Estimation of Post Burn Survival Duration – A Predictive Equation Model and a New Perspective with Reference to detailed Histomorphological Changes of Kidney in Fatal Burn Injuries – An Autopsy based Study in a Tertiary Teaching Hospital of Eastern India

Nirmalya Chakrabarti[1], Uma Banerjee[2], Arghya Bandyopadhyay[3], Aniruddha Das[4], Dipankar Guha Roy[5]

ABSTRACT

Introduction: "Burn" – the thermal injury is a very serious health issue having both medical and legal implications. This multifaceted pathological condition involves both skin and other vital internal organs through different pathobiological mechanisms. In the present study an attempt has been made to find out the detailed histomorphological changes in kidney and statistical relationship among different non modifiable and modifiable pathomorphological parameters, with an aim to reach a predictive equation model for estimation of duration of post burn survival.

Material and methods: This study was conducted at Burdwan Medical College. The study population consisted of 53 cases, between February 2013 and January 2014. The cases with mixed injuries, decomposed bodies and death autopsy interval more than 12 hours were excluded. In the present study, the parameters analyzed, were socio-demographic profile of the victims of burn injuries, percentage of total body surface area burnt, interval between incidence of burn and death, gross and histopathological changes of kidneys. Statistical methodology by student's t – test, Chi – square test or Fisher's exact test along with bivariate correlation analysis were performed with significance accepted at $P < 0.05$.

Results: Out of 53 cases 33 cases (62.26%) had 61-90% TBSAB, 18 (33.96%) cases had > 90% TBSAB. 34 victims (64.14%) died within initial 96 hours after burn injury and 15 cases (28.30%) survived after 144 hours post burn.

Conclusion: This predictive equation model in turn may provide an aid to solve different medicolegal litigations.

Keywords: Burn, Survival Duration, Kidney Changes, Predictive Equation

INTRODUCTION

Burn is best defined as "an injury which is caused by application of heat or chemical substances to the external and internal surfaces of body, which causes destruction of tissues".[1] Burn is a serious global health issue as well as a medicolegal problem. India has annual burn incidence of 6-7 million where 70% of the victims are from the age group of 15-40 years and majority of them are from poor socioeconomic condition.[2]

The pathology of burn injury is multi factorial. It has a complex pattern and the pathology goes beyond the skin, reaching into various internal organs. Different pathological, biochemical, immunological and microbiological responses drive the case of burn and ultimately lead to systemic inflammatory response syndrome, finally multi organ dysfunction syndrome and death. Among the internal organs kidney is the one of the most vulnerable targets of burn which has been demonstrated in various research works in past.

The current study is aimed for an approach for the pathologists and the medicolegal experts to correlate the changes of the microscopic level with the clinical parameters and to reach in such an objective way that the histopathology becomes an important tool to estimate post burn survival duration.

In the present study we analyzed the histopathological parameters of kidney changes in respect to total body surface area burnt (TBSAB) and post burn survival duration to develop a predictive equation model and a scoring system which might tell the post burn survival period from the histopathological data and the TBSAB calculated in the mortuary which in turn helps in the administration of justice.

MATERIAL AND METHODS

This study was conducted after getting the permission letter from Institutional Ethics Committee, Burdwan Medical College. The study population consisted of 53 cases, between February 2013 and January 2014, with fatal burn injuries brought to mortuary, Burdwan Medical College, with death autopsy interval not more than 12 hours. The cases with mixed injuries, decomposed bodies and death autopsy interval more than 12 hours were excluded.

In the present study, the parameters analyzed, were socio-demographic profile of the victims of burn injuries, percentage of total body surface area burnt, interval between incidence of burn and death, gross and histopathological changes of kidneys.

[1]Assistant Professor, Department of Pathology, College of Medicine and J.N.M Hospital, West Bengal, [2]Professor and Head, Department of Pathology, Medical College, Kolkata, [3]Assistant Professor, Department of Pathology, Burdwan Medical College, [4]Assistant Professor, [5]Professor and Head, Department of Forensic Medicine and Toxicology, Calcutta National Medical College, India

Corresponding author: Dr. Nirmalya Chakrabarti, 170/3427 Fingapara By Lane Left, Fingapara, Near Rajlaxmi Balika Vidyamandir; P.O – Fingapara, District – North 24 Parganas, PIN – 743129, West Bengal, India.

