OPEN ACCESS

Research Article

Transdermal fluid loss in severely burned patients

Transdermaler Flüssigkeitsverlust bei Schwerbrandverletzten

Abstract

Introduction: The skin protects against fluid and electrolyte loss. Burn injury does affect skin integrity and protection against fluid loss is lost. Thus, a systemic dehydration can be provoked by underestimation of fluid loss through burn wounds.
Purpose: We wanted to quantify transdermal fluid loss in burn wounds.
Method: Retrospective study. 40 patients admitted to a specialized burn unit were analyzed and separated in two groups without (Group A) or with (Group B) hypernatremia. Means of daily infusion-diuresis-ratio (IDR) and the relationship to totally burned surface area (TBSA) were analyzed.
Results: In Group A 25 patients with a mean age of 47±18 years, a mean TBSA of 23±11%, and a mean abbreviated burned severity index (ABSI) score of 6.9±2.1 were summarized. In Group B 15 patients with a mean age of 47±22 years, a mean TBSA of 30±13%, and a mean ABSI score of 8.1±1.7 were included. Statistical analysis of the period from day 3 to day 6 showed a significant higher daily IDR-amount in Group A (Group A vs. Group B: 786±1029 ml vs. –181±1021 ml; $p<0.001$) and for daily IDR-TBSA-ratio (Group A vs. Group B: 40±41 ml/% vs. –4±36 ml/%; $p<0.001$).
Conclusions: There is a systemic relevant transdermal fluid loss in burn wounds after severe burn injury. Serum sodium concentration can be used to calculate need of fluid resuscitation for fluid maintenance. There is a need of an established fluid removal strategy to avoid water and electrolyte imbalances.

Keywords: water-electrolyte imbalance, burns, dehydration, critical care, intensive care

Thomas Namdar[1]
Peter L. Stollwerck[1]
Felix H. Stang[1]
Frank Siemers[1]
Peter Mailänder[1]
Thomas Lange[1]

1 Department of Plastic Surgery, Hand Surgery, Burn Unit, University Hospital Schleswig-Holstein, Lübeck, Germany

Zusammenfassung

Einführung: Die intakte Haut dient als Schutz vor Flüssigkeits- und Elektrolytverlust. Die thermisch geschädigte Haut kann diese Funktionen nicht mehr vollständig erfüllen Eine Unterschätzung des transdermalen Flüssigkeitsverlust in Verbrennungswunden kann eine systemische Dehydratation provozieren.
Fragestellung: Wie hoch ist der transdermale Flüssigkeitsverlust in Relation zur verbrannten Körperoberfläche (VKOF) bei oberflächlichen Verbrennungen mit Blasenbildung?
Methoden: Retrospektive Studie an 40 Schwerbrandverletzten. Aufteilung des Gesamtkollektivs in Gruppe A (normonatriäm) und Gruppe B (hypernatriäm). Das tägliche Infusions-Diurese-Verhältnis (IDV) in Relation zur VKOF wurde analysiert.
Ergebnisse: In Gruppe A (n=25) wurde ein Durchschnittsalter von 47±18 Jahren, eine VKOF von 23±11%, und ein abbreviated burned severity index (ABSI) von 6,9±2,1 ermittelt. In Gruppe B (n=15) betrug das mittlere Alter 47±22 Jahre, die VKOF 30±13%, und der ABSI 8,1±1,7. Für den Zeitraum vom 3. bis 6. Tag nach Brandverletzung zeigten sich ein signifikant höheres IDV ($p<0,001$) und IDV-VKOF-Verhältnis ($p<0,001$) in Gruppe A.

Schlussfolgerung: Bei Schwerbrandverletzten kann der transdermale Flüssigkeitsverlust systemisch relevant werden und sollte im individuellen Infusionsregime berücksichtigt werden.

Schlüsselwörter: Elektrolytentgleisung, Verbrennung, Dehydratation, Intensivmedizin

Introduction

The skin is the largest human organ composed by dermis and epidermis and protects against fluid and electrolyte loss [1]. Burn injury is a dynamic process that peaks at about 3 days after thermal trauma and does affect the skin partially or completely [2], [3]. Partial thickness burns usually blister and protection against fluid loss is lost. Lehnhardt et al. [4] demonstrated, that a patient's entire amount of serum proteins accumulates in burn wound fluid within approximately 24 h after a 20% TBSA burn injury. Additionally, in relation to the totally burned surface area (TBSA) a systemic relevant fluid loss can occur.

