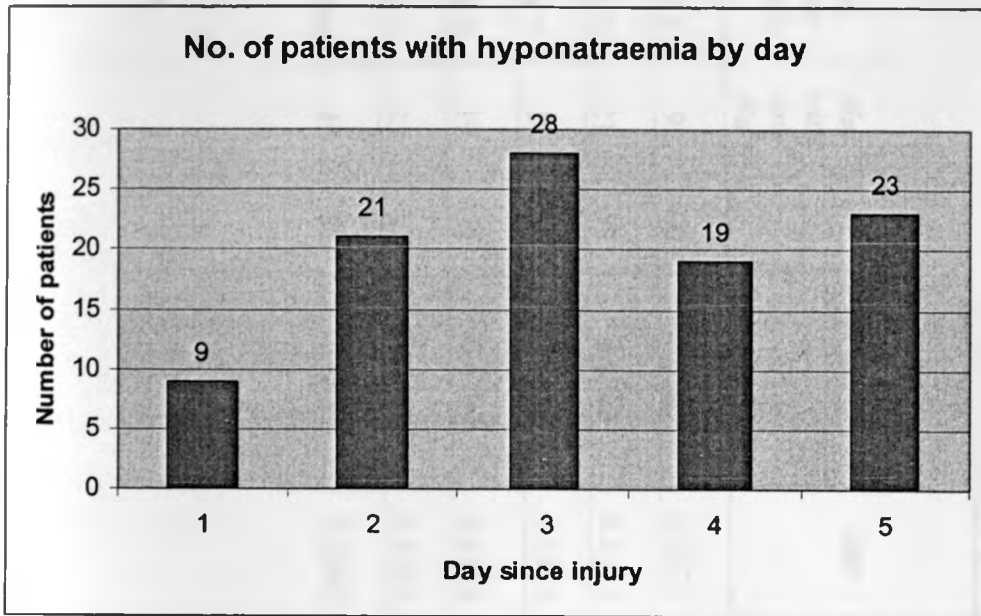


Thus, there was a drop in mean Na⁺ up to day 3, and then a gradual rise was noted thereafter.

Hyponatraemia was more common than hypernatraemia. Indeed, hypernatraemia was noted in only 8 patients all on day 1. Of note is that all these patients were referrals from peripheral hospitals. In contrast, there were 100 episodes of hyponatraemia in the 60 patients during the 281 observations taken. The highest incidence was recorded

on day 3 and the least on day 1. Hyponatraemia was thus not analysed because of the rare occurrence in this study.

Figure 12: Frequency of Hyponatraemia by Day since injury



When patients who survived for the 5 days of observation were considered (53 patients), it was noted that at least one episode of hyponatraemia was registered in 45 patients (85%), in contrast to 8(15%) who registered no single episode of hyponatraemia.

The two groups were analyzed for differences with respect to the following variables;

- a) Age in years
- b) gender
- c) Calculated mean rehydration factor (MRF)
- d) Depth of Burn-full versus partial thickness
- e) % BSA
- f) Inhalation injury
- g) Outcome at day 7 (dead or alive)

Table12: Respective p-values when patients with hyponatraemia were compared with those with no hyponatraemia for differences in various variables

Day since injury	No. with low Na⁺	N	Full thickness	Age	Sex	MRF	Open flame	% TBSA	Inhalation injury	Time delay
Day 1	9	60	0.6303	0.0231	0.4559	0.0038	0.3990	0.6407	0.3283	0.1244
Day 2	21	57	0.4262	0.1123	0.1150	0.9073	0.5011	0.5090	0.492	0.3806
Day 3	28	56	0.1386	0.4453	0.1424	0.9675	0.1135	0.2011	0.027	0.7646
Day 4	19	55	0.4794	0.8152	0.1943	0.4291	0.1996	0.2600	0.0519	0.3071
Day 5	23	53	0.3652	0.8703	0.0958	0.1601	0.5001	0.8521	0.460	0.2926
Overall	45	53	0.6085	0.3060	0.581	0.2794	0.5958	0.8096	0.651	0.909

The mean age for group with hyponatraemia (18.01 ± 14 years) was lower than group with no hyponatraemia (23.4 ± 10.24 years) which translated to a T statistic 1.0341, p-value 0.3060.

There were no significant differences in terms of sex for the 2 groups –

Hyponatraemia group - 21 males, 24 females

Non- hyponatraemia group - 4 males and 4 females

Fisher exact 0.581.

The hyponatraemia group had a slightly higher mean rehydration factor 0.96 compared to 0.80 for group without hyponatraemia, p-value 0.2794 and T – statistic 1.0933. Eighteen (40%) patients in group with hyponatraemia had full thickness burns while there were 3(37.5%) patients with full thickness burn in group without (Fisher exact 0.609).

