CLINICAL REVIEW

The Care of the Adult Burn Patient

Chidinma Chima-Melton, MD, Cynthia Kim, MD and Maryum Merchant, MD

Case

A 55-year-old man with no prior medical history was transported to the ER via ambulance after an industrial gas explosion at work. He complains of face and chest pain but denies any dyspnea. On admission vital signs are unremarkable except sinus tachycardia to a heart rate of 125. Trauma workup is negative. He sustained 32% total body surface area (TBSA) second and third-degree flame burns to his face, neck, anterior chest and bilateral digits.

He is prophylactic intubated in the ER. After evaluation by burn surgeons, he is determined to be a candidate for surgery and you are consulted to help with his medical management.

How do you proceed?

Introduction

The care of the adult inpatient with partial or full thickness burns presents several unique challenges. When possible, these patients should be rapidly stabilized then transferred to an American Burn Association (ABA) certified Burn Center where they receive comprehensive care by a multidisciplinary team. Burn patients present with a number of complex medical issues including fluid shifts, increased susceptibility to infections, airway edema, ventilation, heat loss, electrolyte balance, and circulatory instability.

The ABA has established criteria for referring patients to a burn center[1]:

- Partial thickness burns of greater than 10% total body surface area.
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
- Third-degree burns in any age group.
- Electrical burns, including lightning injury.
- Chemical burns.
- Inhalation injury.
- Burn injury in patients with comorbid illnesses that could complicate recovery.
- Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality.
- Burn injury in patients who will require special social, emotional or rehabilitative intervention.

Epidemiology

Burns injuries are common. In the United States in 2016, there were over 40,000 hospitalizations related to burn injury and approximately 60% of these were admitted to one of the 128 burn centers.[2] Therefore, approximately 40% of burns are managed in hospitals without burn centers with a good chance that physicians without burn experience will manage them. Burn patients managed in burn centers have greater than 95% survival. Demographics of patients hospitalized with burns include: 68% male, 59% Caucasian, 20% African-American and 14% Hispanic. Flame burns are most common, representing 43%, 34% scald injuries, 9% contact or thermal burns, 4% electrical, and 3% are chemical burns.[3]

Burn Injury Pathophysiology

Burn injuries produce an initial hypermetabolic phase followed by a catabolic response which may trigger several maladaptive responses. The hypermetabolic dysregulation following an acute burn is broken down into an early ebb or hypodynamic phase lasting 24 to 72 hours and a later flow phase.[4] The ebb is characterized by decreased cardiac output, reduced metabolic rate and hyperglycemia due to increased gluconeogenesis and increased peripheral insulin resistance. Initially a sustained release of catecholamines and increased vascular permeability causes fluid shifts from the intravascular space into the interstitium. This leads to intravascular volume depletion necessitating volume resuscitation.

The flow phase in contrast may last for several weeks to months after the burn, and in some cases years. It is hyperdynamic with increased metabolic rate.[5] There is accelerated protein catabolism and breakdown of skeletal muscle that can cause severe protein wasting.[6]

Clinical Signs and Symptoms

Following the burn injury, there is initially increased blood pressure and heart rate, peripheral insulin resistance, as well as increased protein and lipid catabolism. This leads to increased resting energy expenditure, elevated body temperature, total body protein loss, muscle wasting, and stimulated synthesis of acute-phase proteins. Hypotension due to the intravascular volume depletion may cause lactic acidosis, acute renal failure and other types of end organ dysfunction. Hemoconcentration and tachycardia is often seen. Due to vascular leak, pulmonary edema can quickly develop causing hypoxia.

Initial Management

The initial management of the burn patient involves the usual steps of trauma resuscitation and assessment. The first step is to use the ABCDE survey: sequential assessments of the Airway, Breathing, Circulation, Disability (neurologic status), and Environment. The secondary survey of burn injuries involves completely exposing the patient to identify and score the depth and total Body Surface Area (tBSA) of the burns. This can be estimated using a "Lund and Browder chart.[7] The secondary survey should be done quickly, usually with nursing staff and surgeons. It may be staged to prevent loss of body heat and risk of hypothermia.