In severely burned patients posttraumatic changes release systemic effects. Microvascular integrity is lost, and a plasma-like fluid leaks into the interstitial space, producing edema. During burn shock plasma volume must be maintained to ensure tissue oxygen delivery. The time after injury at which capillary integrity is restored varies individually [5]. For initial fluid resuscitation several well established formulas exist, that differ in kind and amount of infusion regimen [1], [6], [7], [8], [9], [10]. After acute phase due to recovering of vascular integrity the circulating fluid volume has to be normalized. Hereby extensive water and electrolyte shifts can provoke systemic dehydration [11]. There is a lack of knowledge in fluid loss amount due to burn wounds and post burn shock fluid regimen.

Study purpose: Through this study, we wanted to determine daily transdermal fluid loss in burn wounds.

Materials and methods

Patients admitted to our specialized burn unit were analyzed retrospectively for the incidence and duration of hypernatremia (>146 mmol/l) as a sign for dehydration. Patients with an age >18 years and a TBSA >10% were included. Split skin grafting to deep dermal burn wounds was performed within 7 days after trauma. Burn victims with a TBSA <10% and with a body temperature <36.5° or >37.8° Celsius were excluded. Neither body-weight-, nor thermodilution-, nor central venous pressure measurements were performed regularly.

All patients were treated during the first 24 hours after burn injury guided by the Parkland Formula. No colloids and no catecholamines were administered within the first 48 hours after burn injury. Inhalation injury was diagnosed on admission by bronchoscopy. Fluid equilibration during post-burn shock phase was achieved by application of colloids, albumin, high-caloric parenteral nutrition and Ringer's solution. Fatty acids, amino acids, vitamins and zinc were infused additionally. All burn wounds received an initial debridement and were covered with moist antiseptic dressing, which was renewed daily. A urinary catheter and a fecal collector were placed in all patients. A naso-jejunal tube was placed in all patients for high-caloric enteral alimentation.

Patients' characteristics (age, sex, TBSA, ABSI) and means of daily infusion-diuresis-ratio (IDR) were compared. Infusion volume summarizes saline and colloid infusion, as well as enteral nutrition/oral fluid supply. Diuresis and body temperature were measured by permanent urine catheter. Also means of IDR-TBSA-ratio were analyzed to investigate a potential relationship between burned surface area and transdermal fluid loss. Daily perspiration was calculated with an amount of 500 ml. The time period from day 3 to day 6 was analyzed. Split skin grafting to deeply injured areas was performed on day 7 after trauma.

Statistics were performed with SPSS 15.0 (SPSS Inc. Chicago, USA). To estimate significance the Mann-Whitney-U-Test was performed. Results are written in mean value ± standard deviation.

Results

Forty patients with a mean age of 47±19 years, a TBSA of 26±12%, and a mean ABSI score of 7.3±2, were included. Patients were separated in two groups without (Group A) or with (Group B) hypernatremia.

In **Group A** 25 patients (7 female; 18 male) with a mean age of 47±18 years (minimum: 14 years; maximum: 73 years), a mean TBSA of 23±11% (minimum: 10%; maximum: 60%), and a mean ABSI score of 6.9±2.1 (minimum: 3; maximum: 11) were summarized. The partial-thickness TBSA was 19.4±8.3% and the full-thickness TBSA was 3.8±4.8%. Ten patients were mechanically ventilated on admission. Eight patients showed an inhalation injury. Escharotomy was necessary in two individuals.

In **Group B** 15 patients (5 female; 10 male) with a mean age of 47±22 years (minimum: 17 years; maximum: 83 years), a mean TBSA of 30±13% (minimum: 12%; maximum: 70%), and a mean ABSI score of 8.1±1.7 (minimum: 5; maximum: 11) were included. The partial-thickness TBSA was 23.1±14.8% and the full-thickness TBSA was 7.2±7.1%. Escharotomy was necessary in 1 individual. Fourteen patients were mechanically ventilated on admission. Five individuals showed an inhalation injury. Hypernatremia occurred on day 5±1.4 and persisted for 4.6±2.7 days (Table 1).