How to cite this article: Nirmalya Chakrabarti, Uma Banerjee, Arghya Bandyopadhyay, Aniruddha Das, Dipankar Guha Roy. Estimation of post burn survival duration – a predictive equation model and a new perspective with reference to detailed histomorphological changes of kidney in fatal burn injuries – an autopsy based study in a tertiary teaching hospital of Eastern India. International Journal of Contemporary Medical Research 2017;4(1):279-283.

		Post Burn Survival Duration							Total (n=53)
		<24 hours	>24-48 hours	>72-96 hours	>120-144 hours	>144-168 hours	>336-504 hours	>504-672 hours	
TBSAB	30-60%	0	0	0	0	2	0	0	2
	61-90%	0	9	11	0	0	7	6	33
	>90%	11	3	0	4	0	0	0	18
Total		11	12	11	4	2	7	6	53

Table-1: Distrinution of post burn survival duration according to total body surface area burnt

Observed histopathological changes of kidney	Incidence (n = 53)
Tubular cast	10 (18.87%)
Necrosis of tubular epithelium	53 (100%)
Desquamation of tubular epithelium	51 (96.23%)
Regeneration of tubular epithelium	19 (35.85%)
Congestion of interstitium	37 (69.81%)
Interstitial chronic inflammatory infiltrate	8 (15.09%)
Interstitial edema	24 (45.28%)

Table-2: Incidence of different histological parameters of affected kidneys in fatal burn injuries

Socio-demographic profile was assessed from the history taken from the police officials, inquest papers and the relatives accompanying the victim. The total body surface area burnt (TBSAB) was calculated by using the 'Wallace rule of nine method'. Post burn survival duration was found from the inquest. Gross and histopathological examinations of both kidneys were performed using the standard autopsy protocol.

STATISTICAL ANALYSIS

All the values were expressed as mean with standard deviation (continuous variables) or percentage (categorical variable). Statistical methodology by student's t – test, Chi – square test or Fisher's exact test (where applicable) along with bivariate correlation analysis were performed with significance accepted at $P < 0.05$. All gross and histopathological variables were then included in linear regression analysis method to test the hypothesis that the variables employed affected the post burn survival duration. This analysis was performed using SPSS v.19.

RESULTS

53 cases of fatal burn injury, brought to the police morgue of Burdwan Medical College, Burdwan, West Bengal, India, between February 2013 and January 2014, were included in the study. Among 53 victims, 47 (88.68%) were females. The mean age of 53 cases was 31.42 years (range 16-76 years) with standard deviation of 13.071. 40 cases (75.47%) were Hindu in religion and 44 (83.02%) were married. 42 victims (79.25%) were the residents of rural areas.

Out of 53 cases 33 cases (62.26%) had 61-90% TBSAB, 18 (33.96%) cases had > 90% TBSAB. 34 victims (64.14%) died within initial 96 hours after burn injury and 15 cases (28.30%) survived after 144 hours post burn. Correlation analysis, performed between post burn survival duration and TBSAB, shows that Pearson's correlation coefficient (r) is – 0.494 and $P = 0.00$ (Table-1).

The mean length of right kidney was 104.91 mm (range 90.90-116.20 mm) with standard deviation of 7.86. The mean length of left kidney was 104.54 mm (range 88-115.10 mm) with standard deviation of 7.86. The mean cortical thickness of right kidney