Fluid Resuscitation

Fluid resuscitation is based on the Parkland formula and with resuscitation goals within the first 24 hours.[8] Lactated ringers is the mainstay of fluid resuscitation since normal saline is associated with hyperchloremic metabolic acidosis. Overhydration can be dangerous and "fluid creep" represents one of the challenges in managing burn patients who have been over-exuberantly resuscitated. Over-resuscitation can lead to downstream issues including pulmonary edema, pleural effusions, tissue edema causing poor wound healing, CHF exacerbations, renal failure and bowel edema.

Dressing Changes and Wound Care

Appropriate wound care during the initial evaluation and between surgical debridement is essential for healing. There are a wide variety of dressings, ointments, and coverings available and usage may vary from center to center. Dressings should be capable of absorbing high amounts of exudate from the burn injury, maintain adequate humidity for granulation, assist in epithelialization, provide a barrier against bacterial infection, and accommodate movement. The location and depth of the wound, phase of healing, and bacterial burden all influence dressing choice and frequency of change. The dressings are often combined with ointments, solutions, and rinses that can assist in debridement, granulation/healing, and infection control.[9] Silver is commonly included in both the dressings and the ointments as an antimicrobial.[10]

Inhalation Injury

The oropharynx is capable of absorbing high amounts of heat energy and under typical circumstances thermal energy is typically dissipated before it reaches the lung or lower airway. However, significant heat exposure can cause rapid edema to the posterior oropharynx, tongue, vocal cords and subglottic space. Therefore, the treating physician should maintain a high degree of suspicion for such injury in patients with burns involving the face, nose, or mouth and in patients with prolonged smoke exposure in confined spaces.

We recommend that patients be evaluated by flexible fiberoptic laryngoscopy if they do not have alternate indications for intubation such as respiratory failure, large full thickness burns, inability to protect their airway, or signs of obstruction. Patients with a normal glottis and no evidence inhalation injury may be managed safely with frequent re-evaluations without intubation.[11]

Few reliable clinical indicators predict the presence of lower airway inhalational injury, so fiberoptic bronchoscopy is recommended for burn patients requiring intubation.[12] The presence of inhalational injury is an independent predictor of mortality in burn patients. This injury ranges from mild edema and minimal soot deposition to frank burns with ulcerations and epithelial sloughing resulting in airway casts.[13] Just as skin burn injury can evolve with time, inhalational injury can progress. Serial imaging and bronchoscopies may be required.

Initial evaluation should also include an arterial blood gas (ABG) with co-oximetry for detection of carboxyhemoglobin (COHb). Since carbon monoxide has 200 times the affinity for hemoglobin as oxygen, organs with the highest oxygen dependency are at greatest risk with carbon monoxide poisoning, the brain and cardiovascular system. HbCO levels higher than 3% in a non-smoker and 10% in a smoker indicate carbon monoxide exposure. If carbon monoxide poisoning is suspected, immediate application of 100% oxygen decreases the elimination half-life from 310 min at room air to 74 min.[12,14] Hyperbaric oxygen can also be used, if available. Patients exposed to burning textiles, paints, or plastics are also at risk for cyanide poisoning which can result in severe lactic acidosis and a narrowing of the arterial-venous oxygen gradient. A direct cyanide binder, such as hydroxocobalamin, should be used to treat cyanide poisoning in the setting of concomitant carbon monoxide poisoning.

Burn Surgeries

The standard of care for full thickness burn injury is an autograft or a split-thickness skin graft from an intact donor site on the same patient. Donor skin can be meshed and expanded to cover larger areas. Early excision and grafting of wounds may be associated with prevention of late stage contraction, decreased blood loss, infection, reduced length of hospitalization and increased graft acceptance.[15] After autografting, both the donor and the graft site require dedicated wound care. Donor site bleeding and pain can add morbidity to the autografting process. Electrical burns have unique pathology with soft tissue injury that may be deeper than initial appearance.