The hyponatraemic patients had a slightly lower %TBSA (mean $34.53 \pm 12.54\%$) as opposed to $35.61 \pm 4.17\%$ for the non-hyponatraemic groups – p value 0.8096.

37.8 % of patients with hyponatraemia had inhalation injury as compared to 37.5% of those with no hyponatraemia, Odds Ratio 1.0119, Fisher exact 0.65. There were 3(6.7%) fatalities for hyponatraemia group as compared to 1(12.5%) in group without, Fisher exact 0.491.

Thus, on the overall these variables (age, MRF, burn depth, %TBSA and inhalation injury) though different among the 2 groups did not attain statistical significance.

However, when the two groups were examined on individual days for the same variables, it was noted that age, and calculated MRF were significantly different on day 1, while inhalation injury was on day 3.

The mean age for group with hyponatraemia on day 1 was 10.8 ± 15 years, and 21.7 ± 12.6 years for group without hyponatraemia, p-value 0.0231. Thus hyponatraemic patients were younger than those without hyponatraemia. The MRF was higher for hyponatraemia group, 1.2 ± 0.53 , compared to 0.9 ± 0.20 for group with

no hyponatraemia, p-value 0.0038. It is however important to note that children (<10years), had a higher MRF than those over 10 years. However on bi-variate analysis, the two factors were found to be independently significant.

On day 3, hyponatraemia group had lower incidence of inhalation injury compared to group with no hyponatraemia, p-value 0.027. There was a similar trend on day 4 but did not reach statistical significance (p-0.0519).

Thus, only 2 variables achieved statistical significance. The rest of the analysis is as depicted in the table 12.

DISCUSSION

There were over 100 admissions with severe burns over the study period. However, only 60 met the inclusion criteria. Those who were excluded were mainly referrals from other institutions that arrived beyond 24 hours from time of injury.

The gender composition of the studied population was evenly matched, M: F = 1:1.08, albeit with a very slight female predominance. This ratio does not appear to vary much for the age-groups, under ten (1:1.3) and over 10 years (1:1.05). Other studies have found a slight male predominance in the younger age group due to their inquisitive nature ^{2, 4, 5, 6, 12}. Nthumba in a study of 1157 patients found a male to female ratio of 1:1.08 ⁴¹. Nderitu, Mungara and Okonjo found a slight female predominance in studies involving paediatric burns. Boys are more predisposed than girls due to their clumsiness and inquisitive nature ²⁷. The involvement of women in domestic fires may account for their predominance in domestic burns.

There was a bi-modal distribution of burns reflecting 2 peaks of 0-10 years and the 21-30 year groups. Burns in the young are mainly scalds and in the older population, open flame predominate and this difference may account for the 2 peaks ^{3, 7, 8}. Nderitu found a similar bi-modal distribution in his study of burns in KNH ². Open flame burns contributed more to severe burns than the scalds, 75% and 18.3% respectively in the study. They tend to have associated complications of inhalation injury.

Patients in the study had a mean time delay to admission of 5.38 hours. Indeed, only 1 patient was admitted within the golden hour. This has been reciprocated in another study by Mogire who found a mean time delay of 5.7 hours ⁴³. This has an influence on the amount of fluid that patients receive since they must be infused rapidly to cover the deficits in time. Indeed a negative correlation exist between time delay to admission and MRF, see figure 8.

Developed countries have elaborate patient evacuation and rescue programs and very well trained paramedics, something obviously lacking in our country ³. 48 patients (80%) presented within the first 8 hours (first resuscitation phase) as compared to

12(20%) who came in the second phase. These delays impact on fluid management and electrolyte balance, as was demonstrated in this study.

Those patients who presented “early” had better rehydration; mean factor of 0.9848, in contrast to 0.8092 achieved for those who came in phase 2. This almost achieved statistical significance, T statistic 1.9069, p value 0.0615. This however did not translate to week one survival advantage (Fisher exact 0.6179). The late presenters were mainly referrals from other hospitals mainly due to financial issues or lack of facilities in primary health care centres. Nguyen found late referrals to have inadequate fluid resuscitation in 363 Vietnamese children ⁶.

The bigger proportion had partial thickness burns (second degree burns – 55%). Third degree burns were 45%. This latter group was caused by open flames and electrical injury. There were no fourth degree burns encountered. This is in keeping with data from other sources that open flame burns tend to be deeper than scalds ^{1, 7, 8, 10}. As expected, those patients with immersion scalds suffered deeper burns (second degree deep) as compared to splash scalds. This is due to the longer duration of contact for immersion burns and hence the deeper burns.