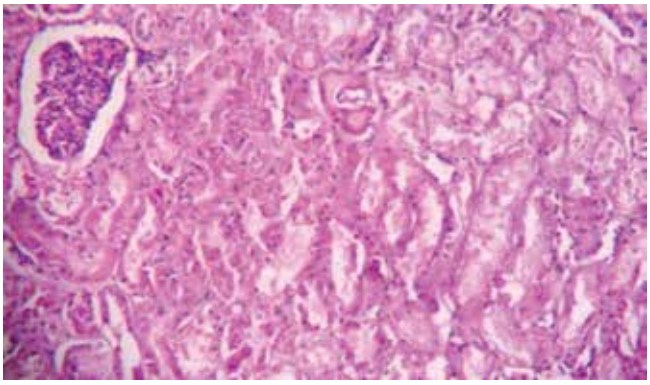

Figure-1: Photomicrograph showing extensive tubular necrosis [Hematoxylin and Eosis, 100X]

was 7.90 mm (range 6- 10.20 mm) with standard deviation of 1.197 and the mean cortical thickness of left kidney was 7.82 mm (range 5.7-11.0 mm). Bivariate correlation analysis between post burn survival duration and kidney length and thickness shows Pearson's r for right kidney length and cortical thickness are -0.058 and -0.051, whereas Pearson's r for left kidney length and cortical thickness are -0.002 and -0.540 respectively.

Surface congestion of kidneys on gross examination was present in 38 cases (71.7%) among which 20 cases (37.74%) had 60-90% TBSAB and rest 18 cases (33.96%) had > 90% TBSAB ($r = 0.511$).

Table-2 shows the incidence of different histopathological changes of affected kidneys in fatal burn injury.

All 53 cases showed tubular necrosis (Figure-1) in microscopic level. 16 cases (30.19%) had severe tubular necrosis involving > 50% tubules of which 11 cases had > 90% TBSAB and rest 5 had < 90% TBSAB ($P = 0.001$, Pearson's $r = 0.483$). 11 cases of severe tubular necrosis survived < 72 hours post burn and 5 cases survived > 72 hours post burn ($P = 0.019$, Pearson's $r = - 0.336$). Among 51 cases showing desquamation of tubular epithelium (Figure-2), 31 (58.49%) had 61-90% TBSAB and 18 (33.96%) had > 90% TBSAB (Pearson's $r = 0.112$). 11 cases (20.75%) with desquamation died within initial 24 hours post burn, 12 (22.64%) within 24-48 hours, another 11 cases within 72-96 hours period and rest 17 survived beyond initial 120 hours (Pearson's $r = - 0.334$). Out of 10 cases having tubular cast (Figure-3), 6 (11.32%) had > 90% TBSAB (Pearson's $r = 0.269$). All 10 died within initial 48 hours (Pearson's $r = - 0.421$). Among 37 victims having interstitial congestion, 18 (33.96%) had > 90% TBSAB and rest 19 (35.85%) had 61-90% TBSAB (Pearson's $r = 0.525$). Out of 19 cases, 15 cases who had survived beyond 144 hours post burn showed histopathological evidence of regeneration of tubular epithelium (Pearson's $r = 0.840$). Among 8 cases showing interstitial chronic inflammatory infiltrate, 6 cases (11.32%) survived beyond 168 hours post burn and 2 cases within 168 hours ($P =$

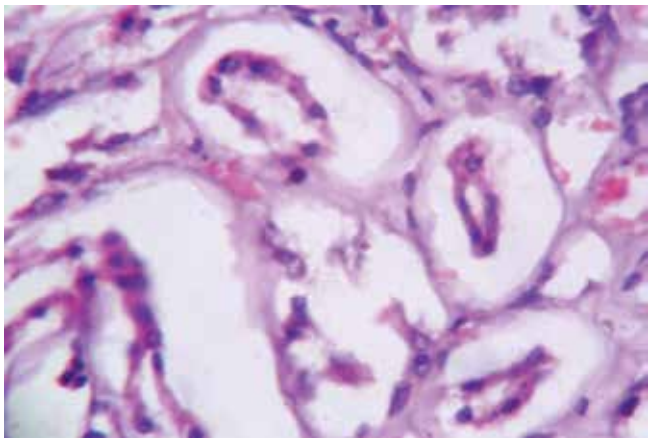

Figure-2: Photomicrograph showing desquamation of tubular epithelium [Hematoxylin and Eosin, 400X]