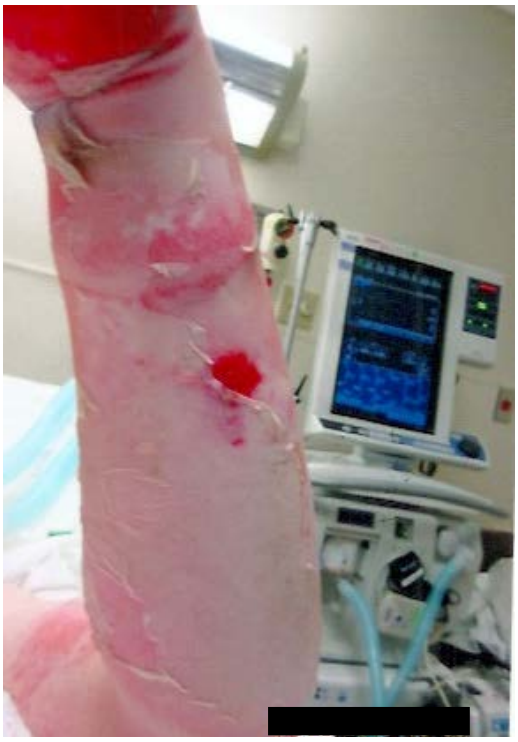

Figure 1: Full thickness circumferential burns to right arm

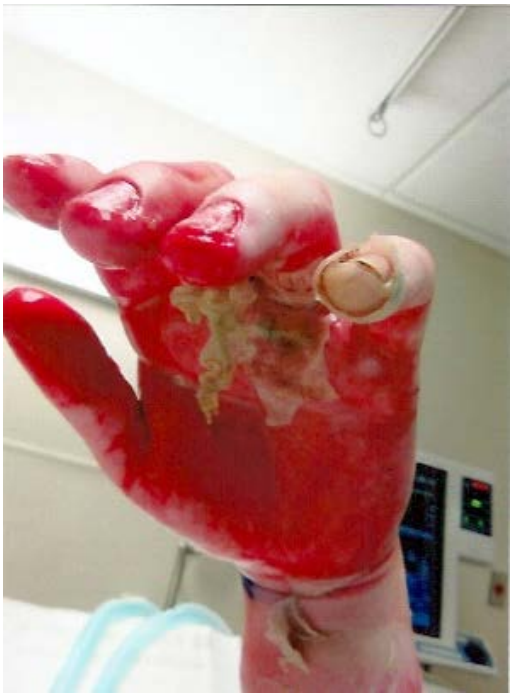

Figure 2: Degloving full thickness burn to right hand

Depending on the depth, a wound may require repetitive surgical debridement before readiness for autograft. In this case, allografts or homografts are used as temporary wound covers. Allografts or homografts are created from cadaveric donor tissue and promote re-epithelialization and more rapid wound healing when compared to dressings alone. Allografts can also be used as a protective cover over widely meshed autografts. Xenografts are created from different species and can be used as an intermediary cover until the wound bed is capable of accepting an autograft. Donor species for xenografts have included frogs, lizards, rabbits, dogs and pigs. Biosynthetic skin can also be used as temporary covers prior to autograft or as partial coverage in large burn areas. Limited evidence suggests biosynthetic skin substitutes have comparable efficacy as allografts.[9]

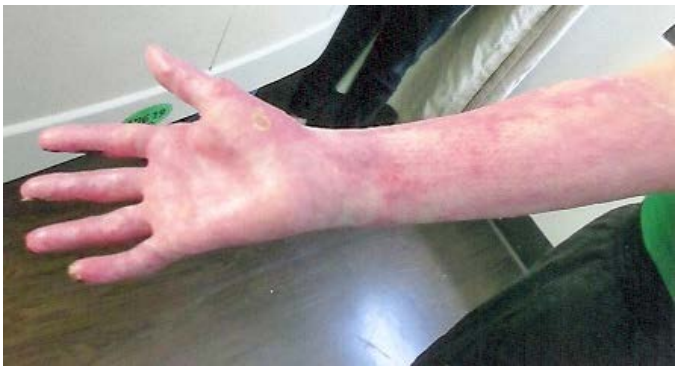

Figure 3: Post graft surgery results (Same patient from Figures 1 & 2)

Infection

One of the skin's most important roles is as a barrier against infection. Routine surveillance cultures are important in burn wound care and have been shown to decrease nosocomial infection. The wounds are typically first colonized with gram-positive organisms, later with susceptible gram-negative bacteria. Delayed wound closure increases risk for colonization with yeast, fungi and resistant bacteria.