Table 1: Patients' characteristics written in mean ± standard deviation (sex, age [years], totally burned surface area (TBSA) [%], ABSI, burn shock fluid [ml/kg/%-TBSA], daily infusion-diuresis-ratio (IDR) [ml] and daily IDR/TBSA [ml/%] for day 3 to 6

	Group A (n=25)	Group B (n=15)	p-value
Sex (female/male)	7/18	5/10	
Age [years]	47±18	47±22	n.s.
TBSA [%]	23±11	30±13	n.s.
ABSI	6.9±2.1	8.1±1.7	n.s.
Burn shock fluid [ml/kg/%-TBSA]	4.9±3	5.8±3	n.s.
daily IDR (day 3 to 6) [ml]	786±1029	–181±1021	p<0.001
daily IDR/%-TBSA (day 3 to 6) [ml/%]	40±41	–4±36	p<0.001

No significant difference in ABSI between both groups was found (p=0.078). There was no significant difference between both groups for fluid resuscitation amount within the first 24 hours (Group A vs. Group B 4.9±3 ml/kg/%-TBSA vs. 5.8±3 ml/kg/%-TBSA; p=0.32). Statistical analysis of the period from day 3 to day 6 showed a significant higher daily IDR-amount in Group A (Group A vs. Group B: 786±1029 ml vs. –181±1021 ml; p<0.001 (Figure 1)) and for daily IDR-TBSA-ratio (Group A vs. Group B: 40±41 ml/%-TBSA vs. –4±36 ml/%-TBSA; p<0.001 (Figure 2)). Burn victims with the need of a mechanical ventilation showed an increased risk for hypernatremia (Pearson chi-square 11.1 (p=0.001); Odds ratio 21.0 (Confidence interval: 2.4–186)). Inhalation injury did not show a relationship to a hypernatremic state.

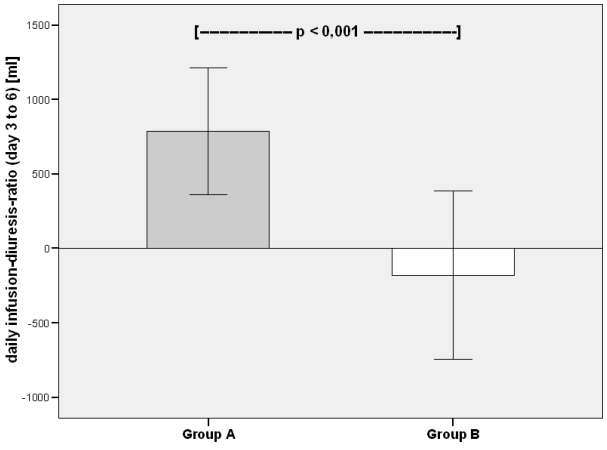

Figure 1: Daily infusion-diuresis-ratio (IDR) from day 3 to day 6 [ml] divided in Group A (without hypernatremia) and Group B (with hypernatremia)

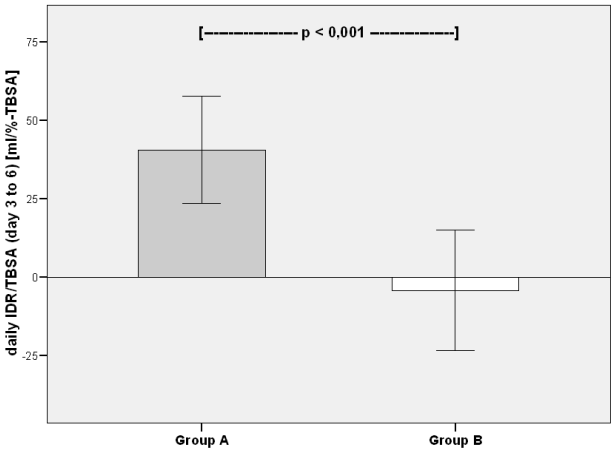

Figure 2: Daily infusion-diuresis-ratio (IDR)/totally burned surface area (TBSA)-ratio from day 3 to day 6 [ml/%-TBSA] divided in Group A (without hypernatremia) and Group B (with hypernatremia)

Discussion

Severe burn injury induce a capillary leak characterized by fluid dysregulation, electrolyte imbalance, loss of proteins and circulatory insufficiency [1], [4]. Fluid resuscitation within the first 24 hours after burn injury is commonly monitored by measuring urinary output, haematocrit and mean arterial pressure. After the burn shock induced capillary leak is closed the majority of the infused saline has to be removed and a systemic fluid equilibrium has to be maintained.