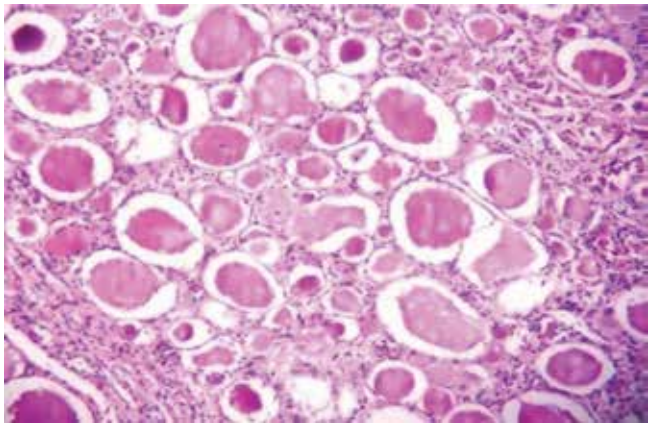

Figure-3: Photomicrograph showing extensive cast formation within tubular lumen [Hematoxylin and Eosin, 100X]

0.002, Pearson's r = 0.495). 24 cases showed histopathological evidence of interstitial edema. Among these 24 cases, 7 cases (13.21%) had the survival period of < 24 hours, 5 (9.43%) had >24-48 hours, 6 cases (11.32%) survived for >72-96 hours and rest 6 cases (11.32%) had the survival period of >504-672 hours (Pearson's r = - 0.090).

All the independent variables except gender, residence and marital status are combined using linear regression analysis method and a predictive equation model is generated for estimation of post burn survival duration:

Post burn survival duration = *23.665 + 0.521 (TBSAB) – 0.11 (length of left kidney in mm) – 0.076 (cortical thickness of right kidney in mm). + 0.042 (age in years) – 1.609 (tubular cast) + 4.086 (regeneration of tubular epithelium) – 1.013 (tubular necrosis) – 2.302 (interstitial congestion) – 1.849 (interstitial chronic inflammatory infiltrate) – 0.939 (desquamation of tubular epithelium)*

For scoring of tubular necrosis '0' is to be put for absence, '1' for involvement of < 25% tubules, '2' for 25-50% tubular involvement and '3' for > 50% tubular involvement. For other histopathological parameters '0' should be put in case of absence and '1' for presence. For TBSAB 1 should be put in case of 30-60% TBSAB, 2 in 61-90% TBSAB and 3 in > 90% TBSAB.

For interpretation of final value of post burn survival duration from the predictive equation the following is to be followed: 1= < 24 hours, 2= 25-48 hours, 3= 49-72 hours, 4= 73-96 hours, 5= 97-120 hours, 6= 121-144 hours, 7= 145-168 hours, 8=169 – 336 hours, 9= 337-504 hours, 10= 505 – 672 hours and 11= > 672 hours.

DISCUSSION

Burn injuries are one of the most devastating injuries and constitute a major global public health hazard. Burns are the fourth most common type of injury worldwide after road traffic accidents, falls and interpersonal violence.[3] In the present study the peak incidence (77.36%) of fatal burn injuries occurred in the age group of 20 to 40 years followed by 40 to 60 years age group (9.43%) which is similar to the study of Chakraborty et al[4] and Gupta et al[2] where the predominant affected age groups were 20 to 39 years and 15 to 40 years respectively. In the present study females were the major victims of burn injury (88.68%) and 83.02% were married following the common trend in the Indian subcontinent. This observation is almost similar with the study of Batra[5] (female 80.8%, 82.4% married) and Shinde and Keoliya[6] (female 85.45%, 81.91% married). The majority of the victims were of young age group in our study as well as in various previous studies because the young persons are engaged in house hold works in kitchen (in case of females) thus exposed to cooking on open, unguarded flame such as stove, chullha, cooking gas and in factories with fire or electricity and chemicals (in case of males). So there is more chance of accidental burn injuries in this group of people than in children or in older persons. Young females are also subjected to dowry related homicidal deaths in Indian society than the older women. 79.25% victims in the present study were from rural areas where the use of kerosene lamps (for lighting), weakly designed kerosene stoves, polyester mixed fabrics (worn by the rural women), ill-designed huts and scanty medical facilities all contribute to the increased fatalities of burn injuries.