Burn patients are unique in that they are both very susceptible to colonization as well as serve as a major source of bacteria. Cross-contamination is a special concern in areas used by multiple patients such as hydrotherapy, hyperbaric chambers and stretchers. Providers should use standard barrier precautions with all patients due to risk of cross contamination. Severe burn injury often requires invasive catheters, which also serve as nidus of infection. Diligent routine maintenance and early removal of these devices are essential parts of burn patient care. Patients with inhalational injury or intubated for very large burns are at increased risk for pneumonia.

Despite precautions, sepsis remains a common cause of death in severe burns. However, the detection of infection is difficult because the typical SIRS response is a normal part of burn injury. A consensus panel by the America Burn Association developed specific guidelines for the diagnosis of sepsis in burn patients that acknowledge burn patients' baseline hypermetabolic state.[16] This is a consensus definition due to the paucity of available data.

American Burn Association guidelines for Diagnosis of Sepsis	Sepsis
Temperature > 39C or < 36.5C HR >110 RR >25 Plt <100 Indications of insulin resistance or feeding intolerance + Documented presence of infection or response to antimicrobials	2 SIRS criteria Temperature >38C or <36C HR >90 PaCO2 <32 or RR >20 WBC >12 or < 4 + Documented source of infection

Procalcitonin, a precursor to calcitonin that increases in response to bacterial infection, can also be elevated in response to trauma and surgery. Data supporting its use for detection of bacterial infection in burn patients is variable but suggests it is a useful adjunctive test with levels >2ng/mL positively associated with severe bacterial infection.[17-19]

Nutrition

Nutrition is the cornerstone of burn care treatment from early resuscitation phase until the end of rehabilitation. Per European Society for Clinical Nutrition and Metabolism (ESPEN) recommendations, enteral feeding is preferred in major burn patients (> 20% TSBA burns) and should be started within 12 hours of injury.[20] High quality studies suggest early enteral nutrition is associated with clinical advantages including: attenuation of stress and hyper metabolic response; reduction of stress ulcers; and reduced risk of malnutrition. Protein requirements in major burn patients are typically higher than other critically ill patients and recommended at 1-2g/kg/day in adults and 1.5-3g/kg/day in children. Optimal glucose control between 100-150mg/dl is associated with better graft take, less infectious complications, and decreased mortality.

Patients with major burns also have increased micronutrient requirement. Major burns need early supplementation of supra nutritional amounts of zinc, copper, selenium as well as Vitamin B1, C, D and E to prevent deficiency related complications. Trace elements deficiencies can develop early in most severe burn patients. Other therapies to reduce hypermetabolic and hypercatabolic states are advocated including keeping the ambient temperature warm, early excisional surgery and occlusive dressings of the burn wounds.

Prealbumin levels are a better nutritional marker than albumin and appears to correlate well with graft healing.[21] However, prealbumin and albumin are also acute phase proteins and their values may be less sensitive to nutritional repletion during the acute phase of burn injury. However, as the acute phase subsides and adequate nutrition is maintained should result in gradual increase in pre-albumin levels. Persistently low prealbumin levels in the presence of normalizing C reactive protein may be a sign of protein or calorie deficiency. We typically trend prealbumin levels every 72 hours in our burn ICU patients.