Children sustained more scalds and hence, partial burns as compared to adults who had deeper burns (chi square 90.0645, DF 64, p 0.0176- by single table analysis). Most of the paediatric burns tend to be scalds ^{2, 4, 6, 13}. They occur due to accidents within the house during cooking or at bath-time.

There were only three electrical burns registered reflecting either high fatality for this type of injury or lower access to electrical appliances by the Kenyan population. All the three were industrial accidents. There was only one alkali burn that was caused by assault by a jilted lover. Chemical injuries tend to follow industrial or farm accidents, but assault has also been implicated ²⁷.

Inhalation injury was observed in 26 (43%) patients, all with open flame burns. This is a very severe injury with a high case fatality rate – 31% in this study. It needs a high degree of suspicion and aggressive intervention. All the patients with suspected or confirmed inhalation injury were intubated and put on supplemental oxygen as is

recommended. It is significant that just as many studies have shown, there is high mortality amongst those with this injury as compared to those without (in this study Fisher exact 0.0329) ^{1, 5, 8, 10,16,28}. Nthumba found a 68% mortality rate in 138 patients with inhalation injury at KNH ⁴¹. The injury was suspected on account of burns in an enclosed space, respiratory distress, bronchorrhoea, or facial burns with burnt vibrissae. No patient had bronchoscopic examination or blood carbon monoxide levels ascertained in this study. Where available, this is desirable to confirm and grade the injury ¹⁶.

It was noted that urine output records were poorly kept; only 15% patients had urine output records. This situation was worse in Burns Unit compared to ICU. In fact, only one child in ICU did not have output records. This was attributed to lack of paediatric size urethral indwelling catheters. There were attempts at urine out-put records in terms of registering the number of times a patient passed urine. This is however inadequate. Adequate urine output is one of the primary end-points of fluid resuscitation in burns management ^{1, 7,23,26,44}. For those whose records were kept, their MRF was greater than one, reflecting the usefulness of urine out-put as a means of assessment of fluid needs. The mere attainment of input equivalent to Parklands formula is not enough. The calculated fluid need in a patient with inhalation injury is high, 5.7mls/Kg/%TBSA ^{1, 28, 30}. Children also need relatively larger volumes than adults. Thus adequate urine output remains the best measure of adequacy of resuscitation.

There were 11 deaths (18.3%) during the study by week one. They had slightly high mean age as compared to those who survived. There were no male: female differences in mortality. Women have been known to fair poorly in other studies. The dead had significantly a higher %TBSA and higher incidence of inhalation injury. Although statistically insignificant, those who succumbed had deeper burns, and lower mean rehydration factors. They also had longer time delay to admission. Nguyen in his study in Vietnam found a significant association between inadequate initial management and mortality ⁶.

This trend is reflected in other studies and indeed, the Tobiasen abbreviated burn severity injury score has age, sex, % TBSA, depth of burn and inhalation injury as the

common predictors of survival or mortality⁴². The older patients, females, those with deep burns, extensive %BSA and inhalation injury fair poorly than their counterparts^{50, 53, 54}.

The mortality noted in this study was still higher than what is observed in specialized centres in the west but compares to other centres in Africa⁵⁵. This may be a reflection of the level of development of Burns units. Patients in KNH arrive late, they lack onsite care, (especially inhalation injured patients) and this may impact on the mortality. This figure of 18% deaths may be a gross underestimation of mortality, bearing in mind the fact that this was only week one mortality. In developed centres, the %TBSA with an expected 50% mortality (LD₅₀) has risen to 80% TBSA. Nthumba found LD₅₀ to be 35% in KNH. Mortalities in the acute phase have progressively diminished in developed centres⁴¹. Indeed in the west, infections are the commonest cause of delayed mortality.

We have our fair share of burn wound infections as was documented by Wanjeri in 1995. He followed up 347 patients and noted an infection rate of 40%. The mortality rate in his study was 87.5% in those with burns greater than 50% TBSA³⁹.

The burn patient is subjected to severe stress to both the renal and cardiovascular systems. Fluid and electrolyte balance poses a very big challenge for this category of patients. Besides the loss of the skin which forms a protective barrier against fluid loss, these patients are perpetually in a catabolic state and therefore, need hyper caloric feeds to shift them to an anabolic phase^{11,12,14,33,47}.

They may also need frequent administration of blood, plasma, albumin, sodium bicarbonate to counter acidosis, total parenteral solutions and antibiotics all of which increase electrolyte load to the patients. Fever in septic patients increases free water losses, both cutaneous and respiratory and may cause hypernatraemia. Urea diuresis from protein loading and breakdown of proteins from burn catabolism liberating osmotically active particles, add further to free water losses. All these factors are common in burn patients, especially in setting of sepsis, and are common beyond resuscitation phases. They may however influence electrolyte balance in the burns patient.