Urinary output is an unreliable guide for the patient's hydration status >48 hours after burn injury. Respiratory water losses, osmotic diuresis secondary to accentuated glucose intolerance, high protein feeding, and derangements in hormone mechanisms contribute to increased fluid losses despite an adequate urine output [12]. The transdermal fluid loss after burn injury is terminated by complete reepithelization or after skin grafting surgery. In patients with a TBSA >10% transdermal burn wound fluid loss can have systemic effects. There is no formula describing the amount of transdermal fluid loss in burned patients.

In burned patients serum sodium concentration is reported as a guide for controlling fluid replacement >48 hours after burn injury and should be in normal range [1]. In-hospital hypernatremia is commonly caused iatrogenically and can be a sign for systemic dehydration [13], [14], [15]. Risk factors for a serum sodium dysregulation are unconsciousness, parenteral feeding, age, renal insufficiency [14], [16]. In severely burned patient hypernatremia is frequently observed due to the extensive initial fluid resuscitation conditioned by capillary hyperpermeability [5], [7], [17]. In critically ill patients elevated serum sodium concentration is associated with an adverse outcome [18], [19].

There are several well established algorithms of burn shock resuscitation within the first 24–48 hours [5], [7], [17]. In this study, we tried to quantify transdermal fluid

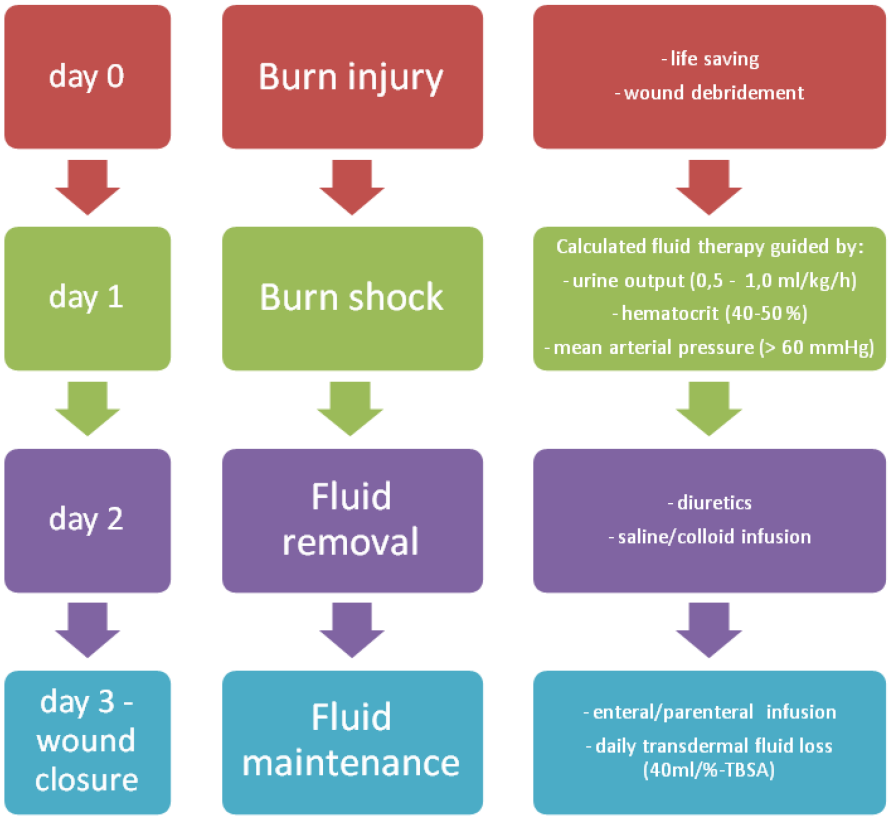

Figure 3: Time table of phases after burn injury and description of therapeutic strategy