Anemia and Transfusion of Blood Products

Severe burns significantly change hematologic parameters and anemia is common in patients with more than 10% TBSA burns. There is no consensus on when to transfuse. Most burn specialists prefer to maintain hemoglobin levels above 10g/dl, based on conventional theory that it promotes wound healing.[22] However, recent studies suggest that mild to moderate anemia has no effect on graft success if perfusion is maintained with fluid resuscitation.[23] A cross sectional study of patients admitted to a burn center in Iran reported a trend towards two-fold increased mortality in patients who received blood products compared to those who did not receive any blood transfusion.[24] A multi-center randomized trial of 345 patients with > 20% TBSA burns were randomized to a restrictive or liberal transfusion strategy. Patients assigned the restrictive strategy received 50% less blood (a median of eight units) compared with the liberal strategy (a median of 16 units). They reported similar rates of bloodstream infections, ventilator days, wound healing, organ problems and mortality in the two groups. They concluded the restrictive transfusion strategy made better use of blood resources without affecting health outcomes.[25]

Adjuvant Medications

Administration of high dose vitamin C (0.66 mg/kg/h for 24 h) has been shown to stabilize the endothelium, reducing capillary leak and fluid resuscitation requirement by about 30% in patients with major burns.[26] Several pharmacological methods have been shown to mitigate the hypermetabolic response to burns. Non-selective beta-blockers like propranolol, lower the heart rate and metabolic rate in severe burns.[27-29] Propranolol dose is titrated to reduce basal heart rate by 20% which decreases cytokines and stress hormone release.[29]

Oxandrolone administration has been associated with decreased mortality and length of hospital stay in major burn patients. Additional reported benefits include decreased protein catabolism and improved healing time. It is proposed that Oxandrolone be started after the acute resuscitation phase, typically at the end of first week with close monitoring of liver function.[30-31]

Gut Dysfunction in Burn Patients

Burn patients are uniquely susceptible to opioid induced constipation and opioid induced bowel dysfunction. We routinely start burn patients on scheduled stool softeners and gentle stimulant laxative on admission with holding parameters. Resistant opiate-induced constipation may require agents such as methylnaltrexone. Correction of electrolyte imbalance, adequate enteral intake, and early mobilization are also essential. Other gut complications include bacterial translocation from gut and abdominal compartment syndrome are common in major burn patients and are associated with increased

morbidity and mortality. Treatment options are limited and there is a need for rigorous studies to guide practice in these areas.[32]

Mobilization

Mobilizing patients with burns can be challenging because of their unique needs. However, early mobilization is essential, as burn patients can lose function and flexibility quickly.[33] Immobilization of a burned extremity is a common practice after debridement and grafting to prevent graft loss. This can increase joint stiffness and functional impairment. Several studies have shown that it is safe and feasible to implement early mobility of patients with major burns, however it requires participation of bedside nurses, working in close collaboration with physical therapists and surgeons. Burn patients have several unique factors that impacts PT participation: edema caused by inflammation from burn injury and fluid resuscitation; pain which can affect patient's vital and functional performance; skin integrity, including graft status; and psychological fears. These can be modified by use of proper splinting and positioning devices, orthotics to minimize loss of mobility, improving edema with compression, as well as reducing pain through activity and motion. Involving the patient and family throughout the treatment process also decreases fear and anxiety.[34]

Psychological Factors

As the survival of patients with major burns improves, it raises awareness of psychological challenges facing these patients. Most burn centers employ social workers, vocational counsellors and psychologists as part of the multidisciplinary burn team. Symptoms of depression and anxiety are very common during the recovery phase and PTSD, measured one month after injury affects burn victims more commonly than victims from other forms of injury. Compared with general population, burn patients have a higher rate of pre-existing psychological conditions, most commonly depression, substance misuse, and personality disorders which can adversely impact the outcomes with longer hospital stay and exacerbation of their preexisting conditions. During the recovery phase, patients begin to experience grief, as they become acutely aware of the impact of burn injuries on their lives, including loss of job, personal properties, change in appearance, loss of joint mobility and physical disability. Short term psychological counseling with pharmacotherapy can help with depression and anxiety during this phase.

