

## Short Communication

# RISK FACTOR OF METABOLISM ALTERATION IN BURN PATIENTS

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## ABSTRACT

Severe burn causes a catabolic response with profound effects on glucose and muscle protein metabolism. Our aim is to determine whether a changes of metabolism and inflammatory protein like serum albumin and CRP and if their level can predict mortality in burn patients. Twenty seven burn patients were included in this study and compared with twenty seven healthy donors. A significant differences ( $p < 0.001$ ) between burn patients and control group were observed in total protein, albumin, globulin, high sensitive C reactive protein, urea, and uric acid. While a non significant increase ( $p > 0.05$ ) in glucose level was observed in burn patients. On the other hand a non significant difference ( $p > 0.05$ ) between burn degrees in same parameters were observed. We conclude from the present study that the metabolic changes occur in patients with varying degrees of burns and its values didn't depended on burn degree, although its values can be used to predict the mortality of burn patients.

**Keywords:** Burn, hypermetabolism, albumin, risk factor, glucose.

## INTRODUCTION

Burn injuries are among the most destructive of all injuries and a major global public health problem (Peck *et al.*, 2008). The changes in patient metabolism following a major burn may be seen for more than 12 months after the initial injury (Norbury *et al.*, 2006). Hypermetabolism is occur in response to severe burns patient (Williams *et al.*, 2009; Machado *et al.*, 2011). It is characterized by hyperdynamic circulatory, physiology, catabolic, immune system responses, and increased risk for infection (Hart *et al.*, 2000). These changes are responsible for much of the morbidity and mortality seen with such an injury (Norbury *et al.*, 2006). The use of prognostic factors has been attempted in burn patients, such as sex, age, total burned body surface area (BSA), full-thickness injuries, and serum albumin levels (Tobiasen *et al.*, 1982; Hörbrandt *et al.*, 2003). However early identification of patients with the greatest risk is essential for their management, and their overall treatment (Colleen, 1998). Severe burn causes a catabolic response with profound effects on glucose and muscle protein metabolism. This response is characterized by hyperglycemia and loss of muscle mass, both of which have been associated with significantly increased morbidity and mortality (Ballian *et al.*, 2010). Plasma C-reactive protein (CRP) is a biomarker commonly used to assess the inflammatory response, and increases are associated with increased inflammation, infection, or sepsis (Lavrentieva *et al.*, 2007). Hypoalbuminemia is a common clinical deficiency in burn patients and is associated with complications related to increased extravascular fluid, including edema,

abnormal healing, and susceptibility to sepsis (Aguayo-Becerra *et al.*, 2013). Urea is a measure of the major end product of protein metabolism (Grove *et al.*, 1995). Uric acid is an end product of purine metabolism. Uric acid is more toxic to tissue than xanthine or hypoxanthine (Yadav, 2010).

Some prognostic tools do not include biochemical parameters, whereas others consider them together with comorbidities it would be ideal to have a biomarker to predict the risk of developing severe infection. The purpose of this study was to determine whether a changes of metabolism and the inflammatory protein like serum albumin and CRP and if their level can predict mortality in burn patients.

## MATERIALS AND METHODS

A total of 27 patients with burn attending Al-Kindy Hospital in Baghdad city were participated in this study. We obtained general information about each patient, including age, sex, etiology, location of burns and degree burn. As a control of 27 healthy individual with matches were included in this study. Five ml were collected from healthy donors and patients. The blood sample was centrifuged at 3000 rpm for 5 min after allowing the blood to clot at room temperature. Serum separated and transferred into test tube, and stored at  $-20^{\circ}\text{C}$  until being used. Total protein was measured by Biuret method, and albumin levels were measured by (Bromo Cresol Green) BCG method. High sensitive CRP and IL10 were measured using Enzyme Linked Immuno Assay (ELISA) kits.

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The statistical software (SPSS v 15; Chicago, IL, USA) was used. The data were analyzed using unpaired *t*-test and person correlation coefficients. Differences were considered significant when  $P < 0.05$ .

## RESULTS AND DISCUSSION

Demographic study of burn patients presented that among 27 burn patients 66.6% were female, 33.3% were male of 14-66 years age. Including 40.7% of burn patients were second degree, 40.7% were third degree, and 18.5% were mixed of second and third degree. Burn wounds were caused by flame in 23 cases, by hot water in 2 cases, by electricity in 2 cases (Table 1).

Hypermetabolic response in burn patients occurs by altering the physiological and biochemical environment characterized by increased metabolic rates, multi-organ dysfunction, muscle protein degradation, blunted growth, insulin resistance (Atiyeh *et al.*, 2008).