With all these assaults on the burn patient, it is prudent that electrolyte monitoring be given appropriate attention. The release of intracellular potassium from burn-destroyed cells may cause a transient hyperkalaemia state. This is commonly seen in patients with extensive burns or with high-voltage electrical injuries^{19,27,48}. This may be exacerbated by use of K⁺ rich solutions. However, where saline-based solutions have been used, this is not a common finding²⁵.

Electrical injuries however cause extensive necrosis of tissue with k⁺ release from intra-cellular stores²⁷. Indeed hyperkalaemia was noted in 2 patients; one had 80%TBSA and the other electrical injuries. The low incidence of hyperkalaemia in this study may be due to the relatively lower mean %TBSA (38%) for this study group. Besides, a few patients especially the referrals had received K⁺ deficient fluids especially 0.9% Normal saline by the time of admission.

On the contrary, hypokalaemia was a common occurrence in this study. Ringers lactate is the fluid recommended in Parklands formula^{1,8,16,44}. This fluid contains K⁺ but less than serum levels. Enthusiastic use of this solution even beyond the resuscitation period (beyond 48 hours) may account for the high incidence of hypokalaemia. The rising incidence of hypokalaemia in these patients as days progressed may not be accounted for by K⁺ losses. Diuretics were used in only 2 patients on day 2 who died same day. Vomiting was only reported on day 5 in 2 patients. All the patients were dressed with 1% silver sulphadiazine and none with 0.5% silver nitrate. This latter compound is associated with increased transechar K⁺ loss²⁰.

Hypokalaemia was found to be more in those with inhalation injury. Inhalation injury may lead to hyperventilation and attendant respiratory alkalosis and thus intracellular shift of K⁺ leading to lower serum K⁺ levels³⁵. This may account for the slightly higher incidence in this selected group. The use of K⁺ depleted fluids may also account for hypokalaemia. This is reflected by the fact that there are no guidelines in KNH on the use of fluids beyond 48 hours. This is left at the discretion of the attending doctor. If normal saline was selected, then Na⁺ levels would rise, while K⁺

levels would continue to drop as days progressed, like what was observed in this study (fig. 9 and 10).

Children were more prone to hypokalaemia than adults, and this may partially be explained by the higher mean rehydration factor obtained for them. However on bivariate analysis the two were found to be independently significant. Patients with normal MRF are expected to have normal electrolytes since Ringers lactate is nearly the composition of plasma albeit with a lower sodium concentration. Even if Parklands formula was standard, Ringers lactate is still deficient of adequate K^+ . Besides, the immature nature of young kidneys may not adequately handle electrolyte imbalance challenges³³. Much as those with hypokalaemia had higher rehydration factor, the adherence to recommended fluid choices is not guaranteed as only amounts in fluids charts and occasionally fluid types are charted. This may lead to a high fluid delivery, but that is K^+ depleted. In those with adequate and appropriately selected fluid input, electrolyte derangements are uncommon.

In deed, the higher incidence of hypokalaemia in referred patients may be a manifestation of this variation in choice of fluid for resuscitation. There was no statistical difference in mortality between those with potassium derangement and those without. It is however known that cardiac events are common in K^+ deranged patients^{45, 46, 48}. These patients with hypokalaemia had just slightly lower K^+ than normal. The lowest mean was 3.51mmol/L, on day 5.

Sodium is an electrolyte whose concentration in body fluids is tightly regulated. Being the predominant extra-cellular cation, its concentration influences the dynamics of extra-cellular fluid. Lactated Ringers solution is the standard resuscitation fluid in Parklands formula. This solution has relatively lower levels of Na^+ (130mmol/l) compared to normal saline (154mmol /l). Its use in burns and in large volumes therefore places the burn patient at a significant risk of hyponatraemia. Indeed there were 100 recorded episodes of hyponatraemia in the study group.

On the other hand, the burn patient has a strong challenge of fluid conservation in the presence of the denuded protective skin cover. With loss of body water, there is a real risk of hypernatraemia, the extent and degree of which will be influenced by the

actual amount of water lost^{44,45,48,49}. Through humoral mediators, the body makes all attempts to conserve body water. Morgan et al demonstrated a ten-fold rise in antidiuretic hormone following burns⁵⁶. The sodium balance will therefore be a factor of the interplay of these opposing processes. This is further complicated by the additional sodium in load in diet and non-resuscitation fluids e.g. antibiotics⁴⁸.