In the present study duration of survival is significantly and negatively correlated with percentage of total body surface area burns which indicates that with increased percentage of TBSAB, the time of survival of the victim decreases significantly. This result is consistent with the study of Olaitan and Jiburum.[7] An increase in burn size is associated with hypermetabolism, with persistent inflammation, with catabolism, with changes in body composition, with increased stress hormone production, with marked organ dysfunction, with increased rate of sepsis and ultimately death.[8]

We had selected length, cortical thickness of both kidneys along with surface congestion as the parameters on gross examination. Both length and cortical thickness showed strong negative correlation with post burn survival duration which is similar to the study of Moghazi S et al[9] and Mounier- Vehier C et al.[10] This may be due to the fact that with increased survival there is development of systemic sepsis which is responsible for the development of renal failure, thus causing decrease in the renal lengths due to both proximal and distal tubular necrosis. The difference of vascularization between the cortex and the medulla, as well as the redistribution of blood flow from the cortex to the medulla due to hypoperfusion created by hypovolemic shock in fatal burn injuries especially in higher percentages of TBSA burns, could explain cortical thinning which ultimately resulting into cortical atrophy. We found that with higher TBSAB we found higher number of kidneys with surface congestion, which was similar to the animal model study

of Thailand by Aengwanich and Simarsks.[11] Fatal burn injuries induce tremendous stress responses in human body that lead to increased heart rate and increased blood flow to muscle, brain and heart which may be the cause of gross surface congestion of kidney. Besides this high blood pressure resulting from an autonomic nervous system response as a transient effect of high percentages of TBSA burns, may cause circulatory rupture leading to hemorrhage in various internal organs such as lung, kidney, liver and heart.

Burn injury affects kidney by creating hypovolemic and intense pro-inflammatory states in the earlier phases of survival whereas in the later days systemic sepsis takes the upper hand for the development of the organ dysfunction. Tubular necrosis, desquamation of tubular epithelium, tubular cast, interstitial congestion and interstitial chronic inflammatory infiltrate are the histopathological findings in kidney in the present study that is close to the studies of Sevitt[12], Argamaso[13], Rathod et al[14] and Schrier et al.[15] Necrosis of tubular epithelium, desquamation of tubular epithelium, tubular casts and congestion of interstitium – all were positively correlated with TBSAB among which tubular necrosis had an statistical significant correlation with TBSAB. Tubular necrosis, desquamation of tubular epithelium, tubular casts and interstitial edema were positively correlated with duration of survival post burn whereas regeneration of tubular epithelium, interstitial chronic inflammatory infiltrate had negative correlation. Here also tubular necrosis and interstitial chronic inflammatory infiltrate had statistically significant correlation with post burn survival duration. Hypovolemia in fatal burn injury leads to reduced renal blood flow and renal vasoconstriction due to release of pro-inflammatory cytokines and stress related hormones, ultimately producing acute renal injury which is manifested by tubular necrosis, desquamation of tubular epithelium and formation of tubular casts by the accumulated desquamated and necrosed epithelial cells in tubular lumen. Interstitial congestion which is produced due to stress response also leads to hypoxia in tubules and further worsens the degree of tubular necrosis. This renal hypoxia deranges ATP dependent sodium pumps on cell membrane of tubular epithelial cells and causes influx of sodium and water in cytoplasm in early post burn phases. Ischemic insult also stimulates bone marrow derived hematopoietic and mesenchymal stem cells which exert a paracrine action that facilitates the repair process by reducing the inflammation and injury in those victims who survived for a prolonged period.[16] We found this pathological basis of repair in our study as a form of regeneration of tubular epithelium in late post burn phases. Interstial edema and chronic mononuclear inflammatory infiltrate were seen in this setting as the response of tubular injury.