Total protein concentration was measured in the sera of control and burn patients according to biuret method. The results in table 2 showed the presence of a highly significant decrease in protein concentration in sera of burn patients in comparison to that of the control group and this decrease was due to the overwhelming protein losses by bleeding, where with bleeding the protein is lost along with the blood so the more deeper the burn is, the more bleeding and more protein-rich fluid leaks from the open burn wounds causes a high decrease in protein concentration (Lehnhardt *et al.*, 2005). This result was agreement with the results obtained by Manelli *et al.* (1998) who showed that low values of protein observed in burn patients are caused by several factors including microvascular hyper-permeability and inflammatory processes. Also the result was agreement with the results obtained by Lenhardrat *et al.* (2005) who found that protein levels in serum were significantly lower as compared to physiological levels.

A highly significant decrease of albumin level ( $p < 0.001$ ) was observed in the present study in sera of burn patients compared to control group. This result in line with the fact that skin is the major storage for albumin so whenever the skin got burned the albumin level will decrease (Aguayo-Becerra *et al.*, 2013). Burns affecting >20% of the body surface cause a major loss of extracellular fluids, thereby inducing shock by increasing vascular permeability and reducing plasma albumin from the wound exudations (Lehnhardt *et al.*, 2005). The results of the present study was agreement with Aguayo-Becerra *et al.* (2013) who suggests that hypoalbuminemia has a deleterious effect on patient survival but does have some limitations. Miquet-Rodríguez *et al.* (2013) reported a mortality rate of <10% in severely burn patients (2/23) in whom hypoalbuminemia was frequently observed,

demonstrating a significant association between the extent of the burn and the serum albumin level.

Globulin level in this study showed a highly significant decrease ( $p < 0.001$ ) in sera of burn patients compared to control group. This decrease is due to blood loss via damaged skin (Steven *et al.*, 2008). This result was agreement with Muhammad and Hayder (2011) who found that the total serum protein, albumin and globulin of male and female burn patients shows significant decrease ( $p < 0.01$ ).

Table 1. Demographic study of burn patients.

Category	Number (%)
Age in year	
0-19	6(22.2)
20-39	13(48.1)
40-59	7(25.9)
$\geq 60$	1(3.7)
Gender	
Male	9(66.6)
Female	18(33.3)
Burn degree	
Second II	11(40.7)
Third III	11(40.7)
Mix(II & III)	5(18.5)
Etiology	
Thermal	23(85.1)
Hot water	2(7.4)
Electricity	2(7.4)

The acute phase response develops in a wide range of acute and chronic inflammatory conditions. The physiological role of CRP is to bind to phosphocholine expressed on the surface of dead or dying cells in order to activate the complement system. Measuring CRP level is a screen for infectious and inflammatory diseases (Du Clos, 2000). A highly significant increase in hsCRP level ( $p < 0.001$ ) was observed in the present study in sera of burn patients compared to control group. This result was agreement with Jeschke *et al.* (2013) who found that significantly higher levels of CRP were found in large burns, and CRP values significantly correlated with burn size, survival and gender. Pileri *et al.* (2009) showed in their study that CRP levels were higher in septic than in non septic patients ( $p < 0.05$ ), but until day 15 day CRP values did not distinguish survivor from non survivor septic patients.

For many years a condition of hyperglycemia among patients suffering major burn injury was considered as a normal and desired response (Holm *et al.*, 2004; Hemmila *et al.*, 2008; Chatham *et al.*, 2008). However the present study showed a non significant increase of glucose level in sera of burn patients in compared to control group. This result may be due to insulin administration as therapy

Table 2. Mean values of serum total protein, albumin, globulin, hsCRP, glucose, urea, and uric acid.

Parameters	Control Mean± SD	Burn patient Mean± SD	P Value
Total protein(g/dl)	8.0484±1.1927	4.990±1.1783	p<0.001
Albumin(g/dl)	3.7413±0.73	2.552±0.6977	p<0.001
Globulin(g/dl)	4.3067±1.539	2.394±1.178	p<0.001
hsCRP(µg/dl)	87.0792±121.05	337.8393±134.163	p<0.001
Glucose(mg/dl)	96.7248±18.555	109.25±34.329	p<0.05
Urea(mg/dl)	15.197±3.5743	35.7363±16.1967	p<0.001
Uric acid(mg/dl)	5.7933±0.7573	2.9233±1.4191	p<0.001

Table 3. Mean values of different parameters in sera of burn patients at II,III, and mix (II&amp;III) degrees.