The initial stress of burn may induce a moderate hypernatraemia due to extra-cellular water depletion due to changes in cellular permeability. This may be exaggerated in patients with SIADH or where electrolytes are leached in dressing materials especially 0.5% silver nitrate. Administration of electrolyte free fluids may pronounce hyponatraemia to symptomatic levels.^{1,20}

The study yielded a mean Na^+ level that was low normal, reflecting more on the choice of resuscitation fluids. The sodium levels were lowest on day 3, perhaps implying continued use of Na^+ depleted fluids beyond the resuscitation phase. The immediate post-burn mean sodium level (138 mmol/L) were within normal and gradually declined to a level of 134mmol/l on day 3 and subsequently starting to gradually rise to a mean of 135.2 on day 5.

The rise may be a manifestation of recovery of the patient from immediate stress facilitating a return of normal gut function, with Na^+ being availed from the diet. This rise may also be due to administration of Na^+ rich fluids e.g. 0.9% normal saline since a corresponding K^+ for the latter days is low suggestive use of K^+ deficient fluids. Onset of sepsis may also account for the gradual rise in Na^+ levels from day 3³⁴. Onset of fever may mean an increased evaporative water loss reflected as a relative increase in sodium concentration.

Patients with major flame burns and inhalation are more prone to developing hypernatraemia. They are incapacitated and have a lower electrolyte free water intake and therefore staff in Burns Unit should be aware of the need to replace this deficit.

Mohammed in 2002 demonstrated this increased need and the positive correlation between a rising sodium amongst septicaemic patients. Indeed, they showed that the rise in sodium preceded septicemia by a few days³⁴.

Subtle renal dysfunction may lead to high urine output due to renal tubular malfunction and diminished concentrating ability. Dudley et al reported fatal hypernatraemia related to a mannitol-induced osmotic diuresis used in the management of haemoglobinaemia in extensive burns. In this study, diuretics were only used in day 5 in 2 patients and thus their influence was not significant in this study. Vomiting and diarrhoea were reported in 3 patients who succumbed on the day of onset of the same. Vomiting is not very common following burns, and when it occurs, older patients have a higher incidence³⁷. These events may not account for the derangements observed.

Hyponatraemia appeared to be a more common derangement than hypernatraemia in this study. This contrast with what has been seen elsewhere⁴⁴. This may be due to major departure from the norm in fluid management e.g. the continued use of Ringers Lactate as a fluid even after the acute phase of burns is over. Over-enthusiastic replacement of insensible loss with Na⁺ depleted fluids may account for the observed hyponatraemia. Besides, the observed hyponatraemia was strongly related to a lower age and higher mean rehydration factor.

Looking at the former variable, one may deduce that the lower age is related to kidney immaturity and poorly developed sodium reabsorption function. The mean age for the hyponatraemic was 10.8 ± 15 years compared to 21.7 ± 12.6 years for the non-hyponatraemic. A high mean rehydration factor may mean higher level of Na⁺ depleted fluids administration and this is typified by the strong association of MRF to sodium levels especially on day 1, p-value 0.038, when rehydration under Parklands formula (using Ringers Lactate) is very intense. This association waned as days progressed. On the overall the presence of sodium derangements did not directly impact negatively on mortality, p-value 0.49110.

From the foregoing it is apparent that perhaps the strongest association of either Na⁺ and K⁺ derangement appear to be age and mean rehydration factor and perhaps to a lesser extent, inhalation injury. This strong association was only observed in days one and two implying a more standard approach to fluid management over this period. It is however clear that with the many parameters that come into play in patients with

burns as far as electrolyte balance is concerned; it is very difficult to predict who is at risk of a specified derangement.

This is further compounded by the fact that in spite of these reported derangements; the urea and creatinine were within normal values (means of 5.7 ± 2.6 mmol/L and 76.8 ± 23.4 ummol/L respectively). This means that a full electrolyte, urea, and creatinine panel is important in assessing renal status in a patient. Besides, assessment of these two electrolytes cannot be used to gauge the adequacy of resuscitation or fluid management. This lack of prediction therefore means that those derangements must be actively sought and addressed. For the patients who had hypokalaemia, KCL supplementation was always successful where replacement was actively done.

This is difficult to comment for sodium since the administration of sodium chloride solutions as part of fluid management is in itself a correction attempt for hyponatraemia³³. The mere attainment for a normal mean rehydration factor (MRF) is not enough for a patient. There were electrolytes derangements in patients who received even higher than calculated fluid needs. One needs therefore to pay attention to quality (electrolyte composition) of the administered fluid in addition to the quantity.

CONCLUSIONS

Burn injuries are a significant cause of morbidity and mortality in our set up.

Scalds injuries are the predominant thermal injury seen in children while open flame injuries are more common in adults.

Patients in KNH with severe burns rarely make it to hospital early enough for the urgent institution of appropriate care, with a mean time delay of 5.4 hours.