loss until burn wound closure. The highest amount of fluid loss via thermally injured skin is expected during day 1 to day 6 after trauma. Within the first 48 hours after burn injury it is difficult to quantify transdermal fluid loss, because of the extensive amounts of infused/removed volume. Therefore we analyzed IDR differences between Group A and Group B for the time period of day 3 to day 6.The fluid resuscitation during burn shock (day 1) in our study was guided by Parkland Formula [8], and fluid removal (day 2) was performed adjusted by measurement of urine output, hematocrit, central venous pressure and blood pressure. We observed a close relationship between hypernatremia and negative daily IDR. We found in Group B a significantly lower amount of daily IDR ($p<0.001$) and IDR-TBSA-ratio ($p<0.001$). In our opinion, a balanced daily IDR is not sufficient for patients with severe burn injury (>10% TBSA). The transdermal fluid loss in second degree burned skin areas has to be kept in mind. The incidence of an in-hospital hypernatremic state after severe burn injury – even in patients with a normal diuresis (0.5–1 ml/kg body-weight/hour) – should be noticed by specialized physicians and needs to be treated immediately to avoid pronounced systemic dehydration and its neurologic sequelae and consequences in wound healing [18], [20], [21].

Even in specialized high-volume burn centers the incidence of post-burn hypernatremia is not avoidable. Only little attention is paid to the post-resuscitation phase and there is still a lack of fluid removal algorithms. Our findings show, that there is a relevant transdermal fluid loss after severe burn injury. We found in Group B (hypernatremic) a significant lower amount of daily IDR. A negative daily fluid balance with a hypernatremic state indicates a relevant systemic dehydration [13], [14]. Our data suggest that a daily transdermal fluid loss due to second degree burn wounds of approximately +40 ml/kg/%-TBSA should be calculated after burn injury. The transdermal fluid loss will be reduced in relation to burn wound closure. In our opinion, severely burned patients do have a transdermal fluid loss which should be replaced for fluid maintenance until burn wound closure is achieved (Figure 3).

Limitations

Because of the small population and sometimes different diuresis protocols used further investigation is recommended to figure out a safe method of fluid removal after burn injury. Choice and amount of administered diuretics, as well as a definition of daily IDR to minimize dehydration risk are not described in the literature.

Conclusions

There is a systemic relevant transdermal fluid loss in second degree burn wounds after severe burn injury. Serum sodium concentration can be used to calculate need of fluid resuscitation for fluid maintenance. There is a need of a well established fluid removal strategy in severely burned patient to avoid water and electrolyte imbalances.

Notes

Competing interests

The authors declare that they have no competing interests.