This subjectivity of histopathological examination of kidney in fatally burnt victims has guided us to a new concept of objectivity of histopathological examination which is estimated by linear regression analysis as a predictive equation for determining post burn survival period from different gross and histopathological parameters. No known previous study has reported this kind of scoring system and importance of post burn survival period estimation from objective histopatholgical examination. In Legal Medicine and Forensic science, the post burn survival period has a paramount importance because proper estimation of post burn survival period may guide us if the victim was capable to give dying declaration or dying deposition which helps in administration of justice in India. Revalidation of post burn survival duration also solve the issues of professional negligence of a physician while treating the patient according to the accepted protocol or refusing to treat a fatally burnt patient in emergency. By proper gross and histopathological examination we can comment on the crucial initial time frame in which proper medical management is needed for a fatally burnt victim. If a doctor refuses the patient to treat in the emergency in this phase it may be taken as an evidence of negligence because in this early crucial phase the patient might survive if he got the proper management. On the other way the doctor may be escaped from the false allegation of negligence if it is proven that the victim was brought to the hospital late when despite resuscitation multi-organ failure has become inevitable. It can also help to judge the issues of bride burning where the in - laws have kept the burnt victim for a prolonged period in home and made unnecessary delay to the treatment leading to the inevitable death.

CONCLUSION

Beside the age old descriptive histopathology of renal changes in fatal burn, in this present study we have made an effort to estimate approximate true post burn survival duration which can be helpful to the medicolegal field. Revalidation of the autopsy examination is made in cases of burn injury along with objective histopathological examination which reestablishes its profound importance with a new perspective.

REFERENCES

1. Vij K. Textbook of Forensic Medicine and Toxicology, Principles and Practice. 3rd ed. New Delhi, India: Elevier; 2009.
2. Gupta JL, Makhila LK, Bajaj SP. National programme for prevention of burn injuries. Ind J Plastic Surg. 2010; 43(suppl):S6-S10.
3. World Health Organisation Website. [Online]. [cited 2014 April 16. Available from:
4. http://www.who.int/violence_injury_prevention/child/injury/world_report/Burns_english.pdf
5. Chakraborty S, Bisoi S, Chattopadhyay D, Mishra R, Bhattacharya N, Biswas B. A study on demographic and clinical profile of burn patients in an Apex Institute of West Bengal. Indian J Public Health. 2010;54:27-29.
6. Batra AK. Burn mortality: recent trends and sociocultural determinants in rural India. Burns. 2003;29:270-275.
7. Shinde AB, Keoliya AN. Socio-demographic characteristics of burn deaths in rural India. Int J Healthcare and Biomedical Research. 2013;1:227-233.
8. Olaitan PB, Jiburum BC. Analysis of burn mortality in a burns centre. Ann Burns Fire Disasters. 2006;19:59–62.
9. Jeschke MG, Mlcak RP, Finnerty CC, Norbury WB, Gauglitz GG, Kulp GA et al. Burn size determines the inflammatory and hypermetabolic response. Critical Care. 2007;11:R90.
10. Moghazi S, Jones E, Arya K, Hennigar RA. Correlation of renal histopathology with sonographic findings. Kidney International. 2005;67:1515-1520.
11. Mounier-Vehier C, Lions C, Devos P, Jaboureck O, Willoteaux S, Carre A, et al. Cortical thickness: An early morphological marker of atherosclerotic renal disease. Kidney International. 2002;61:591-598.

12. Aengwanich W, Simaraks S. Pathology of heart, lung, liver and kidney in broilers under chronic heat stress. Songklanakarin J Sci. Technol. 2004;26:417-424.
13. Sevitt S. Distal tubular and proximal tubular necrosis in the kidneys of burned patients. J Clin Pathol. 1956;9:279-294.
14. Argamaso RV. Pathology, Mortality and Prognosis of Burns: A Review of 54 Critical and Fatal Gases. Canadian Med Ass J. 1967;97:445-449.
15. Rathod MR, Goswami H, Jankar D. A study of histopathological changes in burn deaths at civil hospital Ahmedabad-Gujarat. Int J Res Med. 2014;3:42-45.
16. Schrier R, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 2004;114:5–14.
17. Humphreys BD, Bonventre JV. Mesenchymal stem cells in acute kidney injury. Annual Rev Med. 2008;59:311–325.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 06-01-2017; **Published online**: 17-02-2017