This time delay may translate to lower mean rehydration factor which may individually affect fluid and electrolyte balance.

Hypokalaemia and hyponatraemia are the commonest derangements seen in this study.

Age and mean rehydration factor were most predictive of these two derangements.

There is no standard protocol on fluid management in post-resuscitation phase and the choice is left at the discretion of the attending physician. This leaves room for error and possibly may account for the observed derangements.

Open flame burns by virtue of their association with higher burn depth and inhalation injury tend to be associated with electrolyte derangements.

There was a positive association between derangement and mortality, though not statistically significant, with higher mortality registered in those with derangements.

The level of urea and creatinine was not predictive of electrolyte derangements as all these derangements occurred in the setting of normal parameters.

There are many factors that can potentially alter electrolyte balance, making it difficult to track a particular group of patients for the disturbances. It is therefore prudent to remain on high alert and to actively seek for these disturbances.

RECOMMENDATIONS

Patients need to be educated in the need for early treatment of burn patients.

Peripheral institutions need to be well equipped with personnel and physical resources for initiation of appropriate therapy and means of referrals to specialized centres.

There is need to adhere to laid down protocols of fluid administration to make it easy for protocols to function.

Urine output monitoring is a cheap and effective assessment of resuscitation success and is easily reproducible. Paediatric catheters should be readily available and proper charting instituted for the success of this important aspect of burn management.

Airway management in KNH burn patients is up to date. However, diagnostic measures like blood carbon monoxide assays and bronchoscopic examination need to be introduced if this trend is to be improved.

One cannot be able to accurately predict who is likely to suffer a particular derangement and thus efforts should be made to have ISE machines available and accessible to physicians for regular Na^+ and K^+ monitoring.

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Appendix I Lund and Browder Chart



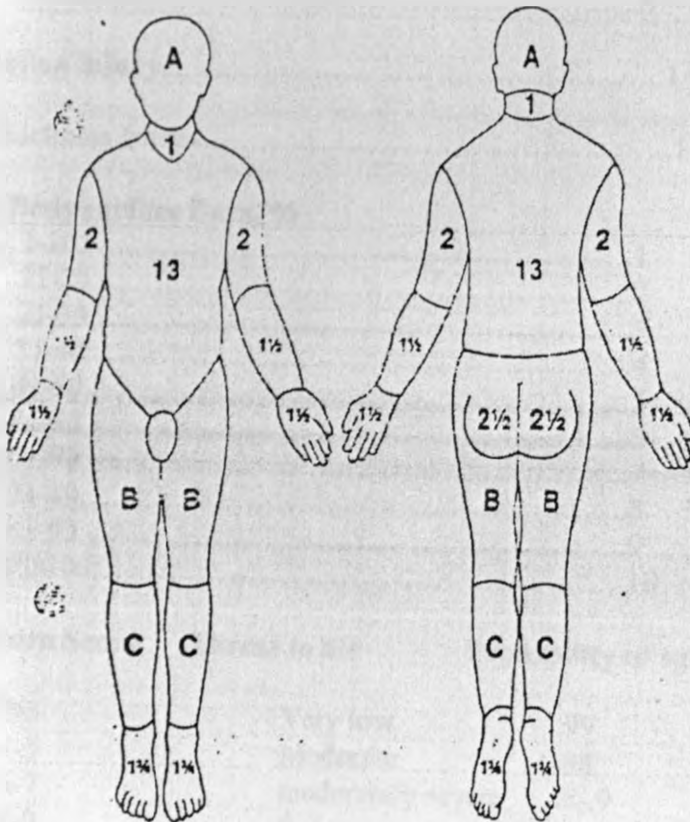
BURN RECORD

NAME _____
 ADDRESS _____
 AGE: _____ M/F _____
 ADMISSION WEIGHT _____ WARD _____

BURN	DATE	TIME OF ADMISSION	TYPE OF BURN
REGISTERED			INHALATION INJURY YES/NO
			PLACE AND TIME OF BURN
			INFORMANT

LUND AND BROWDER CHARTS

IGNORE
SIMPLE ERYTHEMA



Superficial
 Deep

REGION	%
HEAD	
NECK	
ANT. TRUNK	
POST. TRUNK	
RIGHT ARM	
LEFT ARM	
BUTTOCKS	
GENITALIA	
RIGHT LEG	
LEFT LEG	
TOTAL BURN	

RELATIVE PERCENTAGE OF BODY SURFACE AREA
AFFECTED BY GROWTH

AREA	AGE 0	1	5	10	15	ADULT
A = 1/2 OF HEAD	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2	3 1/2
B = 1/2 OF ONE THIGH	2 3/4	3 1/4	4	4 1/2	4 1/2	4 3/4
C = 1/2 OF ONE LEG	2 1/2	2 1/2	2 3/4	3	3 1/4	3 1/2