References

1. Herndon T. Total burn care. Third ed. Philladelphia: Saunders; 2007.
2. Gamelli RL, Paxton TP, O'Reilly M. Bone marrow toxicity by silver sulfadiazine. Surg Gynecol Obstet. 1993;177(2):115-20.
3. Jarrett F, Ellerbe S, Demling R. Acute leukopenia during topical burn therapy with silver sulfadiazine. Am J Surg. 1978;135(6):818-9. DOI: 10.1016/0002-9610(78)90173-3
4. Lehnhardt M, Jafari HJ, Druecke D, Steinstraesser L, Steinau HU, Klatte W, Schwake R, Homann HH. A qualitative and quantitative analysis of protein loss in human burn wounds. Burns. 2005;31(2):159-67. DOI: 10.1016/j.burns.2004.08.015
5. Pham TN, Cancio LC, Gibran NS. American Burn Association practice guidelines burn shock resuscitation. J Burn Care Res. 2008;29(1):257-66. DOI: 10.1097/BCR.0b013e31815f3876
6. Murison MS, Laitung JK, Pigott RW. Effectiveness of burns resuscitation using two different formulae. Burns. 1991;17(6):484-9. DOI: 10.1016/0305-4179(91)90077-T
7. Baxter CR. Fluid volume and electrolyte changes of the early postburn period. Clin Plast Surg. 1974;1(4):693-703.
8. Baxter CR, Shires T. Physiological response to crystalloid resuscitation of severe burns. Ann N Y Acad Sci. 1968;150(3):874-94. DOI: 10.1111/j.1749-6632.1968.tb14738.x
9. Shirani KZ, Vaughan GM, Mason AD Jr, Pruitt BA Jr. Update on current therapeutic approaches in burns. Shock. 1996;5(1):4-16. DOI: 10.1097/00024382-199601000-00004
10. Rackow EC, Falk JL, Fein IA, Siegel JS, Packman MI, Haupt MT, Kaufman BS, Putnam D. Fluid resuscitation in circulatory shock: a comparison of the cardiorespiratory effects of albumin, hetastarch, and saline solutions in patients with hypovolemic and septic shock. Crit Care Med. 1983;11(11):839-50. DOI: 10.1097/00003246-198311000-00001
11. Zawacki BE. Reversal of capillary stasis and prevention of necrosis in burns. Ann Surg. 1974;180(1):98-102. DOI: 10.1097/00000658-197407000-00015
12. Warden GD, Wilmore DW, Rogers PW, Mason AD, Pruitt BA Jr. Hypernatremic state in hypermetabolic burn patients. Arch Surg. 1973;106(4):420-7.
13. Janz T. Sodium. Emerg Med Clin North Am. 1986;4(1):115-30.
14. Lin M, Liu SJ, Lim IT. Disorders of water imbalance. Emerg Med Clin North Am. 2005;23(3):749-70, ix. DOI: 10.1016/j.emc.2005.03.001
15. Snyder NA, Feigal DW, Arieff AI. Hypernatremia in elderly patients. A heterogeneous, morbid, and iatrogenic entity. Ann Intern Med. 1987;107(3):309-19.
16. Adrogué HJ, Madias NE. Hypernatremia. N Engl J Med. 2000;342(20):1493-9. DOI: 10.1056/NEJM200005183422006
17. Heimbach D, Engrav L, Grube B, Marvin J. Burn depth: a review. World J Surg. 1992;16(1):10-5. DOI: 10.1007/BF02067108
18. Fisher LA, Ko N, Miss J, Tung PP, Kopelnik A, Banki NM, Gardner D, Smith WS, Lawton MT, Zaroff JG. Hypernatremia predicts adverse cardiovascular and neurological outcomes after SAH. Neurocrit Care. 2006;5(3):180-5. DOI: 10.1385/NCC:5:3:180
19. Polderman KH, Schreuder WO, Strack van Schijndel RJ, Thijs LG. Hypernatremia in the intensive care unit: an indicator of quality of care? Crit Care Med. 1999;27(6):1105-8. DOI: 10.1097/00003246-199906000-00029
20. Hoorn EJ, Betjes MG, Weigel J, Zietse R. Hypernatraemia in critically ill patients: too little water and too much salt. Nephrol Dial Transplant. 2008;23(5):1562-8. DOI: 10.1093/ndt/gfm831
21. Kuroda T, Harada T, Tsutsumi H, Kobayashi M. Hypernatremia deepens the demarcating borderline of leukocytic infiltration in the burn wound. Burns. 1997;23(5):432-437. DOI: 10.1016/S0305-4179(97)00016-8
22. Perel P, Roberts I. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev. 2007;(4):CD000567. DOI: 10.1002/14651858.CD000567.pub3

Corresponding author:
Thomas Namdar, M.D.
Department of Plastic Surgery, Hand Surgery, Burn Unit, University Hospital Schleswig-Holstein, Campus Lübeck, Ratzeburger Allee 160, 23538 Lübeck, Germany, Fax: 0049-451-500-2190, Phone: 0049-451-500-2061
thomas.namdar@uk-sh.de

Please cite as
Namdar T, Stollwerck PL, Stang FH, Siemers F, Mailänder P, Lange T. Transdermal fluid loss in severely burned patients. GMS Ger Med Sci. 2010;8:Doc28.
DOI: 10.3205/000117, URN: urn:nbn:de:0183-0001173

This article is freely available from
http://www.egms.de/en/journals/gms/2010-8/000117.shtml

Received: *2010-06-11*
Revised: *2010-09-24*
Published: *2010-10-26*