Long term recovery typically begins after discharge from the hospital, when patients begin to reintegrate into society. For most patients with major burns, this stage may involve continued outpatient physical therapy, dressing changes, learning to compensate for their physical limitations and deal with social stressors such as return to work, change in body image, family strains and disruptions in daily life. Social support is very important during this time and there is evidence that adjustment to burn injuries improves over time. Groups such as the Phoenix Society provide peer support, community engagement and advocacy for burn survivors.[35]

REFERENCES

1. **American Burn Association**. Advanced burn life support Course, Provider Manual 2018 update. http://ameriburn.org/wp-content/uploads/2019/08/2018-abls-providermanual.pdf.
2. **American Burn Association**. National Inpatient Sample (HCUP-NIS: 2010 data); National Hospital Discharge Survey (2010 data); recent 100% hospitalization data from several states. http://ameriburn.org/who-we-are/media/burn-incidence-fact-sheet/
3. **American Burn Association**. ABA National Burn Repository 2015.
4. **Snell JA, Loh NH, Mahambrey T, Shokrollahi K**. Clinical review: the critical care management of the burn patient. *Crit Care*. 2013 Oct 7;17(5):241. doi: 10.1186/cc12706. Review. PubMed PMID: 24093225; PubMed Central PMCID: PMC4057496.
5. **Wolfe RR**. Review: acute versus chronic response to burn injury. *Circ Shock*. 1981;8(1):105-15. Review. PubMed PMID: 7016359.
6. **Hart DW, Wolf SE, Mlcak R, Chinkes DL, Ramzy PI, Obeng MK, Ferrando AA, Wolfe RR, Herndon DN**. Persistence of muscle catabolism after severe burn. *Surgery*. 2000 Aug;128(2):312-9. PubMed PMID: 10923010.
7. **Lund CC, Browder NC**. The estimation of areas of burns. *Surgery Gynecology and Obstetrics*. 1944;79:352.
8. **Mehta M, Tudor GJ**. Parkland Formula. [Updated 2019 Feb 28]. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2019 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537190/
9. **Pham C, Greenwood J, Cleland H, Woodruff P, Maddern G**. Bioengineered skin substitutes for the management of burns: a systematic review. *Burns*. 2007 Dec;33(8):946-57. Epub 2007 Sep 7. Review. PubMed PMID: 17825993.
10. **Atiyeh BS, Costagliola M, Hayek SN, Dibo SA**. Effect of silver on burn wound infection control and healing: review of the literature. *Burns*. 2007 Mar;33(2):139-48. Epub 2006 Nov 29. Review. PubMed PMID: 17137719.
11. **Muehlberger T, Kunar D, Munster A, Couch M**. Efficacy of fiberoptic laryngoscopy in the diagnosis of inhalation injuries. *Arch Otolaryngol Head Neck Surg*. 1998 Sep;124(9):1003-7. PubMed PMID: 9738810.
12. **Cancio LC**. Airway management and smoke inhalation injury in the burn patient. *Clin Plast Surg*. 2009 Oct;36(4):555-67. doi: 10.1016/j.cps.2009.05.013. Review. PubMed PMID: 19793551.
13. **Vaughn L, Beckel N**. Severe burn injury, burn shock, and smoke inhalation injury in small animals. Part 1: burn classification and pathophysiology. *J Vet Emerg Crit Care* (San Antonio). 2012 Apr;22(2):179-86. doi: 10.1111/j.1476-4431.2012.00727.x. Review. PubMed PMID: 23016809.