Parameters	II degree Mean± SD	III degree Mean± SD	Mix(II&III)degree Mean± SD
Total protein(g/dl)	5.0765±1.124	4.7216±.962	5.423±1.8965
Albumin(g/dl)	2.6151±0.8068	2.4134±0.6809	2.725±0.464
Globulin(g/dl)	2.4614±0.3172	2.3082±0.2811	2.698±1.432
hsCRP(µg/dl)	299.829±164.96	350.646±141.28	366.35±86.12
Glucose(mg/dl)	98.758±32.843	112.80±39.48	124.548±20.88
Urea(mg/dl)	37.819±21.22	35.00±10.60	32.77±16.52
Uric acid(mg/dl)	3.1045±1.63	2.901±1.53	2.575±0.518

where it is well known that insulin has anti-hyperglycemic action, reduction in infections, promotes muscle anabolism and regulates the systemic inflammatory response (Ballian *et al.*, 2010).

Burns cause a reduction of blood flow to the kidney which lead to, build up of nitrogen waste products, such as creatinine and urea in the body (azotemia). Prerenal azotemia is the most common form of kidney failure in hospitalized patients (Yu ASL 2007). Sabry *et al.* (2009) conclude from their study that acute renal failure complicates burn patients and is related to the size and depth of burn and occurrence of septicemia. A highly significant increase in urea level (p< 0.001) was observed in the present study in sera of burn patients compared to control group.

Uric acid results of the present study showed a highly significant decrease (p< 0.001) in their levels in sera of burn patients in compared to control group. This decrease is due to increase the fractional excretion of uric acid (Peretz *et al.*, 1983). Conflicting results for uric acid level in burns were observed where Nagane *et al.* (2003) indicated in their study significant increase of uric acid in burn patients due to increased activity of xanthine oxide. While Yadav (2010) showed in his study a significant decrease in serum uric acid of burn patients in compared to control group.

The overall analysis of different parameters study in sera of burn patients at degree II, degree III, and mixture degree (II & III) were presented in table 3. As shown

from the Mean values there were a fluctuation in total protein, albumin, and globulin between these degrees. While a non significant increase in hsCRP and glucose levels (p>0.05), and a non significant decrease (p>0.05) in urea and uric acid levels were observed.

## CONCLUSION

We conclude from the present study that the metabolic changes occur in patients with varying degrees of burns and its values did not depend on burn degree, suggesting that its value can be used to predict the mortality of burn patients.

## REFERENCES

- Aguayo-Becerra, OA., Torres-Garibay, C., Macías-Amezcuca, MD., Fuentes-Orozco, C., Chávez-Tostado Mde, G., Andalon-Dueñas, E., Espinosa Partida, A., Alvarez-Villaseñor, Adel S., Cortés-Flores, AO. and González-Ojeda, A. 2013. Serum albumin level as a risk factor for mortality in burn patients. *Clinics (Sao Paulo)*. 68(7):940-945.
- Atiyeh, BS., Gunn, SW. and Dibo, SA. 2008. Metabolic implications of severe burn injuries and their management: a systematic review of the literature. *World J Surg*. 32(8):1857-69.
- Ballian, N., Rabiee, A., Andersen, DK., Elahi, D. and Gibson, BR. 2010. Glucose metabolism in burn patients: the role of insulin and other endocrine hormones. *Burns*. 36(5):599-605.