Appendix 2

ABBREVIATED BURN SEVERITY INDEX

Sex Score

Female.....	1
Male.....	0

Age, years

0-20.....	1
21-40.....	2
41-60.....	3
61-80.....	4
81-100.....	5

Inhalation injury.....1

Full thickness burns.....1

Total Body surface Burn, %

1-10.....	1
11-20.....	2
21-30.....	3
31-40.....	4
41-50.....	5
51-60.....	6
61-70.....	7
71-80.....	8
81-90.....	9
91-100.....	10

Total Burn Score Threat to life Probability of survival

2-3	Very low	.99
4-5	Moderate	.98
6-7	moderately severe	.8-.9
8-9	Serious	.5-.7
10-11	Severe	.2-.4
12-13	Maximum	< .1.0

INPATIENT STATISTICS

BURNS UNIT ADMISSIONS 20

	WARD	BEDS	COTS	INC.	TOTAL BEDS	ADM	DISCH	DEATHS	D+D	WPD	OBD	ABD	VI
<i>7. SPECIAL UNITS</i>													
	BU	12	10	0	22	197	118	91	209	1087	6129	8030	1
	<i>Subtotal</i>	<i>12</i>	<i>10</i>	<i>0</i>	<i>22</i>	<i>197</i>	<i>118</i>	<i>91</i>	<i>209</i>	<i>1087</i>	<i>6129</i>	<i>8030</i>	<i>1</i>
	<i>Grand Total</i>	<i>12</i>	<i>10</i>	<i>0</i>	<i>22</i>	<i>197</i>	<i>118</i>	<i>91</i>	<i>209</i>	<i>1087</i>	<i>6129</i>	<i>8030</i>	<i>1</i>

Source: Medical Records Department

Date: 7-4-06



INPATIENT STATISTICS

WARD	BEDS	COTS	IHC	TOTAL BEDS	ADM	DISCH	DEATHS	C+D
4.51 (DEPT GENERAL)								
SA	51	0	0	51	1930	1704	109	1893
SB	45	0	0	45	2219	2032	134	2226
SC	32	0	0	40	1555	1547	62	1660
SD	52	0	0	52	2128	1953	118	2111
Subtotal	180	0	0	180	8292	7717	422	8339
Grand Total	180	0	0	180	8292	7717	422	8339

Source: Medical Records Department

Note:



Appendix 3b

WFO	OBID	ABD	VBD	%OCC	ALOS	TCB	TCR	OTHRATE
0	17294	18015	1321	82.8	8.1	37.1	0.7	5.7
0	18979	16425	0	115.5	8.5	49.5	0.0	6.0
0	17313	14505	0	118.6	6.1	47.7	0.0	3.2
0	18070	10380	718	95.3	6.7	40.0	0.3	5.5
0	71856	69620	2031	104.7	8.8	42.3	0.2	5.1
0	71856	69620	2031	104.7	8.0	42.3	0.2	5.1

INPATIENT STATISTICS

WARD	BEDS	COTS	INC.	TOTAL BEDS	ADM	DISCH	DEATHS	D+D	WPD	OBD	ABD	VBD	%OCC	ALOS	TOB	TOI	OTHRATE	
3.SURGERY(SPECIAL)																		
4A	9	36	6	51	1916	1852	64	1916	10080	15200	18615	3407	81.7	7.9	31.6	1.8	3.3	
4B	37	4	0	41	540	483	61	544	1368	12041	14865	2924	80.5	22.1	13.3	5.4	11.3	
4C	38	7	0	40	652	544	93	637	4755	14475	14000	125	99.1	22.7	15.9	0.2	14.3	
4D	24	28	0	50	823	835	18	853	3819	17354	18250	896	85.1	20.3	17.1	1.1	2.2	
Subtotal	108	68	6	182	3931	3714	236	3950	20022	58078	66430	7352	80.9	15.0	21.7	1.9	6.0	
7.SPECIAL UNITS																		
42	31	0	0	31	206	204	0	204	108	437	11315	10878	3.9	2.1	6.6	53.3	0.0	
Subtotal	31	0	0	31	206	204	0	204	108	437	11315	10878	3.9	2.1	6.6	53.3	0.0	
Grand Total	139	68	6	213	4137	3918	236	4154	20130	58515	77745	18230	76.8	14.3	18.5	4.4	5.7	

Source: Medical Records Department

Date:



Appendix 4

RESEARCH TOOL (QUESTIONNAIRE)

