Study of alterations in Lipid profile after Burn injury.

Dr. Asha Khubchandani^{1*}, Dr. M. F. Shaikh², Dr. Pankaj Gaadhe³

¹Associate Professor, ³Resident Doctor, Department of Biochemistry, B. J. Medical College, Ahmedabad.

²Professor, Department of Burns & Plastic Surgery, Civil Hospital, Ahmedabad.

Abstract:

Introduction: After burn injury, changes in lipid profile occur in body. Dyslipidemia after burn injury is one of the important alterations. **Objective**: To check alterations in lipid profile after burn injury. **Materials and Method**: It was cross sectional study which was carried out on 250 burns patients of both sex, with an age group of 18-45 years, and varying burns percentage of 20-80% of total body surface area (TBSA). Serum cholesterol, serum LDL, serum HDL and serum triglyceride level were measured on XL-640 fully-auto biochemical analyser. Serum LDL and HDL were measured by Accelerator Selective Detergent Method. Serum cholesterol and triglyceride were measured by Trindor's method. **Results**: Results showed decrease in serum cholesterol, serum LDL and serum HDL, while increase in serum triglyceride level in burns patients compared to normal subjects. **Conclusion**: This study clearly showed the importance of measuring serum cholesterol, TG, LDL and HDL in burn patients and targeting changes that occur in their levels along the burns course, which may have beneficial effect in protection from organ damage, increasing survival rates and improving burn outcome.

Keywords: Triglyceride (TG), Low-density lipoprotein (LDL), High-density lipoprotein (HDL).

Introduction:

Major thermal injury is associated with, extreme hypermetabolism and catabolism. Dyslipidemia after burn injury is one of the important alterations that results from many factors like, hypermetabolic state in burns, release of hormones and inflammatory mediators.¹

An improved understanding of burn pathophysiology has contributed to improvement in fluid resuscitation, infection control, support of hypermetabolic response to trauma, nutritional support and early healing of the burns wound, and burns outcome in general.²



Thermal injury can cause many changes in the skin as local responses and as general systemic response in the body; the metabolic changes are important one among the systemic responses.³ The degree of metabolic changes experience by burn patients is directly related to the extent of injury. In large burn injuries, cortisol, glucagon and catecholamines are

* Corresponding Author:	markedly elevated. ⁴		
Dr. Asha Khubchandani	Cortisol is s		
E-mail: <u>ashakhub@yahoo.com</u>	Cortisoi is s		

Cortisol is strongly catabolic and is associated

with negative nitrogen and calcium balance, loss of tissue protein and bone minerals. It also stimulates gluconeogenesis, increase proteolysis and sensitizes adipocytes to the action of lipolytic hormones. Catecholamines increase the rate of glycogenolysis, hepatic gluconeogenesis; promote lipolysis and peripheral insulin resistance.⁵

These changes lead to the release of amino acids from muscles, and lipolysis of adipose triglycerides leading to the release of fatty acid into the plasma. The free fatty acids can be used directly by most peripheral tissues for their energy requirements. In burns patients, fat oxidation is increased to obtain endogenous energy substrates. In addition to that, there is increase in the recycling of fatty acids that leads to increase in triglycerides plasma level.⁶

The present study was performed in order to evaluate the occurrence of dyslipidemia after burn injury to improve burn management and outcomes.

Materials and Methods:

This cross sectional study was carried after taking required permission on 250 burns patients of both sex with an age group of 18-45 years and having varying burn percentage of 20-80% of total body surface area (TBSA).

Burns patients can be divided into 4 groups depending on % TBSA; (A) Group 1 : <25% of TBSA (B) Group 2: 26-45% of TBSA (C) Group 3: 46-65% of TBSA (D) Group 4 : >65% of TBSA.

Inclusion criteria:

We have included only those patients with the age group of 18-45 years who reported in the hospital within 12 to 24 hours after burns injury and varying burns percentage of 20-80%.

Exclusion criteria:

We have excluded the patients below the age of 18 years as well as beyond the age of 80 years. We have also excluded those patients whose burns percentage was more than 80% and also <20\% of TBSA.

Subjects involved in this study allocated into two groups:

Group A: (250) burned patients treated according to hospital protocol.

Group B: (250) healthy subjects of both sex and with same age groups as that of patients were selected as control for comparison.