14. **Weaver LK**. Clinical practice. Carbon monoxide poisoning. *N Engl J Med*. 2009 Mar 19;360(12):1217-25. doi: 10.1056/NEJMcp0808891. Review. PubMed PMID: 19297574.
15. **Rowan MP, Cancio LC, Elster EA, Burmeister DM, Rose LF, Natesan S, Chan RK, Christy RJ, Chung KK**. Burn wound healing and treatment: review and advancements. *Crit Care*. 2015 Jun 12;19:243. doi: 10.1186/s13054-015-0961-2. Review. PubMed PMID: 26067660; PubMed Central PMCID: PMC4464872.
16. **Greenhalgh DG, Saffle JR, Holmes JH 4th, Gamelli RL, Palmieri TL, Horton JW, Tompkins RG, Traber DL, Mozingo DW, Deitch EA, Goodwin CW, Herndon DN, Gallagher JJ, Sanford AP, Jeng JC, Ahrenholz DH, Neely AN, O'Mara MS, Wolf SE, Purdue GF, Garner WL, Yowler CJ, Latenser BA; American Burn Association Consensus Conference on Burn Sepsis and Infection Group**. American Burn Association consensus conference to define sepsis and infection in burns. *J Burn Care Res*. 2007 Nov-Dec;28(6):776-90. PubMed PMID: 17925660.
17. **Mann EA, Wood GL, Wade CE**. Use of procalcitonin for the detection of sepsis in the critically ill burn patient: a systematic review of the literature. *Burns*. 2011 Jun;37(4):549-58. doi: 10.1016/j.burns.2010.04.013. Epub 2010 Jun 7. Review. Erratum in: Burns. 2011 Sep;37(6):1085. PubMed PMID: 20537467.
18. **Kim HS, Yang HT, Hur J, Chun W, Ju YS, Shin SH, Kang HJ, Lee KM**. Procalcitonin levels within 48 hours after burn injury as a prognostic factor. *Ann Clin Lab Sci*. 2012 Winter;42(1):57-64. PubMed PMID: 22371911.
19. **Barati M, Alinejad F, Bahar MA, Tabrisi MS, Shamshiri AR, Bodouhi NO, Karimi H**. Comparison of WBC, ESR, CRP and PCT serum levels in septic and non-septic burn cases. *Burns*. 2008 Sep;34(6):770-4. doi: 10.1016/j.burns.2008.01.014. Epub 2008 May 29. PubMed PMID: 18513877.
20. **Rousseau AF, Losser MR, Ichai C, Berger MM**. ESPEN endorsed recommendations: nutritional therapy in major burns. *Clin Nutr*. 2013 Aug;32(4):497-502. doi: 10.1016/j.clnu.2013.02.012. Epub 2013 Mar 14. Erratum in: Clin Nutr. 2013 Dec;32(6):1083. PubMed PMID: 23582468.
21. **Moghazy AM, Adly OA, Abbas AH, Moati TA, Ali OS, Mohamed BA**. Assessment of the relation between prealbumin serum level and healing of skin-grafted burn wounds. *Burns*. 2010 Jun;36(4):495-500. doi: 10.1016/j.burns.2009.05.014. Epub 2009 Sep 18. PubMed PMID: 19766398.
22. **Bains JW, Crawford DT, Ketcham AS**. Effect of chronic anemia on wound tensile strength: correlation with blood volume, total red blood cell volume and proteins. *Ann Surg*. 1966 Aug;164(2):243-6. PubMed PMID: 5915934; PubMed Central PMCID: PMC1477256.
23. **Agarwal P, Prajapati B, Sharma D**. Evaluation of skin graft take following post-burn raw area in normovolaemic anaemia. *Indian J Plast Surg*. 2009 Jul;42(2):195-8. doi: 10.4103/0970-0358.59281. PubMed PMID: 20368857; PubMed Central PMCID: PMC2845364.
24. **Tavousi SH, Ahmadabadi A, Sedaghat A, Khadem-Rezaiyan M, Yaghoubi Moghaddam Z, Behrouzian MJ, Nemati S, Saghafi H**. Blood transfusion in burn patients: Triggers of transfusion in a referral burn center in Iran. *Transfus Clin Biol*. 2018 Feb;25(1):58-62. doi: 10.1016/j.tracli.2017.07.003. Epub 2017 Aug 31. PubMed PMID: 28838856.