Identifying information

1. Serial No.....
2. Name.....
3. Hospital No.....
4. Ward Admitted
 BURNS UNIT...
 ICU
 HDU

Demographic information

5. Sex
 Male
 Female

4. Age in years.....

Clinical information

7. Date of Admission.....
8. Time of injury.....
9. Time lapse in hours before
 A. Admission.....
 B. Not known (x).....
10. Type of burn
 A. Scalds (i) Immersion..... (ii) Splash...
 B. Open flame
 C. Electrical
 D. Chemical
11. Percentage body surface area burnt (%TBSA)
12. Depth of burn (a) second degree superficial
 (b) Second degree deep
 (c) Third degree
 (d) Fourth degree
13. Suspected or confirmed inhalation injury
 A. Yes
 B. No
14. Patient intubated (Endotracheal)
 A. Yes
 B. No
15. Amount of fluid given at first 24 hours (mls).....
16. Urine output at 24 hours (mls).....

17. Calculated mean rehydration factor.....

18. Whole blood Electrolyte level (mmol/L)

Electrolyte (mmol/L)	Sodium	Potassium
Day 1		
Day 2		
Day 3		
Day 4		
Day 5		

19. Correction of disturbance attempted

- A. Yes
- B. No

20. Correction Successful

- A. Yes
- B. No

21. BUN at day 5

- A. Urea.....
- B. Creatinine.....

22. Vomiting/ diarrhoea reported

- A. Yes
- B. No

23. Diuretic used

- A. Yes
- B. No

24. Outcome at day 7

- A. Alive
- B. Dead

Appendix 5

CONSENT EXPLANATION

My name is Dr. David Kabaa Kimani working as a house officer in the Department of Surgery in KNH. As part of my M.Med. Surgery requirements, I am doing a study related to severe burns and some of the complications encountered in their management. Specifically, I am looking at electrolytes and how deranged they get with burns.

The hospital's research and ethics committee have approved the study. Your involvement in the study will be highly appreciated but is not a pre-condition for you to receive due care while in this hospital. Any information will be treated with utmost confidentiality i.e. no information will be divulged to people other than those directly involved in your care.

There is no monetary or material gain for your participation in the study. Patients with similar conditions like you will hereafter benefit immensely from the findings of this study. However, you stand to benefit from the fact that if any abnormality / problem is detected, your primary doctor will immediately be informed to optimise your care.

You have a right to decline recruitment in this study and this will not deny you access to all necessary medical attention that is required by you. You can terminate your participation in the study at any time you wish. I wish you a quick recovery.

Please sign in the space provided below as a sign of your willingness to participate in the study.

Consent Form

Having fully understood the above explanation, I have voluntarily agreed to be enrolled in this study.

Patient's / Guardian's signDate.....

I have explained to the patient/ guardian the nature of the study, risks and benefits.

Investigator.....Date.....

SWAHILI FORMAT

Mimi ni Dr David Kimani kutoka Chuo Kikuu cha Nairobi, Idara ya Upasuaji. Ninafanya utafiti kuhusu madhara ya janga la moto. Haswa ninapima madini yanayokuwa katika damu ili nione kama yanabadirika baada ya kuungua. Nikipata kipimo kisichostahili, nitamjulisha daktari wako haraka sana ili marekebisho yafanywe.

Utafiti huu umeidhinishwa na Tume ya maadili mema katika utafiti ya Kenyatta Hospital. Hakuna madhara yoyote tunayokusudia kutokana na utafiti huu. Mbali, wagonjwa wa maafa ya moto waweza kufaidika baada ya utafiti. Hakuna pesa utakazolipwa kwa kuhusika na zoezi hili.

Ni haki yako kukataa kushirikishwa katika utafiti huu na hili haliwezi kukuzuia kupata matibabu mwafaka ukiwa katika hospitali hii. Aidha waweza kujiondoa kwenye zoezi hili wakati wowote.

Hakuna habari zozote zitakazotolewa kwa watu wengine bila ruhusa uliyotia sahihi mwenyewe. Kila habari kukuhusu zitalindwa kikamilifu. Unaweza kuuliza maswali wakati wowote kama hujaridhika kwa vyovyote. Nakutakia kupona haraka.

Tia sahihi kwenye nafasi uliyoachiwa hapa chini kama ishara kwamba umeelewa na umekubali kushirikishwa katika utafiti huu kwa hiari yako.

Fomu va kusajiliwa

Baada ya maelezo kuhusu utafiti huu, mimi nimekubali kusajiliwa katika zoezi hili.

Mgonjwa ama AnayemtunzaTarehe.....

Ninakili kwamba nimemuelezea mgonjwa au mtunzi wake yote yanayohusu utafiti huu.

Mtafiti..... Tarehe

Contact: Dkt. David K. Kimani – P.O. Box 368 KNH Code 00202 Nairobi, Kenya

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