Random Blood samples were collected in plain vacuette from all the subjects by venipuncture on admission to burns unit within the first 24 hours and this value designated to be of the first week; in addition to that blood samples were taken at the second and third weeks (which is the time of discharge from burns unit); to check the changes in the serum cholesterol, serum triglyceride (TG), serum low density lipoprotein (LDL-D) and serum high density lipoprotein cholesterol (HDL-C) levels. Measurement was done on XL-640 fully-auto biochemical analyser. Serum LDL and HDL were measured by Accelerator Selective Detergent Method. Serum cholesterol and triglyceride were measured by Trindor's method.⁸

Statistical analysis of data was done utilizing student't' test (Paired). For the purpose of this study 'p' value < 0.05 was been taken as significant.

Results:

Results in the Table 1 showed the normal values of serum levels of Cholesterol, TG, LDL-D and HDL-C for normal healthy subjects (Group B); as compared to the values of burns patient (Group A) at different time period.

Table and Image 1 showed that serum Cholesterol level was significantly ($P \le 0.05$) lower in burn patients by 25.94% at the first week compared to healthy subjects. In burns patient serum cholesterol level is non significantly raised in second and third week as compared to the first week by 8% and 13% respectively. [Image 1]

Table 1: Lipid profile values in both groups at different time periods (Mean ± S.D.)

Groups		Cholesterol(mg/dl)	HDL(mg/dl)	LDL(mg/dl)	TG(mg/dl)	
Group A	1 ST week	$108.79 \pm 11.34*$	$28.12 \pm 4.73^*$	$108 \pm 13.40*$	156 ± 14.53	
	2 nd week	118.26 ± 15.10	29.50 ± 7.11	113 ± 12.50	158 ± 13.32	
	3 rd week	125.09 ± 19.67	$24.22~\pm~6.48$	117 ± 14.10	$186 \pm 20.19^*$	
Group B	Control	146.90 ± 7.64	54.40 ± 3.00	128 ± 11.20	164 ± 9.11	
*n value statistically significant (n<0.05)						

*p value statistically significant (p≤0.05)

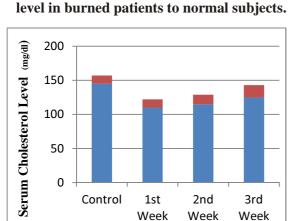
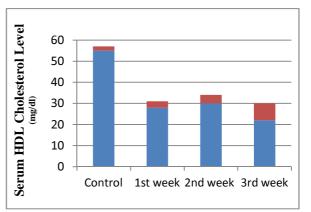


Image 1 : Comparison of serum cholesterol

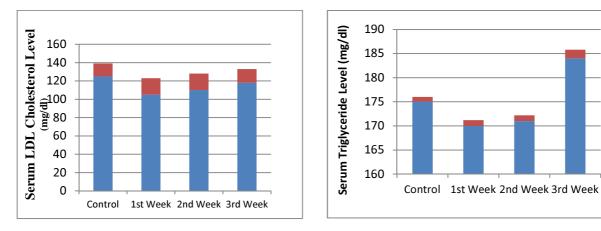
Image 2 : Comparison of serum HDL Cholesterol level in burned patients to normal subjects



There is no significant change in serum TG level of burns patient as compared to normal subjects at first week in spite of a small fall by 4%, but became significantly raised over a period of time due to increase in the level of stress hormones leading to hypermetabolic state. (It increased to 16% in TG level at third week as compared with first week) (P \leq 0.05).Serum TG level increased to 16% at third week as compared to first week

Concerning serum HDL-C level, it clearly showed the significant ($P \le 0.05$) decrease in the first week in burns patient as compared to normal subjects. Image 2 shows that serum HDL-C level decreased by 48.30% in burn patients in the first week and this low level maintained in the second and third weeks.

When LDL level is measured in the first week of thermal injury, significant decrease of 15 % (at p<0.05) was found as compared to healthy subjects – [Image 3]





Discussion:

Severe burn injury is followed by a profound hypermetabolic response. It is mediated by up to 50-fold elevations in plasma catecholamines, cortisol, and inflammatory cells that lead to whole-body catabolism, elevated resting energy expenditures, and multiorgan dysfunction. Dyslipidemia after burn injury is one of the important alterations.

Earlier studies have described these changes in burns patient, the importance of correcting such dysregulation in serum lipids of burns patient and the effect of such targeting on burns outcomes in general.^{8,9}

Studies have described changes in cholesterol and triglyceride level, there is initial decrease in serum level of cholesterol, and an increase in TG level.⁸ The tracking of this lipid profile changes gives some sort of idea regarding organ dysfunction which is beneficial for deciding the line of treatment for the patients with burns injury.