25. **Palmieri TL, Holmes JH 4th, Arnoldo B, Peck M, Potenza B, Cochran A, King BT, Dominic W, Cartotto R, Bhavsar D, Kemalyan N, Tredget E, Stapelberg F, Mozingo D, Friedman B, Greenhalgh DG, Taylor SL, Pollock BH**. Transfusion Requirement in Burn Care Evaluation (TRIBE): A Multicenter Randomized Prospective Trial of Blood Transfusion in Major Burn Injury. *Ann Surg*. 2017 Oct;266(4):595-602. doi: 10.1097/SLA.0000000000002408. PubMed PMID: 28697050; PubMed Central PMCID: PMC5848498.
26. **Tanaka H, Matsuda T, Miyagantani Y, Yukioka T, Matsuda H, Shimazaki S**. Reduction of resuscitation fluid volumes in severely burned patients using ascorbic acid administration: a randomized, prospective study. *Arch Surg*. 2000 Mar;135(3):326-31. PubMed PMID: 10722036.
27. **Herndon DN, Nguyen TT, Wolfe RR, Maggi SP, Biolo G, Muller M, Barrow RE**. Lipolysis in burned patients is stimulated by the beta 2-receptor for catecholamines. *Arch Surg*. 1994 Dec;129(12):1301-4; discussion 1304-5. PubMed PMID: 7986160.
28. **Herndon DN, Hart DW, Wolf SE, Chinkes DL, Wolfe RR**. Reversal of catabolism by beta-blockade after severe burns. *N Engl J Med*. 2001 Oct 25;345(17):1223-9. PubMed PMID: 11680441.
29. **Mohammadi AA, Bakhshaeekia A, Alibeigi P, Hasheminasab MJ, Tolide-ei HR, Tavakkolian AR, Mohammadi MK**. Efficacy of propranolol in wound healing for hospitalized burn patients. *J Burn Care Res*. 2009 Nov-Dec;30(6):1013-7. doi: 10.1097/BCR.0b013e3181b48600. PubMed PMID: 19826272.
30. **Pham TN, Klein MB, Gibran NS, Arnoldo BD, Gamelli RL, Silver GM, Jeschke MG, Finnerty CC, Tompkins RG, Herndon DN**. Impact of oxandrolone treatment on acute outcomes after severe burn injury. *J Burn Care Res*. 2008 Nov-Dec;29(6):902-6. doi: 10.1097/BCR.0b013e31818ba14d. PubMed PMID: 18849836; PubMed Central PMCID: PMC3958934.
31. **Wolf SE, Edelman LS, Kemalyan N, Donison L, Cross J, Underwood M, Spence RJ, Noppenberger D, Palmieri TL, Greenhalgh DG, Lawless M, Voigt D, Edwards P, Warner P, Kagan R, Hatfield S, Jeng J, Crean D, Hunt J, Purdue G, Burris A, Cairns B, Kessler M, Klein RL, Baker R, Yowler C, Tutulo W, Foster K, Caruso D, Hildebrand B, Benjamin W, Villarreal C, Sanford AP, Saffle J**. Effects of oxandrolone on outcome measures in the severely burned: a multicenter prospective randomized double-blind trial. *J*

Burn Care Res. 2006 Mar-Apr;27(2):131-9; discussion 140-1. PubMed PMID: 16566555.

32. **Ng JW, Cairns SA, O'Boyle CP**. Management of the lower gastrointestinal system in burn: A comprehensive review. *Burns*. 2016 Jun;42(4):728-37. doi: 10.1016/j.burns.2015.08.007. Epub 2016 Jan 13. Review. PubMed PMID: 26774605.
33. **Procter F**. Rehabilitation of the burn patient. *Indian J Plast Surg*. 2010 Sep;43(Suppl):S101-13. doi: 10.4103/0970-0358.70730. PubMed PMID: 21321643; PubMed Central PMCID: PMC3038404.
34. **Al-Mousawi AM, Mecott-Rivera GA, Jeschke MG, Herndon DN**. Burn teams and burn centers: the importance of a comprehensive team approach to burn care. *Clin Plast Surg*. 2009 Oct;36(4):547-54. doi: 10.1016/j.cps.2009.05.015. Review. PubMed PMID: 19793550; PubMed Central PMCID: PMC2801053.
35. "Support for Burn Survivors." Phoenix Society, Retrieved from http://www.phoenix-society.org/ on August 17, 2019.