- Chatham, JC., Nöt, LG., Fülöp, N. and Marchase, RB. 2008. Hexosamine biosynthesis and protein O-glycosylation: the first line of defense against stress, ischemia, and trauma. *Shock*. 29(4):431-440.
- Colleen, M. 1998. Objective estimates of the probability of death from burn injuries. *N Engl J Med*. 335(5):362-7.
- Du Clos, TW. 2000. Function of C-reactive protein. *Ann Med*. 32(4):274-8.
- Grove, G. and Jackson, AA. 1995. Measurement of protein turnover in normal man using the end-product method with oral [<sup>15</sup>N]glycine: comparison of single-dose and intermittent-dose regimens. *Br J Nutr*. 74(4):491-507.
- Hart, DW., Wolf, SE., Mlcak, R., Chinkes, DL., Ramzy, PI., Obeng, MK., Ferrando, AA., Wolfe, RR. and Herndon, DN. 2000. Persistence of muscle catabolism after severe burn. *Surgery*. 128(2):312-9.
- Hemmila, MR., Taddonio, MA., Arbabi, S., Maggio, PM. and Wahl, WL. 2008. Intensive insulin therapy is associated with reduced infectious complications in burn patients. *Surgery*. 144(4):629-635.
- Holm, C., Hörbrand, F., Mayr, M., von Donnersmarck, GH. and Mühlbauer, W. 2004. Acute hyperglycaemia following thermal injury: friend or foe? *Resuscitation*. 60(1):71-77.
- Hörbrand, F., Schrank, C., Henckel-Donnersmarck, G. and Mühlbauer, W. 2003. Integration of preexisting diseases and risk factors in the Abbreviated Burn Severity Index (ABSI) *Anesthesiol Intensivmed Notfallmed Schmerzther*. 38(3):151-7.
- Jeschke, MG., Finnerty, CC., Kulp, GA., Kraft, R. and Herndon, DN. 2013. Can we use C-reactive protein levels to predict severe infection or sepsis in severely burned patients? *Int J Burns Trauma*. 8(3):137-43.
- Lavrentieva, A., Kontakiotis, T., Lazaridis, L., Tsotsolis, N., Koumis, J., Kyriazis, G. and Bitzani, M. 2007. Inflammatory markers in patients with severe burn injury. What is the best indicator of sepsis? *Burns*. 33:189-194.
- Lehnhardt, M., Jafari, H.J., Druecke, D., Steintraesser, L., Steinau, HU., Klatte, W., Schwake, R. and Homann, HH. 2005. A qualitative and quantitative analysis of protein loss in human burn wounds. *Burn*. 31(2):159-167.
- Mendonça Machado, N., Gagnani, A. and Masako Ferreira, L. 2011. Burns, metabolism and nutritional requirements. *Nutr Hosp*. 26(4):692-700.
- Manelli, JC., Badetti, C., Botti, G., Golstein, MM., Bernini, V. and Bernard, D. 1998. A reference standard for plasma proteins is required for nutritional assessment of adult burn patients. *Burn*. 24(4):337-345.
- Miquet-Rodríguez, LM., Rodríguez-Garcell, R., Santana-Porben, S. and Cervantes-Flores, R. 2013. Valor Pronóstico del nivel de albúmina sérica inicial en los pacientes quemados. <http://www.portalesmedicos.com/publicaciones/articulos/1108/2/Valor-pronostico-del-nivel-de-albumina-serica-inicial-en-los-pacientes-quemados>.
- Muhammad, O. and Hayder, AH. 2011. Some Physiological Changes in Burn Patients. *Medical Journal of Babylon*. 8(3):303-319.
- Nagane, NS., Bhagwat, VR. and Subramaniam, M. 2003. Increased free radical activity in burns. *Indian J Med Sci*. 57(1):7-11.
- Norbury, WB., Jeschke, MG. and Herndon, DN. 2006. Metabolic Changes Following Major Burn Injury: How to Improve Outcome. *Intensive Care Medicine*. 514-524.
- Peck, MD., Kruger, GE., van der Merwe, AE., Godakumbura, W. and Ahuja, RB. 2008. Burns and fires from non-electric domestic appliances in low and middle income countries Part I. The scope of the problem. *Burns*. 34:303.
- Peretz, A., Decaux, G. and Famaey, JP. 1983. Hypouricemia and intravenous infusions. *J Rheumatol*. 10(1):66-70.
- Pileri, D., Accardo-Palumbo, A., D'Amelio, L., D'Arpa, N., Arnone, G., Grisaffi, C., Amico, M., Brancato, R., Lombardo, C. and Conte F. 2009. Serum Levels of Cortisol, Immunoglobulin, and C-reactive Protein in Burn Patients. *Ann Burns Fire Disasters*. 22(1):3-5.
- Prerenal azotemia | niversity of Maryland Medical Center. <https://umm.edu/Health/.../Prerenal-azotemia>.
- Sabry, A., Wafa, I., El-Din, AB., El-Hadidy, AM. and Hassan, M. 2009. Early markers of renal injury in predicting outcome in thermal burn patients. *Saudi J Kidney Dis Transpl*. 20(4):632-8.
- Steven, L., Stockham, SL. and Scott, MA. 2008. *Fundamentals of Veterinary Clinical Pathology*. (2<sup>nd</sup>edi.).
- Tobiasen, J., Hiebert, JM. and Edlich, RF. 1982. The Abbreviated Burn Severity Index. *Ann Emerg Med*. 11(5):260-2.
- Williams, FN., Herndon, DN. and Jeschke, MG. 2009. The Hypermetabolic Response to Burn Injury and Interventions to Modify This Response. *Clin Plast Surg*. 36(4):583-596.
- Yadav, MK. 2010. A Study On Levels Uric Acid In Burn Patients. MS Thesis. Rajiv Gandhi University of Health Sciences, Karnataka, Bangalore, India.