It has been observed that in the patients with severe burns, serum TG rises, reaching a peak on 4th-6th post burn day, while cholesterol and phospholipids fell drastically; they suggest that the changes observed are reflection of block in the conversion of very low density lipoprotein to low density lipoprotein.⁸ Findings of our study correlate well with this data.

Other studies that support our findings in this regard, showed that severe burn injuries lead to liver damage and changes in lipid metabolism. In burns patients, it is commonly seen that oedema formation in lever leads to cell damage which results in increase of releasing liver enzymes like ALT, AST, ALP etc.^{10,11,12,13} Along with this, sepsis in liver due to burn injury causes intrahepatic cholestesis and ultimately liver damage.

It has been shown that low cholesterol level in burn patients is due to increased catabolism of cholesterol rich lipoprotein.¹³

The decrease in HDL, which regulates LPL activity, will thus result in an 12

accumulation of triglycerides and a reduction in cholesterol due to the fact that LPL will be reduced, which will affect the turnover from VLDL to LDL.⁹

Taken together, the above mentioned evidences clearly documented the beneficial role of measuring serum cholesterol, TG, LDL and HDL in burns patients, also show the importance of targeting changes that occur in their levels along burn course which may have beneficial effect through preventing organ damage and lead to increase in survival rates among burns patient and improve burn outcome.

Conclusion:

From this study we conclude that, changes in serum lipids of burns patients occur; namely decrease in serum cholesterol and serum LDL level, increase in serum TG level and decrease in serum HDL level compared to normal subjects. As severe burn injury is a hypermetabolic response and dyslipidemia after burns is one of the important alterations. Dyslipidemia after burn course we studied till 3rd week has no correlation with burn size, was found in all 20-80% of TBSA patients. Following burns injury there are functional disturbances in liver and pancreas that are reflected in changes of serum level of enzymes like ALT,AST,ALP and Amylase and have good correlation with burn size and so have good prognostic value.

References:

- 1. Coombes EJ, Bastone GF, Shakespeare PG, and Levick PL. Lipid studies after burn injury in man. Burns 1979, 5: 265–268
- 2. Arturson G. Pathophysiology of the burn wound and pharmacological treatment. The Raudi Hermans Lecture, 1995. Burns1996, 22: 255-274.
- 3. Hettiaratchy S, and Dziewulski P. Pathophysiology and types of burns. BMJ 2004, 328: 1427-1429.
- 4. Wolfe RR .Relation of metabolic studies to clinical nutrition the example of burn injury. Am J Clin Nutr 1996, 64: 800-808.
- 5. Gauglitz GG, Herndon DN, Kulp GA, Meyer WJ 3rd, and Jeschke MG. Abnormal insulin sensitivity persists up to three years in pediatric patients post-burn. J Clin Endocrinol Metab 2009,94:1656-64.
- 6. Williams FN, Herndon DN, and Jeschke MG. The hypermetabolic response to burn injury and interventions to modify this response. Clin Plast Surg 2009, 36:583-96.
- 7. Fossati, P. and Prencipe, L. Measurement of serum triglycerides colorimetrically with an enzyme that produce H2O2. Clin. Chem1982, 28:2077-2080.
- 8. Coombes EJ, Shakespeare PG, and Bastone GF. Lipoprotein changes after burn injury in man. J Trauma 1980, 20:971–5.
- 9. Birke G, Carlson LA, von Euler US, Liljedahl SO, and Plantin LO. Studies on burns. Acta Chir Scand 1972,138:321–33.

- 10. Arturson G and Wallenius G. The hepatocellular origin of the circulating enzymes in experimental burns. Acta Chir Scand 1963,123:34–8
- 11. Kamolz LP, Andel H, Mittlböck M, Winter W, Haslik W, Meissl G, and Frey M. Serum cholesterol and triglycerides: potential role in mortality prediction. Burns 2003, 29:810-5.
- 12. Latha B, Ramkrishanan M, Jayaraman V, Babu M.Serum enzymatic changes modulated using trypsin-chymotrypsin preparation during burn wounds in humans. Burns 1997;23:560-64.
- 13. Halkes S, van den berg A, Hoekstra M et al. Transaminase and alkaline phosphatase activity in serum of burns patients treated with highly purified tannic acid. Acta Chir Plast 2002;28:449-53.